Title: Phosphate Loading does not improve 30-km cycling time-trial performance in trained cyclists.

Name(s) of Author(s): Harry Pope, Max Davis, M. Begona Delgado-Charro, Oliver J. Peacock, Javier Gonzalez, James A. Betts

Institutional Affiliation:

Centre for Nutrition, Exercise & Metabolism

Department for Health

University of Bath

Bath, UK.

Running Head: phosphate and exercise performance

Corresponding Author and DoIs:

JAB is an investigator on research grants funded by BBSRC, MRC, British Heart Foundation, Rare Disease Foundation, EU Hydration Institute, GlaxoSmithKline, Nestlé, Lucozade Ribena Suntory, ARLA foods, Kennis Centrum Suiker and Salus Optima (L3M Technologies Ltd); has

completed paid consultancy for PepsiCo, Kellogg's, SVGC and Salus Optima (L3M Technologies Ltd); is Company Director of Metabolic Solutions Ltd; receives an annual honorarium as a member of the academic advisory board for the International Olympic Committee Diploma in Sports Nutrition; and receives an annual stipend as Editor-in Chief of *International Journal of Sport Nutrition & Exercise Metabolism.* J.T.G. is an investigator on research grants funded by BBSRC, MRC, British Heart Foundation, The Rank Prize Funds, The European Society for Clinical Nutrition and Metabolism (ESPEN), Lucozade Ribena Suntory, ARLA Foods Ingredients, Kenniscentrum Suiker and Voeding and Clasado Biosciences; and has completed paid consultancy for PepsiCo and SVGC.

Abstract

Phosphate is integral to numerous metabolic processes, several of which strongly predict exercise performance (i.e. cardiac function, oxygen transport and oxidative metabolism). Evidence regarding phosphate loading is limited and equivocal, at least partly because studies have examined sodium phosphate supplements of varied molar mass (e.g. mono/di/tribasic, dodecahydrate), thus delivering highly variable absolute quantities of phosphate. Within a randomized cross-over design and in a single-blind manner, sixteen well-trained cyclists (age 38 ± 16 y, mass 74.3 ± 10.8 kg, training 340 ± 171 min·week^{-1}; mean ± SD) ingested either 3.5 g·d^{-1} of dibasic sodium phosphate (Na_2HPO_4: 24.7 mmol·d^{-1} phosphate; 49.4 mmol·d^{-1} sodium) or a sodium chloride placebo (NaCl: 49.4 mmol·d^{-1} sodium and chloride) for 4 days prior to each of two 30-km time-trials, separated by a wash-out interval of 14 days. There was no evidence of any ergogenic benefit associated with phosphate loading. Time-to-complete the 30-km time trial did not differ following ingestion of sodium phosphate and sodium chloride (3059 ± 531 s *vs* 2995 ± 467 s). Accordingly, neither absolute mean power output (221 ± 48 *vs* 226 ± 48 W) nor relative mean power output (3.02 ± 0.78 *vs* 3.08 ± 0.71 W·kg^{-1}) differed meaningfully between the respective intervention and placebo conditions. Measures of cardiovascular strain and ratings of perceived exertion (RPE) were very closely matched between treatments (i.e. average heart rate 161 ± 11 *vs* 159 ± 12 bpm; Δ 2 bpm; and RPE 18 [14-20] *vs* 17 [14-20] units). In conclusion, supplementing with relatively high absolute doses of phosphate (i.e. >10 mmol daily for 4 days) exerted no ergogenic effects on trained cyclists completing 30-km time-trials.

Keywords: Disodium hydrogen phosphate; exercise; oxygen; nutrition

Introduction

Phosphorous (P) is one of only six basic elements that comprise >99% of human body mass and occurs naturally in the form of phosphate within all living organisms (Emsley 1998). Phosphates are integral components of anatomical structures at many levels, forming the backbone of DNA within cells, the phospholipid membranes surrounding cells, and are a vital constituent of the skeletal system. Moreover, phosphate serves physiological functions across numerous tissues and organ systems, often involving the phosphorylation of proteins as a mechanism of metabolic regulation – with no compound more central to energy metabolism than that formed when phosphate groups interact with adenine and ribose (i.e. adenosine triphosphate; ATP; Heffernan 2019).

In light of the fundamental importance of phosphorous throughout physiology, it is remarkable that so little research has explored the potential for dietary phosphate supplementation to improve physical function (i.e. performance). Almost 40 years ago, Cade *et al* (1984) first reported that phosphate loading for 4 days can increase both maximal oxygen uptake by 6-12% and resting erythrocyte 2,3-diphophoglycerate (RBC 2,3-DPG) from 13.00 to 13.92 mg/Hb - which would be expected to improve tissue oxygen delivery via a reduction in haemoglobin oxygen affinity, promoting dissociation (see MacDonald, 1977 for a review). Notably, this role of phosphate in relation to exercise metabolism is distinct from the more recent implication of inorganic phosphate accumulation in the fatigue process (Westerblad *et al*, 2002). Whilst several other subsequent studies failed to replicate these initial observations (Duffy & Conlee 1986; Bredle *et al* 1988; Stewart *et al* 1990), other early investigations did echo the increases in maximal oxygen uptake, cardiac function, and blood haemoglobin content, which

translated to improvements in both 5-mile run time and 40-km cycling time-trials (Kreider *et al* 1990; Kreider *et al* 1992). The fact that such effects have not been consistently associated with changes in RBC 2,3-DPG may implicate other potential mechanisms of action. One possibility involves an increase in cardiac output resulting from greater ATP availability in the myocardium. This increases contractility and generates a higher stroke volumes for a given heart rate (O'Connor *et al* 1977). Moreover, inorganic phosphate has postulated direct effects on glycolysis and oxidative phosphorylation (Chasiotis, 1988; Lichtman *et al* 1971). Theoretically, an increase in the concentration of intra- and extra-cellular inorganic phosphate would improve the rate of ATP resynthesis and oxidative energy metabolism (Kreider *et al*, 1992).

