Effects of Ingestion of Bicarbonate, Citrate, Lactate, and Chloride on Sprint Running

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ABSTRACT

VAN MONTFOORT, M. C. E., L. VAN DIEREN, W. G. HOPKINS, and J. P. SHEARMAN. Effects of Ingestion of Bicarbonate, Citrate, Lactate, and Chloride on Sprint Running. Med. Sci. Sports Exerc., Vol. 36, No. 7, pp. 1239–1243, 2004. Purpose: Ingestion of sodium bicarbonate is known to enhance sprint performance, probably via increased buffering of intracellular acidity. The goal was to compare the effect of ingestion of sodium bicarbonate with that of other potential buffering agents (sodium citrate and sodium lactate) and of a placebo (sodium chloride) on sprinting. Methods: In a double-blind randomized crossover trial, 15 competitive male endurance runners performed a run to exhaustion 90 min after ingestion of each of the agents in the same osmolar dose relative to body mass (3.6 mosmol·kg⁻¹) on separate days. The agents were packed in gelatin capsules and ingested with 750 mL of water over 90 min. During each treatment we assayed serial finger-prick blood samples for lactate and bicarbonate. A familiarization trial was used to set a treadmill speed for each runner’s set of runs. We converted changes in run time between treatments into changes in a time trial of similar duration using the critical-power model, and we estimated likelihood of practical benefit using 0.5% as the smallest worthwhile change in time-trial performance. Results: The mean run times to exhaustion for each treatment were: bicarbonate 82.3 s, lactate 80.2 s, citrate 78.2 s, and chloride 77.4 s. Relative to bicarbonate, the effects on equivalent time-trial time were lactate 1.0%, citrate 2.2%, and chloride 2.7% (90% likely limits ±2.1%). Ingested lactate and citrate both appeared to be converted to bicarbonate before the run. There were no substantial differences in gut discomfort between the buffer treatments. Conclusion: Bicarbonate is possibly more beneficial to sprint performance than lactate and probably more beneficial than citrate or chloride. We recommend ingestion of sodium bicarbonate to enhance sprint performance. Key Words: ATHLETE, BUFFER, ERGOGENIC, RUNNING

When continuous exercise is performed at high intensity for ~0.5–5 min, production of energy by anaerobic glycolysis is accompanied by a rise in intramuscular acidity that contributes to fatigue (20). Extracellular bicarbonate helps buffer the acidity in muscle cells by increasing the gradient for efflux of hydrogen ions from the cells. Increasing the body’s buffering capacity by increasing the extracellular bicarbonate concentration should therefore reduce acidosis and thereby delay fatigue. Ingestion of sodium bicarbonate does indeed improve high-intensity performance substantially in most studies (14).

Various researchers (11,16,18,23) have investigated the potential of sodium citrate as an exogenous buffer, because sodium citrate might be associated with less gastrointestinal discomfort than sodium bicarbonate (16).

Sodium citrate does not buffer directly like sodium bicarbonate: the dissociation constant for citrate/citric acid lies well outside the body’s pH range, but the consumption of protons during its oxidation effectively generates bicarbonate (17). McNaughton (15,16) found that ingestion of sodium citrate had a positive effect on work output, but it failed to have a significant effect on performance in other studies (11,18,23). The true effect remains unclear.

Ingestion of other substances could produce an indirect buffering effect similar to that of sodium citrate. One such substance is sodium lactate, which would also consume protons when it is metabolized. Using lactate as a buffer may seem counterintuitive to those who believe that lactic acid causes fatigue, but it must be remembered that intracellular acidity causes fatigue, not the accumulation of lactate ions.

A number of researchers have investigated the influence of polylactate ingestion on endurance performance (2,3,19,22), since Brooks (1) found that lactate can serve as an energy source for exercising muscles. In the study of Fahey et al. (3), the ingestion of 80% polylactate and 20% sodium lactate as a 7% solution in water increased blood pH and bicarbonate compared to ingestion of a glucose polymer drink. These increases occurred via consumption of hydrogen ions when lactate was either oxidized (Equation 1)
In the present study, we have therefore compared the effects on performance of ingestion of the three buffers: sodium bicarbonate, sodium citrate, and sodium lactate. As a placebo, we chose sodium chloride (12) rather than calcium carbonate (24), to control for any effect of sodium intake on performance through changes in intravascular volume (12).

