

Randomized Trial of Acute Changes in Plasma Phosphate After Phosphorus-Standardized Meals in Peritoneal Dialysis



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Introduction: Hyperphosphatemia is associated with increased morbidity and mortality in patients with chronic kidney disease. The aim of this study was to assess whether a meal with high phosphorus content would affect plasma phosphate in the hours that follow among subjects with end-stage kidney disease on peritoneal dialysis.

Methods: This was a single-blinded randomized cross-over trial of 12 subjects on maintenance peritoneal dialysis, in which subjects were randomized to consume a meal with either high or low phosphorus content on 2 separate trial days. On each trial day, plasma phosphate was measured immediately before consumption of the standardized meal and after 1, 2, 3, and 5 hours.

Results: The mean fasting plasma phosphate at baseline was 1.69 ± 0.22 mmol/l. Plasma phosphate was similar between the 2 meals at baseline, as well as at 1, 2, 3, and 5 hours after consumption. The largest observed difference in plasma phosphate between the 2 meals was 0.15 mmol/l, which occurred 5 hours after consumption (high-phosphorus meal 1.75 ± 0.32 mmol/l vs. low-phosphorus meal 1.60 ± 0.14 mmol/l ($P = 0.06$)). Using summary analyses for repeated measures, we observed a significant difference in the plasma phosphate between the 2 meals ($P = 0.03$).

Conclusion: Our results show that in subjects with end-stage kidney disease, a meal with high phosphorus content has only a negligible effect on plasma phosphate compared to a meal with low phosphorus content. Thus, large increases in plasma phosphate cannot be accounted for by a high intake of phosphorus in the hours before blood sampling.

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KEYWORDS: end-stage kidney disease; mineral metabolism; nutrition; peritoneal dialysis; phosphate; phosphorus

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Hyperphosphatemia is believed to have deleterious effects in patients with chronic kidney disease (CKD), because of its association with vascular calcification,¹ bone disease,² and increased mortality.^{3,4} Thus, plasma phosphate is monitored closely in patients with CKD and end-stage kidney disease (ESKD). Treatment strategies to prevent hyperphosphatemia include reducing intestinal phosphate absorption via low-phosphorus diets and phosphate binders, titrated

treatment of secondary hyperparathyroidism with vitamin D analogs and calcimimetics, and, ultimately, intensified dialysis treatment or kidney transplantation.⁵ However, low-phosphorus diets can be difficult to maintain,⁶ and come at the risk of low protein intake, which might outweigh the potential benefits of the diet.⁷ Adherence to phosphate binders is often low because of side effects and high pill burden. Also, calcium-containing phosphate binders may increase the risk of vascular calcification.^{8,9} Therefore, precise and reliable measurement of plasma phosphate and knowledge of factors that influence these levels are important, as they have a major impact on treatment decisions. If a single meal of high phosphorus content consumed shortly before a blood sampling influences

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the measured phosphate levels in a clinically significant way, this must be considered when evaluating the measured phosphate levels, because long-term treatment decisions might therefore be based on the effects of a single meal. However, the acute effects of meals with varying degrees of phosphorus-containing meals on the postprandial levels of plasma phosphate is sparsely described. A few studies have been conducted in healthy subjects^{10–14} and in subjects with CKD stage 3 to 4,¹⁵ but none have been conducted in subjects with ESKD.¹⁶ This study aimed to investigate how a single meal with high phosphorus content acutely affects plasma phosphate compared to a single meal with low phosphorus content in patients with ESKD treated with peritoneal dialysis (PD).

MATERIALS AND METHODS

Study Design

This study was a randomized, single-blind, cross-over trial conducted on 2 separate trial days to investigate the effect of a meal high in phosphorus content on plasma phosphate compared to a meal low in phosphorus content.

Study Participants

Inclusion criteria were age >18 years, maintenance PD treatment for ESKD >3 months, serum ionized calcium between 1.1 mmol/l and 1.4 mmol/l for >3 months, plasma phosphate between 0.7 mmol/l and 3.0 mmol/l for >3 months, and ability to give written informed consent. Exclusion criteria were previous parathyroidectomy, current treatment with hemodialysis or assisted PD, and inability to communicate in Danish or English.

