Ergogenic Effects of Sodium Bicarbonate

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MCNAUGHTON, L.R., J. SIEGLER, and A. MIDGLEY. Ergogenic effects of sodium bicarbonate. *Curr. Sports Med. Rep.*, Vol. 7, No. 4, pp. 230–236, 2008. *Athletes use many different strategies to enhance their performance, including clothing and footwear, training regimes, diets, and ergogenic aids. The use of ergogenic aids is believed to be widespread, with a variety of legal as well as illegal substances being used previously and currently. Among the more popular ergogenic aids is the use of sodium bicarbonate or sodium citrate, collectively recognized as "buffers." These substances potentially provide the body with added resistance against fatigue caused by deleterious changes in acid-base balance brought about by a variety of exercise modes and durations. The popularity of buffering has generated a plethora of research dating back to the 1930s, which continues to date. The issues surrounding buffering revolve around the dosage size, timing of ingestion, and the type of exercise to benefit from the use of buffers. We hope this review addresses these pertinent issues.*

INTRODUCTION

Fatigue, typically defined as the failure to maintain an expected or required force or power output (1), is multifaceted, having both physiological and psychological components (2,3). During various forms of activity, potential contributors to fatigue could be related to muscle energy production, for example, a decline in muscle adenosine triphosphate (ATP) or impaired electrochemical events of muscle contraction/relaxation production (4,5). Alternatively, fatigue could be related to the accumulation of metabolites. During prolonged sub maximal effort (~2 h to multi-day events), energy substrate depletion is generally regarded as the major cause of fatigue, but a number of other factors such as hyperthermia, dehydration, and oxygen transport deficiencies also may contribute in differing amounts.

This review examines the literature regarding the use of sodium bicarbonate and sodium citrate as ergogenic agents to overcome the acute fatigue process in a variety of exercise modes. It builds upon a previous review from our laboratory (6).

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Fundamentals of Acid-Base Balance

In a normally functioning, resting human being, arterial blood pH is approximately 7.4, slightly alkalotic, and human muscle is typically pH 7.0. In a stressed human, one who has been exercising or eating for example, there is a dynamic interplay between those systems that would move pH away from normal and those that would regulate pH toward normal. After strenuous exercise, arterial pH may fall to 7.1, while muscle decreases to pH 6.8. In the body, there is and must be a balance between the formation of hydrogen ions and the removal of hydrogen ions for homeostasis to be maintained.

The body has three basic mechanisms for adjusting and regulating acid-base balance. First, chemical buffers adjust H+ within seconds. Also, pulmonary ventilation excretes H+ through the reaction:

$$H^+ + HCO_3 \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$$

adjusting the H^+ within minutes. Finally, the kidneys excrete H^+ as fixed acid and work on a long-term basis to maintain acid-base balance. The discussion surrounding blood pH regulation during exercise has generally focused upon the role of bicarbonate, since it can accept a proton to form carbonic acid in the following equation:

$$H^+HCO3^- \leftrightarrow H_2CO_3$$

When metabolism produces an acid such as lactic acid, which is much stronger than carbonic acid, a proton is liberated, binds with bicarbonate, and forms sodium lactate and carbonic acid. Eventually, this forms carbon dioxide and water.

The kidneys and the lungs are the most important organs of the buffering process and play an integral part in this regulation, with the bicarbonate buffer, arguably the most important of all extra-cellular fluid (ECF) buffers. While it has been shown previously that muscle cell membranes are virtually impervious to HCO_3^- (7,8), an increase in extracellular HCO_3- increases the pH gradient between the intracellular and extracellular environment.

It is not the intention of this review to excessively delve into the biochemistry of exercise-induced metabolic acidosis, as there are a number of excellent reviews available in this area (9–12). However, many of these reviews suggest that metabolic acidosis may not be caused simply by lactate, but by the imbalance between the rate of proton release and the rate of proton buffering and removal.

