Effects of Bicarbonate, Citrate, and Phosphate Loading on Performance

Craig A. Horswill

Since the 1930s, scientists have attempted to determine if increasing the body’s ability to buffer metabolic acids will enhance physical performance. The buffer of major interest has been bicarbonate; to a lesser degree, citrate and phosphate salts have been investigated. In theory, the buffers facilitate performance by decreasing the accumulation of hydrogen ions that would otherwise presumably inhibit glycolysis and interfere with energy production or impair cross-bridge formation between myofilaments and thereby reduce force production. Literature findings indicate variable results, but overall it appears that bicarbonate salts taken at dosages of 0.3 g · kg⁻¹ may improve performance during repeated sprints or at the end of a progressively more intense exercise test. Athletes are advised of potential ill effects of bicarbonate ingestion, such as gastrointestinal distress. Prior to applying the agents in a competitive setting, athletes should test the effects of buffers on performance during training sessions and consider the sport governing body’s stand on buffer usage.

The fatigue associated with high-intensity efforts that are continuous for 30 s to 3 min or that are repeated intermittently after short periods of rest is largely a function of metabolite accumulation that decreases pH or depletes adenosine triphosphate (ATP), and phosphocreatine. An increase in hydrogen ion (H⁺) concentration concomitant with lactate accumulation is thought to produce fatigue either by inhibiting the cross-linkage between actin and myosin and thereby reducing force production (11, 16) or by inhibiting the activity of phosphofructokinase (PFK) and phosphorylase, enzymes that are critical in regulating glycolysis and resynthesizing ATP (6, 16, 47). The latter mechanism would also block the resynthesis of phosphocreatine and thereby impair performance despite the presence of adequate stores of glycogen and triglyceride fuels.

It is theoretically possible to increase the capacity to buffer H⁺ released from lactic acid or to elevate the phosphagen concentrations in muscle and thereby improve the performance of high-intensity exercise or sport. To effect these changes, the body’s natural buffering capacity could be augmented by ingesting bicarbonate salts (e.g., sodium bicarbonate or NaHCO₃), citrate salts (e.g., tri-sodium citrate or Na₃C₆H₅O₇), or, to a lesser extent, phosphate salts (e.g., K₃PO₄). Other theoretical rationales for ingesting phosphates before high-power exercise are to elevate intramuscular phosphate for recycling ATP and phosphocreatine and to stimulate the breakdown of glycogen for energy.

Craig A. Horswill is with Gatorade Exercise Physiology Lab, 617 W. Main St., Barrington, IL 60010.
This paper reviews the theoretical mechanisms of action whereby bicarbonate, citrate, and phosphate may enhance physical performance at high intensities. A brief review of the literature on the effects of each buffer on various exercise protocols at near-maximal (near 100% peak VO_2) or supramaximal (sprints) exercise is also presented.

Background on Buffers

Endogenous buffers are present in the body and contribute in varying degrees to the maintenance of the acid-base balance. By definition, a buffer is a compound or series of compounds that allow a solution to minimize changes in pH when an acid or base is added to the solution. At rest, pH is maintained close to 7.35 and 7.05 in the blood and muscle, respectively (7); however, during sprinting, pH can temporarily be driven as low as 6.8 in the blood and 6.4 in the muscle (17, 36). The primary intracellular buffers are phosphates and tissue proteins, whereas the major extracellular buffers are bicarbonate, plasma proteins, and hemoglobin. Unfortunately, endogenous buffers are incapable of totally compensating for the large accumulation of metabolic acids during high-power exercise. Thus, inadequate buffering of hydrogen ions can soon lead to exhaustion.

Prior to the performance test or sport competition, subjects ingest the potential ergogenic agents (see Table 1 for a summary of protocols for administering buffers). Ingested bicarbonate elevates the bicarbonate concentration in the extracellular space, whereas the destination and site of buffering action by exogenous phosphate are not clear. Presumably, phosphate enters the intracellular space and acts within the muscle, but there is no research to substantiate this. Endogenous citrate, an intermediate of the tricarboxylic cycle in the mitochondria, typically does not contribute significantly to the buffering systems in the body. Nevertheless, researchers have attempted to increase the body’s buffering capacity by elevating citrate levels directly via citrate salt ingestion and indirectly via consumption of foods high in citrate (18).

