The Effects of Sodium Phosphate Supplementation on Physiological Responses to Submaximal Exercise and 20 km Cycling Time-Trial Performance

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ABSTRACT

The aim of this study was to examine the effects of sodium phosphate (SP) supplementation on physiological responses to submaximal exercise and 20 km cycling time-trial performance. Using a randomised, double-blind, crossover design, 20 endurance-trained male cyclists (age: 31 ± 6 years; height: 1.82 ± 0.07 m; body mass: 76.3 ± 7.0 kg; maximal oxygen uptake [$\dot{V}O_{2max}$]: 57.9 ± 5.5 mL·kg^{-1}·min^{-1}) completed two supplementation trials separated by a 14-day washout period. The trials consisted of 10 minutes of cycling at 65% $\dot{V}O_{2max}$ followed by a 20 km time-trial. Expired air was monitored throughout each trial for the evaluation of $\dot{V}O_2$, minute ventilation ($\dot{V}_E$), and respiratory exchange ratio (RER). Heart rate was monitored during each trial along with ratings of perceived exertion (RPE) and blood lactate concentration. For four days before each trial, participants ingested 50 mg·kg fat-free-mass^{-1}·day^{-1} of either SP or placebo. There were no effects ($p \geq 0.05$) of supplementation on physiological responses during cycling at 65% $\dot{V}O_{2max}$. There were also no effects of supplementation on time-trial performance (placebo: 32.8 ± 2.2 mins; SP: 32.8 ± 2.3 mins). Nevertheless, relative to placebo, SP increased $\dot{V}_E$ (mean difference: 3.81 L·min^{-1}; 95% likely range: 0.16-7.46 L·min^{-1}), RER (mean difference: 0.020; 95% likely range: 0.004-0.036), and RPE (mean difference: 0.39; 95% likely range: 0.04-0.73) during time-trials; as well as post time-trial blood lactate concentration (mean difference: 1.06 mmol·L^{-1}; 95% likely range: 0.31-1.80 mmol·L^{-1}). In conclusion, SP supplementation has no significant effects on submaximal physiological responses or 20 km time-trial performance.

Key Words: Ergogenic; endurance; serum phosphate; 2, 3-diphosphoglycerate

INTRODUCTION

Sodium phosphate (SP) is a legal nutritional supplement that has been suggested to improve athletic performance (Currell et al., 2012). Several mechanisms have been proposed to explain this potential ergogenic effect, including an increase in resting erythrocyte 2, 3-diphosphoglycerate (2, 3-DPG) concentration (promoting oxygen offloading at the muscle via a reduction in oxyhaemoglobin affinity) (Bremner et al., 2002; Cade et al., 1984), an enhancement of myocardial contractility (Kreider et al., 1992), an increase in extracellular hydrogen phosphate (HPO_4^-) concentration (facilitating hydrogen ion buffering) (Buck et al., 2015; Kopec et al., 2016), and an increase in the activity of various oxidative enzymes, such as phosphofructokinase and glyceraldehyde 3-phosphate dehydrogenase (Buck et al., 2013).

Given the above mechanisms, it has been hypothesised that SP supplementation may increase aerobic metabolism, and thus enhance endurance performance (Buck et al., 2013; Fukuda et al., 2010). In corroboration, despite one conflicting report (West et al., 2012), research into SP supplementation has consistently shown an increase in maximal oxygen uptake ($\dot{V}O_{2max}$) (Brewer et al., 2013; Cade et al., 1984; Czuba et al., 2009; Kreider et al., 1992; Kreider et al., 1990; Stewart et al., 1990). Nevertheless, research examining the effects of SP supplementation on endurance performance has produced conflicting results, with some studies reporting significant improvements (Folland et al., 2008; Kreider et al., 1992), while others report no effect (Brewer et al., 2013, 2014; Buck et al., 2014; Kreider et al., 1990). It is also difficult to determine the effects of SP supplementation on physiological responses during endurance exercise, possibly because most investigations have evaluated those responses during self-paced time-trials or incremental tests rather than fixed-intensity bouts of exercise. As such, while some studies have shown a significant increase in oxygen uptake ($\dot{V}O_2$) following SP supplementation (Czuba et al., 2009; Kreider et al., 1990, 1992), others have observed no effect (Brewer et al., 2014; Folland et al., 2008). Similarly, some investigations have demonstrated a SP-induced

71 decrease in heart rate (Czuba et al., 2009), whereas others have reported no effect (Brewer et al., 2013,
72 2014; Folland et al., 2008; Kreider et al., 1990, 1992; West et al., 2012).

