The Effect of 6 Days of Sodium Phosphate Supplementation on Appetite, Energy Intake, and Aerobic Capacity in Trained Men and Women

Jessica S. West, Tom Ayton, Karen E. Wallman, and Kym J. Guelfi

Ingestion of an acute dose of phosphate has been shown to attenuate energy intake in the subsequent meal. This raises the question of whether the practice of phosphate supplementation over a number of days by athletes to enhance performance also influences energy intake. This study investigated the effect of 6 d of phosphate supplementation on appetite and energy intake, as well as aerobic capacity, in trained individuals. Twenty participants completed two 6-d phases of supplementation with either sodium phosphate (50 mg/kg of fat-free mass per day) or a placebo in a double-blinded, counterbalanced design. On Days 1, 2, and 6 of supplementation, a laboratory meal was provided to assess appetite and ad libitum energy intake. All other food and drink consumed during each supplementation phase were recorded in a food diary. After the 6 d of supplementation, peak aerobic capacity (VO_{peak}) was assessed. There was no difference in energy intake at the laboratory meal after an acute dose (i.e., on Day 1; placebo 2,471 ± 919 kJ, phosphate 2,353 ± 987 kJ; p = .385) or prolonged supplementation with sodium phosphate (p = .581) compared with placebo. Likewise, there was no difference in VO_{peak} with phosphate supplementation (placebo 52.6 ± 5.2 ml · kg^{-1} · min^{-1}, phosphate 53.3 ± 6.1 ml · kg^{-1} · min^{-1}; p = .483). In summary, 6 d of sodium phosphate supplementation does not appear to influence energy intake. Therefore, athletes supplementing with sodium phosphate can do so without hindering their nutritional status. However, given that phosphate supplementation failed to improve aerobic capacity, the ergogenic benefit of this supplement remains questionable.

**Keywords**: food intake, nutritional supplementation, maximal oxygen uptake

Phosphate is abundant in the human diet and can be found in many dairy products, meats, cereals, and highly processed fast foods (Uribarri & Calvo, 2003). In the body, phosphate has a number of important roles due to its reversible binding to adenosine diphosphate, creatine, glucose, vitamins, and second messengers in the cell (Berner & Shike, 1988). In particular, phosphate facilitates the release of oxygen from hemoglobin (Benesch & Benesch, 1969), acts as an intracellular buffer, and is central to energy production for muscle contraction and other metabolic processes based on its contribution to the structure and function of adenosine triphosphate (ATP; Berner & Shike, 1988). Given these important roles of phosphate in the body, additional oral consumption of phosphate has been used by athletes to enhance performance, particularly in events relying on aerobic metabolism. This typically takes the form of ingestion of 3–4 g of sodium phosphate per day for a period of 3–6 days. Ergogenic benefits after such supplementation protocols include increased maximal aerobic capacity (VO_{max}; Cade et al., 1984; Czuba, Zacaj, Poprzecki, Cholewa, & Woska, 2009; Kreider, Miller, Williams, Somma, & Nasser, 1990; Stewart, McNaughton, Davies, & Tristram, 1990), increased anaerobic threshold (Cade et al., 1984; Kreider et al., 1992), and improved cycling time-trial performance (Folland, Stern, & Brickley, 2008; Kreider et al., 1992). However, it is important to note that not all studies have observed ergogenic benefits after sodium phosphate supplementation (Ahlberg et al., 1986; Brennan & Connolly, 2001).

Although sodium phosphate may be a useful ergogenic aid to exercise performance, there is recent evidence to suggest that additional phosphate intake may also influence appetite. Obeid, Dimachkie, and Hlais (2010) provided healthy men and women with a liquid solution of water, sucrose, fructose, or glucose, either with or without the addition of 500 mg of phosphorus (as a mixture of potassium and sodium phosphate). Participants were then required to eat ad libitum from a lunch of pizza and water 80 min after consuming the liquid solution. The researchers observed a 25–35% decrease in energy intake from the lunch meal after consumption of the solutions with added phosphate, with the precise reduction depending on the type of liquid solution ingested (i.e., water, sucrose, fructose, or glucose). Although the exact mechanism for the reduction in energy intake resulting from the acute increase in phosphorus consumption was not examined, Obeid et al. suggested that the added phosphate may...
have influenced hepatic ATP status, resulting in afferent signals transmitted to the central nervous system, which in turn may have influenced appetite and acute energy intake. Support for this hypothesis comes from studies demonstrating a reduction in hepatic ATP and an associated increase in energy intake after the administration of substrates such as 2,5 anhydro-D-mannitol (Rawson & Friedman, 1994) or methyl palmoixirate (Friedman, Harris, Ji, Ramirez, & Tordoff, 1999). Of interest, the effect of 2,5 anhydro-D-mannitol on hepatic ATP status and food intake has been shown to be attenuated with the administration of exogenous sodium phosphate (Rawson & Friedman, 1994).

