

Research article

Effect of Sodium Phosphate Supplementation on Cycling Time Trial Performance and VO_2 1 and 8 Days Post Loading

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Abstract

This study examined the effect of 6 days of sodium phosphate (SP) (50 mg·kg·FFM⁻¹·day⁻¹) or placebo (PL) supplementation in trained cyclists on either 100 kJ (23.9 Kcal) (~3-4 min) or 250 kJ (59.7 Kcal) (~10-12 min) time trials performances both 1 and 8 days post-supplementation. Trials were performed in a counterbalanced crossover design, with a 28-day washout period between supplementation phases. No significant differences, moderate-large ES (*d*) or likely (or greater) smallest worthwhile change (SWC) values were recorded for time to completion and mean power output on days 1 and 8 post-supplementation, both within and between SP and PL for either the 100 or 250 kJ (23.9 or 59.7 Kcal) trials. In the 100 kJ (23.9 Kcal) trial (only) first minute VO_2 tended to be higher in SP8 than both PL8 (*d* = 0.60; 88/10/2 SWC) and SP1 (*d* = 0.47; 82/15/3 SWC), as was mean VO_2 (PL8: *d* = 0.77; 93/6/1 SWC and SP1: *d* = 0.84; 90/8/3 SWC). No significant differences were found for heart rate, ratings of perceived exertion and blood lactate post-exercise within or between any trials, while serum phosphate values were not different before or after supplementation with SP or PL. In conclusion, this study showed a tendency for increased VO_2 in a short duration (100 kJ/ 23.9 Kcal: ~3-4 min) cycling test on day 8 after SP supplementation, but no differences in 100 or 250 kJ (23.9 or 59.7 Kcal) time trials performances were observed.

Key words: Serum phosphate, lactate, heart rate, RPE.

Introduction

Sodium phosphate (SP) is an ergogenic aid that has previously been reported to improve aerobic capacity by 3.5-12% (Brewer et al., 2013; Cade et al., 1984; Czuba, et al., 2008; 2009; Kreider et al., 1990; 1992; Stewart et al., 1990), with some benefits also found for endurance performance (Folland et al., 2008; Kreider et al., 1992).

In relation to these benefits, a number of possible mechanisms have been proposed. Specifically, these are: (i) increased 2, 3 diphosphoglycerate (DPG) concentrations, which can ultimately facilitate a greater release of oxygen from haemoglobin (Hb) to the muscle (Cade et al., 1984; Chanutin and Curnish, 1967; Farber et al., 1987; Stryer, 1988); (ii) increased extracellular and intracellular phosphate availability, resulting in greater glycolysis, glycolysis and oxidative phosphorylation, which may increase the rate of ATP production and enhance cellular metabolism during exercise (Chasiotis, 1988; Lichtman et al., 1971; Thompson et al., 1990); (iii) increased intramuscular concentrations of ATP, PCr, and inorganic phosphate, allowing for more rapid restoration of these molecules during aerobic and anaerobic exercise (Chasiotis, 1988; Stryer, 1988); (iv) increased cardiac

muscle contractility (Czuba et al., 2008, 2009; Kreider et al., 1992); and (v) increased buffering capacity of hydrogen ions (Avioli, 1988).

To date, only a few studies have investigated the effect of SP on actual exercise performance, with these typically limited to tasks of prolonged (> 20 min) duration, including a 5-mile run (Kreider et al., 1990), 16.1 km (Folland et al., 2008) and 1000 kJ (238.7 Kcal)/40 km cycling time trials (Brewer et al., 2013; Kreider et al., 1992). Currently, no studies have investigated the effect of SP on exercise tasks of shorter duration, despite the potential for enhanced performance in these types of activities. Based on the proposed mechanisms of SP loading, such as increased H⁺ buffering, increased anaerobic glycolysis, PCr resynthesis and ATP production, athletes may benefit from exercise metabolism during shorter duration, higher intensity exercise. In support of the above mechanisms, we recently reported significantly higher power outputs during the earlier stages of a 1000 kJ (238.7 Kcal) cycling time trial (250 kJ/ 59.7 Kcal and 500 kJ/ 119.3 Kcal time points) following SP supplementation (Brewer et al., 2013). This raises the question of whether SP loading may benefit exercise performance of < 15 min in duration.

