Journal of Sports Sciences
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/rjsp20

Effect of sodium phosphate supplementation on repeated high-intensity cycling efforts

C. P. Brewer[a], B. Dawson[a], K. E. Wallman[a] & K. J. Guelfi[a]
[a] School of Sport Science, Exercise and Health, The University of Western Australia, Perth, Western Australia, Australia
Published online: 10 Dec 2014.

Journal of Sports Sciences, 2015
Vol. 33, No. 11, 1109–1116, http://dx.doi.org/10.1080/02640414.2014.989536

Routledge
Taylor & Francis Group

Effect of sodium phosphate supplementation on repeated high-intensity cycling efforts

C. P. BREWER, B. DAWSON, K. E. WALLMAN & K. J. GUELFI

School of Sport Science, Exercise and Health, The University of Western Australia, Perth, Western Australia, Australia

(*Accepted 14 November 2014*)

Abstract
Limited research has investigated how sodium phosphate supplementation affects exercise performance typical of athletic competition and whether any effects linger in the short term. This study examined the effect of sodium phosphate supplementation on a cycling protocol consisting of repeated-sprint (4 sets of 6 × 15 s) and time-trial (2 × 5 min) efforts on day 1 and 4 post-loading. Trained male cyclists ($\dot{V}O_{2peak}$ 5.3 L · min^{-1}) were randomised to 6 days of sodium phosphate supplementation (50 mg · kg·fat-free-mass^{-1} · day^{-1}; n = 7) or placebo (n = 10). Performance was assessed at baseline and 1 and 4 days post-supplementation on an air-braked cycle ergometer. Compared with baseline, the sodium phosphate group recorded significantly improved ($P < 0.05$) work and mean power output values in both the sprint (baseline, 259 kJ/719 W; day 1, 271 kJ/754 W; day 4, 271 kJ/753 W) and time-trial (baseline, 225 kJ/374 W; day 1, 235 kJ/398 W; day 4, 236 kJ/393 W) aspects of the performance test post-loading. In the placebo group, no differences ($P > 0.05$) in total work or power output were noted in response to supplementation. In summary, sodium phosphate supplementation improved repeated-sprint and time-trial cycling efforts both 1 and 4 days post-loading in trained cyclists.

Keywords: *time-trial, repeated-sprints, cycle ergometry*

Introduction

Sodium phosphate is an ergogenic aid that has been reported to improve maximal aerobic capacity (by 3.5–12%) (Brewer, Dawson, Wallman, & Guelfi, 2013; Cade et al., 1984; Czuba, Zajac, Poprzecki, & Cholewa, 2008; Czuba, Zajac, Poprzecki, Cholewa, & Woska, 2009; Kreider et al., 1992; Kreider, Miller, Williams, Somma, & Nasser, 1990; Stewart, McNaughton, Davies, & Tristram, 1990), but associated benefit to endurance performance (i.e. ~21–56 min cycling and running time-trial efforts) has been equivocal (Brewer et al., 2013; Folland, Stern, & Brickley, 2008; Kreider et al., 1990, 1992). Notably, only one study has assessed the effect of sodium phosphate loading on sprint performance, with no benefit found for peak or mean power output during a 30 s Wingate test in trained male cyclists (Tourville, Brennan, & Connolly, 2001).

The assumption underlying sodium phosphate supplementation is that increased phosphate availability may enhance cellular metabolism. Specifically, phosphate is a basic structural component of phosphocreatine, ATP and rate limiting enzymes such as phosphofructokinase (Eaton, Brewer, & Grover, 1969; Rose, 1970). In red blood cells, phosphate is also a key component of 2,3-diphosphoglycerate (2,3-DPG; Cade et al., 1984; Chanutin & Curnish, 1967), which plays a vital role in oxygen delivery to the working muscles. Importantly, all of these phosphate-related metabolites and enzymes directly affect the metabolic processes involved in ATP production by the phosphagen, glycolytic and oxygen energy systems (Brain & Card, 1972; Chanutin & Curnish, 1967; Lichtman & Miller, 1970). Accordingly, sodium phosphate supplementation has been proposed to facilitate energy metabolism during exercise (Chasiotis, 1983; Lichtman, Miller, Cohen, & Waterhouse, 1971; Thompson et al., 1990), with further potential benefits to exercise performance via additional mechanisms such as increased hydrogen ion buffering in plasma and cells (Avioli, 1988) and enhanced cardiac muscle contractility (Czuba et al., 2008, 2009; Kreider et al., 1992).

Studies supplementing with sodium phosphate have typically used 3–4 g · day^{-1}, split into four equal doses, for 3–6 days, with exercise tests conducted on the following day (Brewer et al., 2013; Cade et al., 1984; Folland et al., 2008; Kreider et al., 1992). However, some research suggests that

Correspondence: Cameron P. Brewer, School of Sport Science, Exercise and Health, M408, The University of Western Australia, 35 Stirling Highway, Crawley, WA 6009, Australia. E-mail: cameronbrewer1@gmail.com

sodium phosphate loading effects may persist for some time after supplementation ends. Cade and colleagues (1984) reported that 2,3-DPG concentrations remained elevated for up to 2 weeks after supplementation with sodium phosphate. However, whether or not any performance benefit is maintained over part or all of this period has yet to be investigated.