METHODS

Subjects and design. Nineteen trained male distance runners (age 31.2 ± 3.2 yr, height 173.6 ± 0.9 cm, mass 73.5 ± 2.3 kg, mean ± SD) volunteered for the study. The subjects were all in training and competed regularly in 5-km or 10-km races at club or national level. We chose distance runners because no sprinters were available at the time of the study, but these runners were accustomed to brief intense efforts in their interval training. All subjects were nonsmokers and were free of injury. Four runners withdrew from the study, two due to injury, and two due to an adverse reaction to the placebo treatment. The project was reviewed and approved by the institutional ethics committee. All subjects gave their written informed consent after a detailed description of the study and the possible risks.

Training logs were completed for the duration of the study, to ensure that training did not change substantially. All subjects were asked to maintain their usual training programs and were asked to report for testing following a rest day or light training day consisting only of jogging. None of the subjects was currently using a buffer as a supplement. We did not monitor diet or use of other supplements, which were considered unlikely to interact with the effect of buffer supplementation on sprint performance.

The design was a randomized double-blind crossover. The order of substances was randomized using a Latin square to aim for balance in the sequence of treatments. On separate days, each subject performed a familiarization test and a performance test for each of the four treatments. All tests for each subject were completed at about the same time of day to reduce any effect of circadian rhythm. The period between testing sessions was at least 2 d and no more than 5 d.

Dosage. The sodium bicarbonate dose of 300 mg·kg\(^{-1}\) of body weight was based on previous studies (10,14,15,21) and guidelines for sodium bicarbonate intake from the Australian Institute of Sport (http://www.ausport.gov.au/ais/nutrition/suppsfs14.htm). Dosages of all other agents were equal in osmotic strength to the dose of sodium bicarbonate (3.6 mosmol·kg\(^{-1}\) of body mass) to control for any feelings of nausea or discomfort arising from the osmotic strength of the buffers. The corresponding dosages of sodium citrate, sodium lactate, and sodium chloride were 525, 400, and 209 mg·kg\(^{-1}\) of body weight, respectively. All capsules were prepared in the laboratory. Subjects ingested between 20 and 60 capsules, depending on the weight of the subject and the molecular weight of the substance, with approximately 750 mL of water (final ionic strength, 360 mosmol·L\(^{-1}\)). We used approximately half the dose of water recommended at the website of the Australian Institute of Sport, because we were concerned that the weight of ingested water would impair performance in a competition. Subjects consumed the gelatin capsules steadily with water over 90 min.

Performance test. Subjects fasted for 3 h before reporting to the laboratory to ingest a given substance and perform the run to exhaustion. Subjects commenced the run 90 min after ingestion of the last capsule (180 min from the start of the capsule consumption). The standardized warm-up consisted of 5 min of running on a treadmill (Quinton Q65, Seattle, WA) at 12 km·h\(^{-1}\), 5 min of stretching, and a 15-s stride-out at each of 4 and 2 km·h\(^{-1}\) below test speed and at test speed, followed by 2 min of standing quietly. The performance test itself consisted of a run to maximum effort on the treadmill at the same fixed speed for each of the four treatments. The speed was set to elicit maximum effort in 1–2 min with the treadmill set at an incline of 2%. (For one subject, the slope was set to 3% to prevent him out-running the speed of the treadmill.) The appropriate speed (range, 19–23 km·h\(^{-1}\)) was determined in a trial-and-error manner for each runner during a familiarization session, as follows. The treadmill speed was first set to a value estimated to elicit fatigue in 1.5 min. The estimate was based on modeled relationships between speed and run time (8) applied to a recent competition time and distance for the runner. If the time to exhaustion was not in the 1- to 2-min range, the runner rested for ~10 min then repeated the test at a new speed based on the previous test and the modeled relationships. No buffering agent was ingested for the familiarization trial.

Three investigators recorded time to exhaustion using stopwatches, and the median value was analyzed. Time was taken from the moment the subject jumped onto the belt, releasing the hands from the side of the treadmill, until he returned his hands to the side of the treadmill to jump off. Subjects reported the intensity of sickness and stomachache by viewing scales on a card at 30-min intervals from the beginning of ingestion until 120 min after the performance test. The scales showed integers from 0 to 10, with descriptors at 0, 3, 6, 9, and 10. The descriptors for feeling sick were not at all, slightly, quite, very, and throwing up; those for stomachache were none at all, dull ache on and off, moderate continuous, severe continuous, and severe doubled up.

