Sodium Phosphate Supplementation and Time Trial Performance in Female Cyclists

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Abstract
This study investigated the effects of three doses of sodium phosphate (SP) supplementation on cycling 500 kJ (119.5 Kcal) time trial (TT) performance in female cyclists. Thirteen cyclists participated in a randomised, Latin-square design study where they completed four separate trials after ingesting either a placebo, or one of three different doses (25, 50 or 75 mg.kg\(^{-1}\) free mass: FFM) of trisodium phosphate dodecahydrate which was split into four equal doses a day for six days. On the day after the loading phase, the TT was performed on a cycle ergometer. Serum phosphate blood samples were taken at rest both before and after each loading protocol, while a ~21 day washout period separated each loading phase. No significant differences in TT performance were observed between any of the supplementation protocols (p = 0.73) with average completion times for the 25, 50 or 75 mg.kg\(^{-1}\) FFM being, 42:21 ± 07:53, 40:55 ± 07:33 and 40:38 ± 07:20 min respectively, and 40:39 ± 07:51 min for the placebo. Likewise, average and peak power output did not significantly differ between trials (p = 0.06 and p = 0.46, respectively). Consequently, 500 kJ cycling TT performance was not different in any of the supplementation protocols in female cyclists.

Key words: Ergogenics, endurance performance, 2,3-DPG.

Introduction
Sodium phosphate (SP) is a nutritional supplement, which has been reported to provide significant ergogenic benefits (5-12% improvement) for aerobic capacity (Brewer et al., 2013; Cade et al., 1984; Czuba et al., 2008; 2009; Kreider et al., 1990; 1992). Furthermore it has also been shown to be effective in improving endurance performance (Folland et al., 2008; Kreider et al., 1992). A number of mechanisms have been proposed to provide benefit following SP ingestion which include an enhanced 2,3-diphosphoglycerate (2,3-DPG) concentration, which allows for greater unloading of oxygen to the peripheral tissues/muscle (Buck et al., 2013; Benesch and Benesch, 1969; Duhm, 1971) and an improved buffering capacity due to increased hydrogen phosphate concentration, which could buffer hydrogen ions produced during intense exercise (Kreider, 1999). Additionally, it is also thought that an improved myocardial efficiency, which is proposed to result in increased stroke volume, a larger cardiac output and consequently greater and more efficient oxygenation of the exercising muscles (Czuba et al., 2009), and greater ATP/PCr synthesis due to increased availability of extracellular and intracellular phosphate, thus providing a larger energy pool (Kreider, 1999), may also contribute to the benefits seen with SP loading. These proposed mechanisms underlie the notion that phosphate supplementation increases serum phosphate levels, thereby allowing processes normally limited by phosphate availability to operate at an enhanced level (Tremblay et al., 1994).

Of the few studies that have assessed the effects of SP supplementation on endurance performance (Brewer et al., 2013; Folland et al., 2008; Kreider et al., 1990; 1992), only male participants were recruited, suggesting that future research should be performed in females. Importantly, due to a number of physiological differences, it is possible that females may respond differently to doses of SP that have been reported to be beneficial in males (Buck et al., 2013; Fukuda et al., 2010; O’Brien, 1985). Females, for example, have a decreased oxygen-hemoglobin affinity compared with males (Humpeler and Amor, 1973; Samaja et al., 1990). Specifically, Samaja et al., (1990) have reported higher P\(_{50}\) values (oxygen tension at 50% oxygen saturation) and increased 2,3-DPG concentrations in females, which may result in females being less responsive to SP supplementation compared with males (Fukuda et al., 2010). Females also have greater and higher fluctuations in estrogen levels compared with males (Janse de Jonge, 2003; Remes et al., 1979), with estrogen acting to decrease renal phosphate reabsorption (Dick et al., 2005). Furthermore, females tend to have smaller hearts, left ventricular masses, stroke volume and reduced red cell mass compared with males (O’Brien, 1985). Related to this difference between the genders is that SP loading has been proposed to improve myocardial efficiency (Czuba et al., 2009; Kreider, 1999), which is thought to lead to a better oxygen supply to the muscles and hence improved exercise performance (Kreider, 1999). However, reduced red cell mass in females (O’Brien, 1985) may limit the magnitude of any oxygen transport benefits gained from enhanced myocardial efficiency.