73

74 It is difficult to attribute the conflicting responses of SP supplementation to methodological
75 differences between studies, since discrepant findings exist regardless of differences in dosing
76 strategies, mode of exercise, or participant training status. Indeed, despite some differences in the
77 duration of supplementation (typically 6 days) the dose range used in previous research is very small
78 (3.3-4.0 $g \cdot day^{-1}$; Buck et al., 2013). Moreover, all studies into SP supplementation have used trained
79 participants; though between-study differences in $\dot{V}O_{2max}$ (50-75 $mL \cdot kg^{-1} \cdot min^{-1}$) support clear
80 differences in levels of ability. The issue of training status is important since well-trained athletes have
81 been shown to have already elevated resting erythrocyte 2, 3-DPG levels (Brodthagen et al., 1985),
82 likely due to an adaptive training response (Mairbäurl, 2013). However, the suggestion that well-trained
83 athletes may be less responsive to the potential ergogenic benefits of SP supplementation seems unlikely
84 considering that previous research has demonstrated that SP can improve time-trial performance in
85 those individuals (Folland et al., 2008; Kreider et al., 1992).

86

87 The factor that explains most likely the discrepant findings regarding the effects of SP
88 supplementation on endurance performance is statistical error associated with the use of small sample
89 sizes (Button et al., 2013). Small sample sizes reduce the chances of finding a true effect as well as
90 reducing the likelihood that a statistically significant finding reflects a real effect (Button et al., 2013).
91 Indeed, of those studies that have investigated the effects of SP supplementation on endurance
92 performance, the largest sample size was 13 (Buck et al., 2014), with most using sample sizes ≤ 10
93 (Brewer et al., 2013, 2014; Folland et al., 2008; Kreider et al., 1990, 1992). The principal aim of this
94 study was therefore to address the issue of sample size in order to examine the effects of SP
95 supplementation on endurance (20 km cycling time-trial) performance. In addition, by examining the
96 effects of SP supplementation on physiological responses during both fixed-intensity submaximal

cycling and time-trial performance, the study also aimed to provide insight into the potential mechanisms behind any ergogenic effect of SP supplementation.

METHODS

Participants

Twenty endurance-trained (De Pauw et al., 2013) male cyclists and triathletes volunteered for the study, which was approved by St. Mary's University Ethics Committee. Sample size calculations were performed based on the results of previous investigations into the effects of SP supplementation on endurance performance (Brewer et al., 2013, 2014; Buck et al., 2014; Folland et al., 2008; Kreider et al., 1990, 1992). Using the associated effect sizes, a power of 0.8, and a *p* value of 0.05, the analyses produced sample sizes ranging from 2 to 20,000. Given the practical limitations associated with recruiting trained participants, a sample size of 20 was chosen as it fell within the range determined from the calculations and was, with one exception (n = 13), at least double the sample size used in previous investigations. Before testing, participants received written and verbal instructions regarding the nature of the investigation and completed a training history questionnaire, which indicated that all had been actively involved in road cycling for at least one year. Time spent training each week was reported as 9.4 ± 3.9 hours. Before commencement of the study, all participants completed a health-screening questionnaire and provided written informed consent. Means ± standard deviation for age, height, body mass, fat-free-mass (FFM), and $\dot{V}O_{2max}$ of the participants were: 31 ± 6 years, 1.82 ± 0.07 m, 76.3 ± 7.0 kg, 67.3 ± 6.3 kg, and 57.9 ± 5.5 $mL \cdot kg^{-1} \cdot min^{-1}$, respectively. Participants were instructed to maintain a consistent training volume throughout the study and to follow the same diet for 24 hours before all trials. Participants were also instructed to avoid food and drink for 1 hour before all trials and to abstain from caffeine, alcohol, and strenuous exercise for 24 hours before all trials.

Experimental overview

Participants were required to complete one preliminary trial followed by two experimental trials. The preliminary trial was used to provide descriptive data and to determine the fixed-intensity submaximal cycling workloads employed during the first part of the experimental trials. The experimental trials were performed in a crossover, randomised, counterbalanced, and double-blinded manner, separated by 14 days to allow for the washout period of SP (Cade et al., 1984). In line with strategies used in previous research (Buck et al., 2013), for four consecutive days before each experimental trial, participants ingested 50 mg·kg FFM^{-1}·day^{-1} of either tribasic SP dodecahydrate (Iron Power, Melbourne, Australia) or placebo (maltodextrin; My Protein, Manchester, United Kingdom). Daily amounts were divided into four equal doses, with each dose administered in an opaque gelatine capsule (My Protein, Manchester, United Kingdom). As in previous research (Brewer et al., 2013, 2014; Buck et al., 2014), doses were ingested at ~4 hour intervals with a meal and ~300 mL of water to prevent gastrointestinal discomfort. Exercise, other than the time trials, was performed on an electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Lode BV, Groningen, The Netherlands). Time trials were performed on a racing bicycle (San Remo, Claud Butler, Brigg, United Kingdom) seated on a motor-braked turbo trainer (Tacx Genius, Aardenburg, the Netherlands). Ergometers of this type have previously been shown to have very good test-retest reliability for 20 km time-trial performance (Peiffer & Losco, 2011). The cycle ergometer and the racing bicycle were fitted with clipless pedals and the participants cycled using their own cycling shoes. Saddle height and handlebar position for each participant were determined during the preliminary trial to enable replication in subsequent trials. Prior to all trials equipment was calibrated in accordance with manufacturer instructions.