Given that ingestion of an acute dose of phosphate has been shown to reduce energy intake in the subsequent meal, this raises the question of whether the practice of phosphate supplementation over a number of days by athletes also influences appetite and energy intake. This may have implications for athletes who are repeatedly phosphate loading to ensure that their energy intake is sufficient to support optimal exercise performance. Therefore, the aim of the current study was to investigate whether the practice of sodium phosphate supplementation (50 mg/kg of fat-free mass per day) affects appetite and energy intake both acutely (i.e., after a single dose) and in response to short-term use (6 days) in trained individuals. A secondary aim was to confirm the previously observed benefits of sodium phosphate supplementation for peak aerobic capacity (VO2peak).

Methods

Participants

Twenty-two healthy, moderately trained men (n = 12) and women (n = 10) were initially recruited to participate in this study. Two participants withdrew during the first supplementation phase due to an unrelated muscle injury and gastrointestinal distress. Therefore, 20 participants (11 men, 9 women) completed the entire experimental protocol (age 23 ± 3 years, body-mass index 22.0 ± 2.9 kg/m², VO2peak 52.6 ± 5.2 ml · kg⁻¹ · min⁻¹). Participants were not taking any supplements or medication that might influence appetite or exercise performance. All women had a regular monthly menstrual cycle, and 1 was on the oral contraceptive pill. The procedures were approved by the institutional human research ethics committee, with written informed consent obtained from all participants before testing commenced.

Experimental Design

Each participant came to the laboratory for one initial session of baseline data collection and familiarization. After this, each completed two supplementation periods with either sodium phosphate or placebo, each continuing for 6 days, in a double-blinded counterbalanced design. A minimum washout of 2 weeks was enforced between supplementation periods (Cade et al., 1984; Folland et al., 2008; Kreider et al., 1990). Female participants supplemented in the follicular phase of the menstrual cycle (supplementation commenced on Day 3 ± 2 of the menstrual cycle) to minimize any additional influence of the phase of menstrual cycle on appetite (Brennan et al., 2009) or aerobic performance (Rechichi, Dawson, & Goodman, 2009), with this resulting in an approximate 3-week washout period between trials. To assess the effect of supplementation on energy intake, a laboratory breakfast was provided on Days 1, 2, and 6 of supplementation, from which ad libitum energy intake was determined. Energy intake from all other food and drink intake during each supplementation period was determined from self-recorded food diaries. To assess the effect of supplementation on VO2peak, an incremental treadmill protocol was performed at the end of each supplementation period (Day 7). After the completion of both trials, participants were asked to report the phase in which they thought they consumed sodium phosphate and why.

Familiarization Session

Before the first supplementation period, participants came to the laboratory for a familiarization session. During this session, body composition was assessed using dual-energy X-ray absorptiometry (Lunar Prodigy, encore 2004, GE Medical Systems, Madison, WI). This procedure was necessary to determine the amount of sodium phosphate to be administered in mg/kg of fat-free mass, in accordance with previous research (Czuba et al., 2009). In addition, a venous blood sample was taken from an antecubital vein for the determination of serum phosphate concentration. Participants were then familiarized with the incremental treadmill protocol to be completed at the end of each supplementation period by running on the treadmill while wearing a nose clip and breathing into a mouthpiece for the collection of expired air. Finally, each participant was presented with a laboratory familiarization breakfast to ensure that the meal was to his or her liking and to minimize novelty on exposure in the first experimental trial (Halse, Wallman, & Guelfi, 2011).

Supplementation

After the familiarization session, each participant was provided with gelatin capsules (The Melbourne Food Depot, East Brunswick, Victoria Australia) filled with either sodium phosphate (50 mg/kg of fat-free mass per day) or placebo (Glucodin Powder Naleant Pharmaceuticals Australasia, Rhodes, NSW, Australia). Capsules were prepared such that participants could consume the daily amount in four equal doses in accordance with previous research (Czuba et al., 2009). Capsules were consumed either with 200–300 ml of water or dissolved in ~200 ml of Powerade (Coca-Cola Amatil, Australia) to disguise the taste and maintain blinding for those who had difficulty ingesting capsules. The method of administration was matched within each participant.
and recorded in a daily food diary (as detailed later) for confirmation. Supplementation began with the ingestion of one dose in the morning before reporting for the first laboratory breakfast on Day 1 and ended after 6 days. Participants were instructed to consume the capsules 60–80 min before breakfast, lunch, afternoon snack, and dinner (capsules were to be ingested even if a participant was to skip a meal).

