Hyperphosphatemia in Children With CKD: A Great Dilemma

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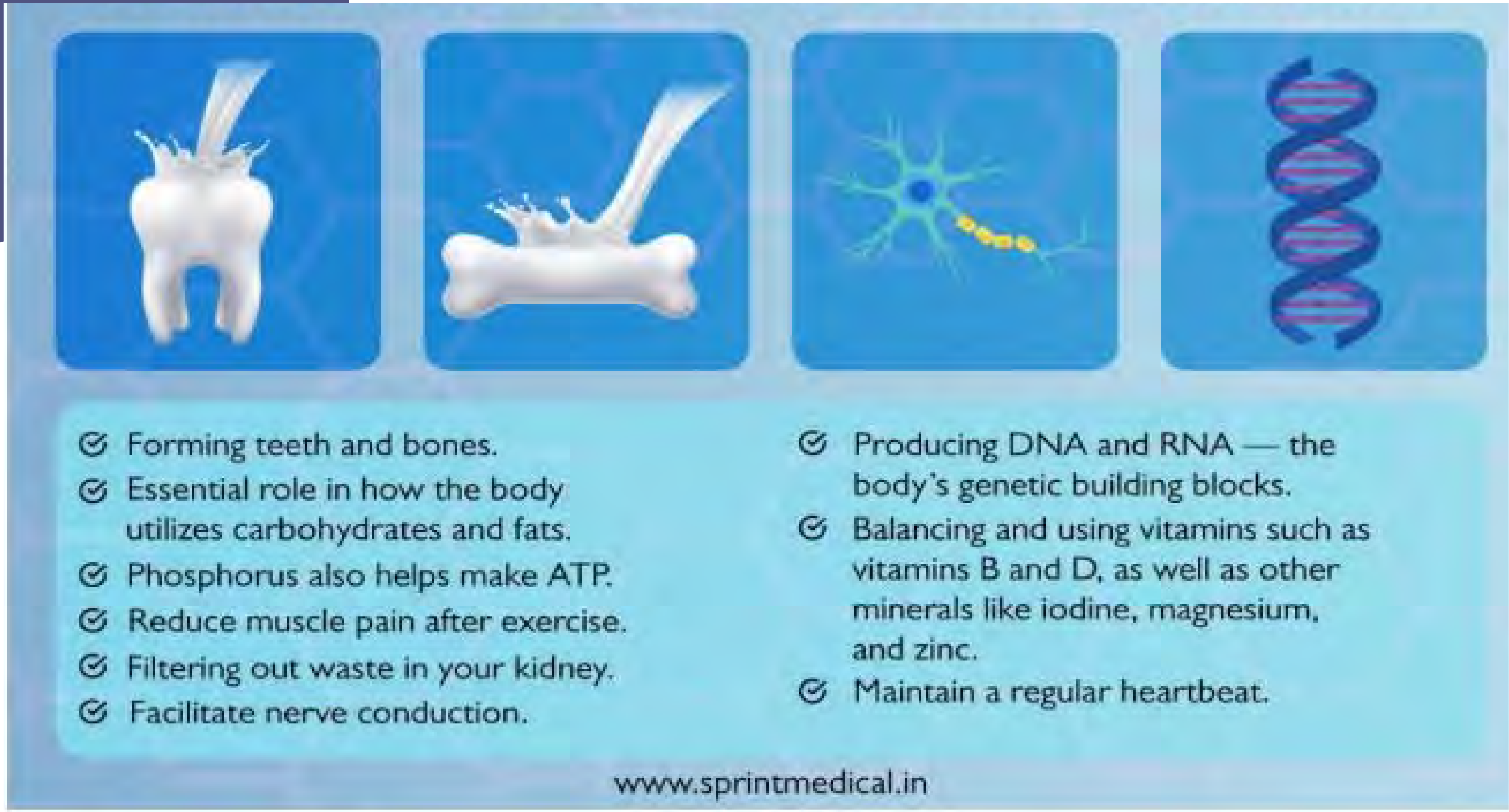
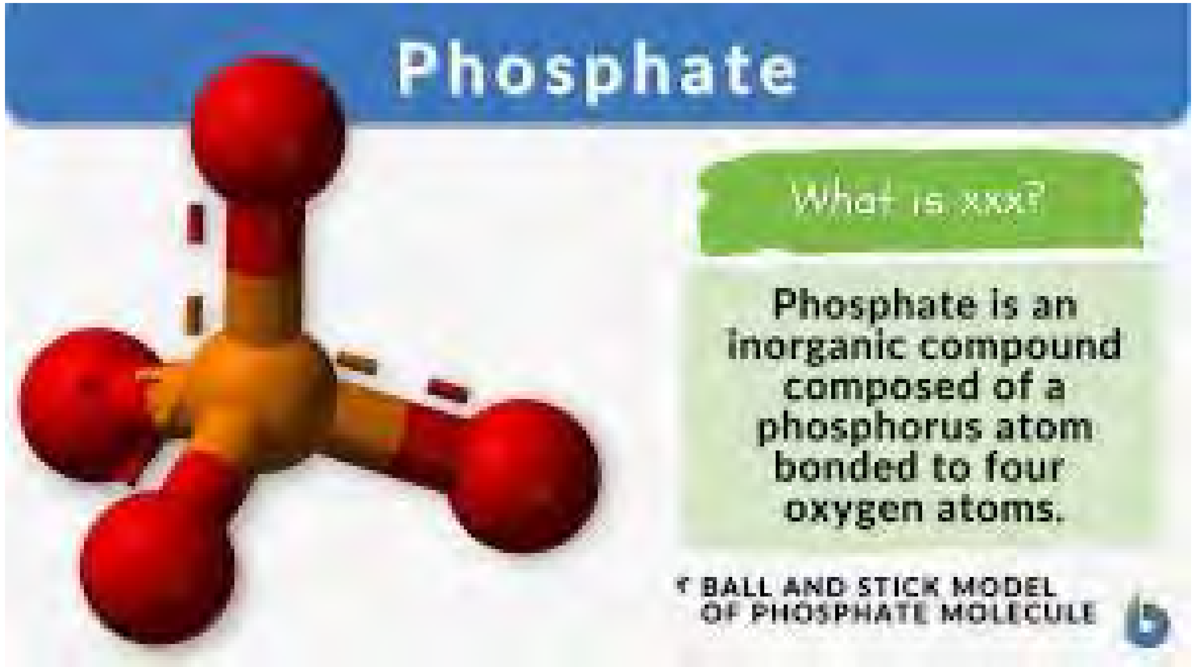
Function of Phosphorus



