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Title: Effect of lactate supplementation and sodium bicarbonate on 40 km cycling time trial performance

Running head: Sodium bicarbonate and lactate time-trial

Authors: Matthew J. Northgraves[1], Daniel J. Peart[1,2] Christian A. Jordan[1] and Rebecca V. Vince[1*]

[1] Department of Sport, Health and Exercise Science, University of Hull, Hull, UK

[2] Biosciences, University Centre, North Lindsey College, Scunthorpe, UK

[*]Correspondence:

Dr Rebecca V. Vince

Department of Sport, Health and Exercise Science, University of Hull, Hull, HU6 7RX, UK

Telephone: +44 (0)1482 463176 / Fax: +44 (0)1482 465149

Email: rebecca.vince@hull.ac.uk

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ABSTRACT

The use of nutritional supplements to improve sporting performance and increase training adaptations is commonplace amongst athletes and is an expanding market in terms of product choice and availability. The purpose of this study was to examine the effects of two ergogenic aids with extracellular blood buffering potential, namely sodium bicarbonate ($NaHCO_3$) and a lactate supplement, during a 40 km cycling time trial. Seven recreationally active males (age, 22.3 ± 3.3 years; height, 182.5 ± 6.5 cm; body mass, 79.2 ± 6.3 kg) completed five 40 km cycling time trials, including a familiarization trial in a randomized blind double placebo design. Subjects ingested either (a) 300 mg per kg body mass $NaHCO_3$ (BICARB), (b) 45 mg per kg sodium chloride (PL-BICARB) as the placebo for the $NaHCO_3$ trial, (c) 1115 mg lactate (LACTATE), or (d) plain flour as the placebo for the lactate trial (PL-LACTATE) 60 minutes before exercise. There was no significant difference in performance between the four conditions ($p > 0.05$). Whilst $NaHCO_3$ ingestion induced significant changes in all the acid-base variables (all $p < 0.05$), no significant change was seen following lactate ingestion ($p > 0.05$). Subjects in the LACTATE condition did have a significantly higher heart rate ($p < 0.05$) without experiencing any greater perceived exertion ($p > 0.05$) than the other three conditions. Neither $NaHCO_3$ nor lactate supplementation appear to improve 40 km cycling time trial performance. However the potential benefits following LACTATE regarding perceived exertion require further research.

Key Words: buffering, alkalosis, ergogenic aid, $NaHCO_3$, acid-base

61 INTRODUCTION

62

63 The use of nutritional ergogenic supplements are commonplace within sport as both
64 recreational and professional athletes aim to improve performance and increase training
65 adaptations(19). Previous research into the benefits of induced metabolic alkalosis on both
66 prolonged continuous and intermittent high-intensity exercise has proved to be equivocal(22).
67 The majority of research has found no significant improvement in endurance performance
68 following induced alkalosis in cycling(1, 32, 36). The exception to these however was
69 McNaughton, Dalton and Palmer(21) who reported a 14% increase in work capacity during
70 60 minutes of high-intensity cycling following the ingestion of sodium bicarbonate
71 ($NaHCO_3$). If the results from such fixed time duration studies could be replicated in a more
72 practical setting such as time trial cycling involving set distance, then $NaHCO_3$ could prove
73 to be an inexpensive ergogenic aid. A negative side effect of $NaHCO_3$ however is the
74 possibility of gastrointestinal (GI) distress(6, 34) which ultimately may offset any possible
75 positive benefits to be gained.

76

77 Decreases in intramuscular pH have previously been reported to inhibit the contractile
78 processes by either a) restricting myofilament function through reducing Ca^{2+} sensitivity(7,
79 11) or b) effecting the excitation-contraction process relating to the uptake and release of Ca^{2+}
80 by the sarcoplasmic reticulum(16, 33). By ingesting $NaHCO_3$ prior to exercise, extracellular
81 bicarbonate (HCO_3^-) reserves are supplemented, resulting in an increased plasma pH and an
82 induced state of metabolic alkalosis(30). The extracellular to intracellular pH gradient
83 therefore increases as HCO_3^- is impermeable to cellular membranes(22) resulting in a greater
84 efflux of H^+ and lactate from active muscles(26). This occurs via either simple diffusion or by

85 the lactate/H^+ co-transporters(17) and has been demonstrated by the higher lactate
86 concentrations post-exercise following $NaHCO_3$ ingestion(1, 28). Increases in plasma HCO_3^-
87 have also been reported following the ingestion of lactate with no reported GI distress(24,
88 39), showing potential for it to be utilized as an alternative exogenous buffer to $NaHCO_3$.