There has been a resurgence of interest in this area over the past 10 years, with some studies reporting improvements in tests of both exercise capacity (i.e. maximal oxygen uptake; (Brewer *et al* 2013; Czuba *et al* 2008; Czuba *et al* 2009)) and exercise performance (i.e. 10-mile cycling time-trial and repeated sprints; Folland *et al* 2008; Brewer *et al* 2015). Again, however, at least as many papers show no clear effects of phosphate loading when using similar tests of either aerobic capacity, repeated sprint ability or high-intensity time-trials of *circa* 4-, 10- and 20-km (Brennan and Connolly 2001; Brewer *et al* 2014; Brown and Glaister 2019; Kopec *et al* 2016; Ploszczyca *et al* 2022; West *et al* 2012). The reasons for these seemingly inconsistent findings are not immediately apparent and many authors have discounted the possibility of dose-dependent effects on the basis that most studies have adopted very similar supplementation protocols – typically prescribing *circa* 3-5 $g \cdot d^{-1}$ of phosphate supplements for 4-6 days. However, the specific source of phosphate prescribed is rarely considered, with past studies reporting use of mono-, di- and tri-basic sodium phosphate, dodecahydrates and dicalcium

phosphate (or failing to identify the source). These different forms of phosphate have quite varied molar masses and therefore the seemingly similar dose of weighed supplement in fact delivers highly variable absolute chemical amounts of phosphate (i.e. studies have typically prescribed 4-24 $mmol \cdot d^{-1}$).

When re-evaluating the balance of available data with due consideration of supplement source (where reported) and therefore molar masses, it appears that higher absolute doses of phosphate are associated with superior mean power outputs during time-trials, with the largest and most consistent effects on performance apparent when the quantity of phosphate ingested exceeds 10 $mmol \cdot d^{-1}$ (e.g. Keider *et al* 1992). Moreover, the most consistent, and largest, ergogenic effects have been observed during longer compared to shorter tests (e.g. Kreider *et al* 1992 ES: 1.22, Buck *et al* 0.031) and so we have chosen to perform a 30-km time-trial. A 30-km time-trial also provides ecological validity as it is a commonly cycled distance during time-trials.

The present study will therefore examine the effects of phosphate loading for 4 days using a relatively large absolute dose of dibasic sodium phosphate (24.7 $mmol \cdot d^{-1}$ phosphate) in relation to 30-km cycling time-trial performance.

Methodology

Approach to the Problem

Consistent with the rationale set-out in the introduction, the present study provided a high absolute dose of phosphate – *i.e.* previous research has provided absolute doses in the range 4-24 $mmol \cdot d^{-1}$, whereas we slightly exceeded the upper-end of that range (24.7 $mmol \cdot d^{-1}$). This was consumed in the form of dibasic sodium phosphate, both because this equates to ingestion of a feasible amount of powdered supplement (3.5 g/day) and because (unlike other forms of sodium phosphate seemingly ingested in other studies) food-grade dibasic sodium phosphate was available. Nonetheless, the product used in this study is a laboratory-grade chemical and we tested for purity in-house via mass spectrometry (spectra were consistent with the powder containing only the specified ingredients).

To achieve the prescribed phosphate intake of 24.7 $mmol \cdot d^{-1}$, phosphate loading was sustained for 4 days, with daily supplements divided equally across 4 doses per day, ingested at 4-h intervals during the wake cycle in each 24-h period. The rationale for this supplementation protocol was based on the hypothesis that phosphate incorporation into RBC 2,3-DPG is subject to a negative feedback loop such that acute ingestion of sodium phosphate in high doses can activate renal phosphate clearance and thus attenuate any net increase in endogenous phosphate status (MacDonald 1977). Division of sodium phosphate dibasic provides an additional practical benefit. Consumption of smaller quantities of sodium phosphate dibasic minimizes the potential for gastrointestinal discomfort increasing the feasibility of supplementation.

Sodium chloride was chosen as the placebo to allow for equimolar amounts of sodium to be consumed in both conditions (49.40 $mmol \cdot d^{-1}$), given the known influence of sodium on numerous physiological responses pertinent to exercise (Thomas *et al* 2016; Munson *et al* 2020). Sodium ingestion maintains osmotic pressure and plasma volume which ultimately contributes to reduced fatigue and loss of homeostasis during exercise (Sawka & Montain, 2000). These physiological mechanisms have the potential to translate to ergogenic effects (*e.g.* Del Coso et al., 2016) and are therefore imperative to control should we wish to isolate the influence of phosphate. Additionally, the consumption of chloride has no known ergogenic benefits and therefore is unlikely to influence results.

The required wash-out period following phosphate loading is uncertain, with most previous studies implementing an interval between repeated trials of between 4 and 14 days (Cade *et al* 1984; Kreider *et al* 1990; Kreider *et al* 1992; Schenck *et al* 1991; Stewart *et al* 1990; Tremblay *et al* 19). Cade *et al* (1984) found serum phosphate remained elevated 1 week after loading and suggested that longer washouts may be necessary to avoid carry-over effects. Therefore we required at least 14 days between participants first and second exercise tests.

The present study tested approximately twice as many participants as most studies in this area and only recruited well-trained cyclists (as verified by training diaries). Most previous sodium phosphate research employed relatively small sample sizes ($n<10$) and/or tested untrained participants. This may further account for some of the inconsistencies in the literature due to the combination of low statistical power, low reliability and potentially treatment effects

that may be small and highly variable. Certainly, other authors have hinted at the possibility of responders and non-responders to phosphate loading (Płoszczyca *et al* 2022). Whilst the necessary replicated controlled trials to test this hypothesis have yet to be conducted (Atkinson *et al* 2015), this suggestion does reflect the fact that responses are at least quite variable and so may warrant larger and/or more homogenously trained participants as are presented here.