We will stress in this review that any ergogenic potential that preexercise metabolic alkalosis may impose depends upon the physiological demands of the activity being sufficient enough to induce a performance-inhibiting level of metabolic acidosis. In reference to short-term, high-intensity exercise, the direct inhibitory influence that H^+ accumulation may incur *in vivo* are complex and not yet fully understood (13). However, the high level of exercise intensity inevitably compromises either preexisting muscle creatine phosphate (CrP) levels, oxymyoglobin stores within skeletal muscle, and/or intracellular ATP concentrations to at or below 4.0 $mmol \cdot kg^{-1}$ wet weight (14,15). Consequently, enzyme activity within glycolytic metabolism is disrupted, sarcoplasmic reticulum (SR) dysfunction occurs as calcium (Ca_2+) resequestering is reduced, and ultimately there is a decrease in force-generating capacity (16).

More than 70 yr ago, a number of researchers began investigations of acid-base balance and exercise (17–19). In an early study (18), the authors used acid salts to make runners more acidic and established that this regimen made them less able to use oxygen efficiently. In turn, this led the researchers to infer that induced alkalosis could have an opposite effect. Slightly later (19), it was demonstrated that runners could have a 1% decrease in running times when alkalotic. The modern study of acid-base balance during exercise essentially began in the 1970s (20). A number of well-conducted studies in the 1980s suggest that ingestion of sodium bicarbonate could be effective in performance enhancement. Two studies (21,22), which conducted well-controlled field work, show that ingestion of buffers could benefit positively elite athletes in their performance of 800-m and 400-m respectively.

SPORT-SPECIFIC ERGOGENIC POTENTIAL

High-Intensity Exercise

Recent research investigating the physiological affect of an induced pre-exercise alkalotic state has had the benefit of improved methodological techniques to assess and compare blood and muscle acid-base balance, often at the cellular level (23). Raymer *et al.* (23) recently published a comparison via ^{31}P-MRS of blood and muscle pH perturbation after ingestion of 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ $NaHCO_3$ using incremental forearm to exhaustion as the exercise model. They reported an attenuation of intracellular acidosis under the alkalotic condition when compared with control, with the prolonged maintenance of acid-base balance in the alkalotic condition providing an increase in time to exhaustion (TTE) and an approximate 12% improvement in peak power output (PO_{peak}). This improvement also was expressed against critical intracellular pH values (estimated by the phosphorylation potential or [ATP]:[ADP]), and as such, critical pH was reached at 60% of PO_{peak} during the control trial as opposed to 70% during the supplemented trial. Additionally, they reported a higher efflux of H^+ from the intracellular medium in the *control* trial. This finding contradicts some of the original proposed mechanisms that attempt to explain the benefit of an increased extracellular HCO_3^- medium observed with $NaHCO_3$ supplementation (24). This recent work (23), as well as the work of others (16), has proposed instead that the intracellular perturbation may have been minimized by the up-regulation of Na^+/H^+ or monocarboxylic (MCT) transporters, or a strong ion difference (SID). In either case, the similar slope and time to critical pH exhibited between blood and muscle pH during $NaHCO_3$ supplementation justifies the continued observation of blood pH kinetics, which is much more practical in the applied sporting environment (25).

In a more sport-specific study, (26) an ergogenic benefit in 200-m freestyle swimming performance after 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ of $NaHCO_3$, 60–90 min before exercise has been reported. In a randomized, double-blind, counterbalanced design, these researchers observed a mean 200-m performance time improvement ($NaHCO_3$: 1:52.2 ± 4.7 s; control: 1:53.7 ± 3.8 s; placebo: 1:54.0 ± 3.6 s) and no negative side effects. Similar to previous reports, blood pH, HCO_3, and base excess (BE) were increased both pre- and post exercise in the alkalotic trial. Similar findings to this study by Lindh *et al.* (26) also have been reported recently (27) using competitive swimmers. Using an interval protocol (2 × 100-m freestyle with 10 min passive rest between bouts) after ingesting 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ $NaHCO_3$ and undergoing a loading sequence of creatine (see methodology of 27), the authors observed an improvement in time to complete the second swim.