Another aspect of SP supplementation that remains unclear is the duration of any ergogenic effect. Studies have typically employed a supplementation protocol involving ingestion of 3-4 g·day⁻¹, split into 4 equal doses, for a period of 3 to 6 days, with an exercise test completed on the following day (Cade et al., 1984; Folland et al., 2008; Kreider et al., 1990; 1992; Wallace et al., 1997). However, some research suggests that the effects of SP loading may persist for some time after ceasing supplementation. For example, Cade et al. (1984) reported a lingering increase in 2, 3 DPG levels up to two weeks post-supplementation. We also recently reported an additional increase in $\text{VO}_{2\text{peak}}$, compared with both baseline and the first phase of supplementation, following a second phase of supplementation with SP. This second phase occurred either 15 or 35 days after the initial loading phase, indicating that the ergogenic effects may be maintained for an extended time and potentially additive upon subsequent supplementation (Brewer et al., 2013). Czuba et al. (2009) also found that when SP supplementation (after initial loading) was continued at lower doses for a further 3 weeks, $\text{VO}_{2\text{max}}$ was maintained at higher than pre-loading levels. Whether SP supplementation can affect exercise performance for up to a week or more post-loading remains to be examined. Therefore, the purpose of this study was to investigate the effects of 6 days of SP

supplementation on short duration (~ 3–4 min and 10–12 min) cycling time trial performance both 1 and 8 days post-loading.

Methods

Participants

Twenty-one competitive male cyclists volunteered for this study, although two failed to complete all testing phases due to unrelated injury. Participants were randomised into two groups, with one group completing a 100 kJ (23.9 Kcal) time trial protocol ($n = 10$, mean \pm SD: age 30.2 ± 9.2 y; body mass 81.9 ± 10.2 kg; height 1.84 ± 0.08 m; body fat 14.2 ± 5.2 %; $\text{VO}_{2\text{peak}}$ 5.29 ± 0.39 L \cdot min $^{-1}$; years of competitive cycling 4.9 ± 2.2 y) and the other a 250 kJ (59.7 Kcal) time trial protocol ($n = 9$, mean \pm SD: age 27.8 ± 8.8 y; body mass 79.6 ± 12.4 kg; height 1.82 ± 0.09 m; body fat 10.7 ± 4.4 %; $\text{VO}_{2\text{peak}}$ 5.38 ± 0.63 L \cdot min $^{-1}$; years of competitive cycling 8.2 ± 5.9 y). The Human Research Ethics Committee of the University of Western Australia (UWA) approved the study and all participants provided written informed consent. None had taken any other nutritional supplements for at least 2 months prior to (or during) this study. The study was conducted during the Western Australian competitive cycling season; each participant was required to maintain a consistent training volume over the study duration. This comprised (mean \pm SD) 271 ± 90 km \cdot wk $^{-1}$ (100 kJ/ 23.9 Kcal group) and 289 ± 188 km \cdot wk $^{-1}$ (250 kJ/ 59.7 Kcal group) of road cycling, with an additional 8.6 ± 5.4 and 12.2 ± 4.1 h per week of cross training in other physical activities, for each group respectively.

Experimental design

Each participant completed an initial familiarisation session, followed 48 h later by a baseline assessment of $\text{VO}_{2\text{peak}}$. Next, participants completed two separate phases of testing in a counterbalanced, double-blind, crossover design over a 3-month period. Each phase involved 6 days of supplementation with SP or placebo (PL), with either a 100 kJ/ 23.9 Kcal or 250 kJ/ 59.7 Kcal cycling time trial completed firstly on day 7 (i.e. the day after cessation of supplementation) and then repeated 7 days later (i.e. 7 days after finishing supplementation). A 28-day washout period, beginning after the last day of supplementation, was completed before the next phase of supplementation and testing commenced.

