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 multi-faceted, 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).

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:

\[
\text{H}^+ + \text{HCO}_3^- \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}_2\text{O} + \text{CO}_2
\]

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:

\[
\text{H}^+ + \text{HCO}_3^- \rightarrow \text{H}_2\text{CO}_3
\]

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 HCO₃⁻ (7,8), an increase in extracellular HCO₃⁻ 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.kg⁻¹ wet weight (14,15). Consequently, enzyme activity within glycolytic metabolism is disrupted, sarcoplasmic reticulum (SR) dysfunction occurs as calcium (Ca²⁺) resequesitered 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 effect 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 ³¹P-MRS of blood and muscle pH perturba-
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·kg⁻¹·bw⁻¹ NaHCO₃ 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·kg⁻¹·bw⁻¹ NaHCO₃) (30-33) and produced increases in venous pH (~7.52) and HCO₃⁻ (~35.3 mmol·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 NaHCO₃ at varying doses (0.3 g·kg⁻¹·bw⁻¹ and 0.5 g·kg⁻¹·bw⁻¹) over a 5-d period was an effective ergogenic aid. Using a standard Wingate test (0.75 g·kg⁻¹·bw⁻¹) at the end of the loading protocol, Douroudos et al. (37) observed an increase in performance in the 0.5 g·kg⁻¹·bw⁻¹ group, but not the 0.3 g·kg⁻¹·bw⁻¹ group. This may indicate that, although 0.3 g·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·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·kg⁻¹·bw⁻¹ twice at 90 and 30 min before training. No difference was observed between groups (NaHCO₃ 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·kg⁻¹·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·kg⁻¹·bw⁻¹ doses studied, the 0.3 g·kg⁻¹·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·kg⁻¹·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·kg⁻¹·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% \( \text{VO}_{2\text{max}} \) followed by completion of 469 kJ total work in an hour time as possible (mean intensity equivalent to 80% \( \text{VO}_{2\text{max}} \)). 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·kg⁻¹·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% \( \text{VO}_{2\text{max}} \), 60 s at 60% \( \text{VO}_{2\text{max}} \) 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 NaHCO₃ 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% $VO_{2\text{max}}$; 20 s passive rest with an additional two repeated sprint bouts [5 × 2 s separated by 35 s at 35% $VO_{2\text{max}}$]). Like the early study (41) from Price and colleagues, authors Bishop and Claudis (29) reported no change in $VO_2$ or RER during exercise, yet an elevated pH and HCO₃ in the NaHCO₃ trial ($2 \times 0.2$ g·kg⁻¹·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·kg⁻¹·bw⁻¹ on intermittent cycling consisting of 1-min work intervals at the power output associated with 95% $VO_{2\text{max}}$ 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).

**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 $VO_{2\text{max}}$ in a bicarbonate or control condition. Sodium bicarbonate was given in a dose of 0.2 g·kg⁻¹·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(CH₂)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·kg⁻¹·bw⁻¹ of NaHCO₃ was matched for osmotic strength (3.6 mosmol·kg⁻¹) against 0.525 g·kg⁻¹ sodium citrate and 0.4 g·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 NaHCO₃ 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 NaHCO₃ 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·kg⁻¹·bw⁻¹ of NaHCO₃ and 0.2 g·kg⁻¹·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 [MWOT]) to exhaustion. Although our pre-exercise pH, HCO₃, 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.

**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·kg⁻¹·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 ($P < 0.05$).
TABLE. A summary of the studies (in or after the year 2000) illustrated in this review.