More conclusive perhaps is the ergogenic potential observed during repeated sprints or multiple effort bouts. Recent work by Bishop and colleagues (28,29) shows improved performance during repeated sprint conditions. In the initial study, this research group used recreational team sport participants to perform a series of five short (6-s) sprints with minimal recovery (~4:1 work-to-rest ratio). As others have reported, they observed lower blood concentrations of H^+ and higher (HCO_3) after supplementing with 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ $NaHCO_3$ (30–33). Although no difference was reported in total work or percent fatigue, they did observe improvements in sprints three through five (28). The authors went on to report no difference between $NaHCO_3$ and control in posttest muscle pH; however, the $NaHCO_3$ trial resulted in significantly higher post-test muscle lactate values. The authors attributed this to be representative of a greater rate of glycolytic flux in the

muscle, or alternatively may have been because equal amounts of Na^+ were used and therefore would not have affected net movement across the cell membrane (28). In contrast to the improved repeated sprint ability (28), dissimilar response in college wrestlers has been reported (34). After supplementing with 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ $NaHCO_3$ in two equal doses at 90 and 60 min before competition, athletes completed eight 15-s intervals of maximal effort arm ergometry (separated by 20 s of active recovery). Aschenbach *et al.* (34) reported increased pre- and post-exercise pH and BE in the supplemented group. However, unlike the previous work of Bishop and colleagues (28,29), these authors (34) did not observe improvements in peak power, total work accomplished, or percent fatigue in any of the eight intervals. Also, no difference in power output (mean or peak) was reported after supplementation in a very recent study (35). Although primarily examining the electrical activity of the muscle (relationship between changes in intramuscular pH and maximum power frequencies), these authors (35) reported no change in performance using moderately trained individuals that performed 10 10-s cycling sprints, with eight 30-s passive recovery. Again, the supplementation protocol was similar to that of other studies (0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ $NaHCO_3$) (30–33) and produced increases in venous pH (~7.52) and HCO_3 (~35.3 $mmol \cdot L^{-1}$) concentrations before exercise. It is interesting to note however, that loading sequences and the timing of the pre-exercise dose tend to be different in most studies, leading to the confusion regarding effectiveness of the various buffering substances.

One loading sequence that has been successful in increasing buffering capacity while minimizing the gastrointestinal (GI) distress has been that of loading over multiple days before an event. Adding to the data published by our laboratory (36), it was recently reported by Douroudos *et al.* (37) that supplementation with $NaHCO_3$ at varying doses (0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ and 0.5 $g \cdot kg^{-1} \cdot bw^{-1}$) over a 5-d period was an effective ergogenic aid. Using a standard Wingate protocol (0.75 $g \cdot kg^{-1} \cdot bw^{-1}$) at the end of the loading protocol, Douroudos *et al.* (37) observed an increase in performance in the 0.5 $g \cdot kg^{-1} \cdot d^{-1}$ group, but not the 0.3 $g \cdot kg^{-1} \cdot d^{-1}$ group. This may indicate that, although 0.3 $g \cdot kg^{-1}$ appears to be the accepted dose for immediate sport performance, it may not be adequate for a prolonged, multiple-day loading sequence. Similar to this study (37), some researchers have investigated the impact upon training improvements using chronic bicarbonate loading (38). The investigators implemented an 8-wk training protocol (3 $days \cdot wk^{-1}$) of 6–12 2-min cycle intervals at 140%–170% of their lactate threshold (LT). The ingestion protocol consisted of 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$ twice at 90 and 30 min before training. No difference was observed between groups ($NaHCO_3$ versus control) for post-training muscle buffering capacity or pH. However, the supplement group had greater improvements in the LT, as well as better performance in a post-training short-term endurance performance test. The authors speculated that although the induced alkalosis may not have a direct impact upon shifting intramuscular pH, the attenuation of H^+during the training may have led to an increase in the overall training stimulus, as observed in the increase in LT (38). Further study is warranted on both the loading sequence and chronic loading for training purposes.

Endurance Performance

Although most of the studies that have investigated the efficacy of sodium bicarbonate loading for enhancing sports performance have typically focused upon short bouts of high-intensity exercise lasting 60 s to 360 s, there have been numerous studies that incorporated more prolonged continuous and intermittent exercise.