Finally, it should be noted that data collection for this study was completed during the peak of the first COVID-19 pandemic in 2021 and thus required field-based trials. This presents several threats to internal validity by limiting some laboratory controls and standardization, but the field-based nature of the work also provides ecological validity to results.

Participants and Sample Size Estimations

Eighteen participants began the study and sixteen participants (males $n = 15$; females $n = 1$; see **Table 1** for characteristics) completed the protocol. Participants were self-reported trained cyclists and voluntarily participated in the study following recruitment from local cycling and triathlon clubs. Exclusion criteria were: aged <18 years; <60 kg body mass, habitual smoker within the past five years, a history of any metabolic or respiratory disease, BMI >30 kg/m^2, and the use of nutritional supplements within 2 months prior to testing. The mean change in individual participant's body mass across trials was ± 0.2 kg suggesting participants were weight stable (0.27% of mean body mass). Ethical approval was granted by The University of Bath Research Ethics Approval Committee for Health (16/10/2020; MSES 19/20-020).

Table 1. Participant characteristics

n	16
Age (years)	38 ± 16
Height (cm)	175.2 ± 6.6
Mass (kg)	74.3 ± 10.8
Training (hours·week^{-1})	5.67 ± 2.85

Data are means ± SD.

Study Design

A repeated-measures, crossover, randomized, single-blind design was implemented as recommended by Betts *et al* (2009) for nutritional research. Simple randomisation was employed, and an equal number of participants were allocated to each condition. A washout period of fourteen days (Cade *et al* 1984) preceded the subsequent supplementation period to minimise any crossover effects from sodium phosphate dibasic. As data collection took place in the peak of the COVID-19 pandemic, the only method of data collection involved the implementation of a field-based experimental design as opposed to the originally planned laboratory setting. Participants conducted the experimental protocol (below) remotely after receiving strict instruction from the experimenters. Protocol is depicted in **Figure 1**.

Data on time-trial completion time, power output, heart rate, and rating of perceived exertion (RPE) were collected. Due to the remote nature of the study, invasive techniques, such as collection of blood samples, were not feasible.

Experimental Protocol

Participants completed two experimental trials separated by a fourteen-day washout period (Cade *et al* 1984) in a single-blinded manner. These trials followed four consecutive days consuming sodium phosphate dibasic (Sigma-Aldrich, UK; **Table 2**) or a sodium-matched placebo (sodium chloride, SAXA, UK; **Table 2**). All supplements were prepared in house using electronic scales (Mukaet, China) and were accurate to 0.001 g. Doses of similar magnitude have elicited the greatest effect sizes in the available literature (Cade *et al* 1984; Kreider *et al* 1992). To minimise gastrointestinal discomfort, supplements (powdered form) were consumed in four doses alongside 300 ml water and food, separated by 4 hours. Gastrointestinal discomfort was assessed following the fourth day of supplementation using a qualitative questionnaire (see Appendix A for post-testing questionnaire).

Table 2. Nutritional supplement information

Nutritional supplement (trial)	Chemical formula	Molecular weight ($g \cdot mol^{-1}$)	Absolute dose ($g \cdot d^{-1}$)	Absolute phosphate dose ($mmol \cdot d^{-1}$)	Absolute sodium dose ($mmol \cdot d^{-1}$)
Sodium phosphate dibasic	Na_2HPO_4	142.0	3.5	24.7	49.4
Sodium chloride (PLA)	NaCl	58.4	2.8	N/A	49.4

Upon commencement of both trials, height (via tape measure) and weight (via electronic scales) were self-recorded. Following this, participants performed a maximal 30-km cycling time-trial using their preferred home set-up. Each participant maintained an identical set up between conditions which had previously been used throughout their training; this included bicycle, turbo trainer, saddle height, handlebar position, cycling shoes, and cooling device (or absence of cooling device n = 4). Equipment was calibrated following manufacturer instructions and set to most closely replicate a flat, outdoor environment. Participants' personal road bicycle and turbo-trainer were used (smart n = 15; standard power meter n =1; see Appendix B for a list of power meters). Similar set ups have been found to have error rates close to that of laboratory-based cycle ergometers (Peiffer and Losco, 2011), acceptable test-retest reliability (CV: 2.6%; Peiffer and Losco, 2011), and adequate level of reliability for the assessment of cycling performance (Hoon *et al* 2016). Speed was determined by several factors including power output and rider weight using Zwift software (Zwift, United States). Participants were free to view speed, power, and distance throughout the time-trial. Riders also monitored total trial distance

248 through the use of Garmins and other cycling computers. Immediately following the time-trial,
249 participants sent all relevant information to researchers. Throughout each time-trial, participants
250 were free to consume fluids ad libitum and were permitted to change cadence and gears ad
251 libitum. Time-trials were performed at the same time of day and in a fed state which was
252 standardised for the second trial. All trials were performed free from verbal encouragement,
253 caloric intake, and music.

254

255 Immediately post time-trial, completion time, average power output, and RPE were
256 recorded by participants. Overall RPE was measured using a 6-20 scale (Borg, 1982). Heart rate
257 data were collected continuously throughout each trial via telemetry. Wireless telemetry has been
258 shown to have high accuracy, resembling the readings of an electrocardiogram most closely
259 (Pasadyn *et al* 2019). Following the completion of both trials, a post-testing questionnaire was
260 administered to assess the effectiveness of blinding methods. Nine participants successfully
261 identified trial condition.