Familiarisation session

Here, medical and training questionnaires were completed, height and body mass were recorded, and body composition (percentage body fat and fat free mass) was assessed using a dual energy X-ray absorptiometry scan (DEXA; Lunar Prodigy, encore 2004, GE Medical Systems, Madison, Wis., USA). Participants then completed the initial stages of a graded exercise test (first three incremental workloads while breathing through a mouthpiece), and following 5 min of recovery, completed either a (100 kJ/ 23.9 Kcal or 250 kJ/ 59.7 Kcal) cycling time trial (depending on their randomisation) while breathing through the same mouthpiece, in order to become accustomed to the tests and procedures to be used during the

subsequent experimental trials. All exercise testing was performed on an air-braked cycle ergometer (Evolution bicycles, Geelong, Australia), linked to a customised computer program for the determination of power output and total mechanical work (Cyclemax version 6.3, School of Sport Science, Exercise and Health, University of Western Australia). This ergometer has fan blades attached to a flywheel, which displaces air as the wheel turns, making resistance proportional to pedalling rate. In addition, the cycling ergometer had six gears which participants were able to adjust, allowing for individual resistance and cadence to be set independently for each performance test undertaken. This setup was intended to maximize ecological validity by replicating the variation in pedal rate and gearing experienced during an actual road cycling time trial.

Assessment of $\text{VO}_{2\text{peak}}$

Peak aerobic capacity was assessed 48 h after the introductory session using an incremental cycling exercise test, starting at 150 W and increasing by 50 W every 3 min, with 1 min of rest between stages to allow for capillary blood sampling for lactate determination (35 μ l; ABL 725, Radiometer, Copenhagen, Denmark). The test was continued until volitional exhaustion or when the participant could no longer maintain the required power output. Heart rate was recorded 30 s prior to the end of each 3 min exercise interval. During exercise, participants breathed through a mouthpiece connected to a computerised gas analysis system, incorporating a ventilometer (Universal ventilation meter, VacuMed, Ventura, California, USA), calibrated before the test using a 1-litre syringe according to manufacturer's instructions. Expired oxygen and carbon dioxide concentrations were analysed using Ametek gas analysers (Applied Electrochemistry, SOV S-3A11 and COV CD-3A, Pittsburgh, PA, USA), which were calibrated immediately before and verified after each test using a certified gas mixture of known concentrations (BOC Gases, Chatswood, Australia). Both the ventilometer and gas analysers were connected to a computer that measured and displayed variables (VO_2 and V_E) every 15 s, which were measured throughout the test. Individual $\text{VO}_{2\text{peak}}$ values were calculated using the sum of the four highest consecutive 15 s VO_2 values recorded during the test.

Supplementation protocol

Participants were given either tribasic sodium phosphate dodecahydrate (Challenge Chemicals Australia, Kwinana, Western Australia; 50 mg \cdot kg \cdot FFM $^{-1}\cdot$ day $^{-1}$) or a placebo (PL) mix of glucose and table salt (ratio 9/1). The PL included salt in order to mask the taste of the supplement ingested, as SP has a slightly salty taste. Each supplement was ground into a fine powder to make them indistinguishable from each other. This daily amount was divided into four equal doses and ingested with meals, to ensure optimal absorption, over the course of each day (4–5 h interval) in opaque capsules (Melbourne Food Depot, East Brunswick, Victoria Australia) for a six-day period until 8 h prior to the scheduled exercise test. Each capsule dose was emptied into a glass and consumed with 15 g of Powerade® powder (Coca-Cola Amatil, Australia) that

had been dissolved with ~ 300 ml of water. This procedure was followed in order to prevent gastrointestinal upset (experienced in prior pilot testing with SP and reported by West et al., 2012) and to mask the taste.

100 kJ (23.9 Kcal) and 250 kJ (59.7 Kcal) time trials protocol

Participants refrained from exercise for 24 h prior to each time trial, with exercise testing taking place at the same time of day (± 1 h) to control for circadian variation. They were also asked to record all food and drink intake, including the type of food, amount consumed and timing of consumption, in a diary for the 24 h prior to each time trial. Copies of this information from the first trial were provided to each participant prior to each subsequent trial, with the requirement for them to replicate this energy intake as closely as possible. Compliance was confirmed upon arrival to the laboratory for each time trial after inspection of the food diaries by the investigator.

As previous research has used a 500 kJ cycling time trial to approximate a distance of 20 km (Peeling et al., 2005), target workloads of 100 kJ (23.9 Kcal) and 250 kJ (59.7 Kcal) were chosen to represent ~ 4 km and 10 km, respectively. When performing each time trial, participants cycled in an isolated room with no external influences (i.e. music or other distractions). A computer screen allowed participants to see the amount of kJ left to complete (which counted down from 100 kJ/ 23.9 Kcal or 250 kJ/ 59.7 Kcal), but kept them blinded to their power output and duration of performance. During exercise, participants breathed continuously through a mouthpiece connected to a computerised gas analysis system (as described previously) for VO_2 measurement.