Currently, there is a lack of research investigating how sodium phosphate supplementation affects actual or simulated exercise performance typical of athletic competition. In cycling road races, repeated maximal sprint and short time-trial efforts are regularly required across a continuous effort lasting for >1 h. Therefore, this study aimed to assess the effect of 6 days of sodium phosphate supplementation on cycling performance (incorporating repeated-sprints and time-trial efforts) both 1 and 4 days post-supplementation. Repeating the test protocol on day 4 following supplementation was included to determine if there was any persisting (short-term) ergogenic effect of sodium phosphate supplementation.

Methods

Participants

Twenty-one competitive male cyclists volunteered for this study: 4 failed to complete all testing phases due to unrelated illness and injury, leaving a final sample of n = 17. After familiarisation, participants were randomised into sodium phosphate and placebo groups (Table I). The study was conducted during the Western Australian competitive cycling season, with each participant required to maintain a consistent training volume over the study duration. This consisted of mean = 367 km · wk^{-1}, s = 144 km · wk^{-1} (sodium phosphate) and mean = 310 km · wk^{-1}, s = 105 km · wk^{-1} (placebo) of road cycling, with an additional mean = 6.3 h · wk^{-1}, s = 6.7 h · wk^{-1} and mean = 3.6 h · wk^{-1}, s = 6.6 h · wk^{-1} of cross-training in other physical activities, respectively (no difference between groups; $P > 0.05$). Participants were not taking any nutritional supplements for at least 2 months before or during the study. The Institutional Human Research Ethics Committee approved the study and all participants provided written informed consent.

Table I. Participant characteristics.

	Placebo (n = 10)	Sodium phosphate (n = 7)
Age (y)	35.6 ± 11.1	33.3 ± 3.3
Height (cm)	176.9 ± 8	178.2 ± 4.8
Body mass (kg)	74.5 ± 8.7	74.5 ± 5.3
Body fat (%)	13.0 ± 5.8	10.8 ± 3.3
Fat free mass (kg)	65.3 ± 8.6	67.4 ± 5.7
$\dot{V}O_{2peak}$ (L · min^{-1})	5.1 ± 1.2	5.6 ± 1.0
Years of competitive cycling	4.5 ± 3.5	6.6 ± 3.8

Note: No significant differences were recorded between groups.

Experimental design

Participants first attended the laboratory for familiarisation and assessment of $\dot{V}O_{2peak}$. Next, baseline cycling performance was measured (i.e. without supplementation), followed by 6 days of supplementation with either the sodium phosphate or placebo (double-blind design), with the exercise test repeated on day 1 and day 4 after ceasing supplementation.

Familiarisation session and assessment of $\dot{V}O_{2peak}$

Medical and training questionnaires were first completed, then height and body mass were recorded and body composition (fat free mass) was assessed using dual energy X-ray absorptiometry (Lunar Prodigy, encore 2004, GE Medical Systems, Madison, Wis., USA). Participants then completed the initial stages of a graded exercise test (first 3 incremental workloads while breathing through a mouthpiece) and after 5 min of recovery, completed the full cycling race simulation (detailed below), 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 (Cyclemax version 6.3, School of Sport Science, Exercise and Health, UWA, Perth, Australia). This ergometer has 6 gear adjustments, allowing for individual resistance and cadence to be set, and the ergometer flywheel has fan blades attached, displacing air as the wheel turns, making resistance proportional to pedalling rate. This set-up reflects the type of resistance normally encountered during road cycling and replicated the variation in pedal rate and gearing commonly experienced during races. All exercise testing was performed in a controlled laboratory environment (~23°C and 47% RH).

After 48 h, participants returned to the laboratory for an assessment of $\dot{V}O_{2peak}$ using the graded exercise protocol previously described by Brewer and colleagues (2013).

Supplementation protocol

Participants were given either tribasic sodium phosphate dodecahydrate (Challenge Chemicals Australia,

Kwinana, Western Australia; 50 mg · kg·fat-free-mass^{-1} · day^{-1}) or a placebo (mix of glucose and table salt: ratio 9/1). The placebo included salt to mask the taste of the supplement, as sodium phosphate has a slightly salty taste. Each supplement was ground into a fine powder to make them indistinguishable from each other. The daily amount was divided into four equal doses and ingested with meals over the course of each day (4–5 h interval) in opaque capsules (Melbourne Food Depot, East Brunswick, Victoria Australia) for a 6-day period until 8 h before the scheduled exercise test as per previous research investigating the ergogenic effect of sodium phosphate supplementation (Brewer et al., 2013; Czuba et al., 2008, 2009). 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 mask the taste and prevent gastrointestinal upset (experienced in pilot testing and reported by West, Ayton, Wallman, & Guelfi, 2012).