Study Procedure

On the day prior to a trial day, the participants underwent overnight fasting (for at least 10 hours). On each trial day, the participants were admitted to our in-hospital research unit at 8:00 a.m. and had a fasting blood sample drawn at 8:20 a.m. (baseline). After this, the participants consumed a meal of either high or low phosphorus content. Participants started the meal at 8:30 a.m. and were encouraged to finish it within 15 to 30 minutes. One, 2, 3, and 5 hours after the start of the meal (at 9:30, 10:30, 11:30, and 13:30), blood samples were drawn (Figure 1). It is well known that phosphate levels exhibit a circadian rhythm,^{17,18} which is why the blood samples were taken at standardized times to eliminate bias. Participants were randomized to receive either the meal of high or low phosphorus content on day 1 and the opposite meal on day 2, and were blinded to the order of the meals. The participants drank water as they pleased during the trial and used their

medication as usual, except for phosphate binders, which they did not consume prior to the meal. The participants did not have in-dwelling peritoneal dialysate fluid during the trial. Participants were randomized by the consecutive opening of sealed envelopes, which had been prepared according to a computer-generated randomization list by a person not involved in the trial.

Blood levels of phosphate, ionized calcium, parathyroid hormone (PTH), magnesium, 1,25-(OH)₂ vitamin D, and fibroblast growth factor 23 (FGF23) were analyzed at every time point (baseline and 1, 2, 3 and 5 hours after the meal), and 25-OH vitamin D was analyzed at baseline.

Peritoneal dialysate and urine were collected 24 hours prior to trial day 2, and were analyzed to measure quality of the dialysis treatment (Kt/V) and phosphate content in dialysate and urine. On both trial days, a food history was collected for the previous 3 days. Nutrient content of the food history and the study meals was analyzed with the program Vitakost (Conava ApS; Kolding, Denmark) with data from the Frida food database (frida.foodwaredata.dk; National Food Institute, Lyngby, Denmark) and consumer product nutrition labeling.

Study Intervention

Two different meals were designed to resemble common Danish breakfast meals with 670 mg of phosphorus in the high-phosphorus meal and 90 mg of phosphorus in the low-phosphorus meal, that is, a difference in phosphorus content of 580 mg. Both meals contained 20% of the daily energy requirements set at 2150 kcal/9000 kJ per day. Nutrient content presented in Table 1. The participants could add up to 24 g of butter to both meals. The 2 meals were isocaloric and consisted of whole foods to better compare with common breakfast meals, and we were therefore not able to avoid large differences in micro- and macronutrients.

Outcomes

The primary outcome of this study was the between-day differences in plasma phosphate levels 5 hours after ingestion of the meals. The secondary outcomes were differences in plasma phosphate at 1, 2, 3, and 5 hours between the 2 treatment days and change in plasma phosphate levels from baseline following each meal. In addition, the levels of ionized calcium, PTH, magnesium, 1,25-(OH)₂ vitamin D, and FGF23 were compared between the 2 days at 1, 2, 3, and 5 hours.

Sample Size

In 2016, mean plasma phosphate levels were 1.71 mmol/l, with a standard deviation of 0.37 mmol/l, for

