Sodium Bicarbonate Supplementation and Ingestion Timing: Does It Matter?

AU1 Jason C. Siegler,[1] Paul Marshall,[1] James Bray,[2] and Chris Towlson[2]

[1]School of Biomedical and Health Sciences, University of Western Sydney, Campbelltown, NSW, Australia; and [2]Department of AU2 Sport, Health, and Exercise Science, University of Hull, Hull, United Kingdom

Abstract

Siegler, JC, Marshall, P, Bray, J, and Towlson, C. Sodium bicarbonate supplementation and ingestion timing: Does it matter? *J Strength Cond Res* 25(X): 000–000, 2011– Although a considerable amount of literature exists on the ergogenic potential of ingesting sodium bicarbonate ($NaHCO_3$) before short-term, high-intensity exercise, very little exists on optimal loading times before exercise. The purpose of this study was to determine the influence of $NaHCO_3$ supplementation timing on repeated sprint ability (RSA). Eight men completed 3 (randomized and counterbalanced) trials of ten 10-second sprints separated by 50 seconds of active recovery (1:5 work-to-rest) on a nonmotorized treadmill. Before each trial, the subjects ingested 0.3 $g \cdot kg^{-1}$ body weight of $NaHCO_3$ at 60 (H1), 120 (H2), or 180 (H3) minutes before exercise. AU4 Additionally, the subjects were assessed for any side effects (gastrointestinal [GI] discomfort) from the $NaHCO_3$ ingestion via a visual analog scale (VAS). Blood buffering was assessed using a 2-way analysis of variance (ANOVA) with repeated measures, whereas repeated sprint performance and GI discomfort were assessed via a 1-way ANOVA with repeated measures. Blood-buffering capacity was not different at preexercise times (HCO_3^- [millimoles per liter] H1: 30.2 ± 0.4, H2: 30.9 ± 0.6, H3: 31.2 ± 0.6; $p > 0.74$). Average speed, average power, and total distance covered progressively declined over the 10 sprints; however, there was no difference between conditions ($p > 0.22$). The incidence of GI discomfort was significantly higher ($p < 0.05$) from preingestion at all time points with the exception of 180 minutes, whereas severity was only different between 90 and 180 minutes. Ingestion times (between 60 and 180 minutes) did not influence the blood buffering or the ergogenic potential of $NaHCO_3$ as assessed by RSA. However, VAS scores indicated that at 180 minutes postingestion, an individual is less prone to experiencing significant GI discomfort.

Key Words blood buffering, loading protocols, metabolic alkalosis, ergogenic

Introduction

The practice of inducing an alkalotic shift in acid-base balance to improve performance before supramaximal exercise has been widespread in elite sport for nearly 30 years (10,11,13). The most common method reported in the scientific literature, and perhaps the most effective, is via the ingestion of sodium bicarbonate ($NaHCO_3$) (13). Ingesting an amount of 0.3 $g \cdot kg^{-1}$ body weight (BW) of this buffer will augment the concentration of the body's primary blood-buffering source, that is, bicarbonate (HCO_3^-), by approximately 5–6 $mmol \cdot L^{-1}$ (16,17,19). This increase in the concentration of blood HCO_3^- is considered to be sufficient to provide a supplemental blood-buffering pool, which in turn may enhance an individual's capacity to combat excessive proton (H^+) production during prolonged high-intensity exercise. Improving the capacity for H^+ removal from the contracting skeletal muscle cell corresponds with the maintenance of intracellular function, specifically adenosine triphosphate resynthesis and Ca^{2+} resequestering within the sarcoplasmic reticulum (1,20). AU5

In practice, metabolic alkalosis via $NaHCO_3$ ingestion is most commonly achieved via the oral administration of the buffer, either through encapsulation or mixing the substance in a palatable liquid (e.g., sport drinks, cordial, flat cola) (3,19). Often, however, inducing such dramatic alterations in acid-base balance is not without consequence. The physiochemical response to relatively large quantities of $NaHCO_3$ entering the stomach results in the Na^+ absorption in the small intestine and increased plasma Na^+ levels (6). This exchange of Na^+ leads to an increase in the stomach PCO_2 and diffusion of CO_2 into plasma and increases the already acidic environment within the stomach (6). This temporary imbalance often results in unfavorable side effects such as stomach bloating, nausea, and diarrhea. The incidence of these side effects appears to vary widely between individuals, ranging from mild discomfort to extreme cases where

AU3 Address correspondence to Jason C. Siegler.