Cycling race simulation protocol

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

The test protocol comprised a combination of 4 sets of 6 × 15 s maximal sprints (with varying recovery durations) and 2 sets of 5-min maximal time-trials (Figure 1). All sets were separated by 3 min of active recovery, where participants were required to maintain 100 W of power output. The first and fourth sets consisted of 6 × 15 s maximal sprints, separated by 45 s of active recovery. The second and fifth sets consisted of 6 × 15 s maximal sprints separated by 15 s of active recovery. The third and sixth sets consisted of the 5-min maximal time-trials. Total trial completion time was 43 min. This protocol was modified from the cycling race simulation used by Vaile, Halson, Gill, and Dawson (2008). When performing the trial, participants cycled in an isolated room with no outside influences. A computer screen allowed them to see the amount of time left to complete each effort (which counted down and reset continuously throughout each test) but kept them blinded to their power output. Total work and mean power output during the whole test, as well as for the specific repeated-sprints and time-trial sets, were determined. In addition, heart rate (Polar Electro Oy Professorintie, Kempele, Finland), blood lactate (35 μL; ABL 725, Radiometer, Copenhagen, Denmark) and ratings of perceived exertion (RPE; Borg, 1982) were measured immediately after each set of the test.

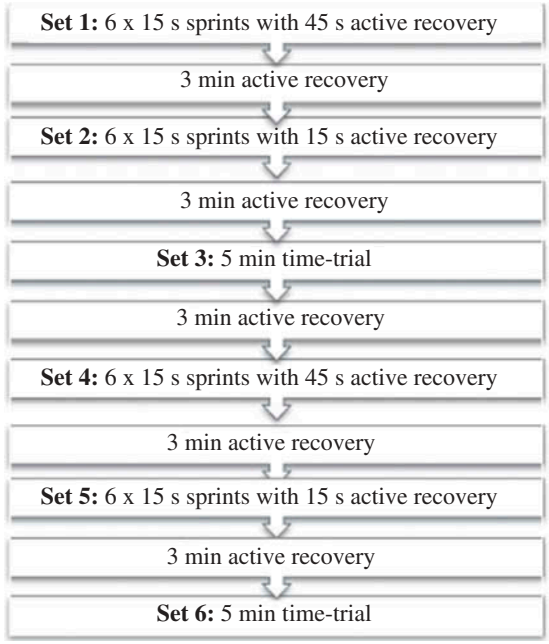

Figure 1. Cycling race simulation protocol.

Determination of serum phosphate

Before each trial, a venous blood sample was taken from an antecubital vein (BD Vacutainer SST II Advance) to determine serum phosphate concentration. Samples were left to clot at room temperature for 60 min before 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, employing the specified Abbott reagents (Abbott Laboratories, Abbott Park, IL 60065, USA). Observed coefficients of variation were 4.2% at a level of 0.95 mmol · L^{-1} and 2.0% at a level of 2.95 mmol · L^{-1}.

Statistical analysis

Independent samples *t*-tests were used to test for physical and physiological differences between groups at baseline. Within groups (on statistical advice, as the sodium phosphate group finished with n = 7), the effect of supplementation on

performance and physiological variables were analysed using one-way repeated-measures (day; baseline, day 1 and day 4 post-supplementation) ANOVA or 2-way repeated measures ANOVA (day × set) as appropriate (SPSS 18.0 for Windows). Where indicated, Fisher's LSD was used for post hoc analyses, with significance accepted when $P \leq 0.05$. Cohen's d effect sizes (Effect size < 0.5, small; 0.5–0.79, moderate; ≥0.8, strong) were also calculated to examine data trends (Cohen, 1988). Only moderate or strong effect sizes are reported here for ease of interpretation. Further analysis identified the smallest worthwhile change in performance scores between sodium phosphate and placebo trials using the method of Batterham and Hopkins (2005). The smallest worthwhile change was set at a Cohen's unit of 0.2, representing the hypothetical, smallest change in performance measures that would benefit the athlete. Where 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 & Hopkins, 2005). Outcomes for smallest worthwhile change are presented as beneficial/trivial/harmful.

Results

At baseline, no significant differences existed between the sodium phosphate and placebo groups for age, height, body mass, body fat, fat-free mass, $\dot{V}O_{2peak}$ or years of competitive cycling ($P > 0.05$; Table I).

Cycling race simulation protocol

The effect of sodium phosphate supplementation on performance is shown in Tables II–IV.

Table II. Total work (kJ) recorded during a cycling race simulation performed at baseline (pre-supplementation) and 1 and 4 days after 6 days of supplementation with sodium phosphate ($n = 7$) or placebo ($n = 10$) (mean ± s).