Therefore, the aim of this study was to assess the effects of a variety of doses of SP supplementation (25, 50 and 75 mg.kg\(^{-1}\) of free fat-mass; FFM) on 500 kJ (119.5 Kcal) cycling time trial (TT) performance in female cyclists. A 50 mg.kg\(^{-1}\) of FFM dose was chosen as this dose has been shown to benefit exercise performance in males (Czuba et al., 2009), while a 75 mg.kg\(^{-1}\) FFM dose was selected as this overall dose is similar to the absolute dose of SP (i.e. ~ 4 g per day) also reported to benefit exercise performance in males (Folland et al., 2006; Kreider et al.,...
1.65 ± 0.05 m, fat-mass 15.0 ± 3.6 kg, FFM 44.9 ± 5.1 kg.

Methods

Participants

Thirteen females who cycled on a regular basis (3.6 h ± 2.1 per week) were recruited to this study based on a G-power analysis (Faul et al., 2009) that determined that this number of participants were needed to detect changes that occurred at an alpha level of 0.05. The physical characteristics of the participants were: VO\textsubscript{2peak} 49.8 ± 7.46 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}, age 25.5 ± 4.4 y, body-mass 62.8 ± 5.5 kg, height 1.65 ± 0.05 m, fat-mass 15.0 ± 3.6 kg, FFM 44.9 ± 5.1 kg. During the study, six participants were taking Levlen ED for birth control, with the remaining participants took no oral contraceptives. Participants took no nutritional supplements for at least two months prior to or during this study. The Human Research Ethics Committee of the University of Western Australia (UWA) approved the study. The Human Research Ethics Committee of the University of Western Australia (UWA) approved the study and all participants provided written informed consent prior to participation.

Experimental design

Participants completed a familiarisation session and four different supplementation protocols over an 18 week period. During the familiarisation session, body composition was determined using dual energy x-ray absorptiometry (Lunar Prodigy, GE Medical Systems, Madison, USA) to determine each participant’s FFM. Additionally, full familiarisation with a 500 kJ (119.5 Kcal) cycling TT protocol was undertaken. On a separate day, participants returned to the laboratory in order to complete a graded exercise test to measure their VO\textsubscript{2peak}. This test was performed on a wind-braked cycle ergometer (Evolution Pty. Ltd., Adelaide, Australia) and consisted of cycling for 3 min intervals, with a 1 min rest period between each interval, until volitional exhaustion was reached. The test began at 150 watts (W), with workload increasing by 50 W for every subsequent interval (Brewer et al., 2013). During this test, expired air was assessed using a gas analysis system consisting of a ventilometer (Universal ventilation meter, VacuMed, Ventura, California, USA) and oxygen and carbon dioxide analysers (Ametek Applied Electrochemistry S-3A/1 and CD-3A, AEI Technologies, Pittsburgh). Before, and after each test, the gas analysers were calibrated using gases of known concentration (BOC gases, Chatswood, Australia) and gas analysers were verified immediately after testing to check for drift. If the post-test calibration demonstrated that ventilatory drift had occurred, then a correction factor (based on the magnitude of the drift) was applied to test data.

Participants were then assigned, in a randomised, Latin-square design, to three SP and one placebo loading protocol. To control for phase of menstrual cycle, the first supplementation protocol began ~3-5 days post the first menstruation (follicular phase) after completing the familiarisation session. All subsequent testing was performed during this same phase, with ~21 days between trials. Notably, 17 days has been proposed to be the minimum washout period for SP (Brewer et al., 2013; Kreider et al., 1992).