89

90 Within exercise metabolism the role of lactate and in particular its production, has been a
91 source of much dispute(4, 11, 15). Debate remains whether the presence of lactate acts as a
92 limiting factor during exercise by inducing acidosis or actually attenuates the onset of fatigue
93 by consuming the excess H^+ responsible for acidosis(4). Many of the studies associating
94 lactate with the development of fatigue tend to be based upon correlational data(4) therefore a
95 cause and effect relationship cannot be ascertained. Furthermore, lactate has the potential to
96 serve as a source of glucose generated from within the body as a substrate for
97 gluconeogenesis(3). Based upon the lactate shuttle theory(2), exogenous lactate
98 supplementation therefore has the potential to increase plasma glucose supplied via
99 gluconeogenesis thus sparing glycogen stores[20]. However to date, research(3, 27, 38) has
100 failed to support this theory.

101

102 The purpose of this study therefore was to determine whether either $NaHCO_3$ or lactate
103 supplementation had any ergogenic potential to improve 40 km time trial performance.
104 Additionally it was designed to establish whether any improvement in performance was
105 associated with changes in acid-base status and buffering capacity. It was hypothesized that
106 the use of either $NaHCO_3$ or a lactate supplement would improve the performance of a 40 km
107 cycling time trial. Furthermore, it was hypothesized that lactate supplementation would

increase both plasma lactate levels and buffering capacity whilst causing less GI distress than is associated with $NaHCO_3$ consumption.

METHODS

EXPERIMENTAL APPROACH TO THE PROBLEM

Using a randomized, double placebo-controlled design; subjects were required to complete a total of five trials, one familiarization trial and four experimental trials. Subjects were instructed to arrive for testing in a rested state having refrained from strenuous exercise and alcohol in the 24 hour prior to testing and had no history of either $NaHCO_3$ or lactate supplementation. Subjects were asked to ingest a minimum of 500 ml of water before arriving at the laboratory to ensure they arrived in a well hydrated state, avoiding caffeine in the 12 hours prior to each trial. They were also asked to consume the same standardized breakfast a minimum of 1 hour prior to arriving for each trial. Subjects performed each of the five trials at the same time of day to control for circadian variation in performance(9). Additionally, each trial was separated by a period of 6 to 9 days in order to ensure an adequate recovery period was attained whilst limiting the opportunity for any improvement being the result of training.

The four experimental conditions were (a) 300 mg per kg body mass $NaHCO_3$ (BICARB), (b) 45 mg per kg sodium chloride (PL-BICARB) as the placebo for the $NaHCO_3$ trial, (c)

1115 mg lactate from a combination of calcium lactate pentahydrate and magnesium lactate dihydrate, equivalent to a mean of 14.1 $mg.kg^{-1}$ body mass per participant based on mean body weight (Sport Specifics, Inc., Chagrin Falls, OH, USA) (LACTATE), and (d) plain flour as the placebo for the lactate trial (PL-LACTATE). All supplements were ingested within gelatine capsules with 500 ml low calorie cordial over a 10 minute period, 60 minutes prior to exercise. Due to the disparity between the $NaHCO_3$ and lactate trials in terms of capsules required, a double placebo design was chosen to improve validity. The use of 300 mg per kg body mass $NaHCO_3$ has been established as the optimal dose for enhanced buffering capacity(22) and has previously been used in a number of studies into $NaHCO_3$ supplementation(5, 25, 33). Furthermore, peak HCO_3^- levels are typically achieved 60 minutes following ingestion(34). The lactate supplement dosage used was as per the manufacturer's instructions. It was felt that this dosage of the supplement should be chosen as consumers who purchase this supplement are unlikely to exceed the recommended dosage.

In terms of performance, the dependent variables of interest were overall performance time, split performance time, heart rate and rate of perceived exertion (RPE). For changes in acid-base status the dependent variables were pH, base excess (BE), HCO_3, lactate and H^+. Changes in overall performance time represent an accurate comparison between trials whilst ultimately being the key variable of interest for competitive cyclists. The use of split times allowed for changes during individual stages to also be identified. The use of pH, BE, HCO_3 and H^+ in research looking at changes in buffering capacity following supplementation is well established (5, 34, 35). Furthermore, as one of the supplements contained exogenous lactate, it was important to establish whether any changes in plasma lactate occurred following its ingestion.