	Baseline sodium phosphate	Day 1 sodium phosphate	Day 4 sodium phosphate	Baseline placebo	Day 1 placebo	Day 4 placebo
Overall total	483 ± 66	506 ± 84[1]	507 ± 77⋆[1]	480 ± 85	479 ± 78	480 ± 86
Overall sprint total	259 ± 31	271 ± 37[1]	271 ± 32⋆[1]	260 ± 50	257 ± 44	261 ± 51
Set 1	76 ± 8	78 ± 8	77 ± 7	77 ± 16	71 ± 12	76 ± 16
Set 2	59 ± 9	62 ± 11[1]	62 ± 9⋆[1]	60 ± 12	61 ± 13	60 ± 12
Set 4	68 ± 9	72 ± 9[1]	73 ± 9⋆[1]	67 ± 14	67 ± 12	69 ± 14
Set 5	56 ± 8	59 ± 10[1]	60 ± 9⋆[1]	56 ± 10	57 ± 11	56 ± 11
Overall time-trial total	225 ± 37	235 ± 48	236 ± 45[1]	220 ± 36	222 ± 36	220 ± 36
Set 3	113 ± 18	118 ± 23	118 ± 22	111 ± 18	117 ± 21	113 ± 19
Set 6	111 ± 19	116 ± 24	118 ± 23 ⋆	112 ± 19	117 ± 22	113 ± 18

Note: ⋆ indicates significant difference from baseline within groups ($P < 0.05$); [1] indicates moderate or strong effect size ($d > 0.5$)/beneficial smallest worthwhile change compared with sodium phosphate baseline.

Table III. Power output (W) recorded during a cycling race simulation performed at baseline (pre-supplementation) and 1 and 4 days after 6 days of supplementation with sodium phosphate ($n = 7$) or placebo ($n = 10$) (mean ± s).

	Baseline sodium phosphate	Day 1 sodium phosphate	Day 4 sodium phosphate	Baseline placebo	Day 1 placebo	Day 4 placebo
Overall	604 ± 195	635 ± 206⋆[1]	633 ± 201⋆[1]	604 ± 216	599 ± 200	605 ± 216
Overall sprint	719 ± 123	754 ± 134⋆[1]	753 ± 119⋆[1]	722 ± 167	713 ± 144	724 ± 166
Set 1	841 ± 88	870 ± 93	851 ± 75	856 ± 175	792 ± 138	843 ± 175
Set 2	656 ± 98	689 ± 121[1]	693 ± 99⋆[1]	666 ± 132	680 ± 148	664 ± 134
Set 4	757 ± 95	797 ± 104[1]	807 ± 95⋆[1]	747 ± 154	747 ± 136	767 ± 153
Set 5	621 ± 85	658 ± 114[1]	663 ± 104⋆[1]	620 ± 114	631 ± 119	622 ± 119
Overall time-trial	374 ± 59	398 ± 78⋆[1]	393 ± 72⋆[1]	367 ± 58	370 ± 60	366 ± 59
Set 3	378 ± 61	394 ± 78	392 ± 75	367 ± 58	370 ± 63	361 ± 54
Set 6	370 ± 62	402 ± 85[1]	395 ± 76⋆[1]	367 ± 61	371 ± 60	371 ± 66

Note: ⋆ indicates significant difference from baseline within groups ($P < 0.05$); [1] indicates moderate or strong effect size ($d > 0.5$)/beneficial smallest worthwhile change compared with sodium phosphate baseline.

Table IV. Heart rate, lactate and rating of perceived exertion (RPE) during a cycling race simulation performed at baseline (pre-supplementation) and 1 and 4 days after 6 days of supplementation with sodium phosphate (n = 7) or placebo (n = 10). Data presented are mean±s.

		Baseline sodium phosphate	Day 1 sodium phosphate	Day 4 sodium phosphate	Baseline placebo	Day 1 placebo	Day 4 placebo
Heart rate (bpm)	Overall	165 ± 11	165 ± 12	165 ± 12	168 ± 10	165 ± 11[a]	166 ± 11
	Overall sprint	163 ± 11	164 ± 12	164 ± 11	166 ± 9	163 ± 10[a]	164 ± 9
	Overall time-trial	168 ± 13	168 ± 12	168 ± 14	172 ± 11	168 ± 11[a]	168 ± 12[a]
Lactate (mmol · L^{-1})	Overall	14.6 ± 3.7	15.2 ± 2.9	13.8 ± 2.6	14.1 ± 2.8	13.5 ± 3.1	13.9 ± 3.5
	Overall sprint	14.3 ± 3.8	15.0 ± 3.1	13.7 ± 2.6	13.6 ± 2.4	12.7 ± 2.7	13.5 ± 3.1
	Overall time-trial	15.2 ± 3.4	15.7 ± 2.5	14.0 ± 2.5[b1]	15.3 ± 3.7	15.1 ± 4.0	14.8 ± 4.5
RPE	Overall	17 ± 1	17 ± 1	17 ± 1	17 ± 1	17 ± 1	17 ± 1
	Overall sprint	17 ± 1	17 ± 1	17 ± 1	17 ± 2	17 ± 1	17 ± 1
	Overall time-trial	17 ± 1	17 ± 1	18 ± 1	17 ± 1	17 ± 2	17 ± 1

Note: [a] indicates significant difference ($P < 0.05$) from baseline within groups, [b] = significantly different from day 1 within groups ($P < 0.05$), [1] = moderate or strong effect size ($d > 0.5$) compared with sodium phosphate day 1.