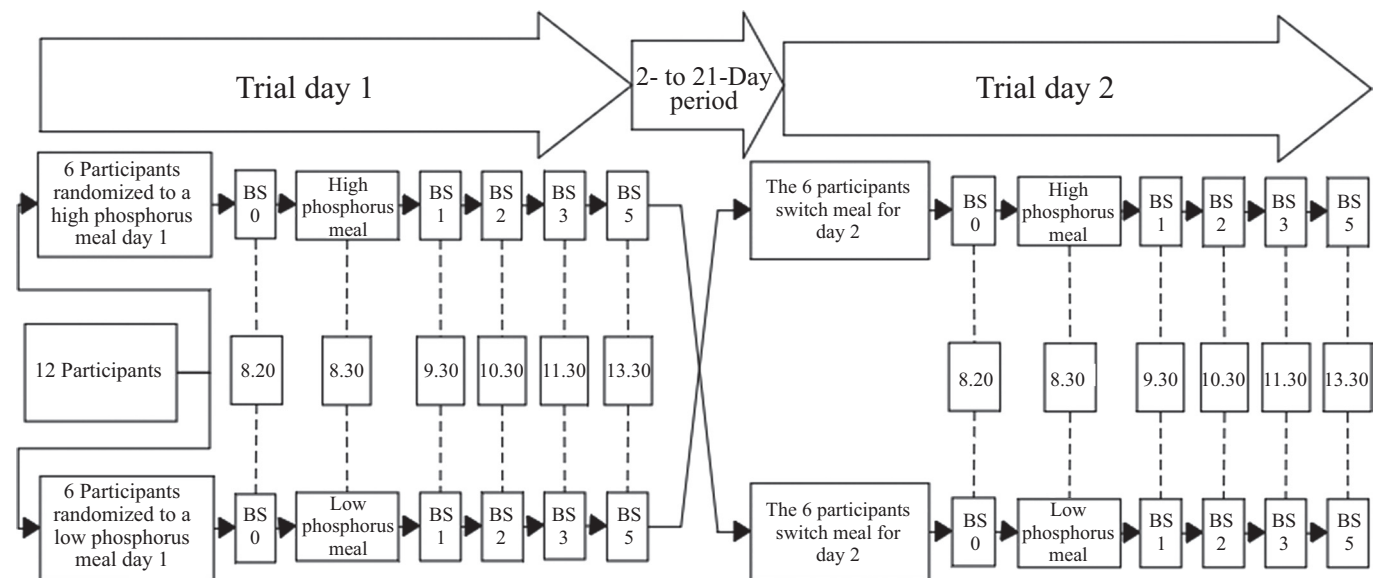


Figure 1. Study procedures. BS, blood sampling.

patients treated with PD at the Department of Nephrology, Herlev and Gentofte Hospital.¹⁹ To detect a difference in plasma phosphate of at least 0.5 mmol/l with $\beta = 0.20$ and $\alpha = 0.05$, a sample size of 11 participants was required.

Laboratory Analysis

All blood samples were analyzed at the Department of Clinical Biochemistry at Herlev Hospital, except plasma 1,25-(OH)₂ vitamin D, and FGF23. Plasma phosphate was analyzed in ethylenediamine tetraacetic acid (EDTA) with standard laboratory methods on a Vitros analyzer (Ortho-Clinical Diagnostic, Raritan, NJ), and PTH was analyzed using the ADVIA Centaur intact PTH assay (Siemens Healthineers, Erlangen, Germany). FGF23 was determined in EDTA plasma using a chemiluminescence immunoassay (CLIA) and 1,25-(OH)₂ vitamin D was measured in EDTA plasma

using CLIA. Both assays were performed on the automated analyzer Liaison XL (Diasorin, Saluggia, Italy). Intermediary precision for the 2 assays was <10%.

Statistical Analysis

Data are presented as mean and standard deviation for data that follow a normal distribution, or as median and interquartile range for data that do not follow a normal distribution. Data were analyzed for period effect and treatment-period effect as recommended for cross-over study designs by Altman.²⁰ The Student paired *t* test or Wilcoxon signed rank test was applied to assess differences compared to baseline on each trial day.

To estimate the mean rate of changes, a simple choice of summary statistic is the regression coefficient of response on time for each subject (i.e., the slope). However, to gain statistical efficiency, we used an optimal linear summary statistic derived from a slope-based analysis of covariance for repeated measures, adjusted for the observed pretreatment levels as expressed by the estimated intercepts (slope-based analysis of covariance [SLAIN]) as described by Frison and Pocock.²¹ The SLAIN coefficients were calculated using R version 3.60.²²

Ethics Considerations

This study was approved by the National Committee of Health Research Ethics of Denmark (H-18063465) and was registered on clinicaltrials.org (NCT03868371) on 11 March 2019.

RESULTS

All participants were recruited from the outpatient peritoneal dialysis clinic at Herlev Hospital, Denmark, between 7 March and 24 May 2019. A total of 81

Table 1. Nutrient content of the high- and low-phosphorous containing study meals

	High-phosphorus	Low-phosphorus
Meal composition	Rye bread 75 g Sliced cheese 60 g Milk 200 g	Wheat toast 105 g Marmalade 45 g
Nutrient content		
Energy (kcal)	379	374
Fat (g)	11.2	3.2
Carbohydrate (g)	37.8	74.6
Protein (g)	28.2	8.5
Fiber (g)	6.4	4.0
Phosphorus (mg)	670	90
Calcium (mg)	709	60
Magnesium (mg)	81	21

Mean ± standard deviation, median with interquartile range, and absolute number with percentage, as appropriate.