Procedures

Preliminary trial

All trials were performed at the same time of day (± 2 hours) in an air-conditioned laboratory maintained at a temperature of 18°C. The preliminary trial began with the calculation of participant

FFM using air-displacement plethysmography (device for measuring volume changes within a body) (BOD-POD, Life Measurement Inc., Concord, CA, USA). Subsequently, participants performed an incremental exercise test which began at 120 W and increased by 20 W every 3 minutes. Participants were given 30 s during the first stage to achieve a comfortable cadence and were instructed to maintain this throughout the remainder of the incremental tests. Each stage was followed by 30 s of passive rest, during which 20 μL of capillary blood was obtained from the earlobe and analysed for blood lactate concentration using an automated analyser (Biosen C-Line, EKF Diagnostic, Barleben, Germany). The test was terminated when a blood lactate concentration ≥ 4 mmol·L^{-1} was attained. After 5 minutes of passive rest, the maximal phase of the incremental exercise test began at 160 W and increased by 20 W every minute. The test was terminated when participants reached volitional exhaustion, at which time a final blood lactate concentration measurement was obtained. Throughout both phases of the incremental exercise test, participants breathed room air through a facemask (Hans Rudolph, Kansas City, MO, USA) that was secured in place by a head-cap assembly (Hans Rudolph, Kansas City, MO, USA). Expired air was monitored continuously using an online gas analyser (Oxycon Pro, Jaeger, Hoechberg, Germany). The analyser was calibrated before each trial using oxygen and carbon dioxide gases of known concentrations (Cryoservice, Worcester, UK), and the flowmeter was calibrated using a 3 L syringe (Viasys Healthcare GmbH, Hoechberg, Germany). All $\dot{V}O_2$ data were filtered to eliminate values that were outside four standard deviations of the local mean (the two breaths preceding and following the breath of interest). Oxygen demand for each of the submaximal incremental stages was determined as the average $\dot{V}O_2$ during the final 30 s of each 3-minute stage. $\dot{V}O_{2max}$ was determined as the highest 30 s average $\dot{V}O_2$ recorded during the maximal phase of the test provided that at least two of the following criteria had been met: (a) a plateau in $\dot{V}O_2$, as determined by an increase of less than 2 mL·kg^{-1}·min^{-1} over the previous stage, (b) a heart rate within 10 b·min^{-1} of age-predicted maximum, (c) a respiratory exchange ratio (RER) ≥ 1.15, and (d) a blood lactate concentration ≥ 8 mmol·L^{-1}. Linear regression and individual power output-$\dot{V}O_2$ relationships were used to calculate the fixed-intensity submaximal cycling workloads required to elicit 65% of $\dot{V}O_{2max}$, to be employed during the experimental trials. After a 10-minute recovery period, where participants cycled at a self-selected low

intensity, a familiarisation 20 km time-trial (same protocol as in the experimental trials) was performed to minimise any potential changes in performance due to learning.

Experimental trials

Prior to each experimental trial, participants rested in a seated position for 5 minutes, after which 300 μL of capillary blood was collected from the earlobe. Blood samples were left to clot at room temperature for 60 minutes before being centrifuged at 4000 rpm for 10 minutes at 4°C. Subsequently decanted serum samples were frozen at -80°C until analysed for serum phosphate concentration using an automated analyser (Monza, Randox, London, UK). Participants then performed 10 minutes of cycling at 65% of the power output required to elicit $\dot{V}O_{2max}$, maintaining the same cadence as in the submaximal incremental test during the preliminary trial. Oxygen uptake, minute ventilation ($\dot{V}_E$), RER, and heart rate (RCX3, Polar Electro Oy, Kempele, Finland) were monitored continuously during cycling at 65% $\dot{V}O_{2max}$ and averaged over the final 30 s of each 5-minute split to provide mean responses at 5 minutes and 10 minutes. Blood lactate concentration and rating of perceived exertion (RPE; 15-point scale; Borg, 1970) were also determined at 5 minutes and 10 minutes during cycling at 65% $\dot{V}O_{2max}$. After 10 minutes of passive rest, participants completed a 20 km time-trial on the turbo trainer with the bicycle rear tyre pressure at 100 psi. The time-trial was performed against a resistance designed to replicate outdoor, level-gradient cycling conditions. No verbal encouragement was provided and all measures of elapsed time were removed from the environment. The only pertinent information visible to participants throughout each time-trial was the distance completed. Participants were free to change cadence and gears throughout each time-trial; however, the gearing chosen during the familiarisation time-trial was noted and used to standardise the starting intensity for the experimental time-trials. Distance completed, power output, and cadence were recorded at 1 Hz throughout each experimental time-trial. Expired air was monitored continuously throughout each experimental time-trial for the evaluation of $\dot{V}O_2$, $\dot{V}_E$, and RER. Heart rate was monitored continuously throughout each experimental