Table 1  Typical Protocols Employed to Administer Buffers

<table>
<thead>
<tr>
<th>Buffer</th>
<th>Dosage</th>
<th>Route</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium bicarbonate</td>
<td>0.1–0.5 g/kg</td>
<td>Oral</td>
<td>Single dose taken 1 hr before performance; repeated doses taken over several hours before performance</td>
</tr>
<tr>
<td></td>
<td>1.2–6.0 mmol/kg</td>
<td>• solution • capsule I.V.</td>
<td></td>
</tr>
<tr>
<td>Sodium citrate</td>
<td>0.1–0.5 g/kg</td>
<td>Oral</td>
<td>Single dose taken 60–90 min before performance</td>
</tr>
<tr>
<td></td>
<td>0.39–1.94 mmol/kg</td>
<td>• solution • fruit</td>
<td></td>
</tr>
<tr>
<td>Phosphate salts</td>
<td>1.2–5.7 g/day</td>
<td>Oral</td>
<td>Single dose 1 hr prior (acute) or multiple doses daily for 3–6 days prior to performance</td>
</tr>
<tr>
<td></td>
<td>9–176 mmol of phosphate/day</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note. kg refers to kilograms of body weight.*
Theoretical Mechanism of Action

The mechanism by which bicarbonate loading exerts its influence may be through the elevation of the extracellular bicarbonate concentrations, which then increases the rate of efflux of \( H^+ \) from the intracellular space. Research indicates that although bicarbonate levels are elevated in the extracellular space, intracellular bicarbonate concentrations are not increased following the ingestion of bicarbonate (8), probably because the cell membrane is fairly impermeable to the bicarbonate ion (45). Other researchers believe that bicarbonate loading produces effects on performance through a more indirect mechanism. Heigenhauser et al. (15) argued that the ingested sodium changes the strong-ion difference, and that this change, not the bicarbonate per se, produces the increase in blood buffering capacity. In addition, these researchers (15) and others (46) have shown that ingested bicarbonate does not directly enter the blood plasma. Rather, the increase in plasma bicarbonate occurs indirectly as a result of a decrease in plasma chloride (\( Cl^- \)), a strong ion that is excreted with excess sodium presented by the sodium bicarbonate dosage (17). Regardless of whether it acts by a direct or indirect mechanism, ingested bicarbonate does increase the plasma bicarbonate concentration and enhances the buffering capacity of the blood.

Citrate works on a principle similar to that of bicarbonate, that is, elevating the buffering capacity of the extracellular space to accelerate the efflux of \( H^+ \) from the intramuscular space. Citrate’s action may be mediated by bicarbonate via (a) citrate metabolism that produces bicarbonate, which becomes elevated in the blood in proportion to the dosage of citrate consumed (38) or (b) the strong ion difference phenomenon (15) caused by sodium (trisodium citrate). Unlike bicarbonate, citrate loading might present a potential conflict in the attempt to enhance power performance. Researchers have speculated that elevating plasma citrate also elevates intracellular citrate, which could inhibit PFK and thereby reduce glycolysis and the supply of ATP (26). Presently, though, no one has used the muscle biopsy to determine if intracellular citrate loading occurs and, if so, whether or not it inhibits anaerobic metabolism.

Phosphate loading may work via a buffering mechanism in either the plasma or the intracellular space. In addition, phosphate loading may stimulate glycolysis or increase levels of inorganic phosphate, which hypothetically could facilitate the resynthesis of creatine phosphate (6). In theory, any of these mechanisms could enhance anaerobic performance. Other mechanisms by which phosphate loading may improve performance are primarily pertinent to altering aerobic metabolism, including (a) inducing an increase in concentrations of 2,3-diphosphoglycerate (2,3-DPG) (5), which can stimulate the release of oxygen from oxyhemoglobin for tissue use, and (b) enhancing myocardial and cardiorespiratory responses to physical exertion (3, 27). Whether these latter physiological changes affect anaerobic performance is unknown; for the most part, changes affecting the delivery and use of oxygen will primarily improve aerobic metabolism and possibly impact submaximal endurance performance more so than sprint performance.

Effects of Buffer Loading on Performance

Substantial research has been done on the effects of bicarbonate ingestion on physical performance in a well-controlled setting where performance is accurately quantified. As well, several review articles (15, 30, 35, 42) have been written on bicarbonate ingestion and physical performance. Using double-blinded, crossover studies in which subjects are randomly assigned to the treatment order, recent studies have overcome the design
limitations of early research by the investigators who fostered the hypothesis of an ergogenic effect of bicarbonate loading (9, 33, 34). Also, in some recent studies, a control trial has been performed in addition to the placebo treatment, the purpose of which is to determine whether a placebo effect may occur (13, 39, 40, 49); interestingly, all of these studies show an ergogenic effect of the buffer treatment.

Figure 1 summarizes the relationship between dosage of sodium bicarbonate and the percent change in performance. To determine these data, mean values from the literature were used (2, 4, 8, 12–14, 19, 20, 22, 23–25, 29, 36, 37, 39, 40, 44, 47–49). The percent change in performance was calculated in most cases using the placebo trial as baseline; however, when performance during control trials (no placebo) was incorporated into the design and data were presented, the placebo and bicarbonate trials were compared to the control trial.