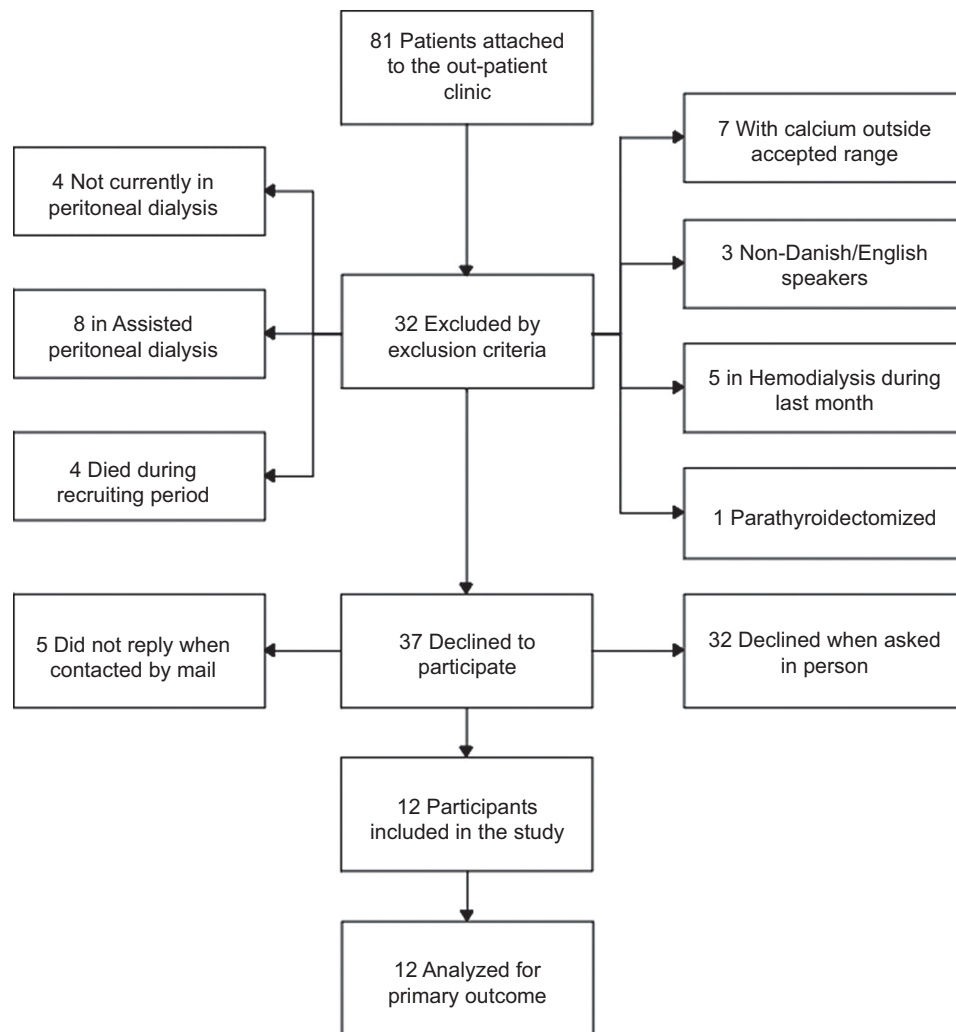


Figure 2. Participant flow diagram/Consolidated Standards of Reporting Trials (CONSORT) study diagram.

patients were treated in the clinic; of these, 32 patients were excluded based on inclusion and exclusion criteria, 37 patients declined participation, and 12 patients agreed to participate in the study (Figure 2). Of the 12 participants, 6 were randomized to the meal of high phosphorus content on day 1 and the meal of low phosphorus content day 2, and 6 were randomized to the meal of low phosphorus content on day 1 and the meal of high phosphorus content day 2. The baseline characteristics of the 12 participants are displayed in Table 2. During the 3 days before the trial day, the average daily phosphorus intake was 958 ± 172 mg, and daily energy intake was 1679 ± 252 kcal.

No period effect or treatment-period interaction was found for mean plasma phosphate at baseline ($P = 0.30$ and $P = 0.33$), mean plasma phosphate at 5 hours ($P = 0.15$ and $P = 0.20$), or SLAIN coefficients for p-phosphate ($P = 0.48$ and $P = 0.51$), and further analysis of data was therefore possible.

Both meals resulted in the same pattern of declining plasma phosphate for the first 3 hours after the meals, followed by an increase from 3 hours to 5 hours, where

the largest difference was observed (1.75 ± 0.32 mmol/l vs. 1.60 ± 0.14 mmol/l, $P = 0.06$). No significant difference was found at any time point (Figure 3, Table 3). Five hours after ingestion of the meals, we observed no significant difference in plasma phosphate compared to baseline (0.038 ± 0.174 mmol/l and -0.068 ± 0.112 mmol/l ($P = 0.06$)) (primary endpoint). Also, we did not observe any significant difference in plasma phosphate at any timepoint compared to baseline (data not shown). We did, however, observe a significant difference between the slopes for plasma phosphate for each meal using the SLAIN coefficient (Table 4).

The levels of ionized calcium, PTH, magnesium, FGF23, and 1,25-(OH)₂ vitamin D at each time point are displayed in Figure 4. Analysis of SLAIN coefficients did not reveal any differences between the slopes for the 2 meals (Table 4).