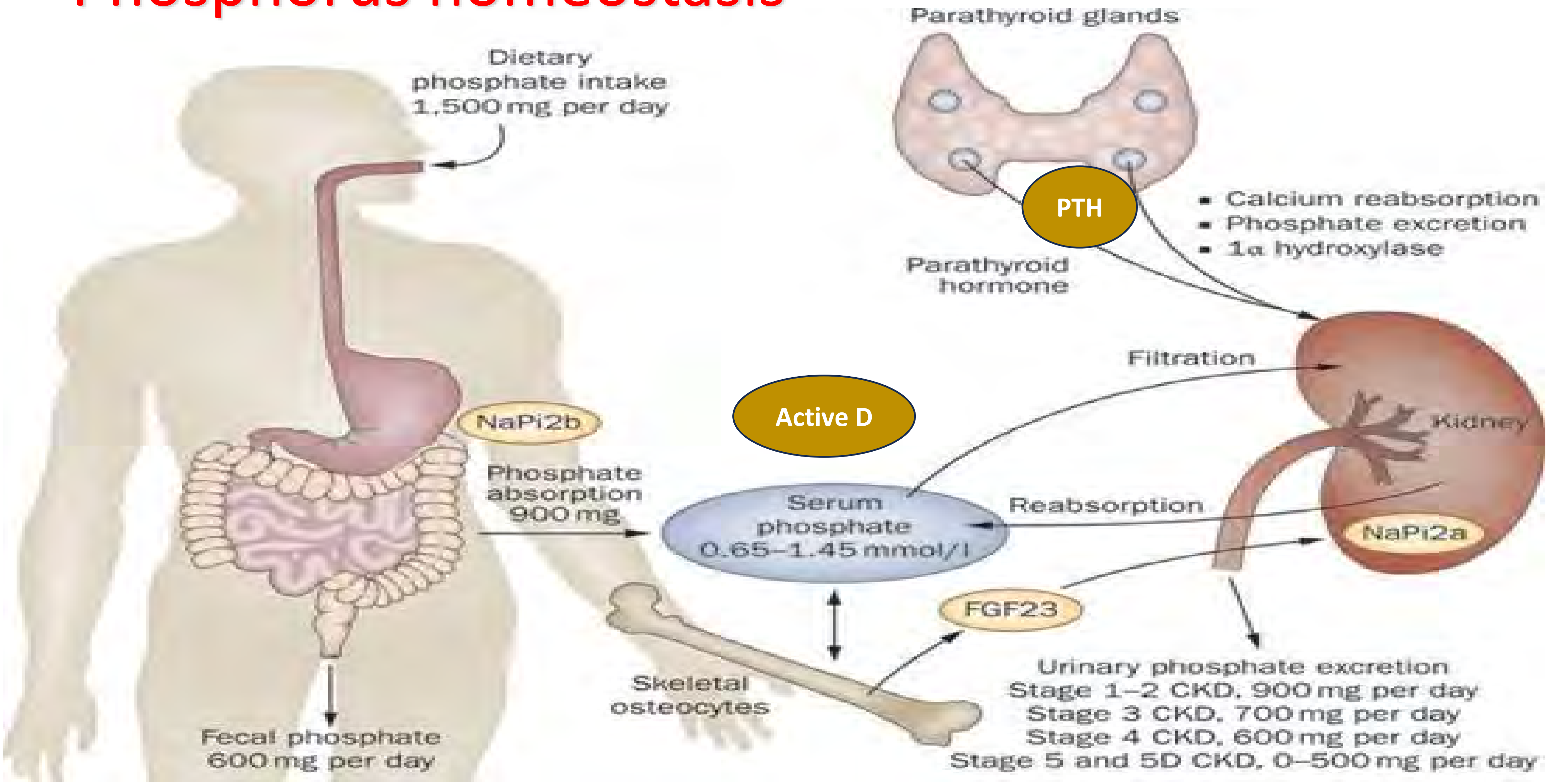

PHOSPHATE
PHOSPHORUS
PHOSPHOROUS


Phosphate
What is xxx?
Phosphate is an inorganic compound composed of a phosphorus atom bonded to four oxygen atoms.
BALL AND STICK MODEL OF PHOSPHATE MOLECULE

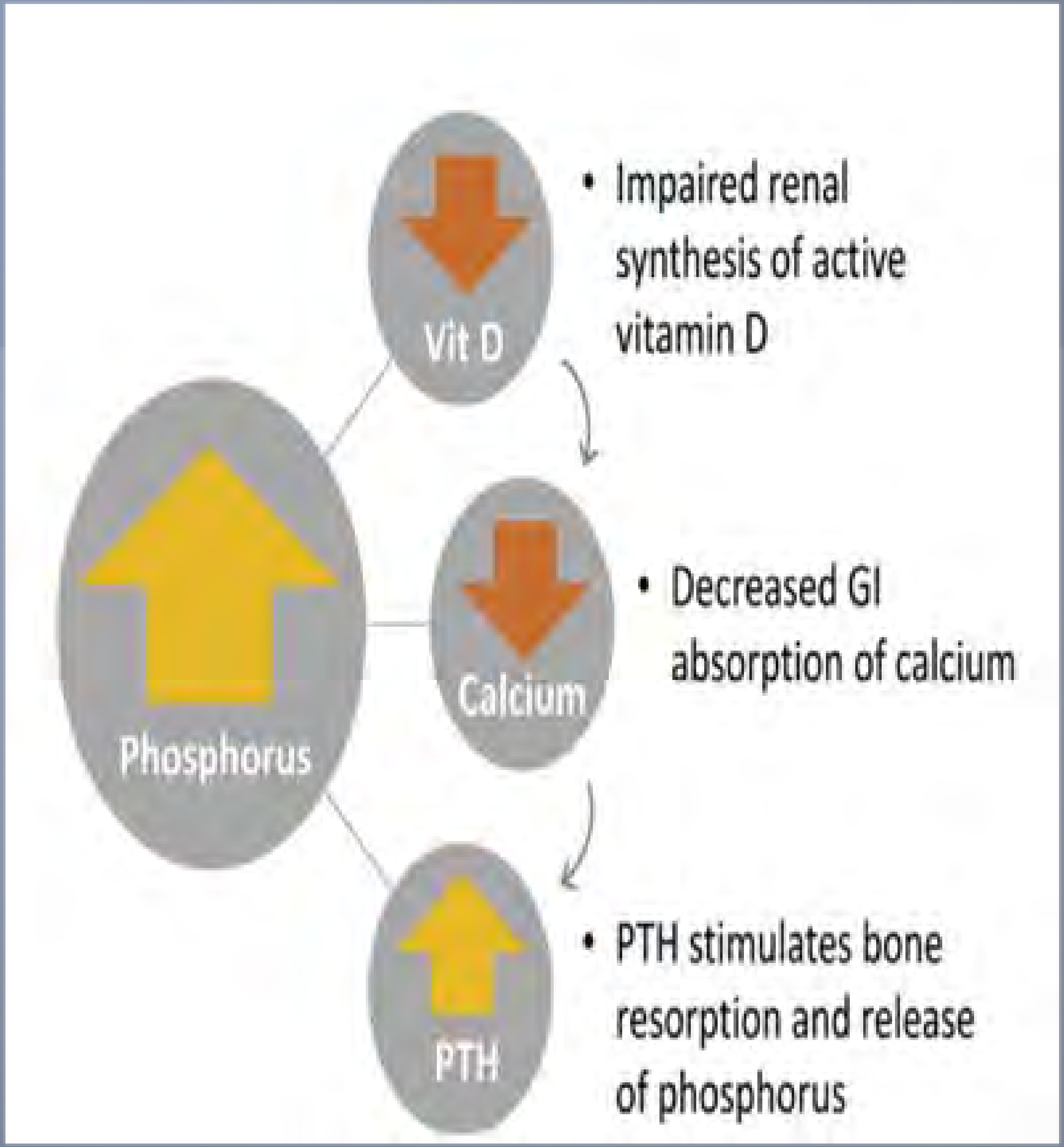
Agenda

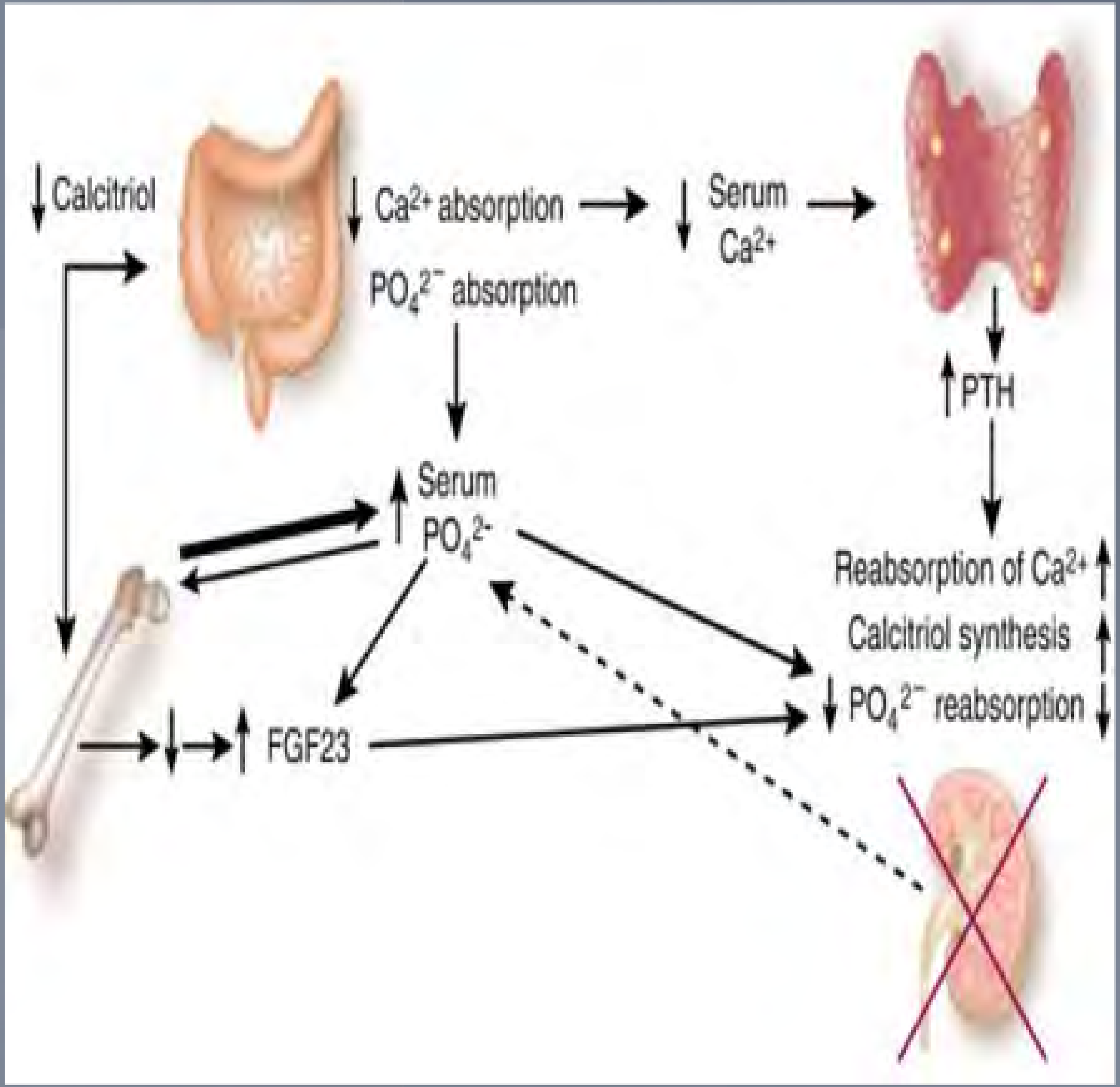
- *Phosphorus homeostasis.*
- *Phosphorus dysregulation in CKD.*
- *Why should we treat hyperphosphatemia?*
- *How do we diagnose hyperphosphatemia?*
- *How do we treat hyperphosphatemia?*