PROLONGED CONTINUOUS EXERCISE

In an early study, the effect of 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$ of sodium bicarbonate ingestion on time to exhaustion at the running velocity associated with a blood lactate concentration of 4 mM was investigated in seven apparently healthy men (39). The sodium bicarbonate trial was associated with a significantly (17%) longer time to exhaustion than the placebo trial (30 min versus 26 min; $P < 0.01$). In a study of the effect of sodium bicarbonate dosage upon a maximal 60-s cycling test, it was earlier reported (30) that out of 0.1, 0.2, 0.3, 0.4, and 0.5 $g \cdot kg^{-1} \cdot bw^{-1}$ doses studied, the 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ dose resulted in the highest total work performed and highest peak power output. The 17% increase in time to exhaustion found by George and MacLaren (39) may therefore have been higher if a higher sodium bicarbonate dose had been used.

The effects of 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ of sodium bicarbonate ingestion on the 1-h maximal effort cycle ergometer performance of 10 well-trained male cyclists in a randomized, controlled trial have been investigated (33). The cyclists performed, on average, 13% and 14% greater total work in the sodium bicarbonate trial compared with control and placebo trials, respectively. In contrast, a more recent study found no difference in sodium bicarbonate (0.3 $g \cdot kg^{-1} \cdot bw^{-1}$) and control trials lasting around 60 min in total, in six well-trained cyclists and triathletes and one cross-country skier (40). The sodium bicarbonate and control trials both consisted of 30 min cycling at 77% $\dot{V}O_{2max}$ followed by completion of 469 kj total work in as short time as possible (mean intensity equivalent to 80% $\dot{V}O_{2max}$). The reason for the contrasting findings between this study and the similar study conducted previously (33) is unclear, but it may have been caused by the small differences in the performance trials or nature of the athletes undertaking the testing protocols.

PROLONGED INTERMITTENT EXERCISE

In a more prolonged intermittent exercise setting, researchers (41) investigated the effects of a 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$of sodium bicarbonate ingestion, in eight healthy men, during a 30-min intermittent cycling protocol. The protocol consisted of repeated 3-min blocks of 90 s at 40% $\dot{V}O_{2max}$, 60 s at 60% $\dot{V}O_{2max}$, and a 14-s maximal effort sprint followed by 16 s

active recovery. Again, as reported elsewhere (30–32), Price and colleagues (41) observed elevated pH and lactate levels during the exercise trial for the $NaHCO_3$ condition. Compared with the placebo, the sodium bicarbonate trial was associated with significantly higher (average) relative peak power output during the maximal sprints ($P < 0.05$) and a significantly higher fatigue index ($P < 0.01$). The improved sprint performance is similar to an older study (42) where the sprint profile consisted of 10 sprints with 6 s recovery between each.

In a similar follow-up to the work by Price and colleagues (41), researchers Bishop and Claudis (29) had field hockey players to complete two 36-min halves on a cycle ergometer, where the intermittent profile was divided into 2-min blocks (4 s sprint, 100 s at 35% $\dot{V}O_{2max}$, 20 s passive rest with an additional two repeated sprint bouts [5 × 2 s separated by 35 s at 35% $\dot{V}O_{2max}$]). Like the early study (41) from Price and colleagues, authors Bishop and Claudis (29) reported no change in $\dot{V}O_2$ or RER during exercise, yet an elevated pH and HCO_3 in the $NaHCO_3$ trial (2 × 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$ taken 90 and 20 min before exercise). No significant differences between the total work performed in the sodium bicarbonate and placebo conditions were seen. Another study examined the effects of 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ on intermittent cycling consisting of 1-min work intervals at the power output associated with 95% $\dot{V}O_{2max}$ interspersed with 1-min relief intervals at 60 W (43). In accordance with the later work (29), this study also found no significant differences in total work completed in the sodium bicarbonate and placebo conditions.

When scrutinizing the exercise pH responses in the three studies cited previously, the study that found a significant effect for sodium bicarbonate also was the study with the lowest pH during the placebo exercise trial (41). The nonsignificant effects for the other two studies (29,43) may therefore have been due to insufficient anaerobiosis during the exercise protocols for the sodium bicarbonate to exert a notable experimental effect (see Table).