DISCUSSION

In this randomized cross-over clinical trial, we observed no significant difference in plasma phosphate

Table 2. Baseline characteristics of study population

Characteristic	n = 12
Male, %	66
Age, yr	76 (61; 81)
Weight, kg	84 (69; 86)
Height, cm	172 ± 9
Body mass index	27 ± 4
Systolic blood pressure, mm Hg	127 ± 14
Diastolic blood pressure, mm Hg	77 ± 9
Cause of kidney disease	
Diabetes	2 (17%)
Hypertension	1 (8%)
Glomerulonephritis	4 (33%)
Polycystic kidney disease	1 (8%)
Other	2 (17%)
Unknown	2 (17%)
Comorbidities	
Hypertension	9 (75%)
Dyslipidemia	9 (75%)
Diabetes mellitus type 2	3 (25%)
Coronary heart disease	4 (33%)
Cerebrovascular disease	2 (17%)
Heart failure	4 (33%)
Gout	5 (42%)
Biochemical variables	
Phosphate, mmol/l	1.69 ± 0.23
Ionized calcium, mmol/l	1.22 ± 0.11
Intact parathyroid hormone, pmol/l	25 (8; 39)
Magnesium, mmol/l	0.93 ± 0.11
25-OH vitamin D, nmol/l	87 ± 23
Fibroblast growth factor 23, pg/ml	2235 (877; 5090)
1,25-(OH) ₂ vitamin D, pmol/l	32 ± 16
Dialysis prescription	
Continuous ambulatory peritoneal dialysis	9 (75%)
Automated peritoneal dialysis	3 (25%)
Dialysis vintage, mo	26 (9;56)
Total kt/V	1.64 ± 0.32
Renal kt/V	0.71 ± 0.54
Dialysis kt/V	0.68 (0.51; 1.6)
24-h Urine volume, ml	1088 ± 728
Daily dialysis phosphate excretion, mmol	4.84 (3.22; 7.15)
Daily renal phosphate excretion, mmol	6.25 ± 4.63
Total daily phosphate excretion, mmol	14.25 ± 5.86
Medication	
Calcium-containing phosphate binders	7 (58%)
Non-calcium-containing phosphate binders	7 (58%)
Calcimimetics	2 (17%)
Alfacalcidol	6 (50%)
Cholecalciferol	10 (83%)
Antihypertensives	7 (58%)
Diuretics	8 (67%)
Erythropoietin	7 (58%)

Mean ± standard deviation, median with interquartile range, and absolute number with percentage, as appropriate.

at any time point during the 5 hours following a meal of high phosphorus content compared to a meal of low phosphorus content, although we did observe an overall higher SLAIN coefficient for plasma phosphate following the high-phosphorus meal. Despite the 7 times higher phosphorus content of the high-

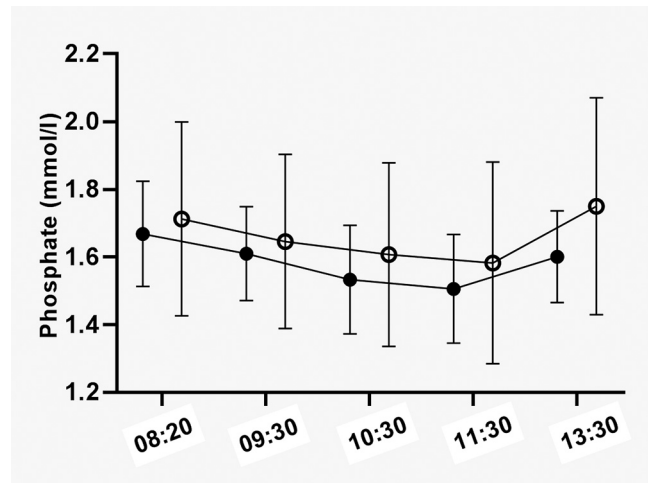


Figure 3. Effect of a meal with high or low phosphorus content on plasma phosphate. Meals were consumed at 8:30 a.m. Mean with standard deviation before the consumption of a high- or low-phosphorus-containing meal (8:20 a.m.) and 1, 2, 3, and 5 hours later. Open circles represent values for the high-phosphorus meal; closed circles represent values for the low-phosphorus meal.

phosphorus meal, the greatest observed difference between the 2 groups was 0.15 mmol/l, which we do not think would have any important influence on the decision to start or change treatment for the individual patient, and far from the 0.5-mmol/l difference, which was the minimal relevant difference that we expected to find.

