The Effects of Chronic Sodium Bicarbonate Ingestion and Interval Training in Highly Trained Rowers

Matthew W. Driller, John R. Gregory, Andrew D. Williams, and James W. Fell

Recent research has reported performance improvements after chronic NaHCO₃ ingestion in conjunction with high-intensity interval training (HIT) in moderately trained athletes. The purpose of the current study was to determine the effects of altering plasma H⁺ concentration during HIT through NaHCO₃ ingestion over 4 wk (2 HIT sessions/wk) in 12 Australian representative rowers (M ± SD: age 22 ± 3 yr, mass 76.4 ± 4.2 kg, VO₂peak 65.50 ± 2.74 ml · kg⁻¹ · min⁻¹). Baseline testing included a 2,000-m time trial and an incremental exercise test. After baseline testing, rowers were allocated to either a chronic NaHCO₃ (ALK) or placebo (PLA) group. Starting 90 min before each HIT session, subjects ingested a 0.3-g/kg body mass dose of NaHCO₃ or a placebo substance. Fingertip blood samples were taken throughout the study to analyze bicarbonate and pH levels. The ALK group did not produce any additional improvements in 2,000-m rowing performance time compared with PLA (p > .05). Magnitude-based inferential analysis indicated an unclear or trivial effect on 2,000-m power, 2,000-m time, peak power output, and power at 4 mmol/L lactate threshold in the ALK group compared with the PLA group. Although there was no difference between groups, during the study there was a significant mean (± SD) 2,000-m power improvement in both the ALK and PLA groups of 17.8 ± 14.5 and 15.2 ± 18.3 W, respectively. In conclusion, despite overall improvements in rowing performance after 4 wk of HIT, the addition of chronic NaHCO₃ supplementation during the training period did not significantly enhance performance further.

Keywords: rowing, VO₂max, performance, alkalosis, pH, NaHCO₃

High-intensity exercise causes a rapid rate of ATP hydrolysis and glycolytic flux, resulting in an increased hydrogen ion concentration ([H⁺]; Robergs, Ghiasvand, & Parker, 2004). The metabolic acidosis caused by the increase in [H⁺] during high-intensity exercise is thought to hinder both metabolic and contractile processes of skeletal muscles through the lowering of myoplasmic pH levels (Chase & Kushmerick, 1988; Favero, Zable, Bowman, Thompson, & Abramson, 1995; Nakamura & Schwartz, 1970; Newsholme & Start, 1973), which may ultimately contribute to muscle fatigue and, therefore, impair exercise performance. There is strong evidence that ingested sodium bicarbonate (NaHCO₃) acts as an extracellular buffer to counter the acidosis caused by the increased [H⁺] during exercise (Carr, Hopkins, & Gore, 2011; Matson & Tran, 1993). Research has shown that acute ingestion of NaHCO₃ results in a significant elevation of pH and bicarbonate ion concentration ([HCO₃⁻]) in the blood, which are thought to play a key role in increasing the efflux of H⁺ and, therefore, maintaining optimal intracellular pH levels for muscle to function (Carr, Hopkins, & Gore, 2011; Matson & Tran, 1993). Furthermore, it has been suggested that chronic manipulation of pH levels during training may also provide long-term benefits to mitochondria, leading to improvements in exercise performance (Edge, Bishop, & Goodman, 2006). Therefore, chronic NaHCO₃ supplementation in a training setting may lead to benefits through two different pathways: It may aid in greater training adaptations after the acute ergogenic effect of NaHCO₃ supplementation, allowing for greater volume and intensity during training sessions, or the chronic manipulation of pH during training via NaHCO₃ ingestion may provide a protective effect on the mitochondria, leading to improved mitochondrial function and, therefore, performance.