Total work

Within groups, there was no significant difference in total work between baseline and day 1 for sodium phosphate, although smallest-worthwhile-change values suggested greater day 1 overall total work (77/22/1), overall sprint total (85/14/1), set 2 (78/21/1), set 4 (83/16/1) and set 5 (85/14/1) work scores. However, 4 days after loading, total work was significantly higher than baseline in the sodium phosphate group, with "likely" chances of benefit, for overall total work (P = 0.033; smallest worthwhile change: 87/13/0), overall sprint total work (P = 0.018; smallest worthwhile change 92/8/0) and work in set 2 (P = 0.007; smallest worthwhile change: 94/6/0), set 4 (P = 0.037; smallest worthwhile change: 92/8/0; d = 0.56), set 5 (P = 0.039; smallest worthwhile change: 91/9/0) and set 6 (P = 0.030; smallest worthwhile change: 89/11/0). Day 4 time-trial total work also recorded a "likely" smallest-worthwhile-change benefit (76/23/1) compared with baseline values. No significant differences were recorded between day 1 and day 4 values in the sodium phosphate group. In contrast, no differences in total work were noted in the placebo group in response to supplementation ($P > 0.05$). However, there was a main effect of set on total work in the sprint sets in both sodium phosphate and placebo ($P < 0.001$), with decreases observed as the test progressed. There was no effect of set on total work during the time-trial component for either group.

Power output

Within groups, significantly greater mean power outputs (with "likely" benefit) were recorded in the sodium phosphate group (compared with baseline) on day 1 for the whole test ($P < 0.001$; smallest worthwhile change: 86/13/1), overall sprint (P = 0.001; smallest worthwhile change: 85/14/1) and overall time-trial (P = 0.031; smallest worthwhile change: 86/14/0) values. Additionally, day 1 set 2, set 4, set 5 and set 6 mean power values also recorded "likely" smallest-worthwhile-change benefits compared with baseline (78/21/1, 83/16/1, 85/14/1, 89/10/1, respectively). The day 4 values in the sodium phosphate group were similar, with significantly greater results and likely chances of benefit (compared with baseline) observed for the whole test ($P < 0.001$; smallest worthwhile change: 90/10/0), overall sprint (P = 0.001; smallest worthwhile change: 92/8/0) and overall time-trial (P = 0.011; smallest worthwhile change: 76/23/1) mean power outputs, as well as for individual sets 2 (P = 0.007; smallest worthwhile change: 94/6/0), 4 (P = 0.037; smallest worthwhile change: 92/7/1), 5 (P = 0.039; smallest worthwhile change: 91/9/0) and 6 (P = 0.030; smallest worthwhile change: 89/11/0). No significant differences in mean power output were found between day 1 and day 4 in the sodium phosphate group. No placebo within-group differences or benefits were found between days. For both sodium phosphate and placebo, there was a main effect of set on power output in the sprint sets ($P < 0.001$), with decreases observed as the test progressed. There was no effect of set on power output during the time-trial component for either group.

Heart rate, blood lactate and ratings of perceived exertion

No significant differences or moderate–strong effect sizes were seen in heart rate in the sodium phosphate group (Table IV), while the placebo group, compared with baseline, had slightly lower heart rates on day 1 for the whole test ($P < 0.05$) and overall sprint components ($P < 0.05$), and both day 1 and

day 4 for the overall time-trial component ($P < 0.05$). For lactate, the only within-group difference for sodium phosphate was a lower concentration on day 4 compared with day 1 for the time-trial component ($P < 0.05$; $d = 0.62$). There were no differences in lactate noted within the placebo group. For RPE, no significant differences were recorded within either the sodium phosphate group or the placebo group ($P > 0.05$).

Serum phosphate

No differences in baseline serum phosphate concentrations between the sodium phosphate (1.38 ± 0.13 mmol $\cdot$ L^{-1}) and placebo (1.18 ± 0.26 mmol $\cdot$ L^{-1}) groups ($P = 0.084$) were recorded. Day 1 ($P = 0.433$) and day 4 ($P = 0.930$) values were also similar between groups. However, compared with baseline, post-loading serum phosphate was lower in the sodium phosphate group on day 1 (1.25 ± 0.13 mmol $\cdot$ L^{-1}; $P = 0.038$) and day 4 (1.16 ± 0.18 mmol $\cdot$ L^{-1}; $P = 0.004$). In comparison, serum phosphate concentrations in the placebo group remained unchanged over time (day 1, 1.19 ± 0.19 mmol $\cdot$ L^{-1}; day 4, 1.17 ± 0.23 mmol $\cdot$ L^{-1}).