Our results are comparable with those of former studies in CKD 3 to 4 and patients with ESKD on hemodialysis. Isakova *et al.*¹⁵ tested the difference between 2 whole-food meals of 250 mg and 500 mg of phosphorus content in 13 patients with CKD stage 3 to 4, and observed that serum phosphate decreased after both meals. The difference in serum phosphate between the 2 meals was just 3%, which is consistent with the results of our own trial. In another study, Mazzetti *et al.*²³ performed an observational study of serum phosphate levels in routine blood samples from hemodialysis patients, compared patients with and without phosphorus intake during the hour previous to blood sampling, and found serum phosphate to be

Table 3. Influence of the study meals on phosphate

Phosphate mmol/l	Influence of study meals on phosphate			P value
	Low-phosphorus meal	High-phosphorus meal	Mean difference (95% CI)	
Baseline	1.67 ± 0.16	1.71 ± 0.29	0.04 (–0.09; 0.18)	0.49
T1	1.61 ± 0.14	1.65 ± 0.26	0.04 (–0.09; 0.16)	0.53
T2	1.53 ± 0.16	1.61 ± 0.27	0.07 (–0.04; 0.19)	0.19
T3	1.51 ± 0.16	1.58 ± 0.30	0.08 (–0.06; 0.21)	0.23
T5	1.60 ± 0.14	1.75 ± 0.32	0.15 (–0.01; 0.31)	0.06

Mean phosphate with standard deviation at baseline and 1, 2, 3, and 5 hours after the consumption of the high- and low-phosphorus-containing meal. Mean difference between the meals with 95% confidence interval (CI).

Table 4. Slope-based analysis of covariance (SLAIN) coefficients for meals with high or low phosphorus content

	SLAIN coefficients		P value
	Low-phosphorus meal	High-phosphorus meal	
Phosphate (mmol/l per hour)	-0.02 ± 0.018	0.007 ± 0.037	0.03
Ionized calcium (mmol/l per hour)	0.006 ± 0.003	0.004 ± 0.005	0.19
Magnesium (mmol/l per hour)	0.007 ± 0.006	0.005 ± 0.005	0.17
Intact PTH (pmol/l per hour)	0.56 ± 0.99	0.76 ± 0.89	0.52
FGF23 (pg/mL per hour)	-35.24 ± 157.91	7.84 ± 79.36	0.49
1,25-(OH) ₂ vitamin D (pmol/l per hour)	0.20 ± 1.03	-0.14 ± 0.93	0.49

Mean ± standard deviation and median with interquartile range, as appropriate. Slope-based analysis of covariance for repeated measures, adjusted for the observed pre-treatment levels (SLAIN) compared between treatment group. The SLAIN analysis determined a small but statistically significant higher level of p-phosphate in the high phosphorus meal group. FGF23, fibroblast growth factor 23; PTH, parathyroid hormone; SLAIN, slope-based analysis of covariance.

0.2 mmol/l higher in the group with phosphorus intake prior to blood sampling. Again, the results are similar to those of our own trial, although the time frame was shorter, and the possible confounders were less well controlled.

Several studies in healthy populations found an acute impact of phosphorus intake on plasma phosphate. Both direct duodenal infusion of inorganic phosphorus²⁴ and intake of soluble phosphorus, with or without meals, increased plasma phosphate significantly as early as 30 minutes after ingestion.^{10–13} The discrepancy between the results of these studies and our own might be the difference in phosphorus source. Kawamura *et al.*¹⁴ compared 2 meals of equal phosphorus content but with different phosphorus sources (phosphorus supplement or natural phosphorus sources). They found a higher plasma phosphate after ingestion of phosphorus supplements compared to that after consumption of a meal with a high natural phosphorus content. This difference is probably due to different bioavailability of phosphorus from different sources. Organic-based phosphorus sources on average have a bioavailability of 60%, animal-based food products around 60% to 80%, plant-based food products around 30% to 40%, and inorganic phosphorus from phosphorus salts >90%.²⁵ Thus, we speculate that the effects of the 2 meals in our study might have had greater impact on plasma phosphate had the phosphorus source of the meals been inorganic or meat based.

The meals in the present study was designed to represent an ordinary Danish breakfast meal. The high phosphorus meal contained phosphorus from organic sources and had a high calcium-to-phosphorus ratio. The calcium-to-phosphorus ratio may influence the bioavailability of phosphorus, with high levels of calcium limiting the intestinal phosphorus uptake²⁶ and

thereby the effect on plasma phosphate. Indeed, calcium carbonate is used as a phosphate binder for treatment of hyperphosphatemia.⁵ A lower calcium-to-phosphorus ratio may have increased the intestinal absorption of phosphorus and thereby led to a higher levels of plasma phosphate. Likewise, slightly higher magnesium content in the high phosphorus meal may have affected the absorption of phosphorus, as magnesium also acts as phosphate binder.²⁷ Perhaps food containing both phosphorus and calcium or magnesium may be less prone to induce hyperphosphatemia. This should be further explored in order to ease restrictions on such foods to expand the food recommendations for patients with ESKD.