Phosphorus homeostasis



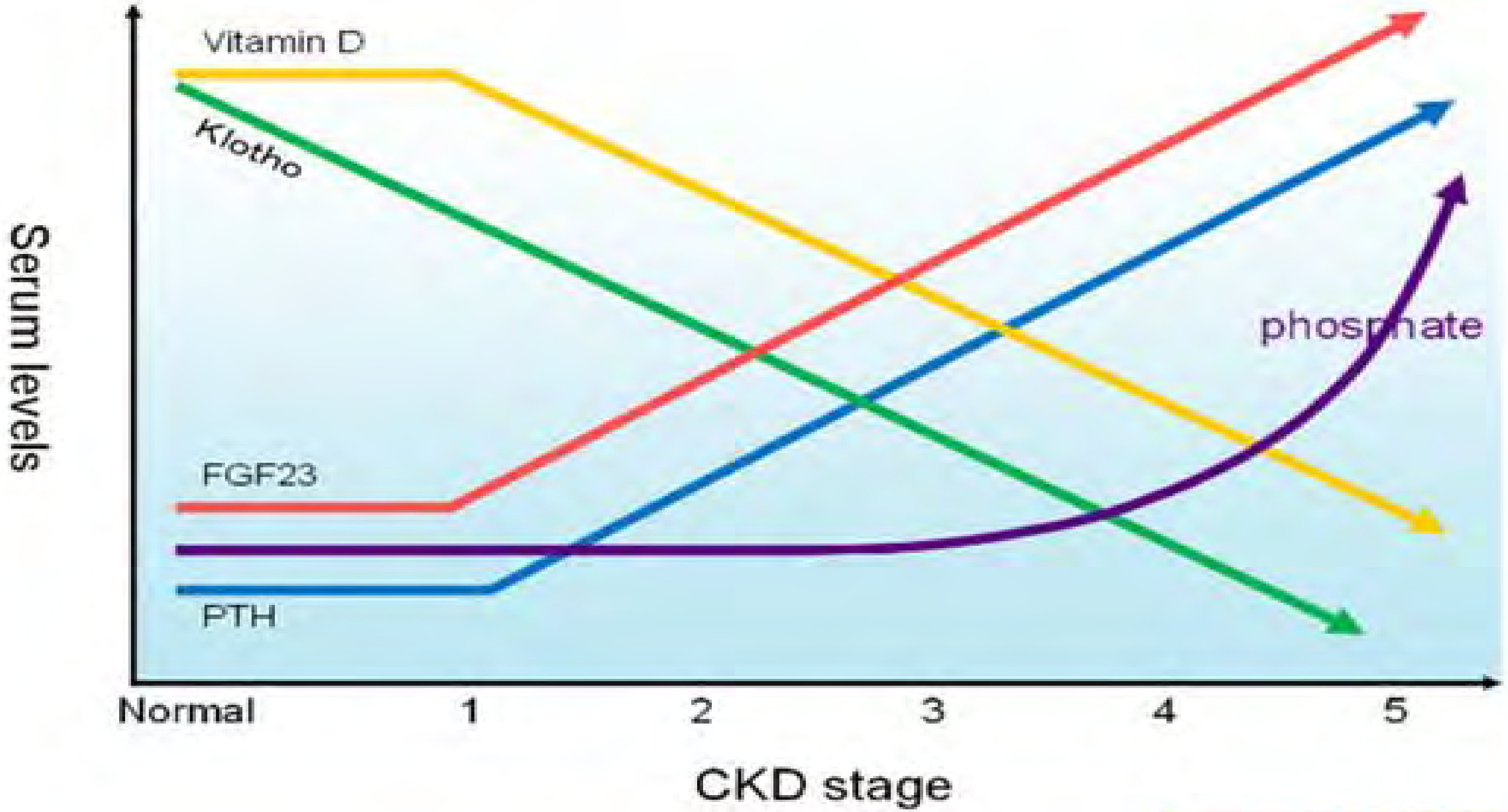
Phosphorus dysregulation in CKD





Phosphorus dysregulation in CKD

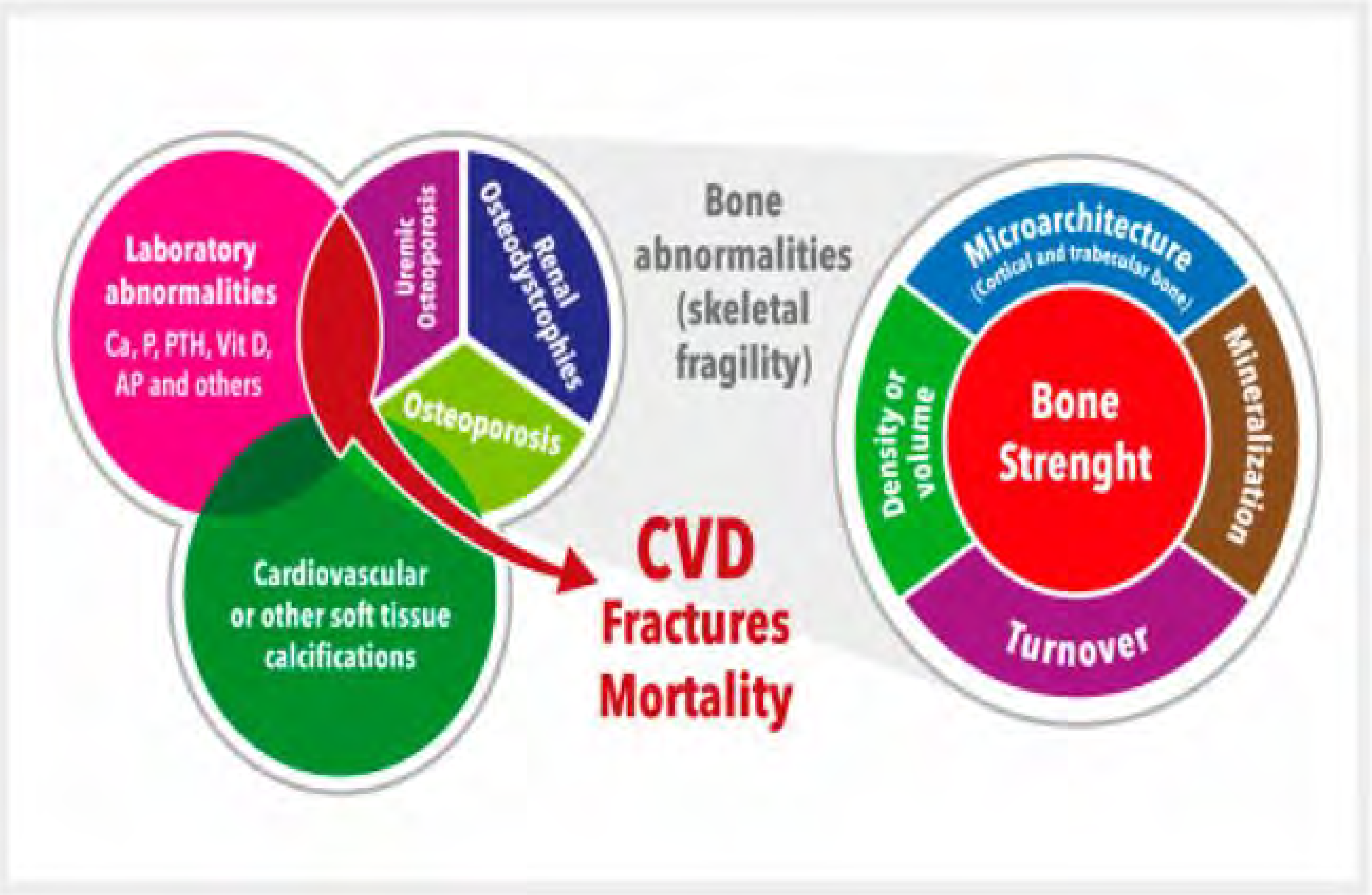
FGF23/iPTH and Phsophorus levels



Wolf M. J Am Soc Nephrol. 2010;21:1427-35

Chronic Kidney Disease–Mineral and Bone Disorder (CKD-MBD)

2017



Prevalence of hyperphosphatemia

5%

G 1-2

20%

G 4

40%

G 5

80%

G 5D

Why should we treat hyperphosphatemia?