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exercise performance may be affected. For example, dosages of 0.3 $g \cdot kg^{-1}$ BW ingested as a single bolus have been reported to cause gastrointestinal (GI) disturbances in some individuals (12,16), whereas in 1992, McNaughton also reported that all subjects experienced side effects at dosages of 0.4 $g \cdot kg^{-1}$ BW and 0.5 $g \cdot kg^{-1}$ BW (12). Consideration of this issue is essential when preparing an athlete for competition under supplemental buffering conditions, because GI disturbances may reduce an individual's perceived physical and mental readiness to perform, and any potential ergogenic benefit of $NaHCO_3$ may be diminished if an athlete is preoccupied or unfocused. With respect to the aforereferenced studies, documentation of the most common side effects associated with $NaHCO_3$ ingestion has been sporadic in the literature (2,3,11,13), and a more objective representation of the relationship between GI discomfort, the loading and dosage sequence, and performance is needed.

One possible approach for reducing the likelihood of GI discomfort having a negative impact on performance might be to either prolong the ingestion time period or increase the time between ingestion and exercise. This practice is not evident in the literature, as a recent review of the $NaHCO_3$ supplementation literature has indicated that a majority of studies (15 out of 16 since the year 2000) had ingestion protocols that required consumption 60–90 minutes before exercise (13). Within these studies, the authors commonly cited original work in this area that reported on the efficacy of various loading doses (8,12). For instance, Horswill et al. reported that dosages of $\leq$0.2 $g \cdot kg^{-1}$ BW were ineffective for improving sprint performance, whereas McNaughton reported that with dosages of 0.1, 0.2, 0.3, 0.4, and 0.5 $g \cdot kg^{-1}$ BW, only 0.1 $g \cdot kg^{-1}$ BW did not significantly enhance sprint performance (8,12). Both of these studies used 60 minutes postingestion as the commencing point for exercise and justified this by indicating that peak blood buffering occurred at this time (8,12). As a result, researchers have consistently presumed that commencing exercise at peak levels of blood buffering is of greater importance in terms of performance outcomes than delaying exercise because of any GI disturbances that may be present. However, to our knowledge, there is no evidence in the literature that suggests that performance is optimized at peak blood HCO_3^- concentrations after supplementation when compared with other time points throughout the loading period.

In light of this gap in the literature, we documented a sustained elevation in blood HCO_3^- after both 0.2 and 0.3 $g \cdot kg^{-1}$ BW for approximately 4 hours postingestion under resting conditions (19). Although slightly lower than peak levels ($\sim$1 $mmol \cdot L^{-1}$), these levels were still 5–6 $mmol \cdot L^{-1}$ above preingestion values during hours 2–4 and well within the performance-enhancing buffering ranges reported by Matson and Tran (11). However, no evidence was provided to demonstrate that performance would still be enhanced 2–4 hours post $NaHCO_3$ ingestion. Moreover, it is reasonable to believe that because symptoms of GI discomfort peak 60–90 minutes post $NaHCO_3$ ingestion, a longer time period (i.e., 2–4 hours) between ingestion and physical performance may minimize GI discomfort while maintaining performance gains. As such, the primary aim of this study was to determine the influence of supplementation timing (60, 120, 180 minutes before) on repeated sprint ability (RSA) after a standard $NaHCO_3$ load (0.3 $g \cdot kg^{-1} \cdot BW$). A secondary aim was to profile both blood-buffering capacity and GI discomfort over these time periods to determine whether any relationship exists between the magnitude of GI discomfort, blood-buffering levels, and RSA. It was hypothesized that (a) RSA would be sustained regardless of the ingestion timing period and (b) peaks in blood-buffering capacity would correspond with maximal GI discomfort.