Blood samples (35 μl) were collected from the earlobe at rest and immediately post-exercise for measurement of lactate using a blood gas analyser (ABL 725, Radiometer, Copenhagen, Denmark). Heart rate (Polar Electro Oy/Professorintie, Kempele, Finland) and rating of perceived exertion (RPE; Borg, 1982) were recorded immediately after each time trial.

Determination of serum phosphate

Prior to the familiarisation time trial and following each 6-day supplementation phase, a venous blood sample was taken from an antecubital vein (BD Vacutainer SST II

Advance) to measure serum phosphate levels. Samples were left to clot at room temperature for 60 min prior to being centrifuged at 1000 g at 4°C for 15 min. The serum obtained was stored at -80°C for later analysis, with serum phosphate determined using an Abbott Architect c16000 analyser, using the specified Abbott reagents (Abbott Laboratories, Abbott Park, IL 60065, USA). Observed coefficients of variation were 4.2% at a level of 0.95 $\text{mmol}\cdot\text{l}^{-1}$ and 2.0% at a level of 2.95 $\text{mmol}\cdot\text{l}^{-1}$.

Statistical analysis

The post-supplementation trials were designated SP1 and PL1 (day 1 post supplementation) and SP8 and PL8 (day 8 post supplementation). The effect of supplementation on time trial performance and associated physiological and perceptual variables (HR, lactate, VO_2 , V_E and RPE) was analysed using 2-way, repeated-measures ANOVA (SPSS 18.0 for Windows). Significance was accepted when $p \leq 0.05$. Cohen's d effect sizes (ES < 0.5, small; 0.5 - 0.79, moderate; ≥ 0.8 , strong) were also calculated to examine performance trends (Cohen, 1988). Further analysis identified the smallest worthwhile change (SWC) in performance/physiological measures between SP and PL time trials using the method outlined by Batterham and Hopkins (2005). The SWC was set at a Cohen's unit of 0.2, representing the hypothetical, smallest change in measures that would benefit the athlete. Where the chances of benefit or harm were both calculated to be > 5%, the true effect was deemed unclear. When clear interpretation was definitively possible, a qualitative descriptor was assigned to the following quantitative chances of benefit: 25-75%, benefit possible; 76-95%, benefit likely; 96-99%, benefit very likely; > 99%, benefit almost certain (Batterham and Hopkins, 2005).

Results

100 kJ (23.9 Kcal) time trial performance

There were no significant differences ($p > 0.05$), moderate or large effect sizes or likely (or better) SWC values found for time to completion or mean power output within and between SP and PL trials (Table 1). There was also no difference in heart rate, RPE and blood lactate values within or between any trials. Oxygen uptake was also similar between trials, although first minute VO_2

Table 1. Mean (\pm SD) 100 kJ (23.9 Kcal) cycling time trial results for supplementation with sodium phosphate (SP) or placebo (PL) 1 and 8 days post-loading (n = 10).

100 kJ (23.9 Kcal)	SP Day 1	SP Day 8	PL Day 1	PL Day 8
Time to Completion (s)	214.5 (34.6)	211.9 (33.3)	216.8 (35.9)	209.2 (38.1)
Average Power (W)	479 (85)	482 (75)	473 (79)	492 (87)
Heart Rate post (bpm)	178 (10)	177 (7)	175 (7)	175 (8)
Rating of Perceived Exertion	18 (2)	18 (2)	18 (2)	18 (2)
Blood Lactate Post ($\text{mmol}\cdot\text{L}^{-1}$)	9.1 (1.2)	9.3 (2.8)	9.0 (2.0)	9.4 (2.4)
Peak VO_2 (Oxygen uptake; $\text{L}\cdot\text{min}^{-1}$)	5.08 (.39)	5.21 (.37)	5.03 (.35)	5.12 (.31)
1st min VO_2 ($\text{L}\cdot\text{min}^{-1}$)	2.95 (.37)	3.14 (.43)	2.88 (.33)	2.86 (.50)
	^a ($d = .47$; 82/15/3 SWC)			^a ($d = 0.60$; 88/10/2 SWC)
Ave VO_2 ($\text{L}\cdot\text{min}^{-1}$)	3.75 (.22)	3.94 (.23)	3.74 (.18)	3.77 (.21)
	^a ($d = .84$; 90/8/3 SWC)			^a ($d = 0.77$; 93/6/1 SWC)
1st min V_E (Ventilation expired; L)	82.5 (21.8)	82.5 (13.8)	80.3 (15.7)	76.3 (18.9)
				^a ($d = 0.37$; 80/18/2 SWC)
Ave V_E (L)	123.6 (15.6)	124.3 (10.4)	121.0 (10.7)	122.3 (15.8)

^a = compared with SP8; d = effect size, SWC = smallest worthwhile change probability.