SUBJECTS

Seven recreationally active non-smoking male subjects (mean ± SD: age, 22.3 ± 3.3 years; height, 182.5 ± 6.5 cm; body mass, 79.2 ± 6.3 kg) with no previous history of supplementing their diets with ergogenic agents volunteered to participate in this study. The subjects consisted of four cyclists, two footballers and a long distance runner, all whom were in a period of regular training at the time of testing. They were all completing a minimum of four hours (6.3 ± 3.3 hours) training per week and all were free from any known cardiac or metabolic diseases. All subjects provided written informed consent, and the study was approved by the Departmental Human Ethics Committee and following the principles outlined in the Declaration of Helsinki.

PROCEDURES

On arrival at the laboratory, the subject had a capillary blood sample taken to establish basal acid-base measurements (pH, BE, HCO_3^-, lactate and H^+) before ingesting the relevant supplement. During the 60 minute post-ingestion period, further capillary blood samples were taken at 10, 20, 30, 45 and 60 minutes post ingestion to evaluate any induced changes in the acid-base variables. All blood samples were collected using 100 µl balanced heparin blood capillary tubes (Radiometer, West Sussex, UK) and immediately analyzed (Radiometer, ABL800, Copenhagen, Denmark).

During the ingestion period, subjects were asked to rate any GI discomfort experienced every 15 minutes using a visual analogue scale (VAS) until the exercise commenced. The potential symptoms listed were: nausea, flatulence, stomach cramping, stomach bloating, stomach-ache, belching, vomiting, bowel urgency and diarrhoea. The VAS scale consisted of nine separate 100 mm scales, anchored at each end with either 'no symptom' or 'severe symptom' and subjects indicated with a vertical mark the severity of each symptom during the ingestion period(5, 34). None of the subjects reported any instances of GI disturbance during the 60 minute pre-exercise period as a result of ingesting BICARB, LACTATE or either placebo.

Following the 60 minute post ingestion capillary blood sample, the subject completed a ten minute warm up at an intensity of 75 watts prior to beginning the 40 km time trial. The time trial was conducted using a Wattbike cycle ergometer (Wattbike Ltd, Nottingham, UK) with heart rate (Polar FS1 HRM, Polar Electro, OY, Finland) and rate of perceived exertion (RPE) recorded at five minute intervals. RPE was monitored using a modified version of Foster et al.(12) perceived exertion scale. Subjects were permitted to drink water ad libitum. During each trial, subjects were blinded to all performance data except the distance countdown. The purpose for this was to minimize any learning effect to be gained from previous trials. Additional capillary blood samples were collected at 20 km, 40 km and at 15 minutes post-exercise.

STATISTICAL ANALYSES

Statistical analyses were completed using IBM PASW statistics 18 (SPSS inc., Chicago, Il). Central tendency and dispersion of all data are displayed as mean ± standard deviation (S.D). Performance time data was compared using a one way analysis of variance (ANOVA) with repeated measures whilst changes in acid-base status, heart rate and RPE were investigated using two way ANOVA with repeated measures. Sidak-adjusted *p* values were used for subsequent pairwise comparisons to establish the significant paired differences when significant *F* ratios were found by the respective ANOVA. Statistical significance was accepted as $p < 0.05$ with effect size reported according to partial eta squared.

RESULTS

PERFORMANCE DATA

The mean times for 10 km, 20 km, 30 km and 40 km along with the individual split times for each 10 km interval are displayed in Table 1. Whilst overall performance time for LACTATE was between 1-3% faster than the other three conditions, the difference was not significant ($p > 0.05$, $\eta_p^2 = 0.20$). Furthermore, the individual split times for each 10 km stage of the time trial were not significantly different between the four conditions ($p > 0.05$). Individual responses to the supplements were varied with 3 subjects performing their fastest trial in the LACTATE condition, whilst 2 performed fastest in PL-LACTATE, and 1 subject completing the time trial fastest in each of the BICARB and PL-BICARB conditions (Figure 1).