Methods

Experimental Approach to the Problem

The study used a randomized (counterbalanced), repeated measures design in which subjects reported to the temperature-controlled laboratory (19–23°C) at the same time of the day between 0900 and 1200 on 4 occasions, each separated by 1 week. The subjects were advised to limit strenuous exercise for 24 hours before testing and to attend each trial adequately hydrated (e.g., a minimum water consumption of 500 ml approximately 3 hours before the trial was recommended to all the subjects). On the initial visit, the subjects undertook a full familiarization session with the nonmotorized treadmill (NMT) and exercise protocol following the methods of Tong and coworkers (9,21). The subjects were also provided instruction on the subjective interpretation of GI discomfort using a visual analog scale (VAS). The VAS questionnaire has been used previously in the $NaHCO_3$ literature (2) and is a commonly accepted tool for documenting subjective pain perception and discomfort (15). The 3 trial conditions (60 minutes $NaHCO_3$ ingestion time [H1], 120 minutes $NaHCO_3$ ingestion time [H2], and 180 minutes $NaHCO_3$ ingestion time [H3]) were implemented in a randomized manner and each separated by 1 week. Because of the varying time ratios (ingestion sequence [60, 120, or 180 minutes] to commencement of exercise) between the 3 conditions, blinding the subjects was not possible. Additionally, including another 3 placebo trials to correspond with the 3 $NaHCO_3$ conditions would have further confounded the repeated measures design with such a small sample size ($n = 8$ was determined sufficient a priori [>0.8] using previously published alkalotic changes in blood buffering after $NaHCO_3$ supplementation [5,22]). Because the primary aim of the study was to distinguish whether different $NaHCO_3$ loading protocols affected performance and *not* the efficacy of $NaHCO_3$ ingestion, this methodological approach was chosen to best match the study objectives. For all trials, the buffer solution (0.3 $g \cdot kg^{-1}$ BW) was dispensed into gelatine capsules and consumed with 750 ml of water over a 15-minute period.

Subjects

Eight recreationally active and healthy male subjects (mean ± *SD*: height, 180 ± 9 cm; body mass, 76.2 ± 9.1 kg; age, 22 ± 2 years) volunteered to take part in the study after being informed verbally and in writing as to the nature and risks associated with the study. The subjects were free of any cardiac or metabolic diseases, involved in regular aerobic training 3 times per week (~4–6 hours total), did not smoke, and refrained from supplementation of all kinds (i.e., vitamins, ergogenic aids, etc.) during the testing period. All the subjects signed an informed consent, and the study was approved by the Departmental Human Ethics Committee and following the principles outlined by the Declaration of Helsinki.

Procedures

Upon arriving at the laboratory for each trial, whole blood (preingestion sample) was collected in a balanced heparin 200-μL blood gas capillary tube for immediate analysis of acid-base balance (pH, bicarbonate [HCO_3^-], base excess [BE]) using a clinical blood gas analyser (OMNI 4 Blood Gas Analyser, Roche Diagnostics Ltd., Sussex, United Kingdom). All measurements were performed in duplicate, and the range of intraclass correlation coefficients (ICCs) was 0.85–0.97, $p < 0.01$ for all dependent blood variables, respectively. AU6 After the preingestion sample was obtained, the subjects were allowed 15 minutes to consume the gelatine capsules and were then required to sit quietly in the laboratory until their performance trial (e.g., either 60, 120, or 180 minutes).