time-trial and RPE was recorded at 5 km intervals. Blood lactate concentration was determined 1 minute before and immediately after each experimental time-trial.

Statistical analyses

All data were analysed using the Statistical Package for the Social Sciences (version 22, IBM SPSS, Armonk, NY, USA). Data are presented as means ± standard deviation, and 95% confidence intervals are provided for all estimates. A paired samples *t*-test was used to determine the effects of supplementation on resting serum phosphate concentration. Two-way (supplement × time) analyses of variance (ANOVAs) were used to determine the effects of supplementation and time on physiological responses ($\dot{V}O_2$, $\dot{V}_E$, RER, heart rate, RPE, and blood lactate concentration) during exercise at 65% $\dot{V}O_{2max}$. Two-way (supplement × 5 km split) ANOVAs were used to determine the effects of supplementation and 5 km splits on 20 km time-trial performance measures (completion time, power output, and cadence) and physiological responses ($\dot{V}O_2$, $\dot{V}_E$, RER, heart rate, and RPE). A two-way (supplement × time) ANOVA was used to determine the effects of supplementation and time on blood lactate concentration prior to and immediately following time-trial performance. Violations to assumptions of sphericity were adjusted using the Greenhouse-Geisser correction factor (Field, 2013). Significant effects were followed up using *post hoc* tests with Bonferroni adjustments (Field, 2013). The significance level was set at $p < 0.05$ for all analyses.

RESULTS

Serum phosphate

Placebo supplementation resulted in a resting serum phosphate concentration of 0.77 ± 0.18 $mmol \cdot L^{-1}$, whereas SP supplementation resulted in a resting serum phosphate concentration of 0.76 ± 0.15 $mmol \cdot L^{-1}$. There was no significant effect of supplementation on resting serum phosphate concentration ($p = 0.762$).

225

226 **Physiological responses to submaximal fixed-intensity exercise**

227 The mean power output during the submaximal exercise bouts was 186 ± 34 W. There was no
228 significant effect of supplementation on $\dot{V}O_2$ (p = 0.694), $\dot{V}_E$ (p = 0.950), RER (p = 0.298), heart rate (p
229 = 0.885), RPE (p = 0.650), or blood lactate (p = 0.375) during cycling at 65% of $\dot{V}O_{2max}$ (Table 1).
230 There was also no significant effect of time on $\dot{V}O_2$ (p = 0.766) or RER (p = 0.656). However, there
231 was a significant effect of time on $\dot{V}_E$ (p = 0.001), heart rate (p < 0.001), RPE (p < 0.001), and blood
232 lactate (p = 0.033). *Post hoc* tests revealed that from 5 min to 10 min, $\dot{V}_E$, heart rate, and RPE increased
233 significantly, whereas blood lactate decreased significantly. There were no significant supplement ×
234 time interactions for $\dot{V}O_2$ (p = 0.982), $\dot{V}_E$ (p = 0.777), RER (p = 0.495), heart rate (p = 0.641), RPE (p
235 = 0.095), or blood lactate (p = 0.573).

236

237 **Time-trial performance**

238 There was no significant effect of supplementation on completion time (p = 0.975), power
239 output (p = 0.777), or cadence (p = 0.503) during the time-trials (Table 2). However, there was an effect
240 of 5 km split on completion time (p < 0.001), with significant differences between all comparisons apart
241 from that between the 5-10 and 10-15 km splits. Similarly, there was a significant effect of 5 km split
242 on power output (p < 0.001). *Post hoc* tests revealed that participants produced a significantly higher
243 power output in the final 5 km of each time-trial in comparison with each of the other 5 km splits. There
244 was also an effect of 5 km split on cadence (p = 0.001), with significantly increased values in the 5-10
245 and 15-20 km splits, when compared with the 0-5 km split. There were no significant supplement × 5
246 km split interactions for completion time (p = 0.505), power output (p = 0.512), or cadence (p = 0.566).