Recent research has suggested that chronic NaHCO₃ supplementation may benefit mitochondrial adaptation (Bishop et al., 2010). The mitochondria are central to the conversion of energy and production of ATP during exercise. Regular endurance-exercise training can result in a rapid increase in the activities of oxidative enzymes (Holloszy & Coyle, 1984), mitochondrial density (Turner et al., 1997), and mitochondrial respiration (Daussin et al., 2008; Starritt, Angus, & Hargreaves, 1999). Recent research has suggested that reducing H⁺ accumulation...
during training (via pretraining ingestion of NaHCO₃) may lead to further improvements in mitochondrial function (Bishop, et al., 2010) and, therefore, exercise performance (Edge, Bishop, & Goodman, 2006). Edge, Bishop, and Goodman reported a significantly greater improvement in lactate threshold and time to fatigue during a constant-load cycling test after a period of high-intensity interval training (HIT) in participants who were supplemented with NaHCO₃ before the HIT sessions, compared with a placebo group. While this was a novel finding, the study investigated only moderately trained participants. Whether the same response would occur in highly trained athletes with already-well-adapted lactate thresholds and mitochondrial function has not yet been investigated. Rowing athletes, as used in the current study, possess elevated buffering capabilities compared with endurance-trained and untrained subjects (Parkhouse, McKenzie, Hochachka, & Ovalle, 1985). Thus, it is possible that the higher initial buffer capacity in these athletes may reduce the potential for improvement of this variable and thereby the potential benefit to performance (Edge, Bishop, & Goodman, 2006). Furthermore, it has been suggested that the long-term implications of repeated exercise-induced metabolic acidosis may not have as great a detrimental effect on mitochondrial function in highly trained athletes as in their less-trained counterparts (Edge, Bishop, & Goodman, 2006).

We have previously demonstrated that, for rowers who are already well trained, improvements in performance are still achievable through HIT (Driller, Fell, Gregory, Shing, & Williams, 2009), but whether these gains can be further enhanced through supplementation with NaHCO₃ before HIT sessions has not been investigated. In our laboratory we have also determined an effective NaHCO₃ loading protocol that minimizes side effects generally associated with its consumption (Driller, Williams, Bellinger, Howe, & Fell, 2012; Driller, Gregory, Williams, & Fell, 2012). Therefore, the aim of the current study was to determine whether chronic NaHCO₃ ingestion during 4 weeks of controlled-duration and -intensity HIT in highly trained rowers could enhance physiological measures and performance. We hypothesized that the combination of NaHCO₃ supplementation and HIT in a highly trained population would enhance performance to a greater degree than a placebo group. competed 2 weeks after finishing the study. A preexercise health-screening questionnaire and a Physical Activity Readiness Questionnaire (PAR-Q) were completed by all subjects before taking part in the study to ensure that there were no contraindications to vigorous exercise. The research was conducted according to National Health and Medical Research Council Guidelines after approval by the institutional human research ethics committee.

**Experimental Design**

The current study was a double-blind placebo-controlled trial. All subjects were familiar with the testing procedures, protocols, and equipment used. Testing consisted of a discontinuous incremental rowing exercise test (GXT) on a rowing ergometer (Concept II, Model-C, Vermont, USA) during which VO₂, blood lactate, and blood-gas concentrations were measured. A 2,000-m rowing-ergometer time trial (TT) was performed 48–72 hr after the GXT. Subjects were instructed to arrive at the testing sessions in a rested and hydrated state after fasting for at least 2 hr and to avoid strenuous exercise in the 48 hr preceding each test session. Food diaries were given to the subjects to record food and fluid consumption for the 24 hr before each test, and subjects were then asked to replicate their dietary intake before each subsequent testing session; however, diet and hydration were not controlled for during the training period. After the pretraining testing, subjects were matched according to their 4-min all-out power output (POpeak) from the final stage of the GXT and then randomly assigned to one of the two groups (n = 6 per group): NaHCO₃ ingestion (ALK) or placebo (PLA). Each subject was tested at approximately the same time of day throughout the study (±1 hr), and performance tests were always conducted on the same ergometer. All other training that the rowers were performing supplementary to their ergometer sessions was the same, as they were all part of the same squad preparing for an upcoming regatta. All training was overseen by the same coaches, and all sessions were recorded in a training diary (12 ± 1 sessions/week).