Blood analysis. Three tubes of capillary blood were taken from seven of the 15 subjects just before the start of the warm-up. All samples were analyzed for blood lactate concentration (YSI 1500 sport, Yellow Springs Instrumentation, Yellow Springs, OH), and pH and bicarbonate (ABL 500, Radiometer, Copenhagen, Denmark).

Data analysis. We quantified the effect of buffers on performance with a repeated-measures analysis using Proc Mixed in the Statistical Analysis System (Version 6.12, SAS Institute, Cary, NC). Treatment was a fixed effect, and order of treatment was included as an additional fixed effect.
to account for continuing familiarization or other order effects. The random effects were identity of subjects and the within-subject variation between trials (the residuals). The same model was used to analyze changes in blood parameters.

Time to exhaustion was log-transformed to reduce non-uniformity of error, and the effects of the buffer treatments were derived by back transformation as percent changes (6). These changes were then converted into approximately equivalent changes in running speed in a constant-distance time trial of similar duration, using a factor derived by differential calculus (8). Briefly, speed (S) has to be expressed as a function f(D) of distance traveled. When the athlete’s fitness changes by a small amount as a result of a treatment, the resulting small change in speed dS for a fixed-distance test or event is related to the small change in distance dD for a fixed-speed test by the relationship \(-dS/dD = f'(D)\), the first derivative of f(D). For tests lasting 1–10 min, the critical-power model provides f(D), as follows. The critical-power model for running is D = a + mT, where T is time to exhaustion and a and m are constants (8). But S = D/T, so S = mD/(D - a) = f(D), and therefore dS/dD = -f'(D) = am/(D - a)^2. Rearranging, 100dS/S = percent change in S = \([a/(D - a)\]100dD/D = \([a(mT)\])\text{percent change in D}. But percent change in D in a constant-speed test = percent change in T in the test; hence, the required factor is \(a/(mT)\). Note that this factor differs from the \(a/(a + mT)\) in Reference (8), which is for a time trial of constant duration, not constant distance. Values for \(a\) (142.5 m) and \(m\) (4.7 m \(\cdot\) \(\text{s}^{-1}\)) came from a study of critical power of runners similar to those in the present study (4), and the value of T was each runner’s time in the placebo trial. The factor has a value of 0.38 for a test duration of 80 s or distance of 500 m.

Measures of centrality and spread are shown as mean \(\pm\) SD. Uncertainty in the estimates of effects is expressed as 90% confidence limits and as chances that the true value of the effect is practically beneficial, trivial, or harmful in relation to performance (5,13). To calculate these chances, we assumed that the smallest worthwhile change in time-trial performance for a competitive sprinter in contention for a medal is 0.5%, which would add 10% to the sprinter’s chances of winning (7). Thresholds for assigning qualitative terms to chances were as follows: <1%, almost certainly not; <5%, very unlikely; <25%, unlikely or probably not; <50%, possibly not; >50%, possibly; >75%, likely or probable; >95%, very likely; >99% almost certain (5,13). Magnitudes of changes in blood pH and concentrations of bicarbonate and lactate were interpreted qualitatively in terms of fractions and multiples of between-subject SD (6).

**RESULTS**

Times to exhaustion (in seconds) were placebo 77.4, citrate 78.2, lactate 80.2, and bicarbonate 82.3 (back-transformed least-squares means; overall between-subject coefficient of variation, 28%). Within-subject variation in time from treatment to treatment (standard error of measurement expressed as a coefficient of variation) was 8.4%. Table 1 shows the improvements in equivalent time-trial performance between treatments and chances of real improvement for elite athletes using one treatment relative to another. Twelve of the 16 subjects improved their time to exhaustion after ingestion of bicarbonate (the most effective buffer) in comparison with the placebo. All buffers produced some improvement relative to the chloride placebo, and the only insubstantial change between any treatments was that between citrate and chloride. The error of measurement in the equivalent time-trial time was 3.2%.

The mean resting values of blood bicarbonate, pH, and lactate after ingestion of each sodium salt immediately before the performance test are shown in Table 2. All buffer treatments produced a very large increase (at least two between-subject SD) in bicarbonate concentration and pH relative to the placebo; chances that these changes were substantial (at least 0.2 SD) were greater than 99%. Resting blood lactate showed a small-moderate decline with each buffer relative to placebo, but in all cases it was possible that the real change was trivial.