Phosphate is a cardiac toxin

Consequences of hyperphosphatemia and elevated levels of the calcium–phosphorus product in dialysis patients

Nathan W. Levin and Nicholas A. Hoenich

Control of serum phosphorus levels is a central goal in the management of patients with chronic renal failure. Inadequate control of serum phosphorus leads to elevated levels of the calcium–phosphorus product. This plays a pivotal role in vascular calcification, cardiovascular disease, calciphylaxis, and death. Elevated phosphorus and elevated levels of the calcium–

Introduction

In early renal failure, the serum phosphorus concentration is maintained in the normal range by an increase in plasma parathyroid hormone (PTH). As renal function declines, hyperphosphatemia becomes increasingly common. For patients receiving regular dialysis for end-stage

Elevated serum phosphorus levels and elevated levels of calcium-phosphorus product has been implicated as a major factor in the development of tissue and arterial calcification and cardiovascular disease.

Curr Opin Nephrol Hypertens 10:563±56 8. 2001 Lippincott Williams & Wilkins.

Serum Phosphorus and Vascular Calcification

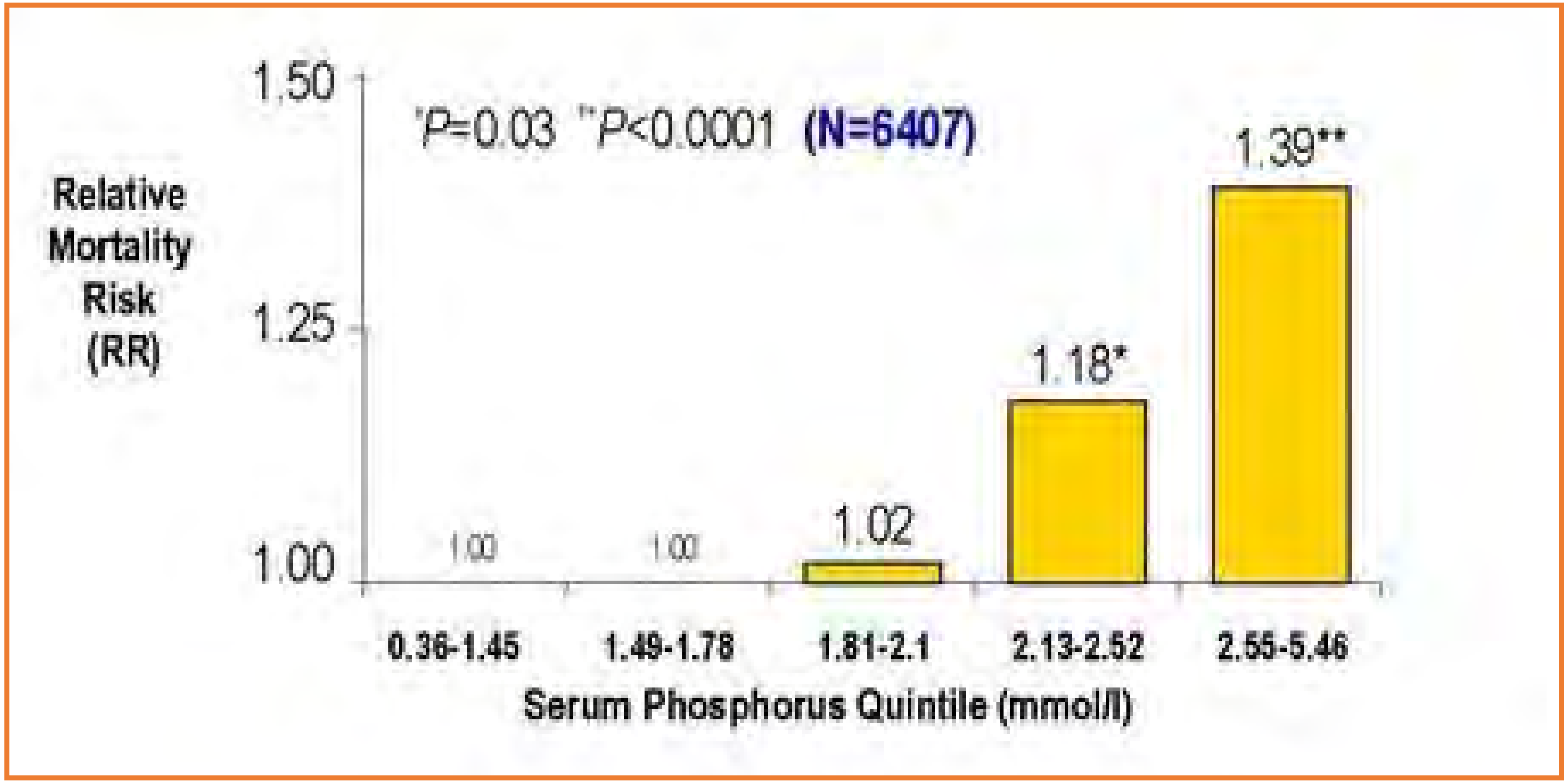
Pediatric studies of surrogate markers of vascular abnormalities

	No of patients	Ca x P	P binders	P	Vit D	PTH
Litwin, 2000 CIMT	37	Y	Y		Y	
Mitsnefes, 2005 CIMT/stiffness LVM	16	Y	Y Y	Y Y	Y Y	Y Y
Civilibal, 2006 CAC	39	Y	Y	Y	Y	Y
Ruiz, 2007 CAC	4				Y	
Shroff, 2007 CAC CIMT	85			Y Y	Y Y	Y Y

Increase in mortality risk

Serum Phosphorus and Mortality

Elevated serum phosphate increases mortality risk

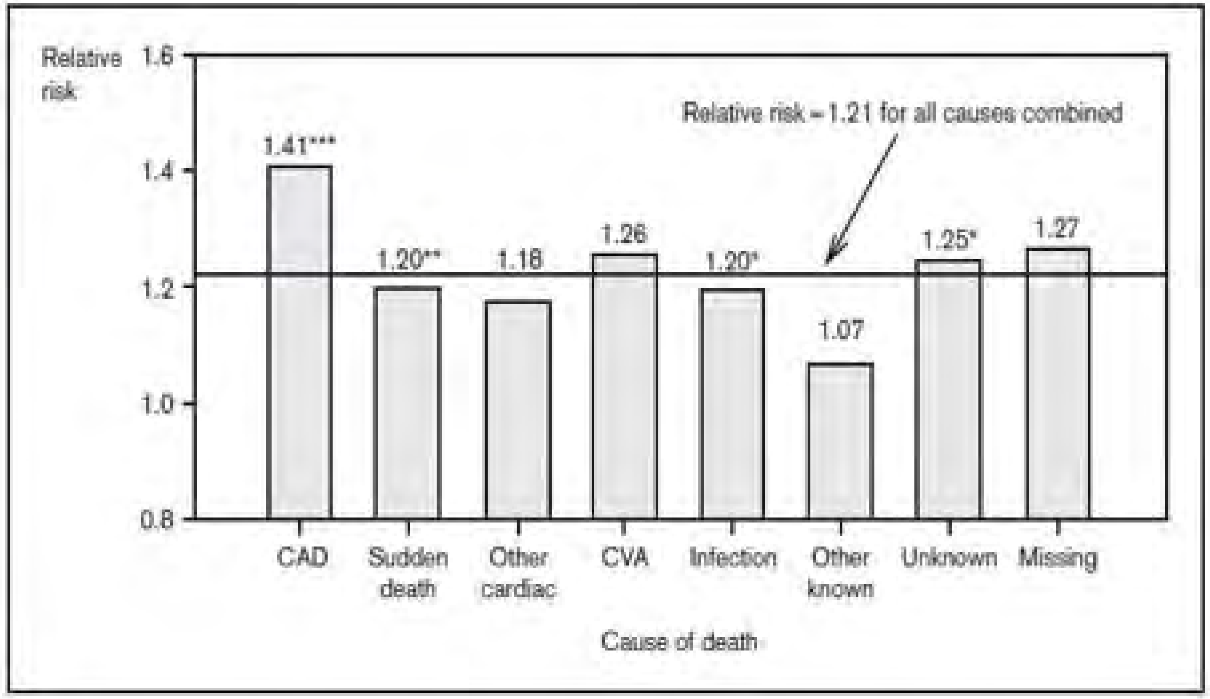


The mortality rate increases by 6% for each 0.3mmol/L rise in phosphorus levels

Serum Phosphorus and Mortality

Figure 1. Adjusted relative risk of mortality by cause of death for patients with serum phosphate levels above 6.5 mg/dl compared with that for patients with serum phosphate levels of 2.4–6.5 mg/dl