**Procedures**

**GXT.** The GXT was conducted to determine VO₂peak, power corresponding to 4 mmol/L blood lactate concentration (LT), mean 4-min POpeak, and peak heart rate. The GXT was performed according to the Australian Physiological Assessment of Rowing Guidelines (Hahn, Bourdon, & Tanner, 2000), to which the subjects were previously accustomed. According to the guidelines, the starting power output and step increments were related to each subject’s most recent TT time, and the drag factor on the rowing ergometer was adjusted to match his weight class. Subjects performed 7 × 4-min incremental steps, with the last step being an all-out effort. They were asked to maintain their target power output during each step of the test, as visually displayed on the rowing ergometer’s monitor. All stages were followed by 1 min of passive
rest during which a fingertip capillary blood sample was collected to determine blood lactate concentration (Lactate Pro, Arkray, Japan). The test–retest reliability of the Lactate Pro has been previously reported, with technical error of measurement results ranging from 0.1 to 0.4 mmol/L at blood lactate concentrations of 1–18 mmol/L (Tanner, Fuller, & Ross, 2010). Cardiorespiratory-metabolic variables were measured throughout the GXT using the Parvo Medics TrueOne 2400 metabolic analyzer (Parvo Medics, Inc., Salt Lake City, UT). The analyzer was calibrated before each test using alpha gases of known concentration according to the manufacturer’s instructions. \(\text{VO}_{2\text{peak}}\) was taken as the highest \(\text{VO}_2\) value recorded during a 1-min period of the final stage in the GXT. The mean power output achieved during the final 4-min stage was deemed as \(\text{PO}_{\text{peak}}\) and was used to set HIT training intensity.

### 2,000-m TT

The TT was also performed on the air-braked Concept IIc rowing ergometer, as this ergometer is believed to effectively simulate the metabolic and biochemical demands of on-water rowing and can be used to assess rowing performance (Maestu & Jurimae, 2001). The subjects were already familiar with the use of this apparatus and the TT testing procedure before taking part in the study. The test–retest reliability of the TT on a Concept IIc ergometer has been previously reported (Schabort, Hawley, & Blum, 1999). All subjects performed a 10-min self-selected warm-up and stretches before the test, which was recorded to ensure exact replication before the posttraining TT. Power output, stroke rate, and 500-m split times were updated continuously on the computer display of the rowing ergometer during the TT, and average values were presented for each measure at the completion of the TT. Time to complete the TT was recorded as the criterion-dependent variable. There was no verbal encouragement given to the subjects during the test to control psychological motivation. There was no ingestion of \(\text{NaHCO}_3\) in either group during the 72 hr before the TT. All subjects were tested individually and were not aware of the performance times achieved by the other subjects.

### HIT

Both the ALK and PLA groups were prescribed two HIT ergometer-training sessions per week for 4 weeks, thus completing eight HIT sessions. At each HIT session, subjects completed 8 × 2.5-min intervals at 90% of \(\text{PO}_{\text{peak}}\). Between intervals, subjects were instructed to row at 40% of \(\text{PO}_{\text{peak}}\) for 3 min. The HIT protocol used in the current study was adapted from our previous study on HIT in rowers (Driller et al., 2009).

### Supplementation

Before each HIT training session, the ALK group ingested \(\text{NaHCO}_3\) (0.3 g/kg body mass) and the PLA group ingested sodium chloride, taken in capsule form (gelatin capsules). The sodium content in each group was matched for equimolar concentration. Both groups began ingesting their capsules 90 min before each HIT training session. The capsules were taken evenly spaced (five doses) with a standardized meal (cereal bar) over a duration of 60 min to reduce the likelihood of gastrointestinal upset (Carr, Slater, Gore, Dawson, & Burke, 2011). On two separate occasions during the study, 100-μl capillary (fingertip) blood samples were taken before and after loading and after the HIT session, with pH and \([\text{HCO}_3^-]\) measured using the i-STAT blood-gas analyzer (i-STAT, Princeton, NJ). The analyzer was calibrated before each test with known controls and according to manufacturer’s recommendations. The reliability of the i-STAT blood-gas analyzer has been described previously, with strong intra- and interclass correlation coefficients observed (.77–.95) over a range of blood variables during rest and exercise (Dascombe, Reaburn, Sirotic, & Coutts, 2007; Silverman & Birks, 2002). Furthermore, subjects were provided with a modified questionnaire on gastrointestinal side effects at the same time that each blood sample was taken to quantify side effects. There were several items describing possible side effects, each with a 10-point Likert scale ranging from 1 = none to 10 = unbearable.