INSERT TABLE 1

INSERT FIGURE 1

Average heart rate during the LACTATE condition (169 ± 9 bpm) was significantly higher than in the other three conditions (BICARB: 160 ± 16 bpm; PL-BICARB: 158 ± 13 bpm; PL-LACTATE: 160 ± 14 bpm respectively; $p < 0.05$, $\eta_p^2 = 0.49$) throughout the duration of the time trials. No significant difference was seen however between the other three conditions. Both heart rate and RPE (both $p < 0.05$, $\eta_p^2 = 0.71$ and $\eta_p^2 = 0.85$ respectively) was seen to increase progressively with each 10 km stage of the time trial (Table 2). No significant main effect for condition or interaction effect between condition and stage (both $p > 0.05$, $\eta_p^2 = 0.18$ and $\eta_p^2 = 0.11$ respectively) was seen for RPE between the four conditions.

INSERT TABLE 2

ACID-BASE BALANCE

Changes in pH, BE, HCO_3^-, lactate and H^+ for the four conditions across the study are displayed in Table 3. There was no significant difference between pre-ingestion levels for any of the blood variables between the four conditions. During the BICARB condition, blood pH was significantly higher at 45 and 60 minutes post ingestion than seen at pre-ingestion ($p < 0.05$, $\eta_p^2 = 0.84$), whilst H^+ levels were significantly lower at the same time points ($p < 0.05$,

243 $\eta_p^2 = 0.67$). By 60 minutes post-BICARB ingestion, BE had increased by in excess of 5
244 mEq/L ($p < 0.05$, $\eta_p^2 = 0.85$) and plasma HCO_3^- by approximately 4.5 mmol/L compared to
245 the pre-ingestion levels ($p < 0.05$, $\eta_p^2 = 0.84$) and significantly increased compared to
246 LACTATE, PL-BICARB & PL-LACTATE (all $p < 0.05$) at the same time point. No
247 significant differences were seen within the other three experimental conditions during the
248 pre-ingestion to 60 minute post-ingestion period. Furthermore, no significant difference were
249 seen for lactate concentration between pre-ingestion and 60 minutes post-ingestion ($p >0.05$,
250 $\eta_p^2 = 0.11$) for any of the four conditions.

251

252 INSERT TABLE 3

253

254 After 20 km and 40 km, pH, BE and HCO_3^- remained elevated and H^+ was lower for the
255 BICARB condition compared to the other three conditions (Table 3). All the differences
256 between the BICARB condition and the other three conditions were significant except for pH
257 and H^+ at 40 km compared to PL-LACTATE (both $p >0.05$) and BE at 40 km compared to
258 the LACTATE condition ($p >0.05$). Whilst at the end of the time trial, plasma lactate was
259 between 2-3 mmol/L higher for the BICARB condition, the difference was only significant
260 compared to PL-LACTATE ($p < 0.05$, $\eta_p^2 = 0.48$). No significant difference was seen for
261 lactate between the LACTATE, PL-LACTATE and PL-BICARB.

262

263 DISCUSSION

264

Whilst mean performance time following the ingestion of the lactate supplement was over 30 seconds faster than the next nearest condition (Table 1) the performance effect was not significant. This mean difference was influenced by subject 2 whose individual time during the supplement trial was around 3 minutes faster than the other three conditions (Figure 1). Additionally ingestion $NaHCO_3$ did not provide any significant ergogenic effect on 40 km time trial performance. Using the same lactate supplement, Peveler and Palmer(27) also found no significant effect on performance of 20 km time trial cycling, heart rate or mean power with the lactate condition actually marginally slower than placebo by ~17.4 seconds on average. They did however fail to show the individual performance times for each condition making it difficult to establish if there was an individual specific response from any of their subjects. Additionally, other forms of lactate supplementation focusing on lactate as a gluconeogenic precursor for endurance exercise have also been previously used unsuccessfully. Both Bryner et al.(3) and Swensen et al.(38) combined lactate and carbohydrate to examine its effect on time to exhaustion (TTE). Bryner et al.(3) found no significant effect on either TTE or peak power using a protocol that involved cycling at 10 beats below target heart rate and ended with a Wingate power test during the last 30 seconds of the trial. Swensen et al.(38) also found no effect on TTE between when cycling at 70% VO_{2max} until exhaustion.

Whilst the current study also supports the majority of research in finding that $NaHCO_3$ did not improve prolonged exercise performance(1, 28, 36) one exception remains(21). McNaughton et al.(21) reported an increase in both overall work (in kilojoules) and average power following the ingestion of $NaHCO_3$ over a 60 minute period of maximal cycling. In this study the 40-km time trial was chosen as it represented a similar duration to that seen in

McNaughton et al.(21) however the use of a set distance as opposed to set time duration provided a greater reflection of competitive cycling.