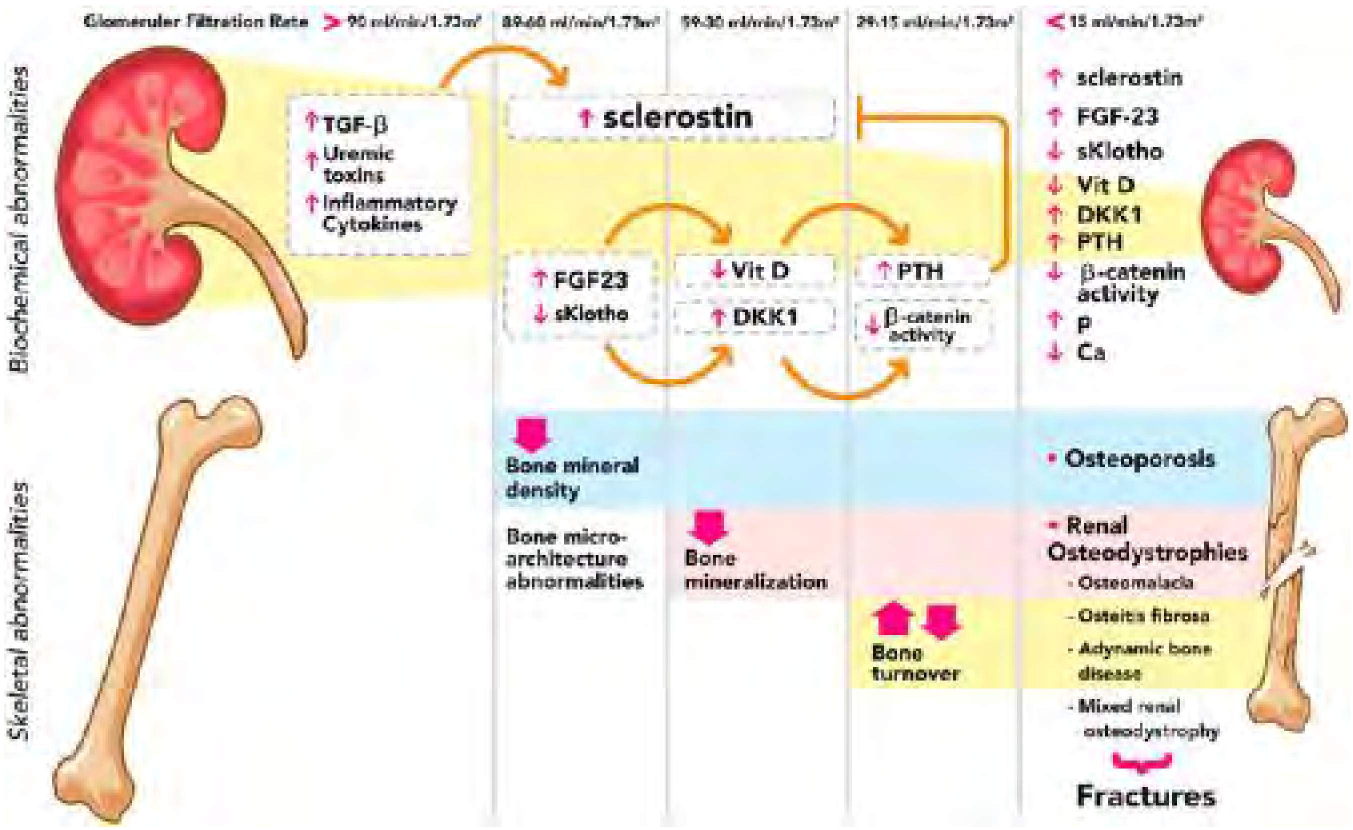
*P < 0.05, compared with a relative risk of 1.0; **P < 0.001, compared with a relative risk of 1.0; ***P < 0.0005, compared with a relative risk of 1.0. CAD, coronary artery disease; CVA, cerebrovascular accident. Reproduced with permission from [2].



Ganesh SK, Stack AG, Levin NW, et al. Association of elevated serum PO4, Ca6PO4 product and PTH with cardiac mortality risk in chronic hemodialysis patients. Am J Kidney Dis

Progression of bone Impairment

Wesseling, Salusky (2013), Pediatr Nephrol 28:617–625



Progression of CKD

CJASN

Association Between Clinical Risk Factors and Progression of Chronic Kidney Disease in Children

Amy D. Staples, Larry A. Greenbaum, [...], and Craig S. Wong

Additional article information

Abstract

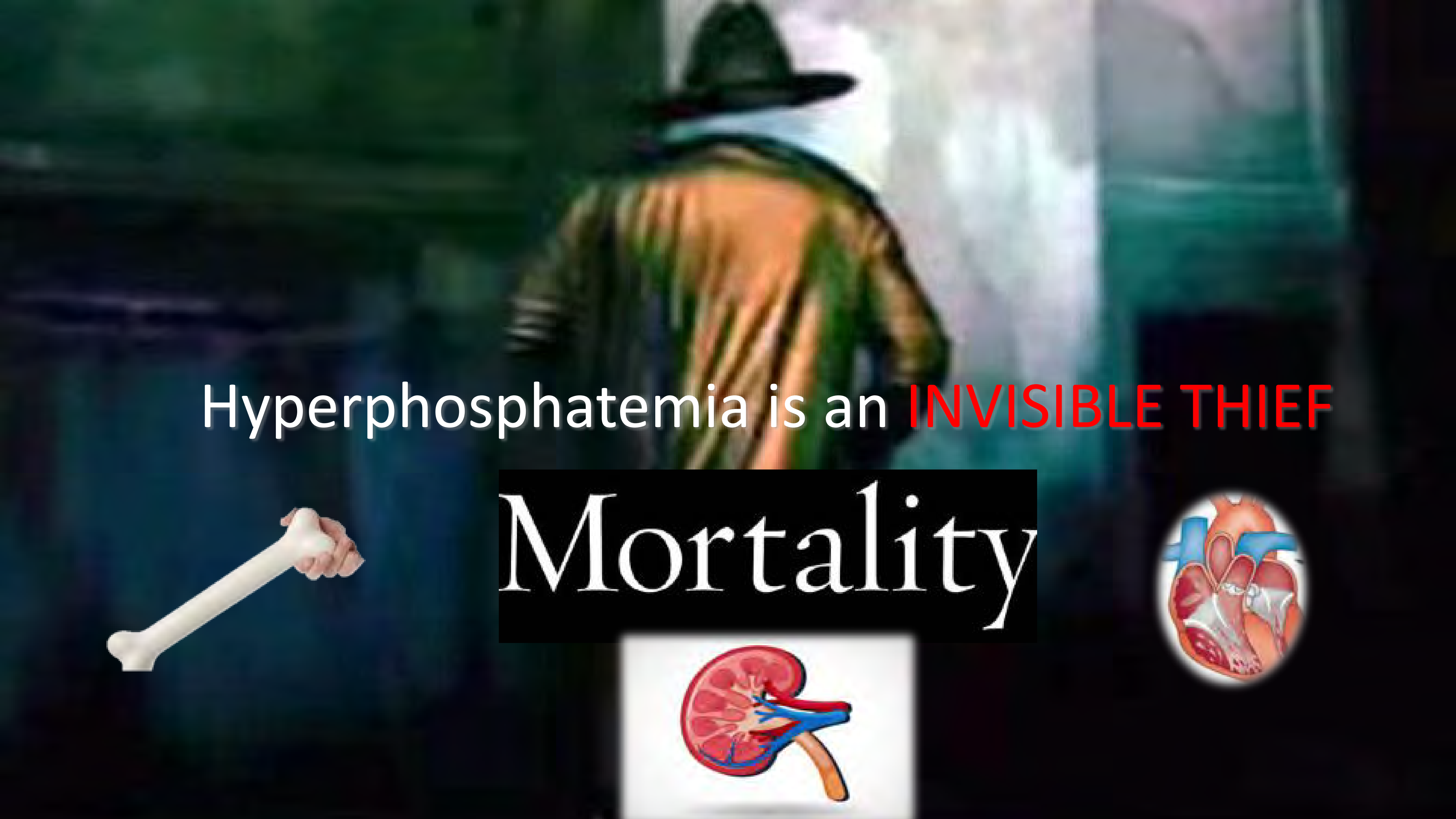
Background and objectives: Children with chronic kidney disease (CKD) have an increased risk of progression to ESRD. There is a need to identify treatments to slow the progression of CKD, yet there are limited data regarding clinical risk factors that may be suitable targets to slow progression.

Design, setting, participants, & measurements: We performed a retrospective cohort study using the North American Pediatric Renal Trials and Cooperative Studies CKD database. There were 4166 pediatric subjects with CKD stages II to IV. Disease progression was defined as a GFR on follow-up of <15 ml/min per 1.73 m^2 or termination in the registry because of dialysis or transplantation. We used Kaplan-Meier and Cox proportional hazards methods to describe progression rates and determine factors associated with CKD progression.

Results: In the univariate analysis, CKD progression was associated with age, gender, race, primary disease, CKD stage, registration year, hematocrit, albumin, corrected calcium, corrected phosphorus, and use of certain medications. Factors that remained significant in the multivariate analysis were age, primary disease, CKD stage, registration year, hypertension, corrected phosphorus, corrected calcium, albumin, hematocrit, and medication proxies for anemia and short stature.

Conclusions: There are multiple risk factors associated with disease progression in the pediatric CKD population. Factors that may be amenable to intervention include anemia, hypoalbuminemia, hyperphosphatemia, hypocalcemia, hypertension, and short stature. Because of the retrospective nature of our study, confirmation of our results from ongoing prospective studies is warranted before recommending prospective interventional trials.