Discussion

This is the first study to assess whether sodium phosphate supplementation could improve performance in a continuous, prolonged exercise protocol including repeated-sprint bouts and short duration time-trials, intended to simulate a cycling road race. In addition, the lingering (short-term) effect of sodium phosphate supplementation was examined by repeating the exercise trial 4 days after loading ceased. Supplementation with sodium phosphate resulted in several significant improvements in work and mean power output (with associated likely chances of benefit) for the whole test, plus overall sprint and time-trial efforts, as well as in individual sets during the exercise protocol on both days 1 and 4 after supplementation compared with baseline. In contrast, in the placebo group, no differences were noted between baseline and day 1 and 4 values. The consistent benefits in work and power output observed within the sodium phosphate group suggest that the supplementation used here was able to enhance both repeated-sprint and time-trial cycling performance. Further, no differences between day 1 and 4 were observed, suggesting that any effect of sodium phosphate supplementation may linger for at least a few days after loading ceases.

Of relevance, the nature of the simulated road cycling race protocol used here (43 min, consisting of repeated-sprint bouts, time-trials and active recovery periods with no passive rests) would have required metabolic contributions from all three energy systems, with a progressively greater emphasis on aerobic metabolism likely for both the repeated-sprint bouts and the time-trial efforts as the exercise protocol continued (Gaitanos, Williams, Boobis, & Brooks, 1993; Gastin, 2001). Notably, the mechanisms proposed to contribute to the benefit of sodium phosphate supplementation for exercise performance relate to all energy systems as discussed below (and reviewed in Buck, Wallman, Dawson, & Guelfi, 2013).

Improved cycling time-trial performance following sodium phosphate supplementation has been previously reported by both Kreider et al. (1992) and Folland et al. (2008). Kreider et al. (1992) proposed that the primary mechanism underlying this benefit was increased phosphate availability contributing to enhanced oxidative metabolism. They also postulated that an increase in extracellular phosphate availability may be reflected by increases in serum phosphate levels following sodium phosphate supplementation. However, their study returned varying results after sodium phosphate loading, with serum phosphate concentrations showing no change before a 40-km time-trial, but significantly higher levels before a $\dot{V}O_{2max}$ test (compared with placebo). Consequently, Kreider et al. (1992) suggested that this measure may not accurately reflect the effects of sodium phosphate loading on intracellular phosphate levels and hence oxidative capabilities. In support, Stewart et al. (1990) found an improved $\dot{V}O_{2max}$ (11%) after sodium phosphate loading without any increases in resting serum phosphate, suggesting that this variable appeared unresponsive to supplementation. These reports are pertinent to our study, as serum phosphate concentration did not increase (but in fact, decreased) following sodium phosphate loading, despite improved work and power in these trials. The reason for this is unclear. Nevertheless, serum phosphate concentrations remained within the normal range for adults and it is still possible that increased cellular phosphate availability via sodium phosphate loading may have played some role in improving exercise performance here.

Increased RBC 2,3-DPG concentration after sodium phosphate supplementation has also been proposed as a contributing factor for improved endurance performance (Folland et al., 2008) and aerobic capacity (Stewart et al., 1990). Studies have reported increases in haemoglobin (Hb) 2,3-DPG concentrations after sodium phosphate loading, which were also associated with increases in $\dot{V}O_{2max}$ (Cade et al., 1984;

Czuba et al., 2009; Stewart et al., 1990). Increased 2,3-DPG concentrations are proposed to decrease the affinity of Hb for oxygen, thus resulting in greater unloading of oxygen to the peripheral tissues (Czuba et al., 2009). As we were unable to measure RBC 2,3-DPG concentrations in the current study (financial constraints), we can only surmise that this mechanism may have played some part in the improved exercise performance seen here.

Improved myocardial efficiency, via sodium phosphate supplementation, is another proposed mechanism which could impart ergogenic benefit during exercise by providing a greater stroke volume (and hence cardiac output), resulting in greater and more efficient oxygenation of the exercising muscles (Kreider et al., 1992). Using cardiac ultrasound and colour flow Doppler technology, these authors reported that sodium phosphate loading significantly improved cardiac function (end diastolic volume, stroke volume and cardiac output) during a maximal exercise test and 40-km cycle time-trial, compared with placebo. These improvements were associated with increases in sodium phosphate time-trial mean power output (17%) and maximal oxygen uptake (9%) (Kreider et al., 1992). Czuba et al. (2009) also reported significant increases in $\dot{V}O_{2max}$ (5.3%) following 6 days of sodium phosphate supplementation compared with placebo and suggested that enhanced myocardial contractility, resulting in increased stroke volume, was a likely mechanism (although this parameter was not measured). They also reported significant decreases in resting and maximal exercise heart rates following sodium phosphate supplementation. In the present study, we found no differences in heart rate after sodium phosphate supplementation, despite greater work and power scores. As heart rate values alone are unlikely to be a reliable indicator of improved myocardial efficiency, whether this mechanism may have benefited exercise performance in the current study remains uncertain.