The SP trials consisted of the consumption of a 25, 50 or 75 mg·kg\textsuperscript{-1} of FFM dose of trisodium phosphate dodecahydrate (Challenge Chemicals Australia, Western Australia), split into four equal doses a day, consumed for six consecutive days. Each dose was placed into an opaque capsule by a blinded researcher. In order to prevent gastrointestinal (GI) upset, each capsule was emptied into a glass and consumed with 15 g of Powerade powder (Coca-Cola Amatil, Australia) that had been dissolved in ~300 ml of water (Brewer et al., 2013; West et al., 2012). Importantly, no side effects were reported. Participants were instructed to separate the ingestion of each capsule by ~4 h. The placebo protocol followed the same loading strategy with a 4 g dose of glucose each day. On the day after the end of each loading protocol, participants returned to the laboratory and completed the same 500 KJ cycling TT. Finally, participants attended the laboratory one day prior to the commencement of each supplementation protocol so that a venous blood sample could be taken to assess serum phosphate, while a second sample was taken at the end of each supplementation protocol, prior to performing the 500 KJ (119.5 Kcal) TT. The timing of each venous blood sample was standardized between supplementation phases.

Participants were instructed to follow their normal training diet and fluid intake during each supplementation protocol and were also required to complete a detailed dietary record for the 24 h period prior to each TT. A copy of the food diary from the first TT was provided to participants with the instructions to replicate this eating pattern before each subsequent TT. Dietary analysis of each participant’s self-reported intake was undertaken on completion of the study using FoodWorks software package (FoodWorks v 4.2.0, Xyris Software, Qld, Australia). Participants were required to maintain a consistent training volume throughout the study and completed a six day physical activity diary during each supplementation period. Participants were requested to replicate their physical activity patterns using this activity diary during each phase of the study.

Cycling time trial

The simulated 500 KJ (119.5 Kcal) cycling TT was performed on a wind-braked cycle ergometer (Evolution Pty. Ltd., Adelaide, Australia), modified with clip-on pedals and racing handle bars. Exercise was self-paced in order to replicate actual TT situations, with participants instructed to complete the 500 KJ (119.5 Kcal) of work in the fastest time possible. A 500 KJ (119.5 Kcal) workload has been shown in previous research to approximate to a 20 km TT distance in trained male cyclists (Peeling et al., 2005). A test-retest of the 500 KJ (119.5 Kcal) time trial in
ten female cyclists resulted in a coefficient of variance of 2.9%.

Participants determined optimal moderate warm-up intensity during the familiarisation session, with this warm-up intensity replicated for all experimental sessions. Participants refrained from exercise for 24 h prior to each experimental session, with all TT completed at the same time of the day to minimise any circadian rhythm effects. No food and/or caffeine intake was permitted for 2 h prior to each TT. Participants consumed 200 mL of water prior to commencing each TT, with no further fluid ingestion allowed until completion of the trial.

During each TT, participants were blinded to their power output and duration of their performance, but had a visual display of the accumulated kJ of work completed. The performance variables recorded during the 500 kJ (119.5 Kcal) TT were time to completion and peak and average power output, which were recorded by a customised computer program (Cyclemax; UWA). These variables were recorded automatically at 125 kJ split times and heart rate (Polar Heart Rate Monitors, Kempele, Finland) was recorded at each 125 kJ (29 Kcal) split. Rating of perceived exertion (RPE - Borg’s 6-20 point scale; Borg, 1982) and blood lactate were measured at the start and end of the 500 kJ (119.5 Kcal) TT. To determine blood lactate, a capillary blood sample was collected in a 35 µL heparinised glass capillary tube from the earlobe. The sample was then immediately analysed for plasma lactate concentration using a blood-gas analyser (ABL 725, Radiometer, Copenhagen, Denmark).

**Venepuncture**

Before and after loading, resting venous blood samples for serum phosphate determination were collected via venepuncture of an antecubital vein in the forearm. A total of 8.5 mL of blood was collected for each sample, which were then 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, employing 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 analyses**