247

248 **Time-trial physiological responses**

Participants completed the placebo and SP experimental time-trials at a mean intensity of 83 ± 8% of $\dot{V}O_{2max}$. There was no significant effect of supplementation on $\dot{V}O_2$ ($p = 0.944$) or heart rate ($p = 0.141$) during the time-trials (Table 3). However, there was an effect of 5 km split on $\dot{V}O_2$ ($p = 0.012$), with significantly increased values in the 15-20 km split, when compared with the 0-5 and 10-15 km splits. There was also an effect of 5 km split on heart rate ($p < 0.001$), with mean values increasing throughout the time-trials and with *post hoc* tests revealing significant differences between all comparisons apart from that between the 5-10 and 10-15 km splits. There were no significant supplement × 5 km split interactions for $\dot{V}O_2$ ($p = 0.701$) or heart rate ($p = 0.111$).

There was an effect of supplementation on $\dot{V}_E$ ($p = 0.042$) during the time-trials (Table 3), with SP resulting in significantly higher values than placebo (mean difference: 3.81 $L \cdot min^{-1}$; 95% likely range: 0.16-7.46 $L \cdot min^{-1}$). There was also an effect of 5 km split on $\dot{V}_E$ ($p < 0.001$), with mean values increasing throughout the time-trials and with *post hoc* tests revealing significant differences between all comparisons apart from that between the 5-10 and 10-15 km splits. However, there was no significant supplement × 5 km split interaction for $\dot{V}_E$ ($p = 0.103$). There was also an effect of supplementation on RER ($p = 0.020$) during the time-trials (Table 3), with SP resulting in significantly higher values than placebo (mean difference: 0.020; 95% likely range: 0.004-0.036). However, there was no significant effect of 5 km split on RER ($p = 0.095$) and no supplement × 5 km split interaction ($p = 0.978$).

There was an effect of supplementation on RPE ($p = 0.030$) during the time-trials (Table 3), with SP resulting in significantly higher values than placebo (mean difference: 0.39; 95% likely range: 0.04-0.73). There was also a significant effect of 5 km split on RPE ($p < 0.001$). *Post hoc* tests revealed a progressive increase in RPE throughout the time-trials with significant differences between all comparisons. However, there was no significant supplement × 5 km split interaction for RPE ($p = 0.632$).

There was a significant effect of supplementation on blood lactate concentration ($p = 0.003$; Figure 1). Blood lactate concentration also significantly increased from pre-time-trial to post-time-trial ($p < 0.001$). Moreover, there was a significant supplement × time interaction ($p = 0.006$). *Post hoc* tests revealed that there was no significant effect of supplementation on pre-time-trial blood lactate concentration ($p = 0.738$); however, relative to placebo, post-time-trial blood lactate concentration was significantly increased with SP (mean difference: 1.06 mmol·L^{-1}; 95% likely range: 0.31-1.80 mmol·L^{-1}; $p = 0.004$).

DISCUSSION

The principal aim of this study was to examine the effects of SP supplementation on 20 km cycling time-trial performance. The key finding was that SP supplementation had no significant effect on time-trial completion time. Supplementation with SP also had no significant effect on power output or cadence during the time-trials. The absence of any significant effect of SP supplementation on time-trial performance is consistent with some reports (Brewer et al., 2013, 2014; Buck et al., 2014; Kreider et al., 1990), but not others (Brewer et al., 2015; Folland et al., 2008; Kreider et al., 1992). However, it is worth noting that the small sample sizes associated with previous research increase the risk of false positives and reduce the likelihood that findings reflect a true effect (Button et al., 2013). Given that the present study was the first to examine the effects of SP supplementation on time-trial performance using a relatively large sample size, the findings of the present investigation add considerable weight to the argument that SP supplementation provides no ergogenic benefit during time-trial performance.

Resting serum phosphate concentrations in the present study were slightly lower than anticipated, but were within the normal range for adults (Buck et al., 2013). Nevertheless, relative to placebo, the present study demonstrated no SP-induced increase in resting serum phosphate concentration. Apart from one exception (Czuba et al., 2009), previous research has also reported no change in serum phosphate concentration following SP supplementation (Brewer et al., 2013; Buck et

al., 2015; Kopec et al., 2016; Kreider et al., 1990; Stewart et al., 1990). Given similarities in the dosing strategies used in these investigations, it seems, as highlighted by others (Buck et al., 2015; Kopec et al., 2016; Kreider et al., 1992; Stewart et al., 1990), that the measure may not be the best indicator of SP loading effects.