Enhanced hydrogen ion (H^+) buffering (Czuba et al., 2009) is another mechanism associated with sodium phosphate loading that may have enhanced exercise performance in the current study, particularly in respect of the repeated-sprint bouts. Evidence for the use of anaerobic glycolysis here is reflected by blood lactate concentrations of ~13–16 mmol $\cdot$ L^{-1} during the exercise protocol. In part, enhanced buffering of H^+ in the current study is indirectly supported by improved overall mean power output and work values following sodium phosphate supplementation, despite no difference in final blood lactate concentrations. These results may reflect an improved buffering capacity and/or a greater aerobic energy contribution (Gaitanos et al., 1993) occurring in the latter sets of the cycling protocol. However, measuring exercising intramuscular pH values, both before and after sodium phosphate loading, is necessary so that more definitive comments can be made about whether enhanced buffering capacity from sodium phosphate supplementation impacts repeated-sprint performance. The only other study to examine the effects of sodium phosphate supplementation on sprint performance (a single Wingate 30 s test, with no effect seen) did not report any lactate or pH values (Tourville et al., 2001).

Lastly, the similar improvements in exercise performance recorded here on day 1 and 4 after sodium phosphate loading suggest that any ergogenic effect lingers for at least a few days after supplementation ceases. Cade et al. (1984) reported that it took approximately 2 weeks for RBC 2,3-DPG concentration to return to baseline following only 3 days of sodium phosphate loading. This suggests that any aerobic benefits associated with this particular mechanism may still be in effect 4 days after sodium phosphate loading, particularly as a longer loading period (6 days) was used here. Evidence for an additive effect of repeated or continued sodium phosphate supplementation has been provided by Brewer et al. (2013) and Czuba et al. (2009). Brewer et al. (2013) employed a second sodium phosphate loading phase, with 14 days separating the end of one loading phase and the beginning of the next (both 50 mg $\cdot$ kg$\cdot$fat-free-mass^{-1} $\cdot$ day^{-1} for 6 days), which resulted in additional improvement in $\dot{V}O_{2peak}$ (~7%) compared with benefits seen following the first loading phase (~4%). Czuba et al. (2009) recorded further improvements in cycling power output after an extended (21 day) loading period, which followed an initial 6 days of supplementation. Further research is needed to probe how long any initial effect of sodium phosphate supplementation may last, as well as the potential benefits of repeated or continued loading on exercise performance.

In conclusion, 6 days of sodium phosphate supplementation resulted in enhanced performance (greater work and power outputs) during a simulated high-intensity road cycling protocol incorporating repeated-sprints and short duration time-trial efforts. These benefits were still evident 4 days after supplementation had finished, with no performance differences found between day 4 and day 1 post-loading. Information from this study may be pertinent to cyclists who compete in road tour events, where competition continues over several days. Future studies should investigate mechanisms associated with these benefits, as well as how long these exercise performance benefits last following sodium phosphate loading.

Acknowledgement

The authors acknowledge the assistance of PathWest Laboratory Medicine, Royal Perth Hospital, Perth, Australia for the analysis of serum phosphate.

References

Avioli, L. (1988). Calcium and phosphorus. In M. Shils & V. Young (Eds.), *Modern nutrition in health and disease* (pp. 254–288). Philadelphia, PA: Lea & Febiger.

Batterham, A. M., & Hopkins, W. G. (2005). Making meaningful inferences about magnitudes. *Sportscience*, *9*, 6–13.

Borg, G. (1982). Psychophysical bases of perceived exertion. *Medicine and Science in Sports and Exercise*, *14*(5), 377–381.

Brain, M. C., & Card, R. T. (1972). Effect of inorganic phosphate on red cell metabolism: In vitro and in vivo studies. *Advances in Experimental Medicine and Biology*, *28*, 145–154.

Brewer, C. P., Dawson, B., Wallman, K. E., & Guelfi, K. J. (2013). Effect of repeated sodium phosphate loading on cycling time-trial performance and VO_{2peak}. *International Journal of Sports Nutrition and Exercise Metabolism*, *23*(2), 187–194.

Buck, C. L., Wallman, K. E., Dawson, B., & Guelfi, K. J. (2013). Sodium phosphate as an ergogenic aid. *Sports Medicine*, *43*(6), 425–435.