During the ingestion period, the subjects were asked to rate any GI discomfort they were experiencing using the VAS every 30 minutes until the commencement of exercise. The symptoms provided to each participant consisted of the following: nausea, flatulence, stomach cramping, stomach bloating, stomach ache, belching, vomiting, bowel urgency, and diarrhea (2). The VASs consisted of 9 separate 100-mm scales, anchored at each end with 'no symptom' on the left hand and 'severe symptom' on the right hand. The subjects were instructed to indicate with a vertical mark the severity of each of the 9 symptoms presented on the scales.

Once the ingestion period concluded, a preexercise capillary sample was obtained, and the subjects completed a standardized warm-up consisting of a 10-minute series of short, high-intensity bouts on a motorized treadmill (Ergo ELG 55, Woodway GmbH, Weil am rhein, Germany) and light, active stretching. The performance trials required a standing start for ten, 10-second maximal efforts on an NMT (Woodway Force, Woodway GmbH, Weil am rhein, Germany). Each 10-second maximal effort was separated by 40 seconds of walking at 5 $km \cdot h^{-1}$, then 10-second standing in preparation for the next maximal effort (work-to-rest ratio 5:1). The performance data were acquired from the treadmill using a customized interface (XPV7 PCB, Interface, Fitness Technologies, Adelaide, Australia) and analyzed at 20 Hz using Force 3 Software (Innervations, Joondalup, Australia). A priori reliability measures for power and speed outputs on the NMT provided a range of ICCs between 0.88 and 0.99, $p < 0.01$, respectively. Upon completing the protocol, a final 1-minute postexercise capillary blood sample was obtained.

Statistical Analyses

All statistical analyses were completed using Statistica Software, Tulsa, OK, USA, and GraphPad Prism 5.0, San Diego, CA, USA. A 2-way analysis of variance (ANOVA) for repeated measures (condition × time) was used to analyze the differences in blood acid-base balance (pH, HCO_3^-, BE). The performance (peak speed [PS] and average speed [AS] in meters per second, peak power [PP] and average power [AP] in watts, and total distance [TD] covered in meters) and GI discomfort (incidence and severity) data for each trial were analyzed using a 1-way ANOVA for repeated measures. The severity data were not normally distributed and thus analyzed using the Friedman statistic and where significant Dunn's posttest for comparison of the sum of ranks between groups. In all other instances, Tukey's honestly significant difference was performed in the event of a significant *F* ratio. Two-tailed statistical significance was accepted at $p < 0.05$. When significant differences are stated, the mean difference plus the 95% confidence interval (CI) of the mean difference are provided. The Cohen effect size (*d*) is also provided, which was calculated and interpreted as recommended by Hopkins (7). Briefly, Hopkins suggests that the effect size be interpreted as 0–0.19 (trivial), 0.20–0.59 (small), 0.60–1.19 (moderate), 1.20–1.99 (large), and 2.0 + (very large) (7).

Results

Blood Acid-Base Response

Table 1 corresponds to the mean ± *SD* values for pH, HCO_3^-, and BE measured in the blood during each of the 3 experimental trials before $NaHCO_3$ ingestion (Pre-Ing), before exercise (Pre-Ex), and 1 minute postexercise (Post-Ex). There were no significant interactions ($p > 0.80$) or main effects for condition ($p > 0.74$) for any of the acid-base variables (pH, HCO_3^-, BE); however, there was a main effect for time for all variables ($p < 0.0001$), respectively. At Pre-Ex time points, buffering capacity was significantly elevated from Pre-Ing as indicated by changes in pH (mean difference = 0.04; 95%CI = 0.08–0.01; $d = 2.65$; $p < 0.05$), HCO_3^- (mean difference = 4.8 $mmol \cdot L^{-1}$; 95%CI = 5.9–3.7; $d = 3.86$; $p < 0.001$), and BE (mean difference = 5.6 $mEq \cdot L^{-1}$; 95%CI = 7.1–4.1; $d = 3.68$; $p < 0.001$). As expected, Post-Ex acid-base balance was significantly depleted compared with Pre-Ex (pH: mean difference = 0.30; 95%CI = 0.34–0.27; $d = 4.83$; $p < 0.001$; HCO_3^-: mean difference = 18.2 $mmol \cdot L^{-1}$; 95%CI = 19.3–17.1; $d = 9.48$; $p < 0.001$; BE: mean difference = 23.1 $mEq \cdot L^{-1}$; 95%CI = 24.6–21.6; $d = 8.99$; $p < 0.001$). T1