There appears to be a positive relationship between bicarbonate dosage and the extent of improvement in performance (Figure 1). This superficial analysis is supported by investigators who conducted a meta-analysis of literature findings and concluded that the ergogenic effects of bicarbonate are indeed real (35). Obviously, a large enough dosage of the buffer is needed to elevate plasma bicarbonate concentration to the point that there is a physiologically significant increase in the buffering capacity of the blood.

Figure 1 — Relationship between sodium bicarbonate dosage (NaHCO₃) and the change in performance as a percentage of the placebo treatment. Data were generated from mean values reported in the literature (2, 4, 8, 12–14, 19, 20, 22, 23–25, 29, 36, 37, 39, 40, 44, 47–49). In the cases where control trials were also run, the control data were used to calculate percent change during placebo trial (0 g NaHCO₃) and the bicarbonate trial. Dotted line indicates the regression line.
It would appear that 0.3 g · kg⁻¹ is the minimum dose; however, several studies have shown positive results with amounts less than this (8, 13, 20, 37). Possibly, in a situation where repeated sprints are performed, a dosage of less than 0.3 g · kg⁻¹ may be effective in increasing power or speed during sprints (8, 13, 37).

A closer examination of the studies is required, though, before it can be concluded that bicarbonate will be effective during the performance of any short-duration, high-power effort. The ergogenic effects of bicarbonate appear to be most consistent either when exercise protocols involve repeated sprints that are interspersed with short recovery periods (8, 13, 29, 37, 48) or when protocols commence at submaximal intensities, become progressively more difficult, and culminate at near-maximum levels, for example, a ramp protocol that ends at 95% of peak VO₂ with a timed endurance bout (20, 22, 47). This is in contrast to inconsistent findings of ergogenic effects when a single sprint is performed; some studies show no effects on performance (14, 19, 23, 24, 36), and others show positive effects (2, 39, 40, 49). Research indicates that acidosis created artificially prior to performance will not adversely affect the power output of a single brief sprint (36). Thus, within a brief single interval of exertion, the normal intracellular milieu may have ample buffering mechanisms. Alternatively, in a single brief sprint, intracellular pH may not be as critical a factor in producing fatigue as is depletion of the initial phosphagen stores in the active muscles.

In research protocols where multiple sprints are performed, the buffering capacity within the muscle may be exceeded by H⁺ accumulation because lactate concentrations can be driven much higher than those typically produced by more prolonged continuous work (17); thus, the facilitation of H⁺ efflux by elevated bicarbonate in the extracellular fluid may speed recovery between efforts so that energy production and force production may be maintained closer to peak levels in the subsequent sprints (31, 32). During performance of the ramp protocol (47), the blood bicarbonate system becomes the primary mechanism for buffering H⁺ only after the subject reaches the anaerobic threshold (1). At that point, artificially elevated plasma bicarbonate would presumably enhance H⁺ efflux from the muscle. Therefore, when the subject exercises at the final intensity level (e.g., 95% of peak VO₂), the intracellular compartment of the muscle would not be as acidic as it would under the placebo condition, and the detrimental effects of H⁺ on performance would be attenuated.

Other factors certainly could enter into the explanation of inconsistent findings among the studies, but such factors have not yet been investigated. The physical fitness of the subjects may affect the results. Subjects with greatest fitness have the greatest performance reliability (i.e., consistency in speed or power), which enhances the sensitivity for detecting an ergogenic effect; however, fit subjects may also have maximized their endogenous buffering capacity so that exogenous buffers have lesser effects. Also, the extent to which the performance test protocol included a warm-up may affect the results. Inclusion of a warm-up may make the exercise protocol more like a series of repeated physical efforts or more like a ramp protocol, both of which often show positive results upon bicarbonate loading. The duration of time between buffer ingestion and performance, and possibly the route of delivery (oral in one bolus vs. oral in serial dosages or gelatin capsules vs. intravenous), may affect the outcome since both factors may influence the final level of plasma bicarbonate prior to performance. Finally, the type of muscle action (e.g., isometric vs. isokinetic or concentric only vs. combined concentric and eccentric) may have some bearing on the effectiveness of bicarbonate as an ergogenic agent.