The increase in buffering capacity achieved by ingesting $NaHCO_3$ prior to exercise has previously been well documented(6, 22, 35) although the benefits are typically associated with events lasting between 30 seconds to 3 minutes(22, 30, 34). Lactate levels during the current study were higher following $NaHCO_3$ ingestion than that of the other three conditions at both 20 km and 40 km although only to a significant level at 40 km over PL-LACTATE (Table 3). It has been suggested that by increasing extracellular levels of HCO_3^-, the efflux of lactate and H^+ from within the muscle is facilitated(13, 21) with similar results having previously been found by Price et al.(28). In theory this could have improved performance by maintaining pH closer to the homeostatic levels(21). McNaughton et al.(21) attributed their significant increase in work during their 60 minute cycling study to the maintenance of pH nearer to resting levels allowing greater contractile performance. Interestingly though, McNaughton et al.(21) also reported plasma lactate levels lower than those of their control (no supplement) and placebo (sodium chloride) trials. Whilst conflicting with the expected higher lactate levels seen in the current study, a difference in protocol may account for the disparity between studies.

Despite the improved acid-base status prior to and during exercise following $NaHCO_3$ ingestion in the current study, the lack of improvement in performance would appear to indicate an alternative factor separate from acidosis was the predominant cause of fatigue. Although using an alternative buffer in the form of sodium citrate, Schabort et al.(32) supported this as during the trial with the highest pH, lactate concentrations were not the

highest showing other factors contributed to the fatigue. Whilst allowing the subjects to consume the same standardized breakfast before each trial was intended to attenuate the effect of glycogen depletion on fatigue its effects cannot be ruled out. Furthermore the accumulation of inorganic phosphate rather than H^+ has also been associated with restricting the contractile processes(40) however as these were not measured in the current study, its role cannot be determined.

Lactate supplementation has also been suggested as an alternative acid-base buffer to $NaHCO_{3(24)}$ however the results from this study fail to support this. Previous research has reported increases in plasma HCO_3^- following lactate ingestion(10, 24, 39) however a lack of reported pre-ingestion HCO_3^- levels mean that the level of increase is difficult to quantifiy(10, 39). Morris et al.(24) reported increases in plasma HCO_3^- levels of approximately 3 mmol/L between pre-ingestion levels and 80 minutes post-ingestion. In the present study, four of the seven subjects experienced an increase in HCO_3^- following ingestion of the lactate supplement although the largest increase seen was just 1 mmol/L between pre-ingestion and 60 minutes post ingestion compared to an average increase of 4.6 mmol/L for the $NaHCO_3$ condition. However, the concentration of lactate supplement in this study was considerably less than the 120 mg/kg body mass of lactate used by Morris et al.(24) or the 320 mg/kg body mass of lactate used by Van Montfoort et al.(39). The reduced dosage in the current study was used as it followed the manufacturers' guidelines and is similar to that previously used by Peveler and Palmer(27).

In the current study, the expected increase in plasma lactate failed to be observed following the ingestion of the lactate supplement. The lactate shuttle theory by which exogenous lactate

supplementation is thought to increase gluconeogenesis and improve performance however is highly disputed(27). An increase in plasma lactate is thought to promote increased plasma lactate oxidation whilst inhibiting intramuscular lactate production(14). Miller et al.(23) regulated lactate plasma levels to 4 mmol/L during exercise of moderate intensity via intravenous infusion. As a result of this, the contribution of glycogenolysis in supplying plasma glucose decreased as increased lactate oxidation compensated potentially sparing glycogen stores. However, in the current study an increase in plasma lactate levels was not observed following ingestion. Neither Morris et al.(24) nor Van Montfoort et al.(39) reported significant increases in plasma lactate despite using considerably larger quantities of lactate. Whilst a change in the rate of lactate oxidation potentially may account for a rise in plasma lactate not being shown, other explanations may exist. It is possible that the oral consumption of lactate was either too small to elicit a change or it failed to increase the lactate availability due to degradation by stomach acid and/or lack of absorption, unlike the direct intravenous method used by Miller et al.(23).