Statistics Package for the Social Sciences Version 16.0 for Windows (SPSS, Inc., Chicago, IL) was used to perform one-way repeated-measures ANOVAs to test for significant differences between the varying doses of SP for each dependent variable. Bonferroni post-hoc tests were applied to determine the location of significant differences. In addition, Cohen’s ℓ effect sizes (Cohen, 1988) (ES < 0.2, small; 0.5 - 0.79, moderate; ≥ 0.8, strong) were calculated to assess the magnitude of difference between experimental trials. Further analysis identified the smallest worthwhile change in performance scores between trials using the method outlined by Batterham and Hopkins (2005). The smallest worthwhile value of change was set at a Cohen’s unit of 0.2, representing the hypothetical, smallest change in physiological measures that would benefit the athlete. Where the chance of benefit and harm were both calculated to be > 5%, the true effect was deemed unclear. When clear interpretation was possible, a qualitative descriptor was assigned to the following quantitative chances of benefit: 25-74%, benefit possible; 75-94%, benefit likely; 95-98%, benefit very likely; > 99%, benefit almost certain (Batterham and Hopkins, 2005). In order to include only pertinent information in this manuscript, only quantitative chances > 25% and moderate to large effect sizes (ES ≥ 0.5) are reported. Pearson correlations were also performed to assess for any relationship between serum phosphate levels and main performance variables. All results are presented as mean±SD.

**Results**

**500 kJ (119.5 Kcal) TT**

There were no significant differences observed between trials for time to completion (p = 0.73; Table 1). Further, there were no ‘possible’ or ‘likely’ benefits, or moderate or large effect sizes found between trials for this variable. The 75 mg·kg⁻¹ of FFM trial resulted in the highest average power and peak power output but this was not significantly different to any other trial (p = 0.06 and p = 0.46, respectively), nor were there any ‘possible’ or ‘likely’ benefits or moderate to large effect sizes associated with the results (Table 1). Notably, four participants recorded their best time following the 50 mg·kg⁻¹ of FFM trial, with an additional four participants performing better on this dose than the placebo (Table 2). In addition, five participants recorded their best TT following the 75 mg·kg⁻¹ of FFM trial, with an additional participant recording a faster time following this dose compared with the placebo (Table 2). Time to completion was slowest and average power output lowest following the 25 mg·kg⁻¹ of FFM trial compared with all other trials, with these results supported by ‘very likely’ detriments in performance when compared with the placebo and the 75 mg·kg⁻¹ of FFM trials (Table 1).

**Table 1. Performance variables for 500 kJ cycling time trial following placebo and sodium phosphate supplementation (n = 13). Data are means (±SD).**

<table>
<thead>
<tr>
<th></th>
<th>Placebo</th>
<th>25 mg/kg Free Fat Mass</th>
<th>50 mg/kg Free Fat Mass</th>
<th>75 mg/kg Free Fat Mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to completion (min:s)</td>
<td>40:39.8 (07:51)</td>
<td>42:21.6 (07:53) a</td>
<td>40:55.2 (07:31)</td>
<td>40:38.4 (07:20)</td>
</tr>
<tr>
<td>Average power output (W)</td>
<td>212 (40)</td>
<td>203 (40) a</td>
<td>211 (42)</td>
<td>213 (40)</td>
</tr>
<tr>
<td>Peak power output (W)</td>
<td>330 (77)</td>
<td>328 (102)</td>
<td>349 (70)</td>
<td>335 (74)</td>
</tr>
<tr>
<td>Post exercise RPE</td>
<td>17.5 (1.3)</td>
<td>17.5 (1.2)</td>
<td>17.8 (1.2)</td>
<td>18 (1)</td>
</tr>
<tr>
<td>Post exercise lactate (mM)</td>
<td>6.4 (1.2)</td>
<td>6.2 (1.6)</td>
<td>7.1 (2.4)</td>
<td>8.5 (2.4)</td>
</tr>
<tr>
<td>Post exercise heart rate (bpm)</td>
<td>170 (7)</td>
<td>165 (7)</td>
<td>169 (5)</td>
<td>169 (8)</td>
</tr>
</tbody>
</table>

a = “very likely” to be detrimental compared with 75 mg/kg FFM trial and the placebo trial.
Table 2. Participant time to completions for 500 kJ time trial following placebo and sodium phosphate supplementation (n = 13). FFM = free fat mass; * = average time for faster participants (n = 8); † = average time for slower participants (n = 5).