There is an urgent need to identify risk factors and develop new methods to halt chronic kidney disease


Hyperphosphatemia is an INVISIBLE THIEF
Mortality

How do we diagnose hyperphosphatemia?

Diagnosis

When (Grade: 2D)

- Beginning of G2

What (Grade: 3A)

- Ca + P + PTH + ALP

Frequency (Not graded)

	Ca & P	PTH	ALP
G3	6-12 ms	Based on baseline levels &CKD progression	
G4	3-6 ms	6-12 ms	12 ms
G5	1-3 ms	3-6 ms	12 ms

More frequent in established abnormalities and receiving treatment.

(Not graded)

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How do we treat hyperphosphatemia?

Novel Paradigm for Hyperphosphatemia Management in CKD-MBD

Treatments should be based on ***SERIAL*** assessments of P, Ca, PTH, and ALP.

Not graded

Conventional Approach

- Dietary intervention
- Phosphate binders
- Active/analog vitamin D
- Calcimimetics

All 3 ***All lab elements*** ount before making changes to the treatment plan, and first-line drug treatment may include a combination of phosphate binders, vitamin D, and calcimimetics.

2017 KDIGO Guide for Key CKD-MBD Labs	
Lab	**Goal**
Calcium	Avoid hypercalcemia
Phosphorus	Reduce toward the normal range
PTH	2x – 9x the upper limit of normal

Grade: 2C

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Reference values for Phosphate

Table 1 Reference values for phosphate and calcium metabolism in children and adults

Age range	Normal range for calcium (mmol/L)	Normal range for ionized calcium (mmol/L)	Daily recommended intake for calcium (mg)	Normal range for phosphate (mmol/L)	Daily recommended intake for phosphate (mg)
Birth-5 months	2.18–2.83	1.22–1.40	210	1.50–2.40	100
6–12 months	2.18–2.75	1.20–1.40	270	1.50–2.40	275
1–5 years	2.35–2.70	1.22–1.32	500	1.50–2.10	460
6–12 years	2.35–2.58	1.15–1.32	800	1.20–1.90	500 until 8 years, 1250 after
13–20 years	2.20–2.55	1.12–1.30	1300	0.70–1.50	1250

For calcium, the conversion factor from mmol/L to mg/dL is to multiply by 4.0. The calculation formula for corrected calcium (CaC, mmol/L) using measured calcium (mmol/L) and albuminemia (g/L) is the following: $CaC = Ca - 0.25 \times (albuminemia - 40)$. If albuminemia is not available, CaC may be calculated with protidemia (g/L) with the following formula: $Cac = Ca/\ (0.55 + P/16)$. For phosphate, the conversion factor from mmol/L to mg/dL is to multiply by 3.1. These data are adapted from [49]

Novel Paradigm for Hyperphosphatemia Management in CKD-MBD

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Dietary management of hyperphosphatemia

15 pediatric nephrologists & renal dietitians from 8 countries

PRNT

Pediatric Nephrology (2020) 35:501–518
https://doi.org/10.1007/s00467-019-04370-z

GUIDELINES

Check for updates

The dietary management of calcium and phosphate in children with CKD stages 2-5 and on dialysis—clinical practice recommendation from the Pediatric Renal Nutrition Taskforce

Endorsed by IPNA & ESPN

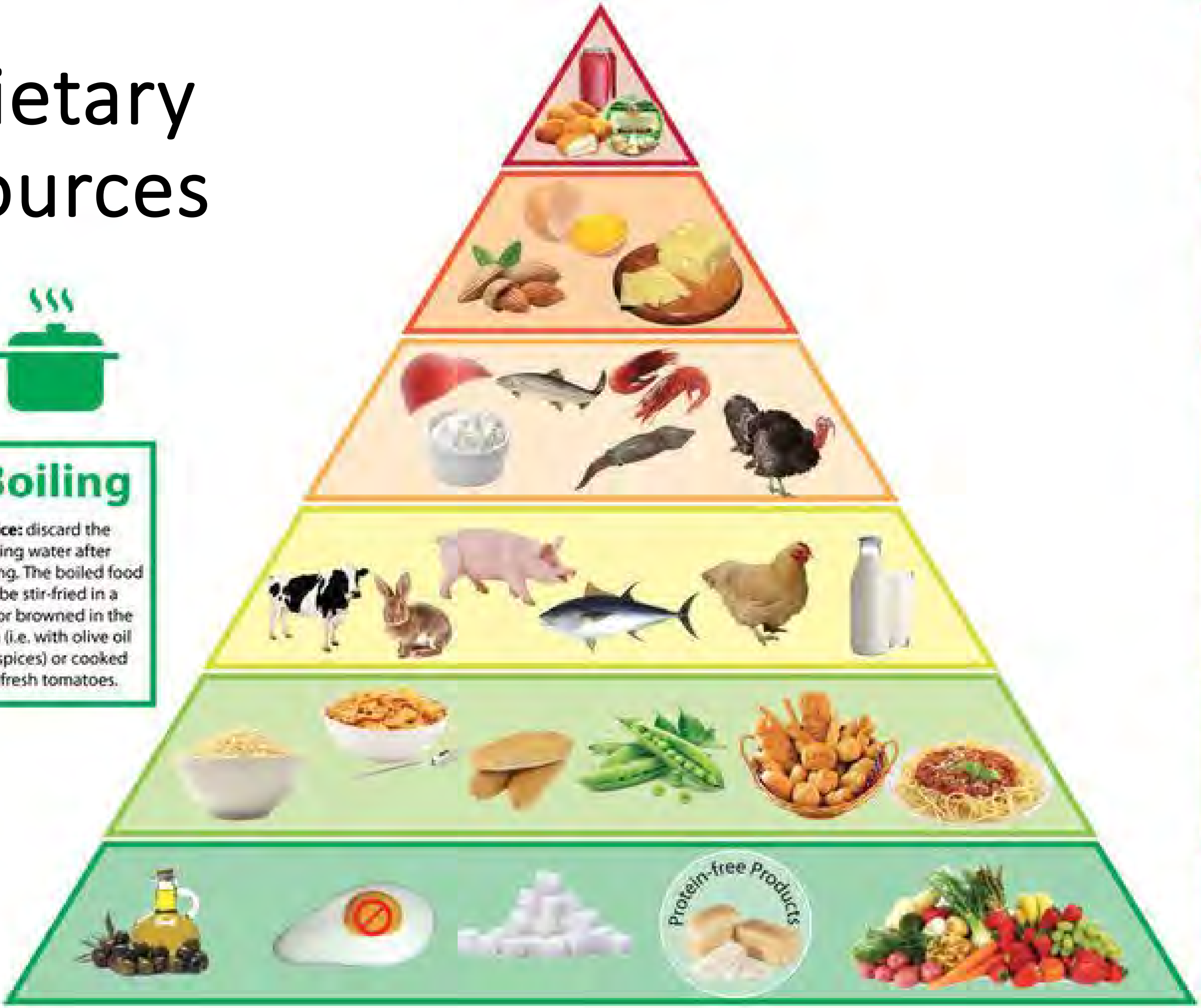
Dietary management of hyperphosphatemia

Dietary management of hyperphosphatemia

Dietary sources

Boiling

Advice: discard the cooking water after boiling. The boiled food may be stir-fried in a pan or browned in the oven (i.e. with olive oil and spices) or cooked with fresh tomatoes.



Beverages and Foods with phosphate-additives (E338-343 E450-458 E540-545): soft drinks (cola in particular), dehydrated milk, processed cheese, processed meat (i.e. chicken nuggets), dessert, instant cappuccino…

Hard cheeses: parmesan, cheddar, emmentaler, pecorino…
Nuts
Yolk

Meat (a): sausages, offal (liver, brain)…
Poultry (a): turkey…
Fish (a): shrimp, squid, salmon…
Soft cheeses: cottage, cream, mozzarella cheese…

Meat (b): rabbit, lamb, ham with no preservatives, pork, veal…
Poultry (b): chicken…
Fish (b): trout, tuna fish, cod, hake, sole…
Milk , yogurt…

Cereals: bread, pasta, rice, cous cous, maize flour, cornflakes…
Legumes: peas, broad beans, beans, chickpeas, lentils, soy…

Egg white
Fruits and vegetables (c)
Olive oil and vegetables fats (d) (i.e. vegetable margarine, corn oil, peanut oil…)
Butter (d)
Sugar (e)
Protein-free products (f)

Main dietary sources of P in children

(Ungraded)

Table 3 Percentage contribution of food types to average daily intake of phosphate (P)

% Total dietary P intake

Food group	Age (years)			
	4–6	7–10	11–14	15–18
Cereal (grain) and cereal products	24	27	26	24
Milk and milk products	35	29	25	23
Eggs and egg dishes	1	2	2	2
Meat and meat products	15	17	19	20
Fish and fish dishes	3	3	3	3
Vegetables, potatoes, and savory snacks	11	12	13	14
Fruit and nuts	2	2	1	1
Sugars, preserves, and confectionary	3	3	4	3