### Statistical Analysis

To interpret the practical significance of the findings in the current study, we employed analysis combining traditional statistical methods with magnitude-based inferences (effect sizes) and precision of estimation (90% confidence limits). Results for all measured performance variables were compared between groups at baseline using independent unpaired \(t\) tests, with statistical significance set at \(p < .05\) for all analyses. A repeated-measures ANOVA was then used to identify any within-group, between-groups, and group interaction effects on all of the investigated variables (SPSS 10.0, Chicago, IL). Each subject’s change score was calculated as a percentage of baseline score via analysis of log-transformed values, to reduce the bias arising from nonuniformity of error. Standardized changes in the mean of each measure were then used to assess magnitudes of effects and provide the likelihood of the true effects being practically positive, trivial, and negative by dividing the changes by the smallest worthwhile change (Batterham & Hopkins, 2006). The qualitative likelihoods are defined by the following scale: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely or probably not; 25–75%, possibly or maybe; 75–95%, likely or probably; 95–99%, very likely; >99%, almost certainly. Measures of reliability known as coefficient of variation (CVs) were halved and used as the smallest worthwhile change for each of the variables (Hopkins, 2004). As identified previously (Schabort et al., 1999), mean power in a 2,000-m TT has a reported CV of 2%, while time to complete a 2,000-m rowing TT has a 1% CV. The CVs for power output at 4.0 mmol/L lactate threshold (LT) and \(\text{PO}_{\text{peak}}\) were 1% and 2%, respectively, as identified from quality-assurance data maintained for testing protocols used at the Tasmanian Institute of Sport (unpublished observations). To make inferences about the true value of an effect between ALK and PLA, the uncertainty in the effect was expressed as 90% confidence.
limits. To assess the overall effect of the HIT, regardless of intervention, the group performance data were pooled and analyzed using paired t tests. Results are shown as $M \pm SD$ unless stated otherwise.

## Results

There were no significant differences between groups for any of the measured performance variables (2,000-m power, $PO_{peak}$, 4-mmol/L LT) at baseline testing ($p > .05$). As identified by the repeated-measures ANOVA, there were no significant Group $\times$ Time differences between the ALK and PLA groups for any of the performance variables after the intervention period ($p > .05$). When using the magnitude-based analytical approach, there was an unclear/trivial effect on 2,000-m power, 2,000-m time, $PO_{peak}$, and 4-mmol/L LT in the ALK group compared with the PLA group (Table 1), as identified by <75% likelihood of a positive effect, paired with >15% likelihood of a negative effect.

Pooled data from two separate training sessions demonstrated that the NaHCO$_3$ supplementation was effective in significantly increasing [HCO$_3^-$] and pH levels after the loading period (Figure 1). In addition, pH levels remained significantly higher ($p = .007$) in the ALK group posttraining (Figure 1[b]). There were no significant differences in the training intensities or volumes between the two groups, and there were no reported incidences of gastrointestinal upset for either group throughout the training period.

When performance data for the two groups were pooled to analyze the change in performance regardless of intervention, there were significant improvements in 2,000-m time ($1.4\% \pm 1.4\%, p = .008$), 2,000-m power ($4.5\% \pm 4.5\%, p = .006$), $PO_{peak}$ ($3.7\% \pm 3.3\%, p = .002$), and LT ($4.6\% \pm 4.9\%, p = .02$) over the duration of the study.

The occurrence of gastrointestinal upset after NaHCO$_3$ loading was minimal in the current study, with only two instances of subjects reporting any side effects on the 10-point Likert scale. Both subjects reported mild bloating (3 and 2) and nausea (2 and 1) after acute supplementation. One of the athletes to report side effects felt it was more appropriate to use such a performance test in the current study.

## Discussion

The current study has shown that despite improvements in performance in highly trained rowers during the training period, the addition of NaHCO$_3$ supplementation before the HIT sessions did not provide any statistically significant additional benefits to rowing performance compared with the PLA group ($p > .05$). The findings were irrespective of clearly influenced blood variables before (pH and HCO$_3^-$) and after (pH) training sessions in the ALK group. When using a magnitude-based inferential approach, as is often used when studying small but highly trained athletic populations (Batterham & Hopkins, 2006), there was an unclear/trivial effect on mean 2,000-m performance in the ALK group.