262

263 Participants were required to perform both trials at the same time of day to account for
264 circadian variation and 'normal' diet and 'normal' training volume were maintained throughout
265 the study. Participants recorded their training and diet for 24-hours immediately prior to the first
266 time-trial and replicated this for the 24-hours preceding the second. Alcohol consumption within
267 the 24-hours prior to trials was prohibited. Caffeine was allowed providing consistency between
268 trials.

269

Statistical Analyses

Descriptive statistics were conducted to summarise data. A Shapiro-Wilk test determined the appropriate measures for central tendency and distribution, and unless stated otherwise, data are presented as (mean ± SD, mean difference (Δ), ± normalised confidence intervals (nCIs)). The effect of treatment on outcome variables was delineated through nCIs. Means whose nCIs overlap by no more than half of one side of an interval would typically be deemed statistically significant ($p \leq 0.05$) through traditional t-testing. The smallest difference in performance deemed to be worthwhile was derived from the recent paper by Borg *et al* (2018), which concluded that a meaningful change in cycling within the context of this experiment would be in the range of 4.1-4.9 %. This roughly equates to a difference in absolute mean power of at least 10 W (〜0.1 W/kg) and therefore approximately 125-150 seconds difference to complete a 30-km time-trial. Pearson *r* or Spearman *R* were used to explore correlations between normal and non-normally distributed data respectively. Analyses were conducted in IBM SPSS Statistics (version 28.0; IBM, Armonk, NY, USA) and Microsoft Excel (version 16.58; Microsoft, Redmond, WA, USA).

Results

Performance Data

Time-trial completion time was not meaningfully different following the consumption of sodium phosphate dibasic when compared to placebo (3059 ± 531 s; 2995 ± 467 s; Δ 64 s; ± 47 s **Figure 2 a & b**). This is unsurprising as absolute mean power (221 ± 48 W; 226 ± 48; Δ 5 W; ± 7 W **Figure 3 a & b**) and relative mean power (3.02 ± 0.78 $W \cdot kg^{-1}$; 3.07 ± 0.71 $W \cdot kg^{-1}$; Δ 0.05 $W \cdot kg^{-1}$; ± 0.09 $W \cdot kg^{-1}$ **Figure 4 a & b**) remained unchanged following supplementation.

Physiological and Perceptual Data

Heart rate data are summarised in **Figure 5** and RPE and gastrointestinal discomfort in **Table 3**. Ingestion of sodium phosphate dibasic did not affect average heart rate (161 ± 11 bpm; 159 ± 12 bpm; Δ 2 bpm; ± 3 bpm **Figure 5**) or maximum heart rate (176 ± 12; 174 ± 12; Δ 2 bpm; ± 2 bpm **Figure 5**). Similarly, RPE did not differ between conditions (median [range]; 18 [14-20]; 17 [14-20]; median difference [range], 0 [-1, 3]; **Table 3**).

Table 3. Perceptual Responses

	Sodium phosphate dibasic	Placebo
RPE	18 [14-20]	17 [14-20]
Gastrointestinal Discomfort	2	2

Data shown are medians [range] for RPE and total number of participants reporting distress for Gastrointestinal Discomfort

Trial Order Effects

Individual performances are illustrated on figures with first and second trials identified separately to enabled consideration of potential trial order effects for each participant. In summary, the mean time-trial performance was 67 ± 124 s (2.2 %) slower for trial 1 than for trial 2, so is very similar to the 63 ± 126 s (2.1 %) difference between experimental treatments that was considered not to be practically meaningful. We also applied the statistical analysis advocated by Welleck and Blener (2012) to explore possible 2-way interactions between experimental condition and trial sequence, revealing no statistically significant main effects or interactions (all $p \geq 0.2$).

Discussion

The primary aim of the current study was to determine whether sodium phosphate supplementation provides a meaningful improvement to 30-km cycling time-trial performance. Contrary to the hypothesis, phosphate supplementation does not provide any practically worthwhile benefit to 30-km cycling performance when sodium intake is controlled for. This was true for all performance, physiological, and perceptual measures obtained throughout the trials; time-to-completion, mean power output (absolute and relative), heart rate (average and maximum), and RPE all remained unchanged.

Time-trial completion and power output (absolute and relative) were the main performance measures assessed in the present study, and all remained unchanged between trials (i.e. the 2% change observed is less than half the 4-5 % effect considered to be practically meaningful). These results are surprising as, whilst data were far from conclusive, our a prior search of the literature suggested there exists a correlation between absolute sodium phosphate supplementation dose and performance enhancements. Previous studies indicated a positive correlation for time-trial completion ($r_s = 0.44$) and average power output ($r_s = 0.58$) with absolute sodium phosphate dose. Further strengthening this notion was the work of Kreider *et al* (1992). This is currently the only other study to use absolute phosphate doses in excess of 20 $mmol \cdot d^{-1}$, and they reported markedly positive findings (time-trial ES: 1.22; average power ES: 2.90).

As previously stated, research considering sodium phosphate is limited and equivocal. The original findings of Cade *et al* (1984) presented a correlation between increases in 2,3-GDP concentration and improved time-trial performance ($p < 0.01$, $r = 0.81$) following provision of 4 g tribasic sodium phosphate for 4 days. Similarly, Kreider *et al* (1992) reported performance benefits which are, to date, the greatest in magnitude. Following supplementation with 4 g tribasic sodium phosphate for 3 days, stark performance enhancement was seen compared to a glucose control. Completion time for a 40-km time-trial was improved by 210 s (ES: 1.22, $p <$ 0.02) and was accompanied by a large increase in $\dot{V}O_{2max}$ (69.3 to 75.4 $ml \cdot kg^{-1} \cdot min^{-1}$) attributed to the reported increases in 2,3-GDP. However, Kreider *et al* (1992) failed to match conditions for sodium and therefore the impact of phosphate cannot be isolated. Conversely, Brewer *et al* (2013) failed to report any significant improvements on 1,000 kJ (~40-km) time-trial performance following 50 $mg \cdot kg^{-1}$ tribasic sodium phosphate supplementation when compared to a control of sodium chloride and glucose. These findings are not isolated, with several other studies reporting no ergogenic effect of sodium phosphate on time-trial performance or average power (e.g. Buck *et al* 2014; Brown and Glastier, 2019; Brewer *et al* 2014). In fact, at doses of 25 mg/kg/day, Buck *et al* (2014) suggested the effects may even be deleterious. The current study adds to the body of literature which suggests sodium phosphate has no meaningful ergogenic effects.