Other mechanisms that may influence plasma phosphate are increases in urinary or peritoneal excretion. We do not have data from our study population on urinary phosphate excretion on the trial days; however, the average daily urinary phosphate excretion was very low (266 mg of phosphorus, i.e., a mean of 55 mg phosphorus for the 5-hour duration of the trial), and large compensatory changes in phosphaturia caused by the meals seems unlikely in our population with very limited kidney function. Our population did not have in-dwelling peritoneal dialysate fluid during the trial and therefore no phosphate excretion in dialysate fluid. Adams *et al.*¹⁰ demonstrated a high intestinal absorption of acute dietary inorganic phosphorus intake and no compensatory increase in urinary excretion in rats with reduced kidney function. The authors speculated that deposition or storage of phosphate in the extravascular compartments may have blunted the rise in phosphate. In our trial, we did not measure intestinal absorption or urinary excretion of phosphate on the trial days, and are therefore unable to assess this possibility.

The high-phosphorus meal seemed to have no short-term effect on magnesium, ionized calcium, PTH, FGF23, and 1,25-(OH)₂ vitamin D. This is evident in the SLAIN coefficient, which accounts for changes adjusted for values at baseline, for which we found no significant difference between the groups for any of these secondary outcomes.

The high-phosphorus meal contained 649 mg more calcium and 60 mg more magnesium than the low-phosphorus meal (Table 1). Despite these marked differences in content, blood levels of ionized calcium and magnesium were not significantly different between the 2 meals. This further strengthens the argument that calcium and magnesium may have acted as phosphate binders, thus impairing not only phosphate absorption, but also absorption of calcium and magnesium in the intestine.