Table 2. Mean (\pm SD) 250 kJ (59.7 Kcal) cycling time trial results for supplementation with sodium phosphate (SP) or placebo (PL) 1 and 8 days post-loading ($n = 9$).

250 kJ (59.7 Kcal)	SP Day 1	SP Day 8	PL Day 1	PL Day 8
Time to Completion (s)	674.9 (112.7)	662.0 (104.2)	673.7 (95.1)	674.4 (117.8)
Average Power (W)	379 (57)	386 (58)	377 (47)	380 (63)
Heart Rate post (bpm)	180 (16)	179 (14)	180 (14)	180 (13)
Rating of Perceived Exertion	18 (1)	18 (1)	18 (1)	19 (1)
Blood Lactate Post ($\text{mmol}\cdot\text{L}^{-1}$)	9.2 (4.2)	9.1 (4.4)	9.2 (3.1)	8.3 (2.8)
Peak VO_2 (Oxygen uptake; $\text{L}\cdot\text{min}^{-1}$)	5.18 (.82)	5.06 (.85)	5.21 (.66)	5.14 (.78)
1st min VO_2 ($\text{L}\cdot\text{min}^{-1}$)	2.54 (.54)	2.54 (.51)	2.53 (.41)	2.42 (.40)
Ave VO_2 ($\text{L}\cdot\text{min}^{-1}$)	4.37 (.63)	4.45 (.60)	4.46 (.51)	4.41 (.60)
1st min V_E (Ventilation expired; L)	68.9 (22.3)	67.7 (18.7)	67.8 (21.9)	68.1 (17.5)
Ave V_E (L)	125.0 (35.3)	128.1 (27.3)	125.5 (25.0)	128.8 (27.1)

Note: No significant differences, moderate or larger effect sizes and likely or better smallest worthwhile effects were found.

tended to be higher in SP8 compared with both SP1 ($p = 0.150$) and PL8 ($p = 0.105$), as was mean VO_2 (SP1: $p = 0.103$ and PL8: $p = 0.052$). Furthermore, first minute V_E was higher for SP8 compared with PL8 ($p = 0.140$).

Of note, the $\text{VO}_{2\text{peak}}$ achieved during the time trials were 96.0 % (SP1), 98.5 % (SP8), 95.1 % (PL1) and 96.8 % (PL8) compared with the $\text{VO}_{2\text{peak}}$ achieved during the incremental exercise test.

250 kJ (59.7 Kcal) time trial performance

There were no significant differences ($p > 0.05$), moderate or large effect sizes or likely (or better) SWC values for time to completion or mean power output within and between SP and PL trials (Table 2), despite a faster (~ 13 s) mean completion time and a slightly higher ($\sim 2\%$) average power output in SP8. Heart rate, RPE, blood lactate and VO_2 responses to exercise were also similar between trials ($p > 0.05$). The $\text{VO}_{2\text{peak}}$ achieved during the time trials were 96 % (SP1; mean time = 674.9 s; mean power = 379 W), 94 % (SP8; mean time = 662.0 s; mean power = 386 W), 97 % (PL1; mean time = 673.7 s; mean power = 377 W) and 96 % (PL8; mean time = 674.4 s; mean power = 380 W) compared with the $\text{VO}_{2\text{peak}}$ achieved during the incremental exercise test.

Serum phosphate

There were no differences ($p > 0.05$) in serum phosphate levels recorded with supplementation for either the 100 kJ (23.9 Kcal) (mean \pm SD: baseline 1.27 ± 0.18 ; SP1 1.27 ± 0.17 ; SP8 1.29 ± 0.12 ; PL1 1.25 ± 0.12 ; PL8 1.29 ± 0.18 $\text{mmol}\cdot\text{l}^{-1}$) or 250 kJ (59.7 Kcal) trials (mean \pm SD: baseline 1.23 ± 0.17 ; SP1 1.24 ± 0.13 ; SP8 1.14 ± 0.08 ; PL1 1.26 ± 0.15 ; PL8 1.24 ± 0.18 $\text{mmol}\cdot\text{l}^{-1}$).