A secondary aim of the present study was to investigate the potential mechanisms behind the ergogenic effects of SP supplementation. If, as hypothesised, SP supplementation improves oxygen offloading at the muscle via an increase in erythrocyte 2, 3-DPG levels (Bremner et al., 2002; Cade et al., 1984), then an enhancement of aerobic metabolism would be expected. However, given that there was not only no SP-induced improvement in time-trial performance, but also no increase in $\dot{V}O_2$ during either fixed-intensity submaximal cycling or time-trial performance, the findings of the present study fail to provide support for the above mechanism. Given that the period of fixed-intensity submaximal cycling was performed at a lower intensity than the time-trials, it seems unlikely that the absence of an effect of SP on $\dot{V}O_2$ could be due to exercise intensity. Although resting erythrocyte 2, 3-DPG levels were not measured in the present study, an alternative explanation for the lack of any increase in $\dot{V}O_2$ is that SP supplementation may not increase 2, 3-DPG concentration in red blood cells. Indeed, of those studies that have measured 2, 3-DPG concentrations following SP supplementation, Cade et al. (1984) and Stewart et al. (1990) reported significant increases, Buck et al. (2015), Czuba et al. (2008), Kopec et al. (2016), and Kreider et al. (1992) reported no change, and Kreider et al. (1990) reported a significant decrease. Once again, the use of a small sample sizes may have influenced these findings along with the fact that erythrocyte 2, 3-DPG levels are already elevated in endurance trained individuals (Brodthagen et al., 1985; Buck et al., 2013) and can change rapidly post-sampling (Llohn et al., 2005).

An alternative mechanism by which SP supplementation has been suggested to increase aerobic metabolism is via an enhancement of myocardial contractility (Buck et al., 2013; Fukuda et al., 2010). Indeed, using cardiac ultrasound and colour flow Doppler technology, Kreider et al. (1992) reported

SP-induced increases in various measures of cardiac function in well-trained athletes, albeit concomitant with increases in time-trial performance (Kreider et al., 1992). Then again, there were few effects of SP supplementation on those same measures when compared at each intensity during an incremental test, or when compared at the same relative intensity (anaerobic threshold). Moreover, although heart rate values alone are unlikely to be a reliable indicator of cardiac function; it is worth highlighting that the present study, and several others have reported no SP-induced change in heart rate during time-trial performance (Brewer et al., 2013, 2015; Folland et al., 2008; Kreider et al., 1990). Overall, considering that the present study found no effect of SP supplementation on $\dot{V}O_2$ or time-trial performance, it appears that any effect of SP on cardiac function does not translate into any ergogenic benefit.

It is difficult to explain how SP increased RER during the time-trial in the present study, but had no effect on RER during fixed-intensity submaximal cycling. Although the latter is supported by research showing no effect of SP supplementation on RER during submaximal incremental exercise (Kreider et al., 1992); previous research investigating the effects of SP supplementation on RER during time-trial performance has contrastingly reported no effect, despite a SP-induced increase in power output (Folland et al., 2008; Kreider et al., 1992). In the present study, a SP-induced increase in RER, in the absence of any change in $\dot{V}O_2$ during the time-trial, would support the corresponding increase in $\dot{V}_E$, as a result of an associated increase in $\dot{V}CO_2$. Then again, it is difficult to reconcile that response in the absence of any change in performance. To add to the confusion; of those studies that observed no SP-induced change in RER despite an increase in time-trial performance, Folland et al. (2008) reported no corresponding change in $\dot{V}_E$, while Kreider et al. (1992) reported an increase. Moreover, Brewer et al. (2014) reported no change in performance and no change in $\dot{V}_E$.

As with the above, it is difficult to reconcile how, in the absence of any change in performance, blood lactate concentrations in the present study increased following SP supplementation in the time

353 trial, but not during fixed-intensity submaximal exercise. Previous research has shown that SP
354 supplementation has no significant effect on post-time-trial blood lactate concentration regardless of
355 whether there was an increase in performance (Folland et al., 2008; Kreider et al., 1992) or not (Brewer
356 et al., 2013, 2014; Buck et al., 2014). In the present study, the increase in post-time-trial blood lactate
357 concentration is consistent with the corresponding increase in RER and $\dot{V}_E$ resulting from the need to
358 buffer associated hydrogen ions. However, an increase in blood lactate concentration generally indicates
359 an enhancement of anaerobic energy provision (Maughan & Gleeson, 2004) which, in the absence of
360 any change in $\dot{V}O_2$, would normally suggest an increase in performance. Similar contradictions in the
361 present study exist concerning RPE, with significant SP-induced increases during the time-trial, despite
362 no change in performance, contrasting with no effect of SP during fixed-intensity submaximal cycling.
363 Moreover, the result is in contrast with previous studies showing that SP has no effect on RPE during
364 (Folland et al., 2008; Kreider et al., 1990) or immediately after time-trial performance (Brewer et al.,
365 2014); though differential effects on performance add to the confusion. Given that the RPE scale was
366 initially validated against heart rate (Borg, 1970), it was unsurprising that the absence of an effect of
367 SP supplementation on heart rate during fixed-intensity submaximal cycling coincided with no SP-
368 induced change in RPE. However, it is unclear as to why the same response was not reflected during
369 the time-trials. One potential explanation for the increase in RPE during the time trials is that it was
370 induced by the corresponding increase in $\dot{V}_E$. In partial support, it has recently been shown that
371 breathing frequency correlates very strongly ($r = 0.89$) with RPE during time-trial performance (Nicolò
372 et al., 2016).