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Given athletes are likely to follow manufacturers recommendations when using supplementations due to safety and overall cost issues(27), both the small change in acid-base status and the absence of an increase in plasma lactate following lactate supplementation, suggests the dosage used does not represent a viable alternative to $NaHCO_3$ for increasing buffering capacity. The use of a chronic dosing of lactate over a number of days may be an option in the future as it has been previously shown to increase acid-base status when using $NaHCO_3$(8, 20). However, given the negligible increases in acid-base status in this current study, each individual dose would possibly need to be increased from current study for any effect to be seen. Future investigations into alternative dosing strategies are therefore warranted

362

363 An increase in RPE over time was observed but this was not different between conditions
364 (Table 2). However average heart rate was higher during the LACTATE condition compared
365 to the other three conditions. Although performance differences were not significant overall
366 in this group, the increased heart rate during the LACTATE condition may therefore have
367 contributed for the faster performance time, although heart rate measurements were only
368 recorded every 5 minutes meaning heart rate for each 10 km stage is based on a total of two
369 or three measurements, which may have affected the results gained. Whilst the increased
370 heart rate in the LACTATE condition occurred without altering perceived exertion, the
371 subjective nature of RPE measure makes it difficult to conclude if the difference was related
372 to the supplement. In a similar study, Peveler and Palmer(27) reported reduced RPE
373 following the ingestion of a lactate supplement although this may have been accounted for by
374 the slower performance time compared to the placebo condition. The effect of induced
375 alkalosis upon the RPE following $NaHCO_3$ and lactate ingestion has been equivocal to date
376 with both positive(18, 31, 37) and negative(13) effects reported. This variety in results
377 however can probably be accounted for by the variety of different exercise protocols,
378 ingestion strategies and subject training statuses used throughout the previous literature(10,
379 13, 18, 31, 37).

380

381 PRACTICAL APPLICATIONS

382

383 The pursuit of legal ergogenic aids continues at both a recreational and professional level(21).
384 Whilst the majority of $NaHCO_3$ supplementation research has focused on either single or

multiple bouts of short duration maximal intensity exercise(22, 30, 34), the research conducted into prolonged continuous and intermittent exercise has proved equivocal(1, 21, 28). The current study has demonstrated there is little ergogenic benefit to be gained by inducing metabolic alkalosis via $NaHCO_3$ supplementation prior to prolonged cycling. Although not significant the LACTATE condition was fastest for three of the seven subjects and was on average approximately 30 seconds faster than the nearest condition. Whilst this figure was influenced by an individual performance of around 3 minutes faster during the lactate than the other three conditions, it raises the possibility that the ergogenic effect is individual specific. Considering the tight winning margins typically associated with time trial competition, any legal supplement that could provide such performance gains obviously would prove beneficial.

Using the dosages seen in the current study, lactate supplementation did not offer a viable alternative to $NaHCO_3$ in terms of improving blood buffering capacity. However, given $NaHCO_3$ ingestion is associated with GI distress(5, 6, 34) which may reduce any ergogenic benefits that may be achieved(34), research into alternative buffering agents is warranted. In this study no GI distress for either supplement was reported, suggesting that lactate supplementation is not associated with GI distress at this concentration and that the response to $NaHCO_3$ is individual specific, as recently alluded to by Price and Simons(29). Future work on lactate supplementation should therefore focus of dosing strategies in order to maximize the potential for an ergogenic effect to be seen on performance. Given any ergogenic effect of lactate supplementation appears to be individual specific, experimentation of the supplement prior to prolonged use is essential to assess the cost-benefit analysis to the individual.