The previous research which has reported large effect sizes with substantial absolute sodium phosphate doses appear to have limitations. Preliminary findings produced by Cade *et al* (1984) suggested that, when compared to a control, sodium phosphate increased $\dot{V}O_{2max}$ ($p <$ 0.02) and time to exhaustion ($p < 0.05$). However, Cade *et al* (1984) used relatively few

participants and the washout period employed was also insufficient, lasting just 4 days. This presents the potential for a crossover effect from sodium phosphate supplementation. Similarly, the work of Kreider *et al* (1992) had an underpowered sample (n = 6) and used a glucose control. The limitations of previous research could have led to unacceptable type I error and our hypothesis may have been based on invalid and unreliable findings. In fact, to our knowledge, the only previous study to satisfy sample size requirements (n > 15) was that of Brown and Glaister (2019), who, like the present study, reported no effect of sodium phosphate supplementation. More sufficiently powered studies with sodium matched controls are required to provide conclusive evidence for the effects of sodium phosphate.

Additionally, average HR in the current study remained unchanged (sodium phosphate dibasic, 161 ± 11; PLA, 159 ± 12 bpm; Δ 2 bpm). This suggests that sodium phosphate dibasic supplementation does not have the proposed effect on myocardial efficiency. Such findings are concordant with many previous studies (Kreider *et al* 1990; Folland *et al* 2008; Brewer *et al* 2013; 2014; Brown and Glaister, 2019). Unsurprisingly, given time-trial completion, heart rate (average and maximum), and average power output (absolute and relative) all remained unchanged, RPE was similar between trials. Almost all of the current literature agrees with this finding, with the exception of Brown and Glaister (2019), who found increased RPE following sodium phosphate supplementation (mean difference: 0.39; 95% CI: [0.04, 0.73]).

Despite the findings of the present study concluding that sodium phosphate dibasic supplementation has no meaningful performance enhancing effects there are several limitations.

One applicable criticism of the sodium phosphate literature was presented by Buck (2015). It was suggested that body size and composition (i.e. FFM) has not been sufficiently accounted for and this may alter the effect of loading protocols on serum phosphate concentration. As the current study administered a standardised, as opposed to relative, dose of sodium phosphate it could be argued that body mass influenced results. Post-hoc Spearman rank analysis indicated that this was not the case. No correlation between baseline body mass and changes in time-trial completion time (r = 0.04) or absolute mean power output (r = -0.25) were found. This suggests that the standardised dose provided was not the cause of the insignificant findings.

Another limitation to the present study was the lack of a familiarisation trial. As our participants were trained cyclists, we would expect them to have deep-rooted, learned pacing strategies which would negate the need for a familiarisation (Mauger *et al*, 2010). This belief is supported by the literature, with previous studies reporting that, even without a familiarisation trial, time-trial completion and mean power are consistent between cycling time-trials in trained athletes (Sporer and McKenzie, 2007; Zavorsky *et al*, 2007). Indeed, no meaningful order effects were present. Whilst trial two was faster than trial one (3060 ± 539 *vs* 2993 ± 456 s), the difference was just 2.2%. This is remarkably similar to the difference between trials (2.1%) which was deemed insignificant, so it was concluded that order effects did not meaningfully impact the results.

Our participants displayed a wide range of fitness levels, with time-trial completion ranging from 2574 to 4764 s. Individuals with higher maximal oxygen uptake values have

greater 2,3-DPG concentrations (Remes *et al*, 1979), which may limit the ergogenic potential of sodium phosphate (Galloway *et al*, 1996; Tremblay *et al*, 1994). However, a correlation analysis suggested that less fit individuals did not respond any better than fitter individuals (time-trial: r = 0.45, p = 0.08; relative average power: r = 0.33, p = 0.21).

There were other limitations presented by the home-based nature of this study which could provide a threat to internal validity. Invasive data collection (such as blood samples) was not feasible and therefore we cannot be confident the current sodium phosphate dibasic protocol had the desired effects on serum phosphate concentration. Whilst Bremner *et al* (2002) found a 30% increase in plasma phosphate and a 25% increase in 2,3-GDP following sodium phosphate loading, we cannot be sure the present protocol had the same effect. The present study administered a large absolute dose of sodium phosphate dibasic (24.70 $mmol \cdot d^{-1}$), which is, to our knowledge, currently larger than any previous study. However, this could have potentially had an adverse influence on performance. Doses on this scale have been avoided in the literature to date. It has been hypothesised that following the provision of such quantity of sodium phosphate dibasic, a parathyroid response may be initiated (Kreider, 1999). In a negative feedback loop, the large rise in serum phosphate concentrations can lead to enhanced clearance by the kidneys, maintaining serum phosphate homeostasis. Whilst it therefore remains a possibility that the present intervention may have been excessive (i.e. eliciting the aforementioned negative feedback loop), the fact remains that previous research using this same absolute dose have detected meaningful effects. Future research should ensure serum phosphate concentration is measured to allow confidence in the efficacy of loading protocol.