<table>
<thead>
<tr>
<th>Participant</th>
<th>Placebo mins</th>
<th>25 mg/kg FFM mins</th>
<th>50 mg/kg FFM mins</th>
<th>75 mg/kg FFM mins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Participant 1*</td>
<td>40:20.2</td>
<td>40:11.8</td>
<td>40:16.8</td>
<td>41:16.6</td>
</tr>
<tr>
<td>Participant 2†</td>
<td>44:25.2</td>
<td>47:25.2</td>
<td>42:45.0</td>
<td>46:50.6</td>
</tr>
<tr>
<td>Participant 3†</td>
<td>43:54.4</td>
<td>42:24.0</td>
<td>45:24.6</td>
<td>41:57.4</td>
</tr>
<tr>
<td>Participant 4*</td>
<td>38:36.4</td>
<td>41:25.8</td>
<td>34:48.0</td>
<td>40:14.6</td>
</tr>
<tr>
<td>Participant 5*</td>
<td>38:52.0</td>
<td>40:19.0</td>
<td>38:33.0</td>
<td>37:53.2</td>
</tr>
<tr>
<td>Participant 6†</td>
<td>56:14.0</td>
<td>57:11.0</td>
<td>54:22.2</td>
<td>52:04.6</td>
</tr>
<tr>
<td>Participant 7†</td>
<td>44:54.4</td>
<td>49:28.6</td>
<td>50:56.8</td>
<td>49:01.4</td>
</tr>
<tr>
<td>Participant 8*</td>
<td>32:07.0</td>
<td>30:25.6</td>
<td>33:51.8</td>
<td>32:10.6</td>
</tr>
<tr>
<td>Participant 9*</td>
<td>36:25.6</td>
<td>40:56.2</td>
<td>38:06.4</td>
<td>36:07.0</td>
</tr>
<tr>
<td>Participant 10*</td>
<td>30:45.4</td>
<td>32:43.6</td>
<td>28:46.6</td>
<td>29:26.2</td>
</tr>
<tr>
<td>Participant 11*</td>
<td>31:34.2</td>
<td>33:44.0</td>
<td>31:27.0</td>
<td>33:45.6</td>
</tr>
<tr>
<td>Participant 12†</td>
<td>56:02.6</td>
<td>57:32.0</td>
<td>54:25.0</td>
<td>51:19.2</td>
</tr>
<tr>
<td>Participant 13*</td>
<td>34:26.0</td>
<td>36:50.0</td>
<td>38:16.0</td>
<td>36:19.2</td>
</tr>
</tbody>
</table>

Average time benefit compared to placebo for those that benefited

- 1 min 7 s
- 1 min 25 s
- 2 min 14s

*Average time for faster participants (n = 8)  †Average time for slower participants (n = 5)

Due to the large differences in TT completion times (28 min 46 s vs 57 min 32 s), a separate analysis of results was performed for those who completed the four trials in ≤ 45 min (n = 8, average VO2peak = 53.1 ml·kg⁻¹·min⁻¹) and those who completed the trials ≥ 45 min (n = 5, average VO2peak = 45 ml·kg⁻¹·min⁻¹) (Table 2), with 45 min representing approximately the half-way point between the fastest and slowest trials. This analysis resulted in no moderate or large effect sizes, no beneficial or detrimental effects or significant differences between the four trials for either the faster group (p = 0.19) or the slower group (p = 0.37).

In addition, analysis of post exercise blood lactate concentrations and ratings of perceived exertion values demonstrated no significant differences between any trials (p = 0.16 and p = 0.72, respectively). It was observed that post TT heart rate was different between trials (p = 0.03), with post hoc analysis showing that HR was lower in the 25 mg·kg⁻¹ trial than both the 50 mg·kg⁻¹ of FFM and the placebo trial (p = 0.04 and p = 0.01, respectively).

Serum phosphate

There were no significant differences between trials for post-loading serum phosphate levels (p = 0.47) or between pre and post-loading values (p = 0.63). Pre and post-loading serum phosphate levels for the 25, 50 and 75 mg·kg⁻¹ of FFM trials were 1.35 ± 0.13, 1.36 ± 0.15, 1.28 ± 0.18, 1.31 ± 0.09, 1.41 ± 0.09 and 1.37 ± 0.1 mmol·L⁻¹ respectively. The pre and post-loading serum phosphate levels for the placebo trial were 1.38 ± 0.1 and 1.37 ± 0.15 mmol·L⁻¹.