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FIGURES

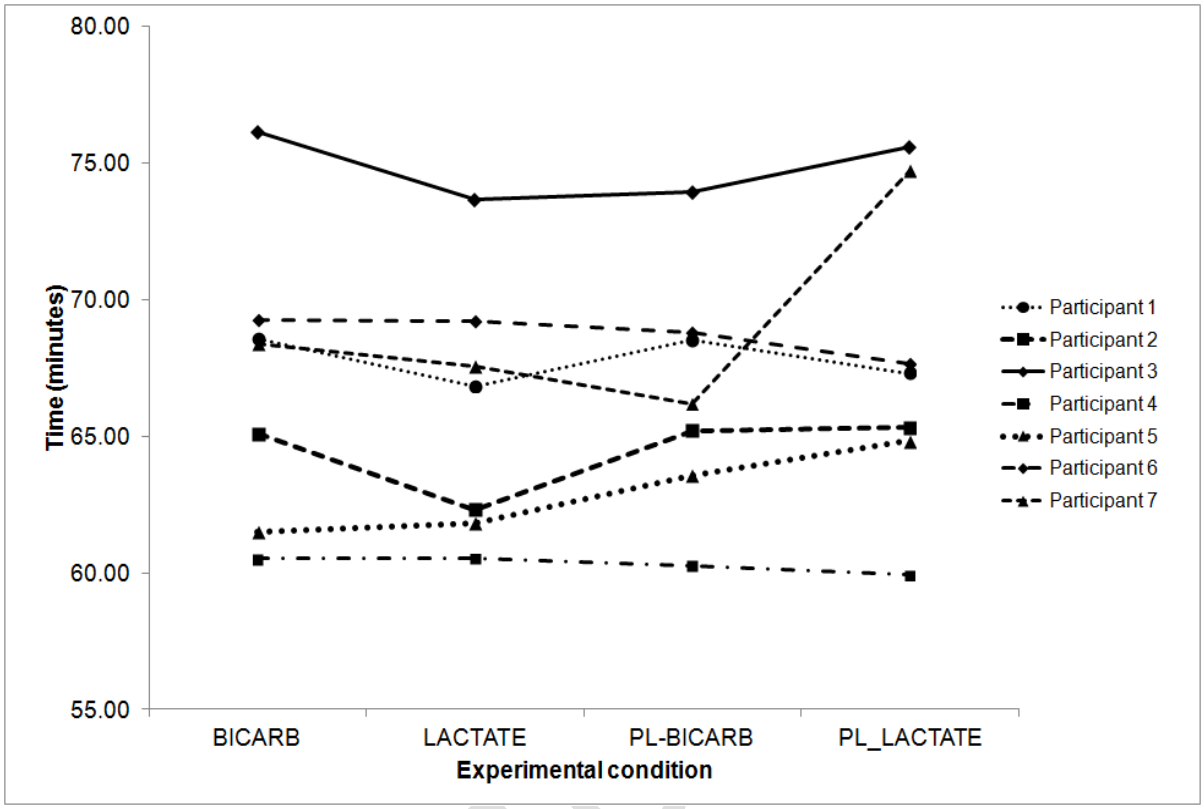

Figure 1. Individual performance times for each of the four experimental conditions

BICARB = Sodium Bicarbonate; LACTATE = Lactate supplement; PL-BICARB = Sodium Chloride; PL-LACTATE = Flour

TABLES

Table 1. Mean 40 km Cycling time trial performance times (minutes) including split times for the four experimental conditions (mean ± S.D) (n=7)

	10 km	20 km	30 km	40 km
BICARB	17.07 ± 1.45	34.06 ± 3.15	50.56 ± 4.29	67.08 ± 5.04
		(17.00 ± 1.32)	(16.50 ± 1.15)	(16.11 ± 1.00)
LACTATE	16.19 ± 1.14	32.55 ± 2.26	49.47 ± 3.40	66.02 ± 4.44
		(16.36 ± 1.15)	(16.52 ± 1.16)	(16.15 ± 1.06)
PL-BICARB	16.48 ± 1.21	33.31 ± 2.24	50.41 ± 3.23	66.41 ± 4.04
		(16.55 ± 1.12)	(16.59 ± 1.09)	(15.59 ± 0.50)
PL-LACTATE	16.57 ± 1.38	33.55 ± 2.59	51.05 ± 4.02	67.54 ± 5.34
		(16.58 ± 1.27)	(17.10 ± 1.08)	(16.49 ± 1.53)

Split times displayed in brackets

BICARB = Sodium Bicarbonate; LACTATE = Lactate supplement; PL-BICARB = Sodium Chloride; PL-LACTATE = Flour

Table 2. Mean heart rate and rate of perceived exertion for the four conditions during each 10 km stage of the time trial (mean ± S.D) (n=7)

	Stage (km)			
	0-10 km	10-20 km	20-30 km	30-40 km
Heart rate* (BPM)				
BICARB	149.3 ± 17.6	157.0 ± 15.0	161.4 ± 13.8	170.3 ± 10.6
LACTATE	162.9 ± 9.5	169.3 ± 6.1	169.7 ± 6.3	176.1 ± 4.3
PL-BICARB	150.9 ± 11.1	155.6 ± 10.5	158.7 ± 12.9	168.0 ± 12.7
PL-LACTATE	152.6 ± 17.6	158.0 ± 13.8	162.7 ± 10.1	168.7 ± 8.1
RPE*				
BICARB	4.4 ± 1.1	5.0 ± 1.0	5.8 ± 0.7	6.7 ± 0.8
LAC TATE	4.7 ± 0.5	5.5 ± 0.9	6.1 ± 1.2	7.6 ± 1.2
PL-BICARB	4.9 ± 1.3	5.6 ± 1.2	6.1 ± 0.5	7.3 ± 1.1
PL-LACTATE	3.9 ± 1.3	4.7 ± 0.9	5.5 ± 0.9	7.1 ± 0.9