Adapted from National Diet and Nutrition Survey (1995 and 2000)

Main dietary sources of P in children

(Ungraded)

Table 4 Average content of phosphate in some usual food products

Food	Portion (g)	Phosphate quantity (mg)
Beaufort, Parmesan, dry goat cheese	30	240
Cheese with cooked pressed dough	30	192
Gouda, Edam, Morbier	30	156
Comté, Mimolette	30	204
Soft cheese (Camembert)	30	132
Soft cheese (St Marcellin ou St Félicien)	30	55
Fresh goat cheese	30	46
Cream cheese (Petit suisse)	100	126
Yogurt	125	115
Cooked fish	100	226
Cooked chicken	100	223
Cooked meat	100	213
Liver	50	212
Egg	100 = 2 œufs	204
Crustaceans	100	150
Pulses and legumes (cooked)	100	140
Muesli	50	157
Brazil nuts	30 g = a handful	197
Walnuts, cashews, almond	20 g = a handful	90
Hazelnut, Pecan nut	20 = a handful	54
Nut spread	15 = a tea spoon	28
Chocolate (milk or dark)	10	24
Cola	200 mL = a glass	20

Unseen sources of P in children

(Ungraded)

Preservatives

Unseen sources of P in children

(Ungraded)

Preservatives

Table 3 Main food additives containing phosphate

Name of additive	Food where the additive can be found	Function of the additive
Orthophosphoric acid (E338)	Cola	Acidification
Sodium orthophosphate (E339)	Pizza, food preparation as « preparation bags» for desserts	Anti-oxidation, acidification, texture
Potassium orthophosphate (E 340)	Cappuccino, soja drink, dessert cream	Acidification regulation, texture, water retention
Calcium orthophosphate (E 341)	Dairy products	Anti-oxidation, stabilization, firming agent
Magnesium orthophosphate (E 343)	Butter, ice cream, breakfast cereals, appetizers	Anti-oxidation, anti-agglomeration, thickening agent, emulsifier
Diphosphate (E 450)	Soft cheese	Modification of the repartition between fat and proteins in the cheese
Triphosphate (E 451)	Chocolate powder	
Polyphosphate (E 452)	Ham	Water retention
Other food additives containing phosphate: E 442, E 626–635, E 101, E 1410, E 1412, E 1413, E 1414, E 1415 and E 541	Cacao and chocolate desserts/chocolate-based sweets	Emulsifier, binding agent, modified starch

Unseen sources of P in children

(Ungraded)

Medications

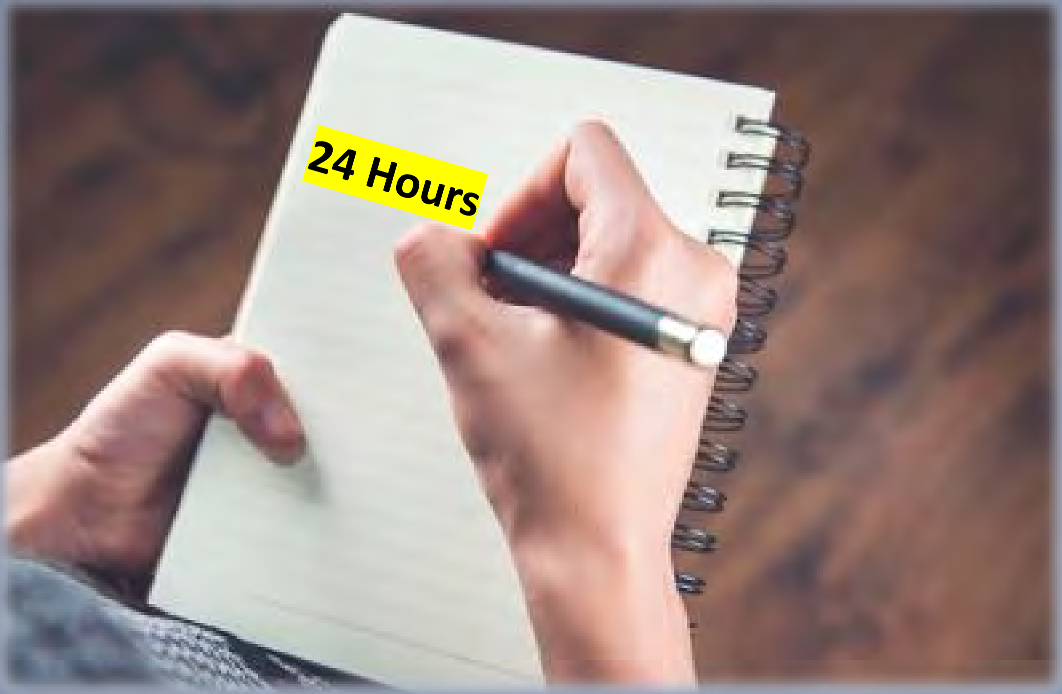
A dearth of data: the problem of phosphorus in prescription medications

- 200 of the most widely prescribed medications in Dialysis Clinic centers in the United States Was examined , found that 23 (11.5%) contained phosphorus.
- The phosphorus content of a generic 10 mg lisinopril (32.6 mg) and a generic 10 mg amlodipine (40.1 mg).

Dietary management of hyperphosphatemia

(Grade C)

Assessment of intake



Dietary management of hyperphosphatemia

Determination of requirements

It is suggested that the dietary P intake of children with CKD should be *within the SDI for age*, without compromising adequate nutrition.

(Grade C)

Suggested dietary intake

Table 7 Summary of SDI (suggested dietary intake) for calcium and phosphate in children with CKD2-5D

Age (years)	SDI calcium (mg)	SDI phosphate (mg)
0–<4 months	220	120
4–<12 months	330–540	275–420
1–3 years	450–700	250–500
4–10 years	700–1000	440–800
11–17 years	900–1300	640–1250

For children with poor growth, reference to the SDI for height age may be appropriate. This is the age that corresponds to their height when plotted at the 50th centile on a growth chart

Dietary management of hyperphosphatemia

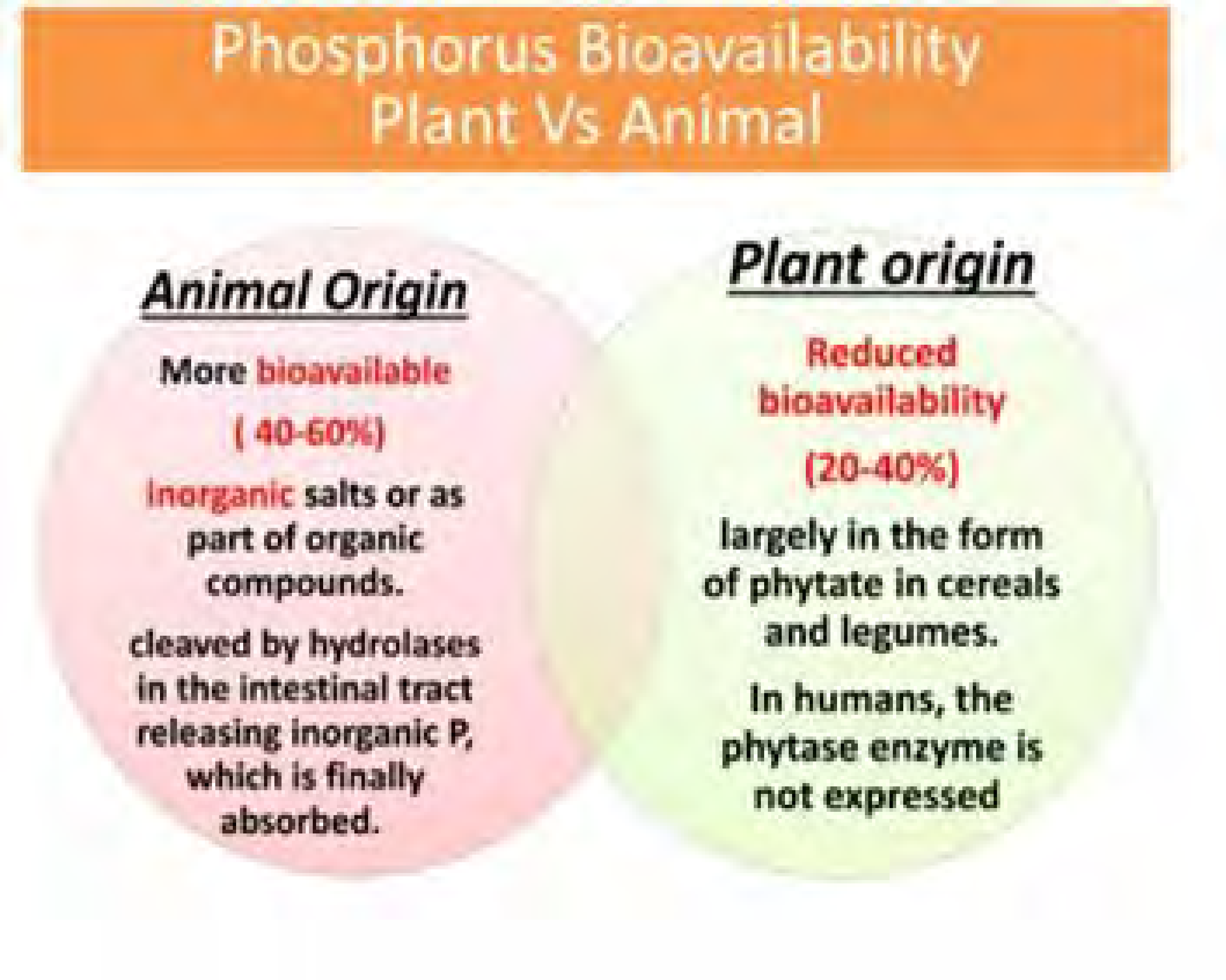
Management of requirements

It is suggested that in children with CKD who have hyperphosphatemia will require further dietary restriction of P, potentially to *the lower limit of SDI for age*, without compromising adequate nutrition.