Discussion

This is the first study to assess the effects of SP supplementation on shorter duration, higher intensity (100 kJ/ 23.9 Kcal, ~ 3 -4 min; and 250 kJ/ 59.7 Kcal, ~ 10 -12 min) exercise performance. In addition, it is the first study to examine exercise performance both 1 and 8 days post supplementation, to determine whether any effect of SP loading persists across this time frame. Results showed that SP supplementation had no significant effect on either 100 kJ/ 23.9 Kcal or 250 kJ/ 59.7 Kcal cycling time trial performance on either day 1 or 8 after loading. In addition, no effect on heart rate, RPE and blood lactate response to exercise was noted. Further, oxygen con-

sumption was also similar between trials and days, although moderate effect sizes and SWC values indicated that first minute and mean VO_2 during the 100 kJ/ 23.9 Kcal time trial tended to be higher in SP8 compared with both SP1 and PL8, with this reflected, in part, by higher first minute mean V_E .

Results from some previous studies have shown benefits to endurance performance following supplementation with similar amounts of SP as used here. Specifically, Kreider et al. (1992) and Folland et al. (2008) have reported significantly faster 40 km (by $\sim 8\%$, 3.5 min) and 16.1 km ($\sim 3\%$, 30-40 s) cycling time trial performances respectively. We also recently found a (NS) decreased time to completion of ~ 60 -70 s ($\sim 2\%$ faster) over a 1000 kJ/ 238.7 Kcal (~ 40 km) cycling time trial (Brewer et al., 2013). In the present study, 250 kJ/ 59.7 Kcal time trial performance was also ~ 13 s ($\sim 2\%$) faster in SP8, but this result was also NS and not associated with any moderate (or better) effect sizes or likely (or better) smallest worthwhile (beneficial) change values.

Collectively, these results suggest that the exercise duration is an important factor with regard to potential ergogenic benefits of SP supplementation. Here, the time trials used took ~ 3 -4 min (100 kJ/ 23.9 Kcal) and ~ 10 -12 min (250 kJ/ 59.7 Kcal) to complete, compared with ~ 26 to ~ 55 min in studies demonstrating an ergogenic effect (Folland et al., 2008; Kreider et al., 1992). Specifically, the shorter duration and higher intensity endurance exercise protocols used here may not have allowed the potential aerobic benefits associated with SP loading (principally, greater unloading of oxygen due to increased 2, 3 DPG levels, enhanced myocardial efficiency and greater availability of phosphate for oxidative phosphorylation) to have any pronounced effect (Chasiotis, 1983; Czuba et al., 2008; Kreider et al., 1990; 1992). Possibly, the potential aerobic exercise benefits from SP supplementation (1-8 days later) may have a greater ergogenic role in longer duration (~ 25 min +), lower intensity distance efforts, where the percentage total aerobic energy contribution is greater, rather than shorter (< 15 min), more intense, exercise tasks. The potential improvements in buffering capacity from SP supplementation may also have limited effects unless there are major disruptions to normal acid-base homeostasis. Here, although pH values were not taken, the indirect evidence of lactate accumulation being only ~ 9 to 10 $\text{mmol}\cdot\text{L}^{-1}$ after the time trials may suggest that only a moderate challenge to acid-base balance was presented by the exercise challenges.

In relation to the potential mechanisms of ergogenic effect with SP supplementation, this study found no difference in serum phosphate levels post-loading between trials. Notably, a review by Tremblay et al. (1994) emphasised the need for a measurable change in serum phosphate post-loading as evidence for an intervention effect to have occurred. While this has been reported in some studies (Czuba et al., 2009; Kreider et al., 1992), others have found improved aerobic capacity and exercise performance with no change between pre and post-loading resting serum phosphate levels (Brewer et al., 2013; Stewart et al., 1990), suggesting that this measure may be inconsequential for any physiological and/or performance changes. The measurement of 2, 3 DPG levels before and after SP loading (unfortunately unavailable for this study) may be more compelling from a mechanistic standpoint than serum phosphate values, which have a wide range of pre loading levels (suggesting large individual variation) reported in the literature (~ 0.8 to 1.4 mmol·L⁻¹; Czuba et al., 2008; Kreider et al., 1990; 1992).