*Significant main effect for stage, $p < 0.05$; BICARB = Sodium Bicarbonate; LACTATE = Lactate supplement; PL-BICARB = Sodium Chloride; PL-LACTATE = Flour

Table 3 Mean acid-base variables at different time points pre- and post- ingestion for the four conditions (mean ± S.D) (n=7)

		Pre-ingestion	10 min post-ingestion	20 min post-ingestion	30 min post-ingestion	45 min post-ingestion	60 min post-ingestion	20 km	40 km	15 min post-exercise
pH	BICARB	7.402	7.416	7.418	7.433	7.443*$^{+}$	7.450**$^{++}$	7.446*°	7.385°	7.460*
	LACTATE	7.399	7.403	7.401	7.402	7.398	7.399	7.366	7.332	7.388
	PL-BICARB	7.397	7.396	7.405	7.404	7.411	7.392	7.374	7.312	7.375
	PL-LACTATE	7.406	7.400	7.398	7.398	7.397	7.393	7.381	7.333	7.377
BE	BICARB	0.2	1.0	2.6	3.6	4.6*$^{+}$	5.7**$^{++}$	1.3*	-3.9$^{\#}$	2.3**
(mEq/L)	LACTATE	0.6	0.7	1.0	0.9	0.7	0.7	-6.0	-9.0	-3.5

	PL-BICARB	1.0	0.5	0.8	0.5	0.8	0.6	-4.6	-9.2	-4.4
	PL-LACTATE	0.6	0.3	0.2	0.6	0.7	0.7	-4.3	-7.7	-3.2
HCO_3	BICARB	24.6	25.3	26.4	27.4	28.3*$^{+}$	29.2**$^{++}$	26.0**$^{++}$	21.6*	26.7**
(mmol/L)	LACTATE	24.8	24.9	25.1	25.0	24.8	24.8	20.0	17.8	21.8
	PL-BICARB	25.0	24.7	25.0	24.8	25.1	24.7	20.9	17.4	21.1
	PL-LACTATE	24.8	24.6	24.5	24.8	24.7	24.7	21.3	18.6	22.0
H^{+}	BICARB	39.7	38.4	38.3	36.9	36.0*	35.5**	35.9**	41.4$^{\#}$	34.6*
(mmol/L)	LACTATE	39.9	39.6	39.7	39.7	40.0	40.0	43.1	46.6	41.0

	PL-BICARB	40.1	40.2	39.4	39.5	38.9	40.5	42.4	48.8	42.2
	PL-LACTATE	39.3	39.8	40.0	40.0	40.1	40.5	41.8	46.5	41.6
La	BICARB	2.2	1.9	2.0	1.9	1.8	1.6	8.6	12.8^	7.5
(mmol/L)	LACTATE	1.9	2.1	1.7	1.8	1.7	1.8	7.8	10.7	5.3
	PL-BICARB	1.8	1.8	1.7	1.8	1.6	1.6	6.6	10.6	5.4
	PL-LACTATE	1.6	1.7	1.8	1.6	1.7	1.7	6.9	9.7	5.0

BICARB = Sodium Bicarbonate; LACTATE = Lactate supplement; PL-BICARB = Sodium Chloride; PL-LACTATE = Flour; BE = Base excess; HCO_3 = Bicarbonate; H^+ = Hydrogen ion; La = Lactate

*Significant difference between BICARB and LACTATE/PL-BICARB/PL-LACTATE, $p < 0.05$, **Significant difference between BICARB and LACTATE/PL-BICARB/PL-LACTATE, $p < 0.01$, ° Significant difference between BICARB and LACTATE/PL-BICARB, $p < 0.05$, # Significant difference between BICARB and PL-BICARB/PL-LACTATE, $p < 0.05$, ^ Significant difference between BICARB and PL-LACTATE, $p < 0.05$, + Significantly different to pre-ingestion levels, $p < 0.05$, ++ Significantly different to pre-ingestion levels, $p < 0.01$.