OTHER RELEVANT STUDIES

There has been little research on the effects of sodium bicarbonate loading upon specific acyclic sports performance. A recent study investigated the effect of 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ of sodium bicarbonate ingestion on simulated judo performance (44). The measure of judo performance consisted of attempting as many throws as possible within three discrete time periods. The nine elite judo competitors performed significantly (5.1%) more throws in the sodium bicarbonate trial than in the placebo trial ($P < 0.01$). With sodium bicarbonate ingestion, the same study also reported a significantly higher relative mean power in the third and fourth bouts of four upper body Wingate tests, each separated by 3 min recovery ($P < 0.05$). In the fourth bout, the peak power also was significantly higher in the sodium bicarbonate trial compared with the placebo trial ($P < 0.05$).

EXCEPTIONS TO THE RULE?

Although most studies report elevated pH before exercise, not all studies report an enhanced performance. Katz *et al.* (7) exercised eight trained men at 125% of their predetermined $\dot{V}O_{2max}$ in a bicarbonate or control condition. Sodium bicarbonate was given in a dose of 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$, while the placebo consisted of NaCl. In the bicarbonate condition, subjects cycled for 100.6 s, while with the placebo, the time of exhaustion was 98.6 s ($P > 0.05$). In another study from the same laboratory (45), researchers found no difference in performance when subjects performed four bouts of intense, 2-min sprint exercise. The pH was elevated after ingestion of sodium bicarbonate, but this increased buffering capacity did not result in improved performance. Data from our laboratory have been equivocal with regards to performance (25,46). One reason for this discrepancy may not be related directly to the metabolic influence of increasing the extracellular buffering potential, but rather due to differing methodological application. Timing sequences with regards to ingestion patterns vary greatly between studies and may be an important issue, especially with regards to single-bout exercise. Attaining peak buffering potential while minimizing the risk of GI distress before exercise is essential, especially if athletes are considering loading before an event. One method that appears to minimize the risk of GI distress is either to load with sodium citrate — $Na(CH_2)_2 COH(COO^-)_3$ — as a substitute, or in combination with sodium bicarbonate (25,47). However, supplementing with sodium citrate does not appear to have the same potential for improving performance as bicarbonate alone. Recently, sodium bicarbonate has been compared with other potential buffers, including sodium citrate, using competitive 5- and 10-k runners (48). In this study, the dose of 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$ of $NaHCO_3$ was matched for osmotic strength (3.6 $mosmol \cdot kg^{-1}$) against 0.525 $g \cdot kg^{-1}$ sodium citrate and 0.4 $g \cdot kg^{-1}$ and sodium lactate (NaLa). Using a treadmill run at maximum effort (fixed speed and 2% grade), they compared TTE between the conditions. Van Montfoort *et al.* (48) show the greatest mean improvement in the $NaHCO_3$ group (2.7%), while sodium citrate and sodium lactate resulted in a mean improvement of 2.2% and 1.0%, respectively. The authors went on to report no difference in feelings of sickness; however, they speculated that the performance discrepancy between the $NaHCO_3$ and sodium citrate condition may have been due to the lower overall dose in the sodium citrate trial (related to balancing of osmotic strength).

Our lab has also shown less of an ergogenic potential by using a combination of 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$ of $NaHCO_3$ and 0.2 $g \cdot kg^{-1} \cdot bw^{-1}$ of sodium citrate 1 h before exercise (25). In this study, we used trained cyclists to compare buffering potential during a supramaximal effort (110% of maximum watt output [MWO]) to exhaustion. Although our pre-exercise pH, HCO_3, and BE were all within normal peaking ranges of other studies, we observed no difference in performance compared with placebo or control trials (25). One potential reason for this is that citrate does not have a pK of an ionizable group within a physiological range (25,47), and therefore rendering its buffering potential to a minimum.

TABLE. A summary of the studies (in or after the year 2000) illustrated in this review.