Assessment of Appetite and Energy Intake

The effect of supplementation on appetite and energy intake was assessed on Days 1, 2, and 6 of each phase. On each of these days, participants were required to attend the laboratory in a fasted state approximately 1 hr after ingesting the first capsules of the day. They were then provided with a laboratory breakfast consisting of a large bowl of warm porridge (~700 g), refrigerated orange juice, and water (~1,000 g each). Participants were instructed to eat and drink ad libitum until satiated. The specific breakfast time was chosen by each individual, with this equating to a time that best represented his or her normal eating patterns, but was standardized between visits. Breakfast items were weighed on electronic scales pre- and post-ingestion to determine the total energy intake (kJ). Data from our laboratory have shown a within-subject coefficient of variation of 10% and a test–retest correlation of .91 for assessing energy intake using this laboratory meal. To assess the effect of supplementation on the perception of appetite, participants were asked to complete a 10-cm visual analog scale comprising four questions to assess hunger, fullness, desire to eat, and prospective food consumption before and immediately after the laboratory breakfast (Flint, Raben, Blundell, & Astrup, 2000). All other food and drink intake during the supplementation period was self-recorded by participants in a food diary to allow for any compensatory eating outside the laboratory to be assessed. Instructions on use (including a 1-day example) and the necessity for accurate and detailed recordings of energy intake immediately after consumption were emphasized. Measuring implements (including measuring cups and spoons) were provided to participants to allow for recordings that were more precise. Daily energy intake was determined from these records using a commercially available software program (Foodworks, Xyris Software, QLD, Australia).

To monitor physical activity during each supplementation period, participants completed a daily physical activity diary and were required to wear an accelerometer (GT1M Activity Monitor, ActiGraph, FL). The total number of steps and estimated energy expenditure per day were determined using ActilLife software (ActiGraph). Before the second supplementation phase, participants were provided with a summary of their physical activity routine from Phase 1 to enable them to repeat similar activity patterns to ensure that the two phases of supplementation were well matched.

Assessment of VO2peak

After each supplementation period (i.e., on Day 7), participants returned to the laboratory for an assessment of VO2peak. Before commencing the test, a venous blood sample was taken for the determination of serum phosphate levels to compare with baseline. Exercise involved incremental treadmill running at progressively increasing intensities until volitional exhaustion (O’Donoghue, Fournier, & Guelfi, 2010). Each stage was 3 min in duration, with 1 min between stages to allow for capillary blood (35 μl) to be sampled from a hyperemic earlobe to determine blood lactate levels using a blood-gas analyzer (ABL 725, Radiometer, Copenhagen, Denmark). During the test, heart rate was monitored (Polar Electro Oy, Kempele, Finland) and participants breathed through a mouthpiece connected to a computerized gas-analysis system. This system included a ventilometer (Universal ventilation meter, VacuMed, Ventura, CA, USA) to calculate the volume of inspired air, in addition to oxygen and carbon dioxide analyzers (Ametek Applied Electrochemistry S-3A/1 and CD-3A, AEI Technologies, Pittsburgh, PA) to measure the percentage of oxygen and carbon dioxide in expired air. Calibration of the ventilometer was completed before each test per manufacturer specifications, and the analyzers were calibrated before use and verified after each test using a standard reference gas of known concentration. Each participant’s VO2peak was calculated using the sum of the four highest consecutive 15-s VO2 values recorded during the test. Strong verbal encouragement was provided throughout the test. Heart rate, blood lactate concentration, VO2, and running speed at each individual’s anaerobic threshold were calculated using the modified Dmax method (Cheng et al., 1992).

Determination of Serum Phosphate Levels

Venous blood samples were left to clot at room temperature for 60 min before being centrifuged at 1,000 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 analyzer with the specified Abbott reagents (Abbott Laboratories, Abbott Park, IL). Observed coefficients of variation were 4.2% at a level of 0.95 mmol/L and 2.0% at a level of 2.95 mmol/L.