373

374 In conclusion, the results of the present study indicate that SP supplementation has no
375 significant effect on time-trial performance. Indeed, the associated increase in RPE suggests that SP
376 supplementation may result in endurance athletes having to work subjectively harder to achieve the
377 same level of time-trial performance. Given that SP has been proposed to improve endurance
378 performance primarily via aerobic mechanisms, the absence of any SP-induced change in $\dot{V}O_2$ or heart
379 rate provides further support for this lack of an ergogenic benefit. Notably, SP supplementation

increased RER and $\dot{V}_E$ during the time-trials and resulted in a higher post-time-trial blood lactate concentration all of which are difficult to explain; particularly given the absence of a corresponding effect during submaximal exercise or any effect on time trial performance.

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Declaration of Interest

The authors declare no conflicts of interest. The authors alone are responsible for the content and writing of the article.

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Table 1. The effects of sodium phosphate supplementation on various physiological responses during 10 minutes of cycling at 65% of the power output required to elicit maximum oxygen uptake (N = 20). Values are means ± standard deviation.

Supplement	Time (min)	$\dot{V}O_2$ ($L \cdot min^{-1}$)	$\dot{V}_E$ ($L \cdot min^{-1}$)	RER	Heart rate ($b \cdot min^{-1}$)	RPE	BLC ($mmol \cdot L^{-1}$)
Placebo	5	3.02 ± 0.47	69.76 ± 12.25	0.88 ± 0.06	128.1 ± 9.0	11.5 ± 1.4	1.76 ± 0.99
	10	3.02 ± 0.40	72.01 ± 11.35	0.88 ± 0.05	130.9 ± 10.2	12.2 ± 1.1	1.55 ± 1.16
SP	5	3.01 ± 0.47	69.99 ± 11.19	0.89 ± 0.06	128.2 ± 11.5	11.1 ± 1.4	1.83 ± 1.01
	10	3.00 ± 0.52	71.93 ± 11.56	0.89 ± 0.04	131.3 ± 12.5	12.3 ± 1.6	1.65 ± 1.33

Note: SP = sodium phosphate; $\dot{V}O_2$ = oxygen uptake; $\dot{V}_E$ = minute ventilation; RER = respiratory exchange ratio; RPE = rating of perceived exertion; BLC = blood lactate concentration.

Table 2. The effects of sodium phosphate supplementation on completion time, power output, and cadence during a 20 km cycling time-trial (N = 20). Values are means ± standard deviation.

Supplement	Distance (km)	Completion time (min)	Power output (W)	Cadence (rpm)
Placebo	0-5	8.40 ± 0.58	285 ± 56	96 ± 9
	5-10	8.18 ± 0.56	293 ± 55	97 ± 8
	10-15	8.21 ± 0.58	290 ± 57	97 ± 8
	15-20	7.97 ± 0.55	317 ± 58	98 ± 9
	0-20	***32.76 ± 2.20***	***296 ± 54***	***97 ± 8***
Sodium phosphate	0-5	8.43 ± 0.61	284 ± 62	96 ± 10
	5-10	8.19 ± 0.58	292 ± 59	98 ± 10
	10-15	8.19 ± 0.61	293 ± 60	98 ± 10
	15-20	7.95 ± 0.60	321 ± 64	99 ± 11
	0-20	***32.77 ± 2.31***	***297 ± 58***	***98 ± 10***

Note: rpm = revolutions per minute.

Table 3. The effects of sodium phosphate supplementation on various physiological responses during a 20 km cycling time-trial (N = 20). Values are means ± standard deviation.