Figure 1 shows that mean ratings for sickness and stom-achache were low (\(<1.5\) on the 10-point scale). However, four of the initial 19 subjects experienced substantial nausea after intake of only part (\(<5–35\%)\) of the dose of sodium chloride. Two of these subjects withdrew from the study. The other two subjects completed the other treatments but not the placebo trial; their data are not included in Figure 1.

**DISCUSSION**

In the present study, we compared the effects of ingestion of three pH-buffering agents (sodium bicarbonate, lactate, and citrate) and a placebo (sodium chloride) on sprint performance. The observed mean effects of all three agents on performance were positive, with bicarbonate very likely and
lactate likely to have a substantially better true effect than chloride. Bicarbonate was also likely to be better than citrate, but the other comparisons of relative effectiveness of the four agents were less clear. The majority of subjects improved their performance following ingestion of bicarbonate in comparison with the placebo.

Precision of estimation of the differences in performance would have been better if the performance test had error of measurement as small as the 1–2% of some of the best sprint tests (8). At least part of the greater error observed with our performance test is likely to be due to individual responses to some of the treatments, which inflate the apparent error of measurement in controlled trials (6). It is also possible that a run to exhaustion lasting 1–2 min is inherently less reliable than self-paced distance or time trials of similar duration, although constant-power tests of longer duration are generally among the most reliable of all performance tests (8). Our subjects would have needed to perform an additional placebo trial to resolve this issue.

Although all three agents produced very large changes in blood pH and bicarbonate concentration, the order of effectiveness of the agents on performance did not mirror the order of changes in the blood. In particular, citrate produced the highest blood pH and bicarbonate concentration. We knew that citrate had the potential to raise blood bicarbonate and pH more than the other agents, because citrate ions have three negative charges whereas lactate and bicarbonate have only one. The molarity of anionic negative charges in a sodium citrate solution is therefore 1.5 times that of equi-osmolar bicarbonate or lactate solutions, so metabolism of the ingested citrate would lead, via consumption of hydrogen ions, to generation of more bicarbonate and a higher pH.

Exactly why performance was not best with citrate is not clear, but there are several plausible explanations. First, performance of short-term high-intensity exercise is probably limited in part by intracellular pH, but extracellular pH may not mirror intracellular pH with the different buffering agents. Second, an increase in intracellular citrate concentration after ingestion of citrate could reduce generation of ATP via inhibition of the enzyme phosphofructokinase (9). The latter explanation may also account for the discrepancy between our findings and those of Parry-Billings and MacLaren (18), who found that the effect of citrate on performance was at least as good as that of bicarbonate. These authors used doses of citrate and bicarbonate that were equal in mass (0.30 g·kg⁻¹). These doses would raise blood pH to approximately the same extent, and at the lower concentration than we used, the citrate might have a negligible effect on generation of ATP. Finally, differences in the timing of administration of the buffering agents between the studies could have resulted in different intracellular concentrations of citrate and consequently differences in inhibition of ATP production. The time course of the effect of each agent on performance following ingestion is a topic for more research.

Several researchers have suggested that sodium citrate might cause less gastrointestinal disturbances than sodium bicarbonate (16,17), but our subjects reported little or no sickness and stomachache with either or these agents or with sodium lactate. The only substance to cause substantial gastrointestinal distress was sodium chloride, and the distress occurred after consuming a partial dose. Kozak-Collins et al. (12) experienced a similar problem, when two of seven subjects reported mild nausea and two others vomited after consuming sodium chloride. We cannot exclude the possibility that performance in the placebo trial was impaired by the slight increase in the feelings of nausea preceding this trial, but the fact that there was little difference in feelings of nausea immediately after the trial makes this possibility remote, in our view. Hydration status was not measured during the current study and may have given more insight into the effect of the amount of water consumed on both performance and nausea.

In conclusion, we have found more evidence that ingestion of sodium bicarbonate is an effective strategy to enhance sprint performance. Sodium lactate is also likely to be effective, although possibly not as effective as sodium bicarbonate. Sodium citrate is probably not as effective as sodium bicarbonate. We therefore recommend ingestion of sodium bicarbonate to enhance sprint performance.
REFERENCES


EFFECTS OF VARIOUS BUFFERS ON SPRINTING

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