As serum phosphate homeostasis is tightly controlled by several mechanisms, there may be reason to investigate the influence of co-ingestion with other ergogenic aids. One regulatory of serum phosphate concentration is blood pH (MacDonald, 1977). In alkalosis, the rate of glycolysis is known to increase which inhibits the action of the enzyme DPG phosphatase. The overall response is an increase in the concentration of 2,3-DPG, potentially increasing the potency of the action of sodium phosphate in alkaline conditions. There appears to be reason to suggest sodium phosphate supplementation may benefit from an ergogenic aid which would increase the pH of the blood. Sodium bicarbonate, one of the most frequently studied and utilised supplements, is believed to do just this. Future research should investigate whether sodium phosphate and sodium bicarbonate co-ingestion could enhance the action of sodium phosphate supplementation.

In conclusion, 4 days of sodium phosphate dibasic supplementation (24.70 $mmol \cdot d^{-1}$) does not improve any measures of 30-km cycling time-trial performance when compared to sodium chloride in trained cyclists. The findings indicates that even at high doses, sodium phosphate supplementation fails to manifest in an ergogenic benefit when sodium is controlled for.

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Figure Legends

Figure 1. Protocol Schematic.

Figure 2. Mean ± nCI 30-km time-trial duration presented as bars. Individually paired data are presented as lines. Circles indicate the first trial; crosses indicate the second.

Figure 3. Mean ± nCI 30-km absolute mean power presented as bars. Individually paired data are presented as lines. Circles indicate the first trial; crosses indicate the second.

Figure 4. Mean ± nCI 30-km relative mean power presented as bars. Individually paired data are presented as lines. Circles indicate the first trial; crosses indicate the second.

Figure 5. Mean ± nCI average (dark bars) and maximum (light bars) heart rate. Individually paired data are presented as lines. Circles indicate the first trial; crosses indicate the second.

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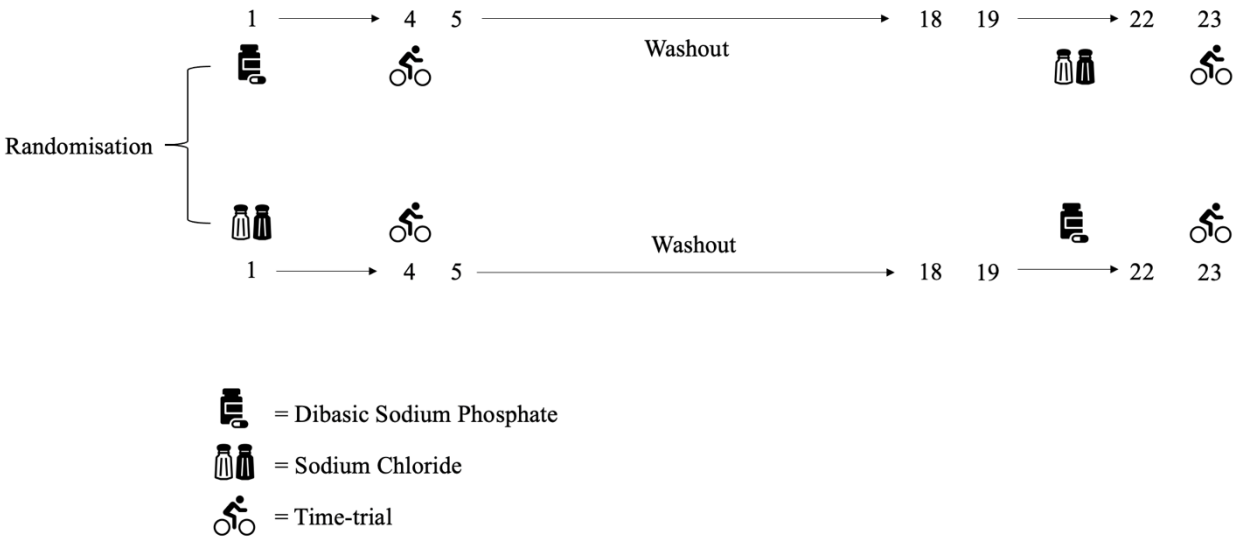