Interestingly, although no effect of SP supplementation on exercise performance was noted in the present study, first minute and mean VO₂ in the 100 kJ (23.9 Kcal) time trial tended to be higher in SP8 (3.14 L·min⁻¹; 3.94 L·min⁻¹ respectively) compared with both SP1 (2.95 L·min⁻¹; 3.75 L·min⁻¹) and PL8 (2.86 L·min⁻¹; 3.77 L·min⁻¹) (based on moderate effect sizes and SWC values). Few SP loading studies have measured VO₂ during the actual performance test, but Kreider et al. (1992) reported an increased mean VO₂ (from 80 to 86% VO₂ max; from 4.13 to 4.82 L·min⁻¹) and ventilation (125.1 to 144.1 L·min⁻¹) throughout a 40 km cycling time-trial and time to anaerobic threshold (14.86 to 16.33 min) during an incremental exercise test. A 17% (220 to 257 W) greater mean power output was also recorded, in conjunction with a significantly faster time trial performance (45.75 to 42.25 min). Folland et al. (2008) have also reported a tendency (p = 0.07) for an increase in VO_{2peak} after SP supplementation during a 16.1 km time trial, in which mean power output was also ~ 10% greater and completion time ~ 3% faster.

Similarly, in our study, mean VO₂ for SP8 was ~76 %VO_{2peak} compared with ~74 %VO_{2peak} in SP1 and PL8 in the 100 kJ/ 23.9 Kcal time trial. However, no other physiological or performance changes were seen in SP8, such that the increased VO₂ cannot be considered as beneficial, particularly as no greater mean power output was evident. Most SP loading studies have measured VO₂ max in a separate incremental (rather than performance) test, with improvements reported both immediately after supplementation (Kreider et al., 1992) and over the next three weeks with continued lower doses (Czuba et al., 2009). Similarly, Brewer et al. (2013) found an increased VO_{2peak} with SP loading, initially by ~ 4%, which then increased to ~8 % after a second loading phase either 15 or 35 days later.

Our rationale for measuring VO₂ during the time trials was to obtain a simple indication of whether oxygen kinetics were altered by SP loading. Theoretically, SP supplementation could potentially facilitate faster oxygen kinetics, via the lowering of oxygen affinity caused by

increased 2, 3 DPG levels (Cade et al., 1984; Chanutin and Curnish, 1967; Kreider et al., 1992). Although the 100 kJ/ 23.9 Kcal time-trial here showed a greater first minute VO₂ in SP8 compared with SP1 (likely SWC) and PL8 (medium ES and likely SWC), this was not similarly matched by a higher mean power output or faster time to completion, therefore no particular focus should be placed on this result. Lack of association between changes in VO₂ found in the current study and improved exercise performance may be due to the relatively greater anaerobic energy contribution required in the short-term, higher-intensity 100 kJ/ 23.9 Kcal effort used here in comparison to previous studies using longer duration exercise tasks (Folland et al., 2008; Kreider et al., 1992). Nevertheless, further research into SP supplementation should continue to explore any potential effects on oxygen kinetics, as well as aerobic capacity.

Conclusion

In conclusion, six days of SP supplementation did not affect shorter duration (<15 min) cycling time trial performance, either 1 or 8 days after loading. While oxygen kinetics (first minute and average VO₂) were slightly greater on day 8 in the 100 kJ (23.9 Kcal) time trial, the absence of any effect on mean power output or completion time suggests that no particular physiological significance should be ascribed to this result. Future studies should investigate the effect of SP loading on repeated sprints and simulated road race performance over extended durations (>30 min), where SP may be likely to have a more beneficial effect (Brewer et al., 2013; Folland et al., 2008; Kreider et al., 1992).

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Key points

- Studies investigating the effects of sodium phosphate loading on shorter duration (<15 min) and higher intensity exercise performance are lacking, as is research on how long any ergogenic effect may last.
- Loading did not improve cycling time trial (~3-4 min and 10-12 min) performance either 1 or 8 days after supplementation.
- Future studies should investigate the effect of sodium phosphate loading on repeated sprints and simulated cycling road race performance over extended durations (>30 min), where it may be likely to have a more beneficial effect.

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