There were no correlations between serum phosphate concentrations and any of the performance variables for the total group or in those participants who had improved their TT completion times following the 50 and 75 mg·kg⁻¹ of FFM dosing protocols. Additionally, when changes in serum phosphate were correlated with changes in TT completion times between the phosphate and placebo trials, no significant relationships were found (r = -0.448 to -0.202 range).

Food diaries

There was no significant differences (p = 0.16) in energy intake for the 24 h prior to the 500 kJ TT for the 25 mg·kg⁻¹ of FFM, 50 mg·kg⁻¹ of FFM, and 75 mg·kg⁻¹ of FFM of SP trials, or the placebo trial (6680 ± 2052 KJ (1629 ± 500 Kcal), 7419 ± 1637 KJ (1809 ± 399 Kcal), 7949 ± 1796 KJ (1938 ± 438 Kcal), respectively).

Discussion

This is the first study to investigate the effects of SP loading on endurance exercise in female cyclists, as well as to assess the effects of a number of SP loading doses on cycling TT performance. Overall, results showed that while the 75 mg·kg⁻¹ of FFM trial resulted in the fastest completion time and highest average and peak power, compared with all other trials, these results were not significant. Similarly, when TT scores were separated into fastest times (TT completion of ≤ 45 min) and slowest times (TT completion ≥ 45 min), there were also no significant differences in results between trials for either subgroup. This suggests that either a 25, 50 or 75 mg·kg⁻¹ of FFM dose of SP ingested for six days has no significant effect on endurance performance in females cyclists, regardless of whether participants were of higher (mean VO2peak = 53.1 ml·kg⁻¹·min⁻¹) or lower fitness (mean VO2peak =45 ml·kg⁻¹·min⁻¹).

Previous studies have reported improved exercise performance in male participants after a relative dose of 50 mg·kg⁻¹ of FFM. Czuba et al. (2008; 2009) reported significant increases (5–5.6%) in cycling VO2max after 6 and 21 days of SP supplementation, while Brewer et al., (2013) reported significant increases in VO2max (3.4% and 7.1%) after an initial and follow-up loading phase using this relative dose of SP, respectively. However, while Brewer et al. (2013) also reported significant improvement in cycling power output 250 KJ (59.75 Kcal) and 500 KJ (119.5 Kcal) split times after SP loading during the second phase of a two phase loading protocol in male cyclists performing a 1000 kJ TT, completion times were not significantly different between trials.
Brewer et al. (2013) suggested that the overall dose used in their study, which equated to ~3.4 g·d⁻¹ over 6 days, may not have been high enough to elicit an ergogenic effect on overall exercise performance. This comment was based on studies by Kreider et al. (1992) and Folland et al. (2006) who reported an 8% and 3% improvement in 40 and 16.1 km cycling TT performance, respectively, following the ingestion of 4 g/d of SP over 3–4 day. Of relevance, the 75 mg·kg⁻¹ of FFM dose of SP used in the current study was similar to the higher absolute dose used by both Kreider et al. (1992) and Folland et al. (2006) (i.e. ~4 g·d⁻¹), yet no benefit was found for endurance performance here.

Differences in results between the current study and those that reported benefit of SP on endurance performance may be related to gender. As noted earlier, females differ to males in respect to oxygen affinity, hormonal concentrations and heart function (O’Brien, 1985), with these factors all associated with mechanisms proposed to result in ergogenic effects following SP loading. These differences either separately or as a whole, could have reduced the magnitude of benefit received by the participants in this study, resulting in no overall benefit of SP supplementation on 500 kJ (119.5 Kcal) cycle performance. It is possible that a higher relative dose may be needed in females for an ergogenic effect to occur in respect to endurance performance.