The current findings do not support those from the only previous human studies on the effect of chronic NaHCO$_3$ loading in conjunction with HIT (Edge, Bishop, & Goodman, 2006). Edge, Bishop, and Goodman reported a novel finding that significantly greater improvements in a time-to-fatigue test occurred after a period of HIT combined with NaHCO$_3$ supplementation when compared with a placebo group (164% vs. 123%, respectively) in 16 recreationally trained female subjects. The improvement in performance was proposed to be a result of a significantly improved LT evident in the NaHCO$_3$ group compared with the placebo group (26% vs. 15%, respectively). As hypothesized by Edge, Bishop, and Goodman, an increase in LT may have been due to the positive effects of reducing H$^+$ accumulation during training on changes in mitochondrial respiration (Ivy, Withers, Van Handel, Elger, & Costill, 1980). Similar findings were reported by Bishop et al. (2010), whereby male Wistar rats that were supplemented with NaHCO$_3$ during 5 weeks of HIT lasted significantly longer in a submaximal time-to-fatigue test than did a placebo group (81.2 $\pm$ 24.7 min and 53.5 $\pm$ 30.4 min, respectively). Bishop et al. concluded that the most likely mechanisms for this performance improvement were reduced mitochondrial protein degradation and/or greater protein synthesis, aided by the higher pH level in the alkalotic condition during the HIT. In the current study, however, there were no significant improvements in 4-mmol/L LT power or 2,000-m rowing time trial in the ALK group. A longer duration performance test may have been more responsive to chronic NaHCO$_3$ supplementation, as the only other previous studies that have shown performance benefits from chronic NaHCO$_3$ ingestion and HIT employed much longer tests (Bishop et al., 2010; Edge, Bishop, & Goodman, 2006). In contrast to our performance test (lasting ~6 min), those previous studies used performance tests that lasted ~20 min, suggesting that chronic NaHCO$_3$ supplementation may be more beneficial to exercise requiring a greater aerobic energy contribution than a 2,000-m rowing time trial. However, given the inherently large CVs often associated with time-to-fatigue tests (Currell & Jeukendrup, 2008) and the specificity of the 2,000-m time trial to rowers, we felt it was more appropriate to use such a performance test in the current study.

Another plausible reason for the lack of difference between the two groups in the current study may be that the athletes investigated would need more than 4 weeks of HIT combined with NaHCO$_3$ supplementation to see any performance improvements in such a highly trained group. Given that Edge, Bishop, and Goodman (2006) used an 8-week intervention period with recreational subjects and Bishop et al. (2010) used 5 weeks of training and supplementation in untrained rats, a longer intervention period may be required to bring about performance
Table 1  Results of the 2,000-m Performance Trial and Graded Exercise Test Pre- and Posttraining in ALK and PLA Groups, $M \pm SD$

<table>
<thead>
<tr>
<th>Variable</th>
<th>ALK</th>
<th>PLA</th>
<th>ANOVA Group × Time interaction, $p^a$</th>
<th>$\Delta ALK – \Delta PLA$ raw difference (90% CL)</th>
<th>Likelihood of ALK being positive, trivial, or negative (compared with PLA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2,000-m power (W)</td>
<td>370.0 ± 18.3</td>
<td>387.8 ± 18.9</td>
<td>367.3 ± 16.5</td>
<td>382.5 ± 16.9</td>
<td>.80 2.6 (–2.8 to 8.0)</td>
</tr>
<tr>
<td>2,000-m time (s)</td>
<td>392.8 ± 6.6</td>
<td>386.8 ± 6.7</td>
<td>393.7 ± 5.8</td>
<td>388.5 ± 5.9</td>
<td>.84 –0.7 (–2.4 to 0.9)</td>
</tr>
<tr>
<td>Peak power output (W)$^a$</td>
<td>398.3 ± 18.7</td>
<td>414.8 ± 22.9</td>
<td>396.5 ± 15.4</td>
<td>408.3 ± 9.5</td>
<td>.60 4.2 (–12.2 to 20.5)</td>
</tr>
<tr>
<td>4-mmol/L LT power (W)</td>
<td>279.2 ± 32.9</td>
<td>293.3 ± 31.4</td>
<td>267.8 ± 28.9</td>
<td>277.0 ± 23.1</td>
<td>.62 5.0 (–9.1 to 19.1)</td>
</tr>
</tbody>
</table>

*Note.* ALK = sodium bicarbonate group; PLA = sodium chloride group; CL = confidence limits; LT = lactate threshold.

*a*Mean 4-min all-out power achieved in the final stage of the progressive exercise test.
improvements, especially in already highly trained athletes. Unfortunately, the constraints associated with studying a highly trained athletic population meant that we were limited by a small sample size, brought together for a short training period in preparation for international competition, and the performance tests used had to comprise a regular part of the prescribed testing for their sport, which could not be altered.