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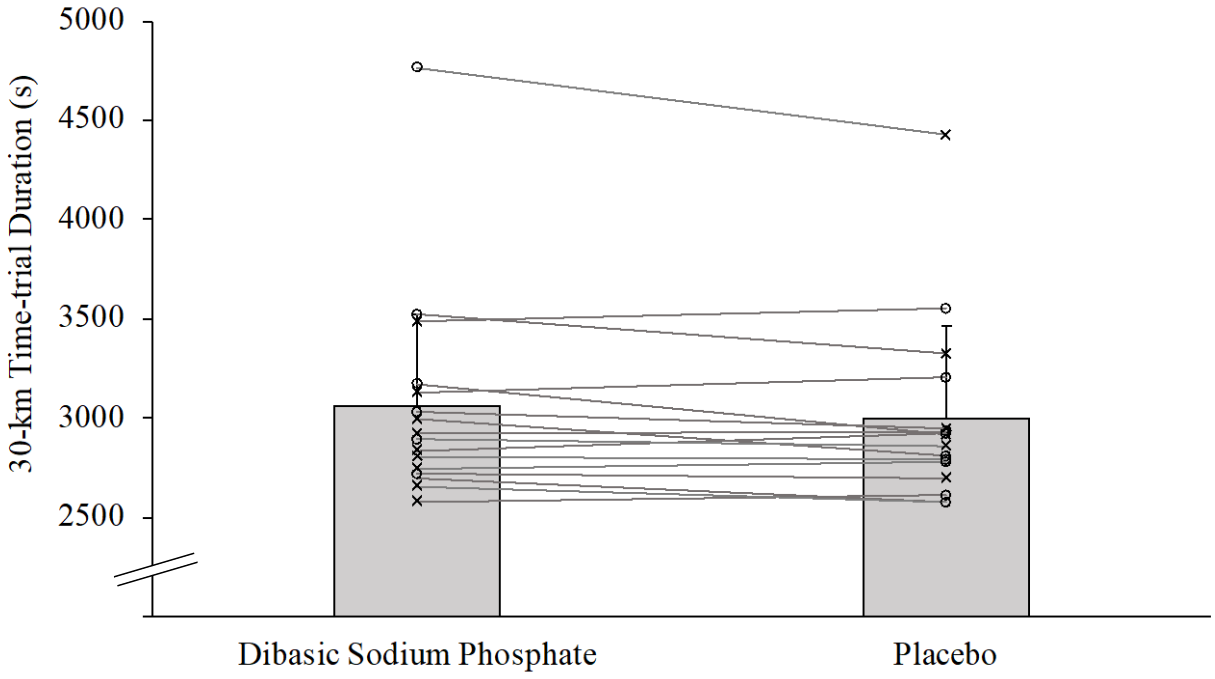
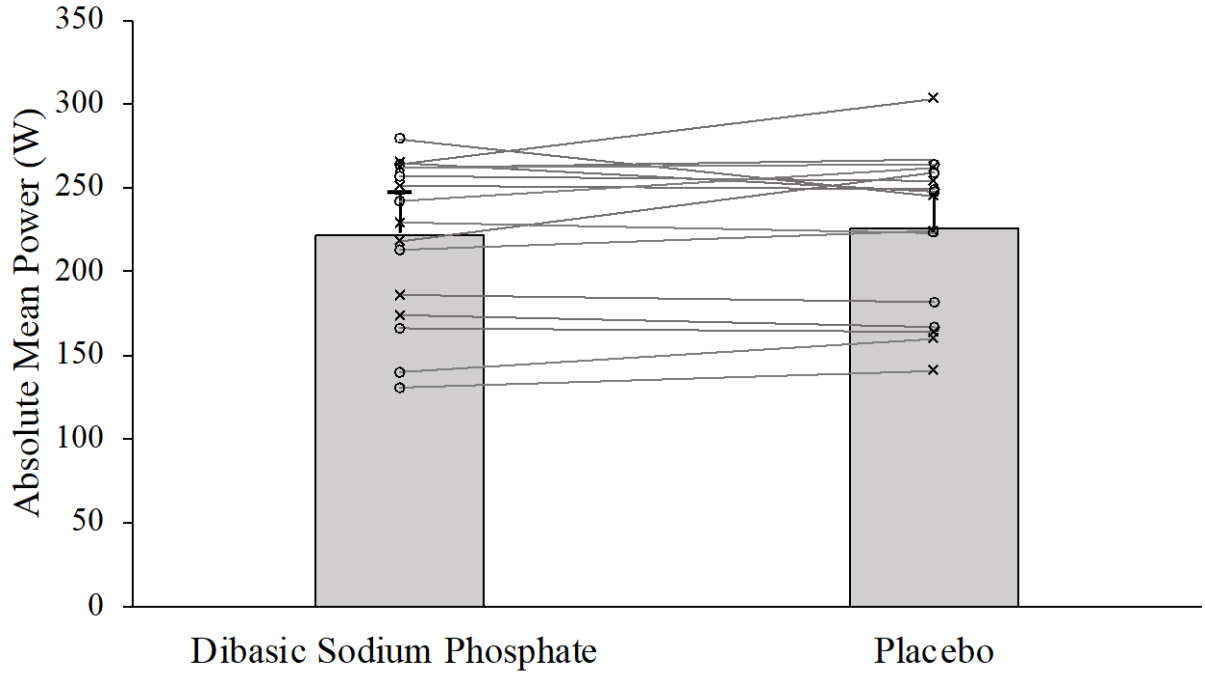
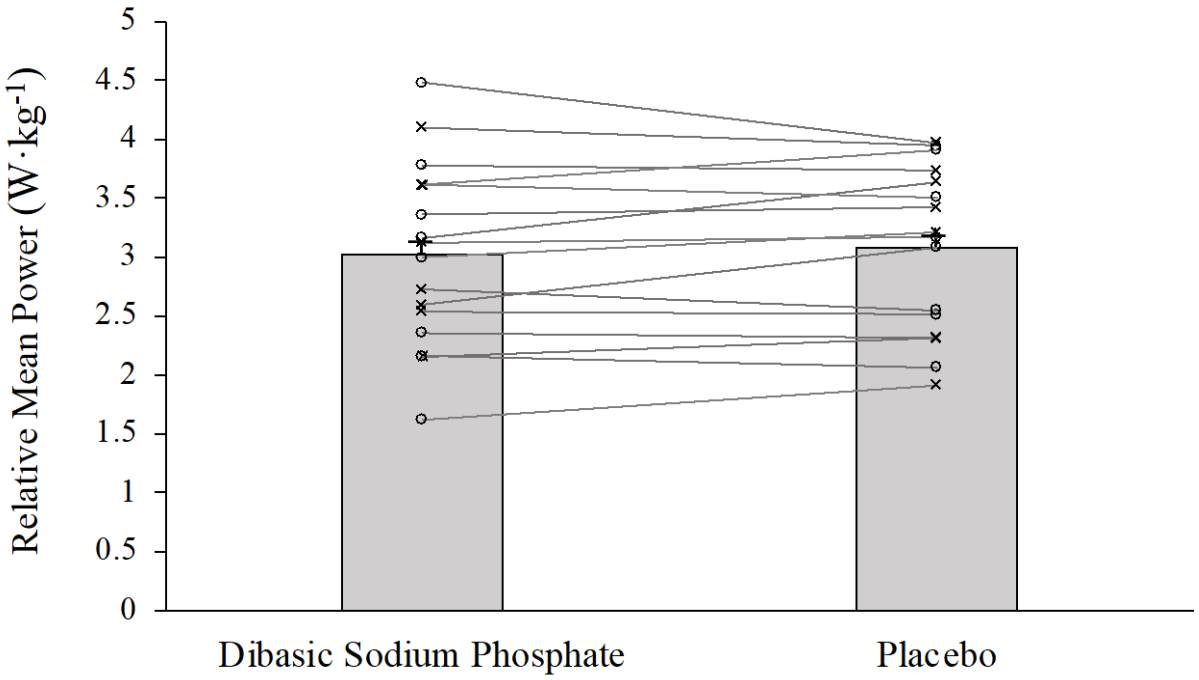
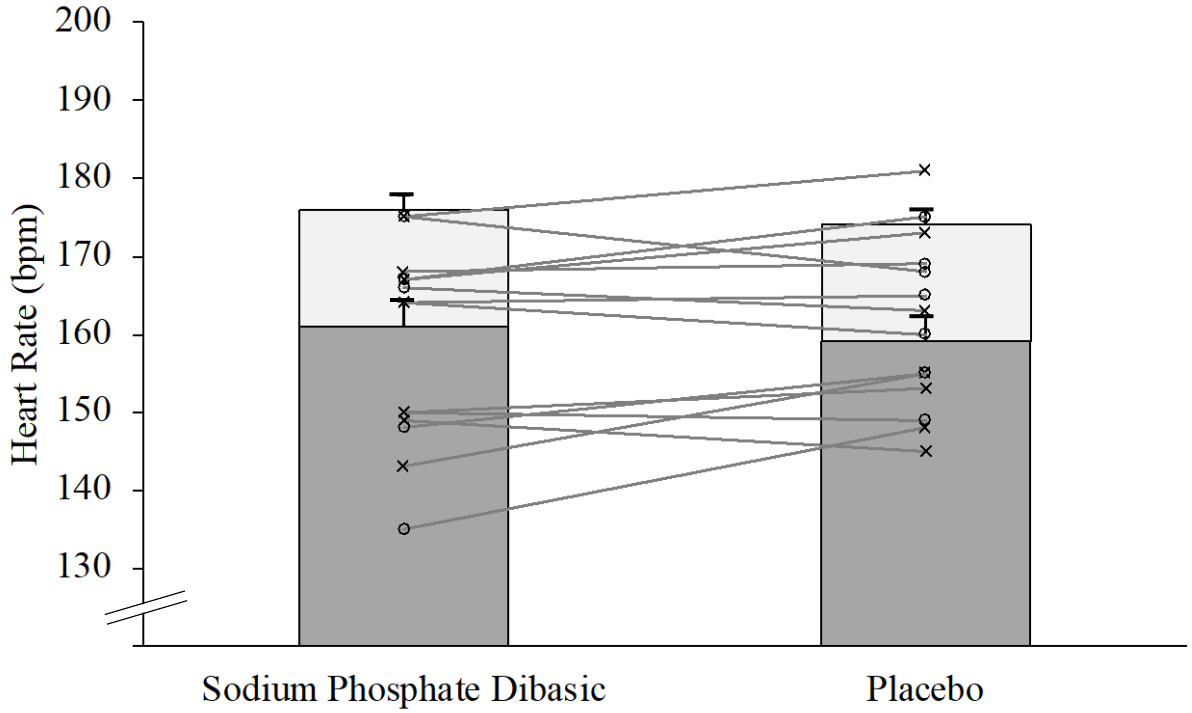