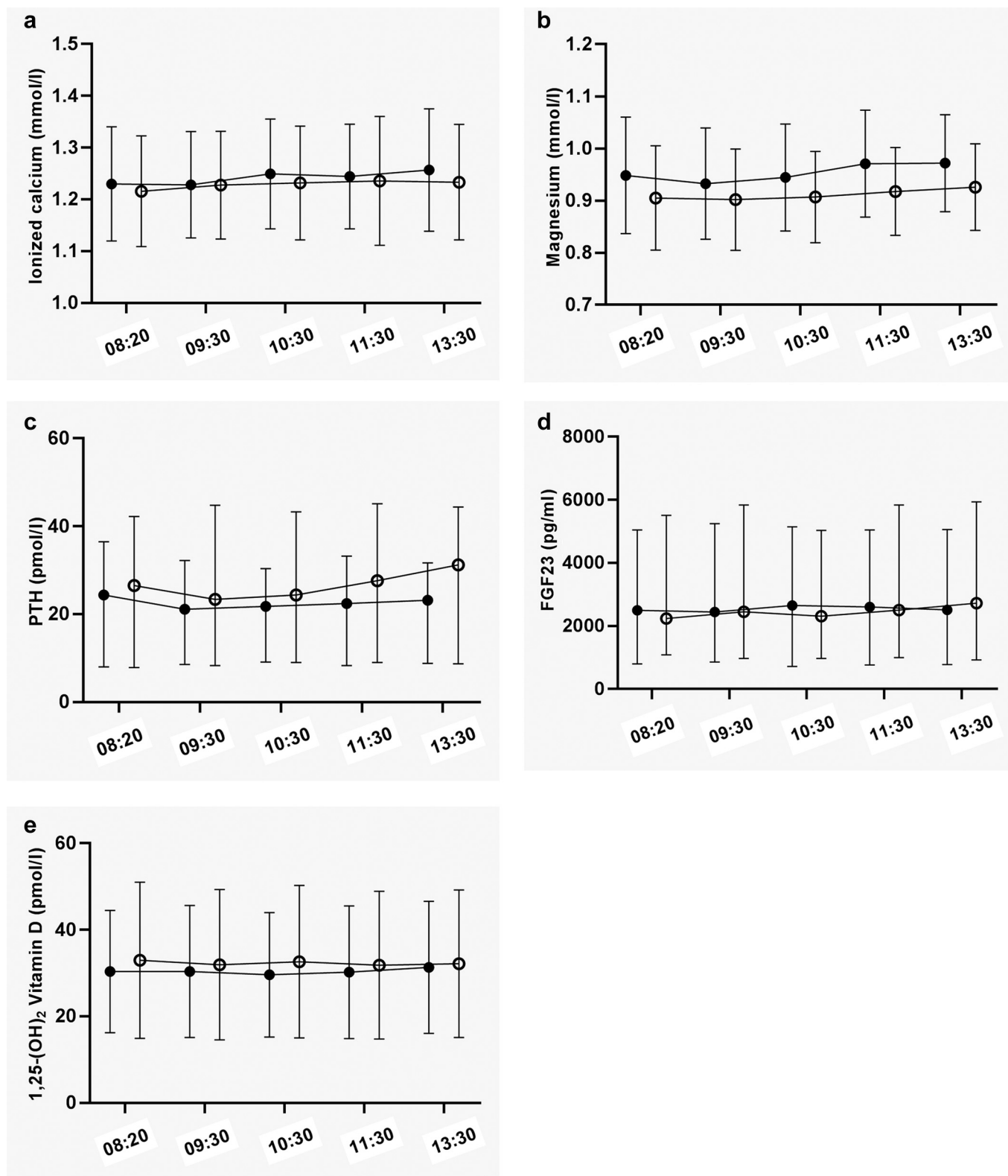


Figure 4. Effect of a meal with high or low phosphorus content on (A) ionized calcium, (B) magnesium, (C) parathyroid hormone (PTH), (D) fibroblast growth factor 23 (FGF23), and (E) 1,25-dihydroxy vitamin D. Meals were consumed at 8:30 a.m. Mean with standard deviation or median and interquartile range before the consumption of a high- or low-phosphorus-containing meal (8:20 a.m.) and 1, 2, 3, and 5 hours later. Open circles represent values for the high-phosphorus meal; closed circles represent values for the low-phosphorus meal.

The main limitation of this trial pertains to the generalizability of the study meals. The results may have been different had we used a high-phosphorus

meal containing higher content of inorganic phosphorus or if the meals had had a lower or equal content of calcium and/or magnesium. In future studies, it

would be of interest to investigate the effect of meals in which the phosphorus source is processed foods with added inorganic phosphorus (as commonly used for preservation) or meals with a lower content of calcium and/or magnesium. Also, we did not measure the exact phosphorus content of the meal, but instead relied on estimates of phosphorus content. The estimates of our population's daily phosphorus intake were low (958 mg) compared to the intake by Danish men and women (1687 mg and 1348 mg) in the 65- to 75-year age group.²⁸ The estimates may have reduced accuracy because of the limited information on actual phosphorus content in consumer products^{29,30} and by patient under-reporting. Finally, we included patients both with and without residual kidney function, and, as only 2 participants had no residual kidney function, we were unable to examine whether this might have affected the results of our trial. The influence of acute phosphorus intake on plasma phosphate may have been more pronounced had we included only patients with no residual kidney function.

The strengths of this trial include its randomized and blinded trial design with standardized observation and a sample size with enough power to detect clinically meaningful changes in plasma phosphate. Another strength was the representativeness of the phosphate and PTH levels of the study population, which was 1.69 ± 0.23 mmol/l and 25 (8 ; 39) pmol/l, respectively, which is close to the Danish average phosphate and PTH levels of 1.61 ± 0.43 mmol/l and 21.8 (11.7 ; 36.7) pmol/l, respectively, in patients treated with peritoneal dialysis.¹⁹ It is, however possible that for patients with higher levels of plasma phosphate, the effect of a meal with high phosphorous content might have a more pronounced effect on plasma phosphate and PTH.

Also, despite the above-mentioned limitations to the high-phosphorus-containing meal, we believe that this meal is representative of the breakfast meals of many Danish patients with ESKD, and thus the results are likely to be valid for patients with ESKD who consume similar breakfast meals.

In general, our results raise the question of whether treatment decisions with regard to reducing phosphorous load can reliably be made based on plasma phosphate. High levels of plasma phosphate do not seem to be explained by the effects of a single meal with high phosphorus content such as the one used in this trial. Instead, other explanations for high plasma phosphate may need to be explored, to prevent clinical inertia and to improve clinical outcomes. This, however, is outside the scope of the current trial.

In conclusion, a meal with high phosphorus content has a negligible effect on plasma phosphate compared

to a meal with low phosphorus content among subjects with ESKD on PD. Whether a meal with a different source of phosphorous (e.g., inorganic or meat based) would have a different effect on plasma phosphate should be explored in future clinical trials.

DISCLOSURE

All the authors declared no competing interests.

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