Performance Outcomes

There was no difference between trials for either PS (H1: 5.47 ± 0.45 $m \cdot s^{-1}$; H2: 5.54 ± 0.73 $m \cdot s^{-1}$; H3: 5.62 ± 0.65 $m \cdot s^{-1}$;

Table 1. Mean ± *SD* data for all acid-base (pH, HCO_3^-, and BE) variables for each of the 3 sodium bicarbonate conditions.*†

Trial	pH			HCO_3^- (mmol·L^{-1})			BE (mEq·L^{-1})		
	H1	H2	H3	H1	H2	H3	H1	H2	H3
Pre-Ing	7.414 ± 0.010	7.413 ± 0.014	7.416 ± 0.023	26.1 ± 0.7	25.7 ± 1.2	26.0 ± 0.8	2.2 ± 1.0	1.7 ± 1.7	1.9 ± 1.1
Pre-Ex	7.454 ± 0.019	7.461 ± 0.024	7.458 ± 0.015	30.2 ± 1.3	30.8 ± 1.7	31.2 ± 1.7	6.9 ± 1.5	7.5 ± 2.0	8.0 ± 1.9
Post-Ex	7.148 ± 0.097	7.173 ± 0.075	7.150 ± 0.107	12.3 ± 2.2	13.0 ± 2.3	12.5 ± 2.7	−15.8 ± 3.1	−14.9 ± 3.4	−15.6 ± 3.8

*Pre-Ing = preingestion; Pre-Ex = pre exercise; Post-Ex = postexercise; BE = base excess; HCO_3^- = bicarbonate.
†For all the trials, the time points (Pre-Ing, Pre-Ex, and Post-Ex) were different from each other for pH, HCO_3^-, and BE ($p < 0.0001$); however, there were no differences between conditions ($p > 0.74$). H1, H2, and H3 represent 60, 120, and 180 minutes postingestion, respectively.

$p = 0.31$) or PP (H1: 984 ± 208 W; H2: 916 ± 131 W; H1: 987 ± 228 W; $p = 0.18$). Although there was no significant interaction or difference between conditions (AS: $p > 0.22$; AP: $p > 0.86$), there was a decline over the 10 sprints in both AS (largest mean difference = 0.12; largest 95%CI = 0.01–0.21; d range = 0.3–0.5; $p < 0.05$; Figure 1) and AP (largest mean difference = 44; largest 95%CI = 0.9–80.1; d range = 0.3–0.5; $p < 0.05$; Figure 1). Similar to PS and PP, the TD covered over the 10 sprints for the 3 trials was not different (H1: 456 ± 35 m; H2: 448 ± 32 m; H3: 448 ± 32 m; $p = 0.22$). F1

Gastrointestinal Discomfort

The incidence of GI discomfort was significantly higher than the Pre-Ing values at all time points with the exception of 180 minutes (largest mean difference = 4.8; largest 95%CI = 0.3–7.3; d range = 1.6–2.1; $p < 0.05$; Figure 2). Although the Friedman test indicated a significant main effect for time ($p < 0.001$), after decomposition, the only difference appeared between 90 and 180 minutes ($p < 0.01$; Figure 2). F2