Compared to the investigations of bicarbonate, less research has investigated the ability of citrate to enhance the body’s buffering capacity. Under conditions of alkalosis
induced by citrate treatment, Parry-Billings and MacLaren (43) failed to see an overall performance enhancement. More recently, McNaughton and co-workers (38, 41) systematically investigated the influence of dosage and exercise duration on the effectiveness of citrate as an ergogenic agent. Evaluating the performance during a 1-min cycle sprint test, McNaughton (38) observed that significant increases in power occurred when subjects ingested at least 0.3 g and as much as 0.5 g of sodium citrate per kilogram body weight. Despite the high dosages, subjects did not experience gastrointestinal distress. Within the limits of additional experiments, McNaughton and Cedaro (41) found that a minimum sprint duration of 120 s was needed to show significant improvements in performance (e.g., power) when a sodium citrate dosage of 0.5 g · kg⁻¹ body weight was ingested prior to performance. Using a ramp protocol in which subjects proceeded from cycling for 20 min at 33% peak \( \dot{V}O_2 \) to cycling for 20 min at 66% peak \( \dot{V}O_2 \) to cycling at 95% peak \( \dot{V}O_2 \) until exhaustion, Kowalchuk et al. (26) found no improvement in endurance time, that is, duration of work at 95% of peak \( \dot{V}O_2 \) when subjects ingested 0.3 g · kg⁻¹ sodium citrate before exercise. In contrast, sodium bicarbonate doses of 0.3 g · kg⁻¹ have produced ergogenic effects when ingested prior to an identical exercise protocol (20, 22, 47). However, because the molecular weight of sodium bicarbonate is less than that of sodium citrate, a lower molar equivalent of citrate (and effectively less buffer) would be administered using dosages of similar mass.

A paucity of data exists on the effects of phosphate loading on high-power performance. There is but one published study on the effects of phosphate loading on high-power performance. Duffy and Conlee (10) tested the effects of acute and chronic ingestion of phosphate salts on sprint performance, leg power, and peak \( \dot{V}O_2 \). Using doses of 1.24 g administered 1 hr prior to the performance test and 3.7 g · day⁻¹ for 9 days prior to performance to study acute and chronic effects, respectively, the authors observed no change in (a) leg power determined using an isokinetic dynamometer, (b) sprint endurance time on the treadmill moving at 214 m · min⁻¹ and at 6% grade, or (c) peak \( \dot{V}O_2 \). No invasive tests were conducted to determine whether plasma levels of phosphate were elevated by the treatment. In fact, the dosage used by Duffy and Conlee (10) was quite low compared to those used in other studies (3, 5, 27, 28). Subsequent studies have used aerobic performance as the outcome variable (e.g., peak \( \dot{V}O_2 \) or submaximal endurance time). Reported increases in peak \( \dot{V}O_2 \) (5, 27, 28) were related to elevated plasma phosphate levels but not necessarily to increased 2,3-DPG concentrations (28). Others have not observed changes in peak \( \dot{V}O_2 \), despite observing physiological changes such as increased \( O_2 \) extraction that might be thought to enhance performance (3).

**Summary**

Fairly convincing evidence exists that the administration of exogenous buffer, particularly sodium bicarbonate, improves high-power performance. When consumed at dosages of at least 0.3 g · kg⁻¹, sodium bicarbonate and sodium citrate may improve performance as a result of speeding the efflux of \( H^+ \) from the muscle and consequently attenuating the adverse effects of acid accumulation on the production of energy or force. The mechanisms underlying any potential benefits of using phosphate salts are not as well researched as those for bicarbonate or citrate. Regardless of the form of buffer, it has not yet been conclusively demonstrated that buffers can improve sport performance (Table 2).
Table 2  Ergogenic Effect of Buffers When Applied to Performance in Field Studies

<table>
<thead>
<tr>
<th>Study</th>
<th>Event</th>
<th>Buffer</th>
<th>Effect$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Johnson and Black (21)</td>
<td>Cross-country race</td>
<td>Citrate; citrate and bicarbonate; phosphate</td>
<td>None for any buffer</td>
</tr>
<tr>
<td>Pierce et al. (44)</td>
<td>Swimming events</td>
<td>Bicarbonate</td>
<td>None</td>
</tr>
<tr>
<td>Wilkes et al. (49)</td>
<td>800-m run</td>
<td>Bicarbonate</td>
<td>Positive (approximately 3 s faster)</td>
</tr>
<tr>
<td>Brien and McKenzie (4)</td>
<td>Rowing (crew)</td>
<td>Bicarbonate</td>
<td>None</td>
</tr>
<tr>
<td>Kindermann et al. (24)</td>
<td>400-m run</td>
<td>Bicarbonate</td>
<td>None</td>
</tr>
<tr>
<td>Kreider et al. (27)</td>
<td>5-mile run</td>
<td>Phosphates</td>
<td>None (tendency for being 11.8 s faster)</td>
</tr>
</tbody>
</table>

$^a$None = no statistical difference between placebo or buffer trial; positive = performance was statistically better ($p < .05$) than placebo trial.

References


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