Data Analysis

Two participants experienced common colds during the early stage of one of the supplementation phases and were therefore excluded from the analysis of energy intake and appetite due to the likely effect on these outcome measures (Eccles, 2005). The effect of sodium phosphate supplementation on energy intake, perception of appetite, aerobic capacity, and anaerobic threshold was assessed using repeated-measures ANOVA (SPSS 18.0 for Windows). In addition, Pearson’s correlation was used to assess the relationship between the change in serum phosphate levels after supplementation and the change
in VO_{2peak} between the phosphate and placebo trials to determine whether greater increases in serum phosphate were associated with greater improvements in VO_{2peak}. All results are reported as $M \pm SD$, with statistical significance indicated by $p \leq .05$.

**Results**

**Energy Intake**

The effect of sodium phosphate supplementation on ad libitum energy intake at the laboratory breakfast is displayed in Table 1. There was no significant effect of an acute dose of sodium phosphate on energy intake from the initial breakfast (i.e., Day 1 of supplementation; $p = .385$). Likewise, there was no effect of supplementation on energy intake from the laboratory breakfast on Day 2 ($p = .225$) or 6 ($p = .911$) of supplementation. Consequently, when the mean energy intakes from all laboratory meals during each period of supplementation were compared, there was no difference between periods ($p = .338$). With respect to the effect of supplementation on daily energy intake from the self-recorded food diaries, there was also no interaction of supplementation type and day of loading on estimated energy intake ($p = .641$; Table 2). Furthermore, there was no significant interaction effect of supplementation type and day of loading on estimated energy expenditure from physical activity ($p = .121$; placebo $2,100 \pm 697$ kJ/day, sodium phosphate $2,058 \pm 810$ kJ/day) or the number of steps per day based on accelerometry ($p = .258$; placebo $9,881 \pm 2,621$ steps/day, sodium phosphate $9,733 \pm 3,139$ steps/day), indicating that physical activity was well matched between trials.

**Perception of Appetite**

The effect of sodium phosphate supplementation on the perception of appetite before and after each laboratory meal is shown in Table 3. There was no acute effect of sodium phosphate supplementation on ratings of perceived hunger ($p = .483$), fullness ($p = .772$), desire to eat ($p = .656$), or prospective food consumption ($p = .504$) in the fasting state before the consumption of the first breakfast meal. Likewise, when all laboratory meals were considered, there was no significant interaction effect of supplementation type, day of loading, and time (pre- or postmeal) on ratings of perceived hunger ($p = .608$), fullness ($p = .489$), desire to eat ($p = .340$), or prospective food consumption ($p = .709$). However, there was a main effect of time (pre- or postmeal; $p < .001$) for each appetite variable, with a decrease in hunger, desire to eat, and prospective food consumption and an increase in feelings of fullness after meal consumption.

**VO_{2peak}**

There was no effect of 6 days of sodium phosphate supplementation on VO_{2peak} ($p = .483$, Table 4). Likewise, there was no effect of supplementation on running speed ($p = .275$, VO_{2} $p = .699$), or heart rate ($p = .344$) at which the anaerobic threshold occurred.

**Serum Phosphate**

There was no significant interaction effect of supplementation type and time (pre- and postsupplementation) on serum phosphate levels ($p = .522$; before placebo supplementation $1.32 \pm 0.20$ mmol/L, after placebo supplementation $1.33 \pm 0.24$ mmol/L, before sodium phosphate supplementation $1.23 \pm 0.17$ mmol/L, after sodium phosphate supplementation $1.31 \pm 0.18$ mmol/L), although there was a tendency for higher serum phosphate after sodium phosphate supplementation compared with baseline ($p = .089$). When the change in serum phosphate was correlated with the change in VO_{2peak} between phosphate and placebo trials, the relationship approached significance ($r = .397, p = .083$). Of note, 14 (70%) of the participants correctly identified the loading phase in which they ingested sodium phosphate. This was partly due to 10 (50%) of the participants experiencing at least one acute episode of mild gastrointestinal distress during sodium phosphate supplementation.

**Discussion**

The primary aim of this study was to investigate the effect of sodium phosphate supplementation on energy intake
both acutely (i.e., after a single dose) and after prolonged use (6 days) in trained men and women. A secondary aim was to confirm the previously observed benefits of sodium phosphate supplementation for VO\textsubscript{2\text{peak}}. Contrary to our expectations, we observed no effect of sodium phosphate supplementation on appetite or energy intake either acutely or over time. There was also no effect of supplementation on VO\textsubscript{2\text{peak}} or the intensity at which the anaerobic threshold occurred. These results suggest that athletes supplementing with sodium phosphate can do so without hindering their nutritional status. However, given the recent finding that an acute dose of phosphate significantly attenuated energy intake at the subsequent meal (Obeid et al., 2010). The reason for this discrepancy between studies is unclear. The acute dosage used in the current study was slightly more than that employed by Obeid et al. (700 ± 179 mg vs. 500 mg). However, Obeid et al. used a mixture of potassium and sodium phosphate, as opposed to sodium phosphate only in the current study. Another possible reason for the lack of effect in the current study may be related to the method of phosphate administration. In the current study, most participants ingested the sodium phosphate in a gelatin capsule with 200–300 ml of water. In contrast, in the study of Obeid et al., participants consumed the phosphate mixture dissolved in a liquid solution (250 ml). The latter method of administration may have assisted phosphate absorption, given the inverse relationship between particle diameter and absorption rate after consumption of drug prepara-