While SP loading resulted in no improvement in TT performance in the current study, 8 of the 13 participants recorded faster times following the 50 mg·kg⁻¹ of FFM trial compared with the placebo trial (1 min 25 s ± 1 min 15 s faster on average), with four of these participants recording their fastest time overall. Furthermore, six participants recorded faster times following the 75 mg·kg⁻¹ of FFM trial, compared with the placebo trial, with five of these participants recording their fastest time overall. Differences in TT results between this highest dose trial and the placebo trial for these participants was 2 min 14 s ± 1 min and 43 s faster on average. These results allude to the possibility of responders and non-responders to SP supplementation, which has been previously described by West et al. (2012). West et al. (2012), noted that changes in serum phosphate levels were correlated with changes in aerobic capacity between a SP and placebo trial and suggested that possibly only certain individuals responded positively to SP supplementation. Individuals should therefore test for the effectiveness of this supplement prior to its use in competition. Furthermore, other studies have also referred to the concept of responders and non-responders in respect to the effects of proposed ergogenic aids on exercise performance. Specifically, when investigating the effects of 5-6 weeks of 4.8 g·day⁻¹ of beta-alanine supplementation on carnosine levels, Baguet et al. (2009) categorized individuals as either high or low responders to beta-alanine supplementation. While the results of the current study support the possibility of responders and non-responders to SP supplementation, correlation analysis of the change in serum phosphate pre and post loading with the change in TT completion trials between the SP and placebo trials did not show significance. However, serum phosphate levels may not be the best indicator for assessing the effect of SP loading on exercise performance (Kreider, 1999). A better measure may be 2,3-DPG concentration, where increases in this substrate are proposed to improve endurance performance. However, due to financial constraints, 2,3-DPG was unable to be measured in the current study, suggesting that this aspect should be considered in future studies.

An unexpected outcome of the current study was that time trial performance and average power output for the 25 mg·kg⁻¹ of FFM trial were ‘likely to be detrimental’ when compared with the placebo and the 75 mg·kg⁻¹ of FFM trials, as determined by qualitative statistical analysis. These results were unexpected because theoretically the explanation of the mechanisms on which the ergogenic benefits of sodium phosphate are based implies that any increase in phosphate levels should enhance these mechanisms and consequently improve exercise performance (Kreider, 1992). While the 25 mg·kg⁻¹ of FFM dose of sodium phosphate was much lower than doses that have previously shown an ergogenic effect on exercise performance (Czuba et al., 2009), this still does not explain the impairment found in exercise performance compared with the placebo and the 75 mg·kg⁻¹ of FFM trials. Furthermore, similar results for pre and post exercise variables relating to serum phosphate and blood lactate concentrations and RPE between the placebo, the 75 and the 25 mg·kg⁻¹ of FFM loading trials do not provide any further evidence to explain this result. Finally, it is unlikely that these results are a consequence of a small cohort due to this study meeting the required number of participants determined by power analysis. Consequently, it is unclear as to why this result occurred.

Conclusion

In summary the present study found no benefit of a 25, 50 or 75 mg·kg⁻¹ of FFM dose of SP on 500 kJ (119.5 Kcal) TT cycle performance in females of varying fitness levels. However, due to the possibility of individual responders to either the 50 or 75 mg·kg⁻¹ of FFM loading protocols, competitive cyclists should trial these doses prior to competition.

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Key points

• No significant benefit of a 25, 50 or 75 mg·kg⁻¹ of FFM dose of sodium phosphate was found on 500 kJ (119.5 Kcal) TT cycle performance in female cyclists.

• Females of differing fitness levels responded similarly to sodium phosphate supplementation.

• Due to the possibility of individual responders to either the 50 or 75 mg·kg⁻¹ of FFM loading protocols, competitive cyclists should trial these doses prior to competition.

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Research interests
Physiological demands of team sports, especially the football codes. Performance analysis in team sports. Environmental physiological demands (heat/humidity, plus altitude). Nutrition and supplements and exercise performance. Sand training vs. firmer surfaces

Kym GUELFI
Employment
Associate Professor at the School of Sport Science, Exercise and Health, the University of Western Australia

Degree
PhD

Research interests
The role of exercise in disease prevention and management (particular obesity and diabetes), as well as the relationship between exercise, appetite and food intake

Lars McNAUGHTON
Employment
Professor and Associate Head of Sport and Exercise Sciences at Edge Hill University

Degree
PhD

Research interests

Acid base balance in exercise

Christopher Buck
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