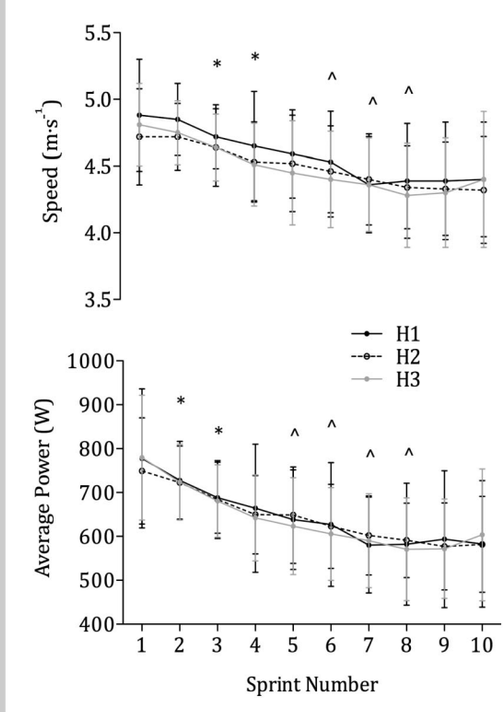

Figure 1. Represented in the following figure are the mean ± *SD* average speed and power data for all the 3 conditions (60 [H1], 120 [H2], and 180 minutes [H3] postingestion of 0.3 g·kg^{-1} BW of sodium bicarbonate). *Indicates significant difference from previous sprint ($p < 0.05$). ^Indicates significant difference from the 2 sprints before exercise (e.g., sprint 4 vs. 6) ($p < 0.05$). AU7

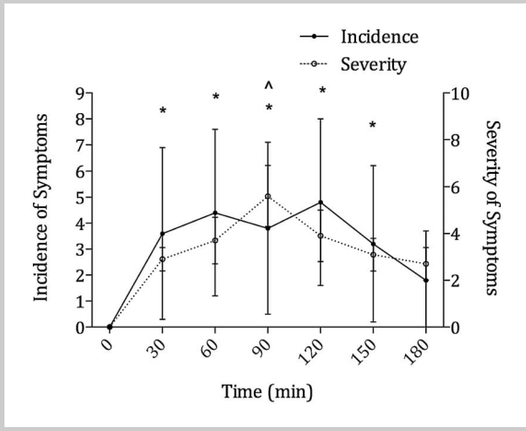

Figure 2. Represented in the following figure are the mean ± *SD* scores for both the incidence and severity of symptoms over 180 minutes after ingestion of 0.3g·kg^{-1} BW of sodium bicarbonate. *Indicates incidence rates that are significantly different from that at 180 minutes. ^Indicates severity significantly different from that at 180 minutes.

Discussion

The primary aim of this investigation was to determine whether ingesting a standard 0.3 g·kg^{-1} BW of $NaHCO_3$ at varying time points would influence RSA. The novel findings of this study were that (a) performance during repeated bouts of high-intensity exercise is not different when ingesting 0.3 g·kg^{-1}·BW of $NaHCO_3$ between 60 and 180 minutes before exercise; (b) metabolic alkalosis (as defined by an elevated blood [HCO_3^-] of ~5 mmol·L^{-1}) is maintained and not statistically different between 60 and 180 minutes postingestion; and (c) the incidence and severity of GI discomfort are reduced at 180 minutes compared with that at all previous time points postingestion, without compromising acid-base balance or performance. The finding that GI discomfort is reduced at 180 minutes postingestion with similar physical performance outcomes is an important consideration when recommending $NaHCO_3$ as an ergogenic substance to athletes.