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Ratings of Appetite on a 10-cm Visual Analog Scale in the Fasted State (Premeal) and After Ingestion of an Ad Libitum Laboratory Breakfast (N = 18, M ± SD)</th>
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<tbody>
<tr>
<td>Rating of Appetite</td>
<td>Day</td>
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<tr>
<td></td>
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<tr>
<td>Perceived hunger (cm)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>2</td>
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<td></td>
<td>6</td>
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<tr>
<td>Perceived fullness (cm)</td>
<td>1</td>
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<tr>
<td></td>
<td>2</td>
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<td></td>
<td>6</td>
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<tr>
<td>Desire to eat (cm)</td>
<td>1</td>
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<td>2</td>
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<td></td>
<td>6</td>
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<tr>
<td>Prospective food consumption (cm)</td>
<td>1</td>
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<tr>
<td></td>
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<td>6</td>
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</tbody>
</table>

*Significant main effect of time (before to after meal ingestion) for each appetite variable (p < .001).

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Peak Aerobic Capacity (VO\textsubscript{2\text{peak}}) and Anaerobic Threshold After 6 Days of Supplementation With Sodium Phosphate or Placebo (N = 20, M ± SD)</th>
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<tbody>
<tr>
<td></td>
<td>Placebo</td>
</tr>
<tr>
<td>VO\textsubscript{2\text{peak}} (L/min)</td>
<td>3.56 ± 0.92</td>
</tr>
<tr>
<td>VO\textsubscript{2\text{peak}} (ml · kg\textsuperscript{-1} · min\textsuperscript{-1})</td>
<td>52.6 ± 5.2</td>
</tr>
<tr>
<td>Anaerobic threshold VO\textsubscript{2} (L/min)</td>
<td>3.37 ± 0.94</td>
</tr>
<tr>
<td>Anaerobic threshold speed (km/hr)</td>
<td>13.6 ± 1.9</td>
</tr>
<tr>
<td>Anaerobic threshold heart rate (beats/min)</td>
<td>176 ± 7</td>
</tr>
<tr>
<td>Anaerobic threshold blood lactate (mM)</td>
<td>4.2 ± 1.0</td>
</tr>
</tbody>
</table>
eral intake may be less responsive to phosphate supplementation, given their lower oxygen–hemoglobin affinity compared with men (Humpeler & Amor, 1973), which may limit any further phosphate-mediated decrease in this parameter, which is one of the proposed mechanisms through which phosphate supplementation may enhance performance (Williams, 2005). Nonetheless, when the male participants of the current study were examined alone, the results remained unchanged (i.e., no effect of phosphate supplementation on appetite, energy intake, VO_{peak}, or serum phosphate levels; results not shown). Alternatively, it may be that only certain individuals respond positively to phosphate supplementation. When the change in serum phosphate concentrations in the current study was correlated with the change in VO_{peak} between phosphate and placebo trials, the relationship approached significance. This highlights the possibility that sodium phosphate supplementation may be appropriate for certain individuals, but its effectiveness should be tested and confirmed for each athlete before use. This is especially important given the acute gastrointestinal discomfort that some individuals experience with supplementation. Of note, only one previous study reported gastrointestinal distress associated with sodium phosphate supplementation.
(Cade et al., 1984). Either previous studies have failed to mention the incidence of this potential side effect or the method of capsule administration has been modified to manage such symptoms. Our own experience suggests that any such side effects can be minimized by dissolving the contents of capsules in a liquid solution before ingestion rather than having participants ingest it in concentrated capsule form.

In summary, this study shows that sodium phosphate supplementation does not influence appetite and energy intake either acutely or after continued ingestion for 6 days. Therefore, athletes supplementing with sodium phosphate can do so without hindering their nutritional status. This is important, given that athletes need to consume adequate energy to maintain body mass and health, as well as maximize training effects (American Dietetic Association et al., 2009). However, given that phosphate supplementation failed to improve VO2peak in the current study, the ergogenic benefit of this supplement remains to be determined.

References