Supplement	Distance (km)	$\dot{V}O_2$ ($L \cdot min^{-1}$)	Heart rate ($b \cdot min^{-1}$)	$\dot{V}_E$ ($L \cdot min^{-1}$)	RER	RPE
Placebo	0-5	3.58 ± 0.61	144.4 ± 11.6	95.4 ± 20.5	0.91 ± 0.06	14.2 ± 1.3
	5-10	3.67 ± 0.55	153.7 ± 12.0	103.0 ± 22.6	0.91 ± 0.05	15.6 ± 1.3
	10-15	3.64 ± 0.56	155.8 ± 13.2	105.0 ± 24.7	0.90 ± 0.05	16.2 ± 1.5
	15-20	3.79 ± 0.56	162.0 ± 13.0	117.0 ± 29.5	0.92 ± 0.06	17.9 ± 1.5
	0-20	***3.67 ± 0.55***	***154.1 ± 11.7***	***105.1 ± 22.7***	***0.91 ± 0.05***	-
Sodium phosphate	0-5	3.55 ± 0.72	144.7 ± 14.9	96.8 ± 22.3	0.93 ± 0.06	14.5 ± 1.2
	5-10	3.67 ± 0.67	155.2 ± 14.0	105.6 ± 23.3	0.93 ± 0.05	15.9 ± 1.1
	10-15	3.65 ± 0.62	158.9 ± 13.9	109.6 ± 24.5	0.92 ± 0.05	16.7 ± 1.3
	15-20	3.79 ± 0.62	164.9 ± 13.1	123.8 ± 28.7	0.95 ± 0.06	18.3 ± 1.3
	0-20	***3.67 ± 0.64***	***156.0 ± 13.1***	***108.9 ± 22.9***	***0.93 ± 0.05***	-

Note: $\dot{V}O_2$ = oxygen uptake; $\dot{V}_E$ = minute ventilation; RER = respiratory exchange ratio; RPE = rating of perceived exertion.

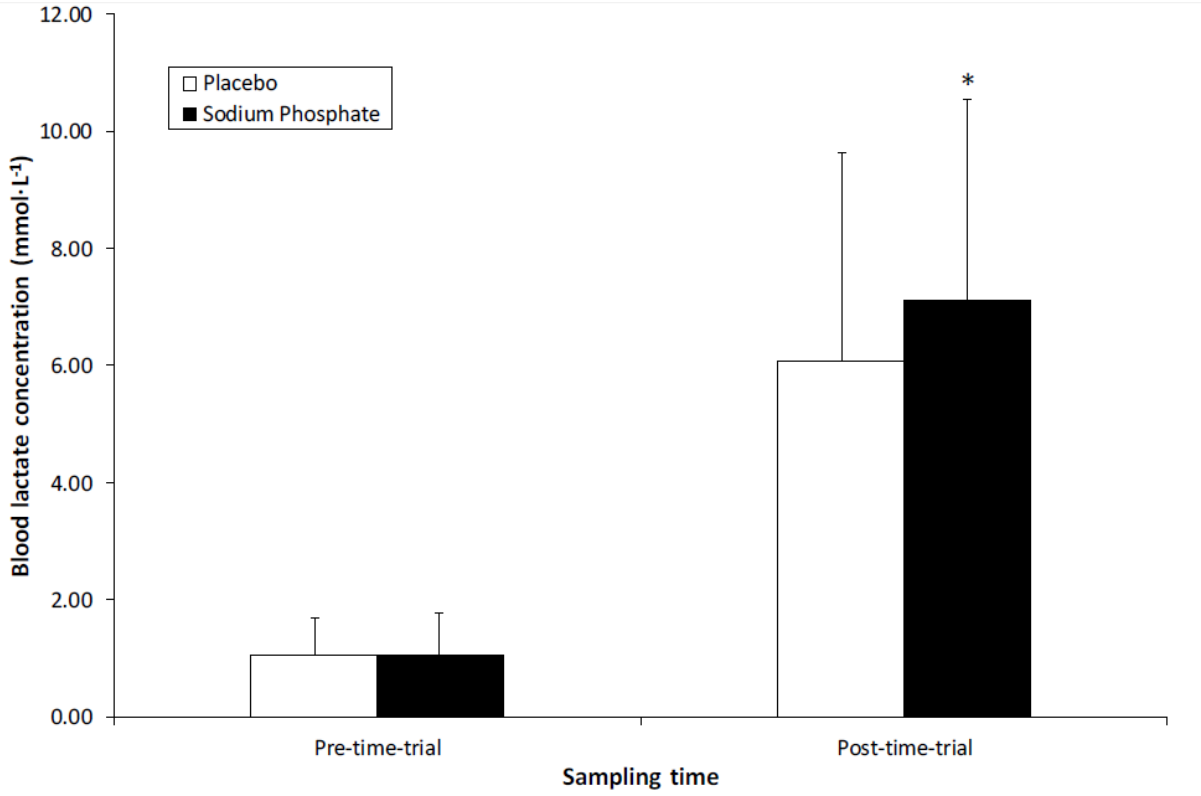

Figure 1. The effects of sodium phosphate supplementation on blood lactate concentration prior to and immediately following a 20 km cycling time-trial (N = 20). Values are means ± standard deviation. * indicates significantly different ($p < 0.05$) from placebo.