The proposed relationship between GI discomfort, $NaHCO_3$ supplementation, and performance is arguably a deciding factor when considering the use of this buffer in a sporting context. The importance of understanding this association may be evidenced by the recent increase in $NaHCO_3$ research related to this very question (2,3,16). At present, however, only the study of Carr et al. has gone beyond simply reporting the symptom-performance relationship to propose alternative loading strategies (e.g., consumption of carbohydrates in conjunction with $NaHCO_3$) (3). In this study, instead of attempting to attenuate symptoms by introducing alternative dietary regimens, we delayed the onset of exercise beyond the typical 60–90 minutes ingestion time sequence (13). Our premise was based on the evidence that blood buffering remains elevated to ergogenic levels after $NaHCO_3$ ingestion of 0.3 g·kg^{-1} BW for a period of 4 hours postingestion (19). Indeed, Pre-Ex blood-buffering levels in this study (~4.8 mmol·L^{-1}; 95%CI = 5.9–3.7) were within these predefined ergogenic ranges regardless of the Pre-Ex time point (60, 120, or 180 minutes) (11). If the approximate 5 mmol·L^{-1} increases in HCO_3^- are sufficient for invoking an ergogenic benefit, then this might also explain the similar fatigue pattern observed during the repeated sprint sequence (Figure 1).

The GI discomfort did not seem to have had a direct impact on RSA during this study. If the GI discomfort experienced by the subjects were to negatively impact the performance, then we would have expected to see an improvement in RSA as ingestion time was prolonged, particularly in the 180-minute condition in which symptoms had become negligible. Alternatively, reduced GI discomfort at 180 minute postingestion with no difference in physical performance subsequent to supplementation is a positive result for the recommendation of $NaHCO_3$ supplementation to trainers and athletes who may otherwise be averse to performing strenuous exercise while symptoms are present. The GI discomfort data must be interpreted with caution, however, because there was a large degree of variability between subjects (as indicated by the large *SD*s in Figure 2). This idea that $NaHCO_3$ supplementation causes differing levels of GI discomfort may also coincide with the often-mentioned 'responders' vs. 'nonresponders' issue in terms of performance (13,16,18). Why the temporary imbalance of carbon dioxide (CO_2) levels in the stomach induced by the $NaHCO_3$ ingestion causes discomfort in some individuals and not in others remains to be determined (6). However, this issue further illustrates the need to determine an optimal $NaHCO_3$ loading sequence. Although it did not affect our participant population of recreationally trained athletes, this might not be the case for all populations (e.g., elite athletes), particularly when considering this application before competition where mental preparation may be just as influential to performance outcomes as a physiologically enhanced blood-buffering state (4,14).

In conclusion, performance during repeated bouts of high-intensity exercise was not different when ingesting 0.3 g·kg^{-1} BW of $NaHCO_3$ between 60 and 180 minutes before exercise. This might have been a direct result of metabolic alkalosis remaining significantly elevated for up to 180 minutes postingestion. Finally, GI discomfort, even when elevated from 30 to 150 minutes postingestion, did not negatively impact performance or compromise the acid-base balance.

Practical Applications

It is essential that both athletes and coaches define the energy requirements of their event before including alkalinizing agents into training and competition. For example, it may be appropriate to apply such practice to high-intensity events (e.g., 400 m up to middle distance) that induce large intracellular and extracellular H^+ accumulation but not within

more endurance-based activity (e.g., 4-hour endurance ride). Considering the optimal loading sequence will require a trial-and-error approach; however, as indicated in both this study (performance related) and other $NaHCO_3$ loading studies (4,13,16,17), there appears to be approximately 3–4 hours after the initial supplementation where the ergogenic benefit will remain present. Delaying the onset of exercise will also minimize the likelihood of GI discomfort (Figure 2) and yet will still provide optimal blood-buffering potential.

Athletes and practitioners should approach this supplement with caution; however, because there is a large degree of within-subject variability in terms of responsiveness (Figure 1). We would advocate experimenting with different loading sequences and strategies before use in competition. We have also recently illustrated that, although not detrimental, it is very common to observe a 'responder,' 'nonresponder' phenomena (4). One reason for this may be that there are many different factors that can contribute to performance (and in this case fatigue); certain athletes may be more susceptible to decreases in pH (or becoming acidic) than are others (14). Another possible reason is the issue of ingestion timing. Getting the balance right between maximizing the extracellular buffering pool while minimizing any potential GI side effects is crucial to determining whether $NaHCO_3$ can become an effective addition to an athlete's training or competition schedule.

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