A potential reason for the unclear findings of the current study may be that previous research has shown that highly trained athletes have more efficient muscle buffering capacity than recreational athletes or sedentary subjects (Edge, Bishop, Hill-Haas, Dawson, & Goodman, 2006; Sahlin & Henriksson, 1984). Furthermore, although it was not measured in the current study, rowing athletes have been shown to have elevated buffering capabilities compared with endurance-trained and untrained subjects (Parkhouse et al., 1985). It has been suggested that gains in muscle buffering capacity can be achieved more readily in untrained subjects than in highly trained athletes (Edge, 2006). Indeed, Edge, Bishop, and Goodman (2006) confirmed this when their subjects with higher pretraining muscle buffering capacity also had smaller increases posttraining than did those with a lower initial muscle buffering capacity. Consequently, there is a possibility that the reason for the improved performance shown by the NaHCO3-supplemented group in the Edge, Bishop, and Goodman study was that, although not significantly different, the ALK group had a lower initial muscle buffering capacity than the placebo group (approximately 145 vs. 157 μmol H+ · g muscle dry weight –1 · pH –1, respectively). The lower initial muscle buffering capacity could lead to larger potential for improvement of this variable, which could have subsequently translated to performance benefits. Therefore, the long-term consequences of repeated exercise-induced metabolic acidosis may not include as much of a detrimental effect on the mitochondrial functions in highly trained athletes than for their less-trained counterparts.

**Figure 1** — (a) Blood [HCO₃⁻] and (b) mean blood pH pre- and postloading and immediately after high-intensity interval training for the NaHCO₃-supplemented (ALK) and placebo (PLA) groups, M ± SD. Data are pooled from two separate training sessions. *Significantly different from PLA (p < .05).
Changes in blood-gas variables during training were similar to previous findings (Edge, Bishop, & Goodman, 2006; Siegler, Keatley, Midgley, Nevill, & McNaughton, 2008), suggesting that the NaHCO3 supplementation and dose regimen was not a limiting factor behind the lack of difference in performance improvements between the two groups in the current study (Figure 1).

Irrespective of which group subjects were in, pooled performance data from the current study showed a mean improvement of 5.6 s in 2,000-m rowing time. Although there was no control group for comparison, the HIT has potentially contributed to this improvement, as witnessed in our previous study in well-trained rowers (Driller et al., 2009). Furthermore, these improvements are even more impressive given the highly trained nature of the athletes in the current study and the peak phase of their training when the study was conducted.

A limitation of the current study was the relatively small sample, which was limited by the number of high-caliber athletes who were brought together for a short training period in preparation for an international competition. While the addition of rowers of a lower standard would have improved the sample size, we were reluctant to add rowers who were not of the same standard, given the primary aim of the current study, as well as previous reports indicating significant differences in power output and skill levels between international- and national-level rowers (Smith & Spinks, 1995).

Future research should employ longer periods of chronic NaHCO3 supplementation (e.g., >8 weeks) to determine any effectiveness in highly trained athletes. Furthermore, the current study matched the training intensity for the two groups, as was the case in the only previous study to investigate chronic NaHCO3 supplementation combined with HIT (Edge, Bishop, & Goodman, 2006). To our knowledge, there have been no published studies examining whether chronic NaHCO3 loading during training might enable athletes to train harder and therefore achieve greater levels of training adaptation. A potential benefit from NaHCO3 loading may only be realized if the training load is not limited, enabling athletes to possibly train harder in the supplemented state. If multiple bouts of acute NaHCO3 loading before training were to lead to increases in training volume or intensity, this increased training dose could lead to enhanced muscular and metabolic adaptations and, therefore, performance. This area clearly warrants future investigation.

Conclusion

The current study showed that 4 weeks of NaHCO3 supplementation before HIT training in highly trained rowers does not provide any statistically significant benefits to performance compared with a control group (p > .05). However, regardless of intervention, these highly trained rowers improved 2,000-m power by an average of 16.4 W over the 4-week training period, which can be possibly attributed to the HIT. The improvement in mean power translates to a 5.6-s decrease in 2,000-m time. These results are impressive, given the level of the athletes and phase of their season when the study took place.

Acknowledgments

The funding for this study was provided by a research grant obtained from the Australian Institute of Sport.

References