<table>
<thead>
<tr>
<th>Author</th>
<th>Exercise Mode or Sport-Specific Exercise</th>
<th>Dose (g·kg⁻¹·bw⁻¹)</th>
<th>Loading Time Before Exercise</th>
<th>Reported Ergogenic Effect</th>
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<tbody>
<tr>
<td><strong>Single-Bout Exercise (Listed by Publish Date)</strong></td>
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<tr>
<td>Lindh et al., 2007 (26)</td>
<td>200-m freestyle swim</td>
<td>0.3</td>
<td>60–90 min</td>
<td>↓ mean performance times in NaHCO₃ trial (~1 s)</td>
</tr>
<tr>
<td>Siegler et al., 2007 (46)</td>
<td>Cycle to exhaustion at 120% of PPO</td>
<td>0.3</td>
<td>60 min</td>
<td>No difference in TTE</td>
</tr>
<tr>
<td>Robergs et al., 2005 (25)</td>
<td>Cycle to exhaustion at 110% of PPO</td>
<td>0.2 NaHCO₃ + 0.2 NaCitrate</td>
<td>60 min</td>
<td>No difference in TTE</td>
</tr>
<tr>
<td>Van Montfoort et al., 2004 (48)</td>
<td>Run to exhaustion (range 19–23 km·hr⁻¹)</td>
<td>0.3 NaHCO₃ or 0.525 NaCitrate</td>
<td>90 min</td>
<td>↑ NaHCO₃ trial (~2.7%)</td>
</tr>
<tr>
<td>Raymer et al., 2004 (23)</td>
<td>Forearm exercise to fatigue</td>
<td>0.3</td>
<td>90 min</td>
<td>↑ NaCitrate trial (~2.2%)</td>
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<td></td>
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<td>↑ NaLactate trial (~1.0%)</td>
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<td></td>
<td>↑ TTE and PPO in NaHCO₃ trial (~12%)</td>
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<tr>
<td><strong>Multiple Bout Exercise</strong></td>
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<tr>
<td>Matsuura et al., 2007 (35)</td>
<td>Ten 10 s RS interspersed with passive recovery (range 30–360 s)</td>
<td>0.3 divided into six ingestion periods every 10 min</td>
<td>60 min</td>
<td>No difference in peak or mean power output</td>
</tr>
<tr>
<td>Artioli et al., 2007 (44)</td>
<td>Simulated judo performance (assessed in number of throws)</td>
<td>0.3</td>
<td>120 min</td>
<td>5.1% more throws in NaHCO₃ trial as well as ↑ average power in Wingate test for upper limbs</td>
</tr>
<tr>
<td>Mero et al., 2004 (27)</td>
<td>Interval swim (2 × 100 m with 10 min passive rest between intervals)</td>
<td>0.3</td>
<td>60 min</td>
<td>↓ Second swim time (~0.9 s) in NaHCO₃ trial*</td>
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<tr>
<td>Bishop et al., 2004 (28)</td>
<td>Series of five 6-s RS (4:1 work-to-rest ratio)</td>
<td>0.3</td>
<td>90 min</td>
<td>↑ in total work and ↑ in work and PO in sprints 3–5</td>
</tr>
<tr>
<td>Aschenbach et al., 2000 (34)</td>
<td>Eight 15-s intervals of maximal forearm exercise (20 s active recovery between sets)</td>
<td>0.3</td>
<td>Split into equal doses at 90 and 60 min</td>
<td>No difference</td>
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<tr>
<td><strong>Endurance Performance</strong></td>
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<tr>
<td>Bishop and Claudius, 2005 (29)</td>
<td>Two 36-min “halves” of intermittent field hockey specific activity</td>
<td>0.2 twice</td>
<td>Split at 90 and 20 min</td>
<td>No difference in total work over 72 min; ↑ work performed in 7 of 18 second half sprints</td>
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<tr>
<td>Price et al., 2003 (41)</td>
<td>Two 30-min intermittent cycling trials</td>
<td>0.3</td>
<td>60 min</td>
<td>↑ average relative PO during maximal sprint efforts</td>
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<tr>
<td>Stephens et al., 2002 (40)</td>
<td>30-min continuous cycling at ~77% VO₂max followed by a performance ride (time to complete 469 ± 21 kJ work)</td>
<td>0.3 (60-min ingestion time)</td>
<td>90 min</td>
<td>No difference in performance</td>
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<tr>
<td><strong>Chronic Loading</strong></td>
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<tr>
<td>Douroudos et al., 2006 (37)</td>
<td>30 s Wingate (0.075 kg·kg⁻¹·bw⁻¹)</td>
<td>0.5 for 5 d</td>
<td>None on day of trial</td>
<td>↑ average power in 0.5 g NaHCO₃ only</td>
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<tr>
<td>Edge et al., 2006 (38)</td>
<td>6–12 2-min cycle intervals at 140–170% of LT (in addition to regimented training)</td>
<td>0.2 twice</td>
<td>90 and 30 min</td>
<td>↑ performance at LT after 8 wk of training on NaHCO₃</td>
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</tbody>
</table>

*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 kg·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.

References