30-km Time-trial Duration (s)
5000
4500
4000
3500
3000
2500
Dibasic Sodium Phosphate
Placebo


Absolute Mean Power (W)
350
300
250
200
150
100
50
0
Dibasic Sodium Phosphate
Placebo


Relative Mean Power (W·kg-1)
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
Dibasic Sodium Phosphate
Placebo


Heart Rate (bpm)
200
190
180
170
160
150
140
130
Sodium Phosphate Dibasic
Placebo

Appendix A: Post-testing Questionnaire

1. Could you tell the difference between the two supplementation trials? Yes or no? *(If yes, please answer question 2, if no, please advance to question 3).*

2. Please list which supplement you believe you took for each of your two trials below. *The supplements provided were sodium phosphate dibasic and sodium chloride.*

3. Did you perceive any performance benefits from either of the two supplements during the two trials? Yes or no? *(If yes, please answer question 4 and 5, if no, please advance to question 6).*

4. In which trial did you perceive the supplement to provide a performance benefit. *Please mark below.*

5. Please list the perceived benefits to performance below.

6. Did you experience any negative side effects during the supplementation or exercise testing phases of either trial? Yes or no? *(if yes, please answer question 7 and 8, if no, please ignore questions 7 and 8).*

7. Within which trials did you experience these negative side effects?

8. Please list below the negative side effects experienced.

Appendix B – Power Meters

Table 4. Equipment (turbo trainers and power meters) used during time-trial performance testing

Turbo trainer model (attached power meter)	No. of participants using turbo trainer (No. of participants using power meter)
Wahoo KICKR CORE Smart Trainer	5
Wahoo KICKR Snap Smart Trainer	1
Tacx Vortex Smart Trainer (Quarq D-zero)	2 (1)
Tacx Neo 2T Smart Trainer	1
Tacx Flux 2 Smart Trainer	1
Elite Direto Smart Turbo Trainer (Garmin Vector 3)	1 (1)
Elite Suito Direct Drive Smart Turbo Trainer	1
Elite Novo Smart Turbo Trainer	1
Elite Qubo Digital Smart Trainer	1
Wattbike Atom	1
Bikemate Indoor Trainer (Garmin Vector 3)	1 (1)