Author	Exercise Mode or Sport-Specific Exercise	Dose ($g \cdot kg^{-1} \cdot bw^{-1}$)	Loading Time Before Exercise	Reported Ergogenic Effect
		Single-Bout Exercise (Listed by Publish Date)		
Lindh *et al.*, 2007 (26)	200-m freestyle swim	0.3	60–90 min	↓ mean performance times in $NaHCO_3$ trial (~1 s)
Siegler *et al.*, 2007 (46)	Cycle to exhaustion at 120% of PPO	0.3	60 min	No difference in TTE
Robergs *et al.*, 2005 (25)	Cycle to exhaustion at 110% of PPO	0.2 of $NaHCO_3$ + 0.2 NaCitrate	60 min	No difference in TTE
Van Montfoort *et al.*, 2004 (48)	Run to exhaustion (range 19–23 $km \cdot hr^{-1}$)	0.3 $NaHCO_3$ or	90 min	↑ $NaHCO_3$ trial (~2.7%)
		0.525 NaCitrate or		↑ NaCitrate trial (~2.2%)
		0.4 NaLactate		↑ NaLactate trial (~1.0%)
Raymer *et al.*, 2004 (23)	Forearm exercise to fatigue	0.3	90 min	↑ TTE and PPO in $NaHCO_3$ trial (~12%)
		Multiple Bout Exercise		
Matsuura *et al.*, 2007 (35)	Ten 10 s RS interspersed with passive recovery (range 30–360 s)	0.3 divided into six ingestion periods every 10 min	60 min	No difference in peak or mean power output
Artioli *et al.*, 2007 (44)	Simulated judo performance (assessed in number of throws)	0.3	120 min	5.1% more throws in $NaHCO_3$ trial as well as ↑ average power in Wingate test for upper limbs
Mero *et al.*, 2004 (27)	Interval swim (2 × 100 m with 10 min passive rest between intervals	0.3	60 min	↓ Second swim time (~0.9 s) in $NaHCO_3$ trial[a]
Bishop *et al.*, 2004 (28)	Series of five 6-s RS (4:1 work-to-rest ratio)	0.3	90 min	↑ in total work and ↑ in work and PO in sprints 3–5
Aschenbach *et al.*, 2000 (34)	Eight 15-s intervals of maximal forearm exercise (20 s active recovery between sets)	0.3	Split into equal doses at 90 and 60 min	No difference
		Endurance Performance		
Bishop and Claudius, 2005 (29)	Two 36-min "halves" of intermittent field hockey specific activity	0.2 twice	Split at 90 and 20 min	No difference in total work over 72 min; ↑ work performed in 7 of 18 second half sprints
Price *et al.*, 2003 (41)	Two 30-min intermittent cycling trials	0.3	60 min	↑ average relative PO during maximal sprint efforts
Stephens *et al.*, 2002 (40)	30-min continuous cycling at ~77% $\dot{V}O_{2max}$ followed by a performance ride (time to complete 469 ± 21 kJ work)	0.3 (60-min ingestion time)	90 min	No difference in performance
		Chronic Loading		
Douroudos *et al.*, 2006 (37)	30 s Wingate (0.075 $kg \cdot kg^{-1} \cdot bw^{-1}$)	0.5 for 5 d	None on day of trial	↑ average power in 0.5 g $NaHCO_3$ only
		0.3 for 5 d		
Edge *et al.*, 2006 (38)	6–12 2-min cycle intervals at 140–170% of LT (in addition to regimented training)	0.2 twice	90 and 30 min	↑ performance at LT after 8 wk of training on $NaHCO_3$

[a]Additional use of Creatine (Cr) supplementation but did not have a Cr only trial included in the methodology.

PPO = peak power output, PO = power output, TTE = time to exhaustion, RS = repeated sprint, LT = lactate threshold.

CONCLUSION

We conclude, on the basis of the amount of data relevant to sodium bicarbonate and sodium citrate as buffering agents, that both are effective. We recommend that coaches and athletes test their response to using buffers to improve their own performance before any competitive event. There would, however, appear to be an optimum ingestible amount of these substances that are ergogenic, that being 0.3 $g \cdot kg^{-1} \cdot bw^{-1}$. Both buffers can contribute to GI upset and may not be tolerated well by all athletes. Our experience suggests that approximately 10% do not tolerate these substances well. It also may be worthwhile to investigate long-term loading to offset any potential GI upset, but the health risks associated with such loading require further investigation. While the research continues, it would appear that both short-term and long-term high-intensity exercise can benefit from the ergogenic effects of these buffers. This includes activities that may not be thought of as typically intermittent (*e.g.*, judo). Finally, some research suggests that high-intensity, longer-duration exercise also may benefit from ingestion of buffers, but this too requires more investigation.

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