Cade, R., Conte, M., Zauner, C., Mars, D., Peterson, J., Lunne, D., … Packer, D. (1984). Effects of phosphate loading on 2, 3-diphosphoglycerate and maximal oxygen uptake. *Medicine and Science in Sports and Exercise*, *16*(3), 263–268.

Chanutin, A., & Curnish, R. (1967). Effect of organic and inorganic phosphates on the oxygen equilibrium of human erythrocytes. *Archives of Biochemistry and Biophysics*, *121*, 96–102.

Chasiotis, D. (1983). The regulation of glycogen phosphorylase and glycogen breakdown in human skeletal muscle. *Acta Physiologica Scandinavica. Supplementum*, *518*, 1–68.

Cohen, J. (1988). *Statistical power analysis for the behavioural sciences*. Hillsdale, NJ: Lawrence Erlbaum Associates.

Czuba, M., Zajac, A., Poprzecki, S., & Cholewa, J. (2008). The influence of sodium phosphate supplementation on VO_{2max}, serum 2, 3-diphosphoglycerate level and heart rate in off-road cyclists. *Journal of Human Kinetics*, *19*, 149–164.

Czuba, M., Zajac, A., Poprzecki, S., Cholewa, J., & Woska, S. (2009). Effects of sodium phosphate loading on aerobic power and capacity in off road cyclists. *Journal of Sports Science and Medicine*, *8*, 591–599.

Eaton, J., Brewer, G., & Grover, E. (1969). Role of red cell 2, 3-diphosphoglycerate in the adaptation of man to altitude. *Journal of Laboratory and Clinical Medicine*, *73*, 603–609.

Folland, J. P., Stern, R., & Brickley, G. (2008). Sodium phosphate loading improves laboratory cycling time-trial performance in trained cyclists. *Journal of Science and Medicine in Sport*, *11*, 464–468.

Gaitanos, G. C., Williams, C., Boobis, L. H., & Brooks, S. (1993). Human muscle metabolism during intermittent maximal exercise. *Journal of Applied Physiology*, *75*(2), 712–719.

Gastin, P. B. (2001). Energy system interaction and relative contribution during maximal exercise. *Sports Medicine*, *31*(10), 725–741.

Kreider, R. B., Miller, G. W., Schenck, D., Cortes, C. W., Miriel, V., Somma, C. T., … Hill, D. (1992). Effects of phosphate loading on metabolic and myocardial responses to maximal and endurance exercise. *International Journal of Sports Nutrition*, *2*(1), 20–47.

Kreider, R. B., Miller, G. W., Williams, M. H., Somma, C. T., & Nasser, T. A. (1990). Effects of phosphate loading on oxygen uptake, ventilatory anaerobic threshold, and run performance. *Medicine and Science in Sports and Exercise*, *22*(2), 250–256.

Lichtman, M. A., & Miller, D. R. (1970). Erythrocyte glycolysis, 2, 3-diphosphoglycerate and adenosine triphosphate concentration in uremic subjects: relationship to extracellular phosphate concentration. *The Journal of Laboratory and Clinical Medicine*, *76*(2), 267–279.

Lichtman, M. A., Miller, D. R., Cohen, J., & Waterhouse, C. (1971). Reduced red cell glycolysis, 2,3-diphosphoglycerate and adenosine triphosphate concentration, and increased hemoglobin-oxygen affinity caused by hypophosphatemia. *Annals of Internal Medicine*, *74*, 562–568.

Rose, Z. (1970). Enzymes controlling 2, 3-diphosphoglycerate in human erythrocytes. *Federation Proceedings*, *29*, 1105.

Stewart, I., McNaughton, L., Davies, P., & Tristram, S. (1990). Phosphate loading and the effects on VO_{2max} in trained cyclists. *Research Quarterly for Exercise and Sport*, *61*(1), 80–84.

Thompson, D. L., Grantham, S., Hall, M., Johnson, J., McDaniel, J., Servedio, F., … Thompson, W. R. (1990). Effects of phosphate loading on erythrocyte 2,3-diphospho-glycerate (2,3-DPG), adenosine 5' -triphoshate (ATP), hemoglobin (Hb), and maximal qxygen consumption (VO_{2max}). *Medicine and Science in Sports and Exercise*, *22*, S36.

Tourville, T. W., Brennan, K. M., & Connolly, D. A. (2001). The effects of sodium phosphate supplementation on anaerobic power in trained male athletes. *Medicine and Science in Sports and Exercise* (Abstract), *33*(5) Supplement 1, S164.

Vaile, J., Halson, S., Gill, N., & Dawson, B. (2008). Effect of hydrotherapy on recovery from fatigue. *International Journal of Sports Medicine*, *29*(7), 539–544.

West, J. S., Ayton, T., Wallman, K. E., & Guelfi, K. J. (2012). The effect of 6 days of sodium phosphate supplementation on appetite, energy intake, and aerobic capacity in trained men and women. *International Journal of Sports Nutrition and Exercise Metabolism*, *22*(6), 422–429.