(Grade C)

Q u a n t i t y

Management of requirements



S
o
u
r
c
e

Management of requirements

Inorganic P

90-100%
Bioavailability

S
o
u
r
c
e

Management of requirements

Table 4 Average content of phosphate in some usual food products

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Low P/protein ratio

Adequate protein content

Source

Management of requirements

Egg

The yolk contains most of the P (largely as phospholipids) with a small amount of protein, while the white part of the egg contains protein (3.7 g for one egg white) with a nearly absent P content.

The egg white is, therefore, a natural source of protein of high biological value, almost free of P.

Low P/protein ratio

Adequate protein content

Source

Management of requirements

Food Science & Nutrition

Maillard reaction products and potatoes: have the benefits been clearly assessed?

Boiling for 30 min reduced phosphorus content up to :

- 42% in beef
- 63 % in chicken breast
- 65% in potato
- 93% in pasta
- 77% in rice

Method of processing :

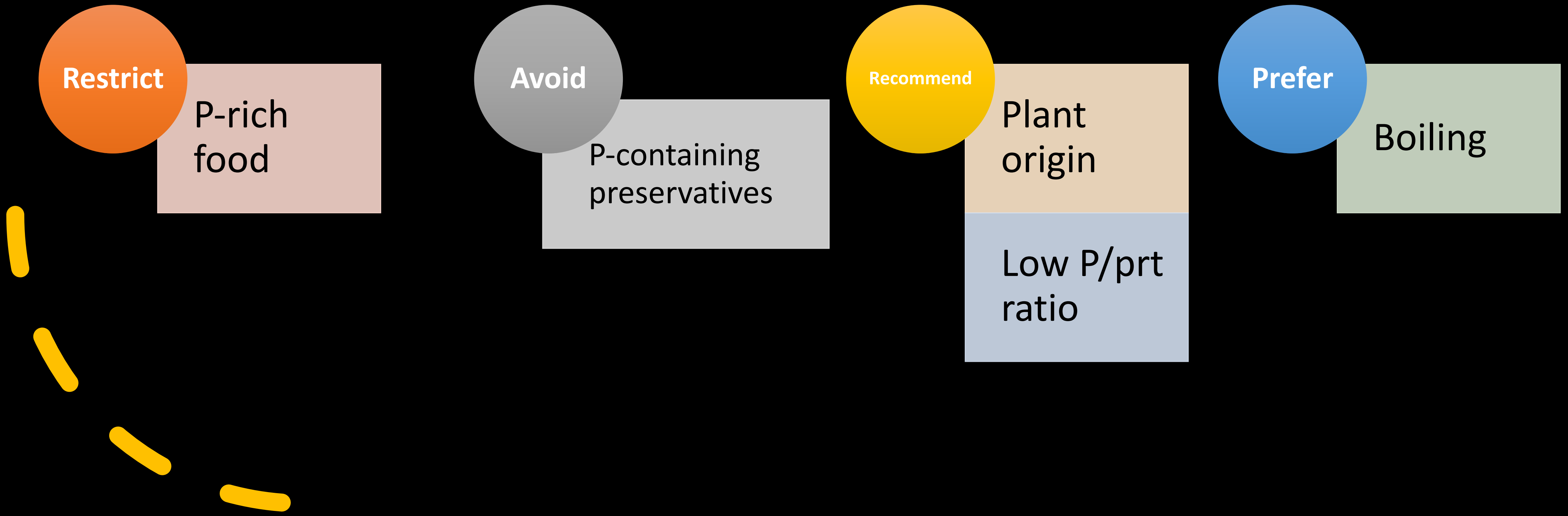
- Frying
- Roasting
- Grilling

reduced phosphorus digestibility and increases fecal excretion of phosphorus in men.

Food Science & Nutrition 2016; 4(2): 234-240

Food Preparation

Management of requirements



Novel Paradigm for Hyperphosphatemia Management in CKD-MBD

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Not graded

Conventional Approach

Dietary intervention

↓

Phosphate binders

↓

Active/analog vitamin D

↓

Calcimimetics

All 3 key labs should be taken into account before making changes to the treatment plan, and first-line drug treatment may include a combination of phosphate binders, vitamin D, and calcimimetics.

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Grade: 2C

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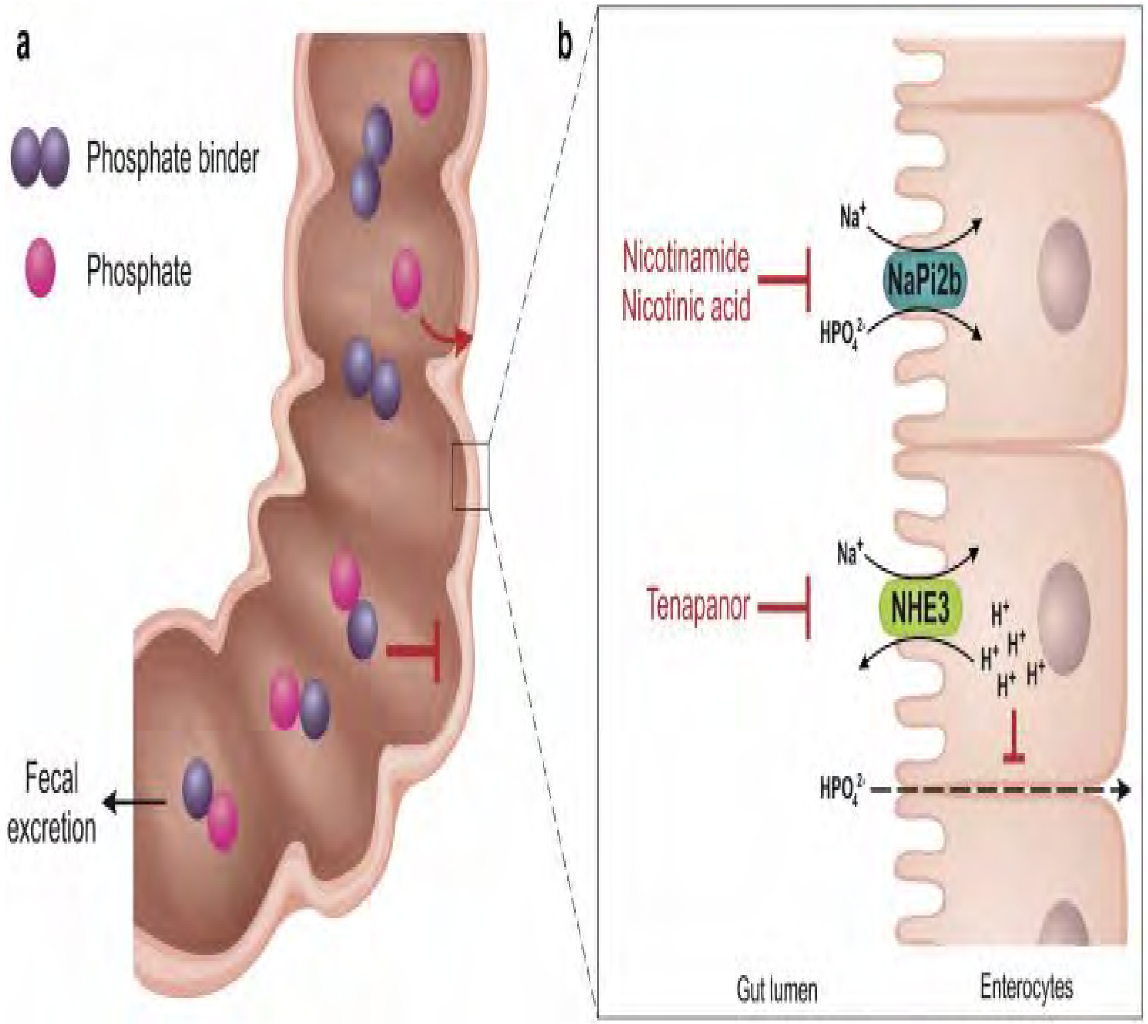
Pharmacological management of hyperphosphatemia

Phosphate binders

Phosphate binders

Mechanism of action



Phosphate binders

Classification

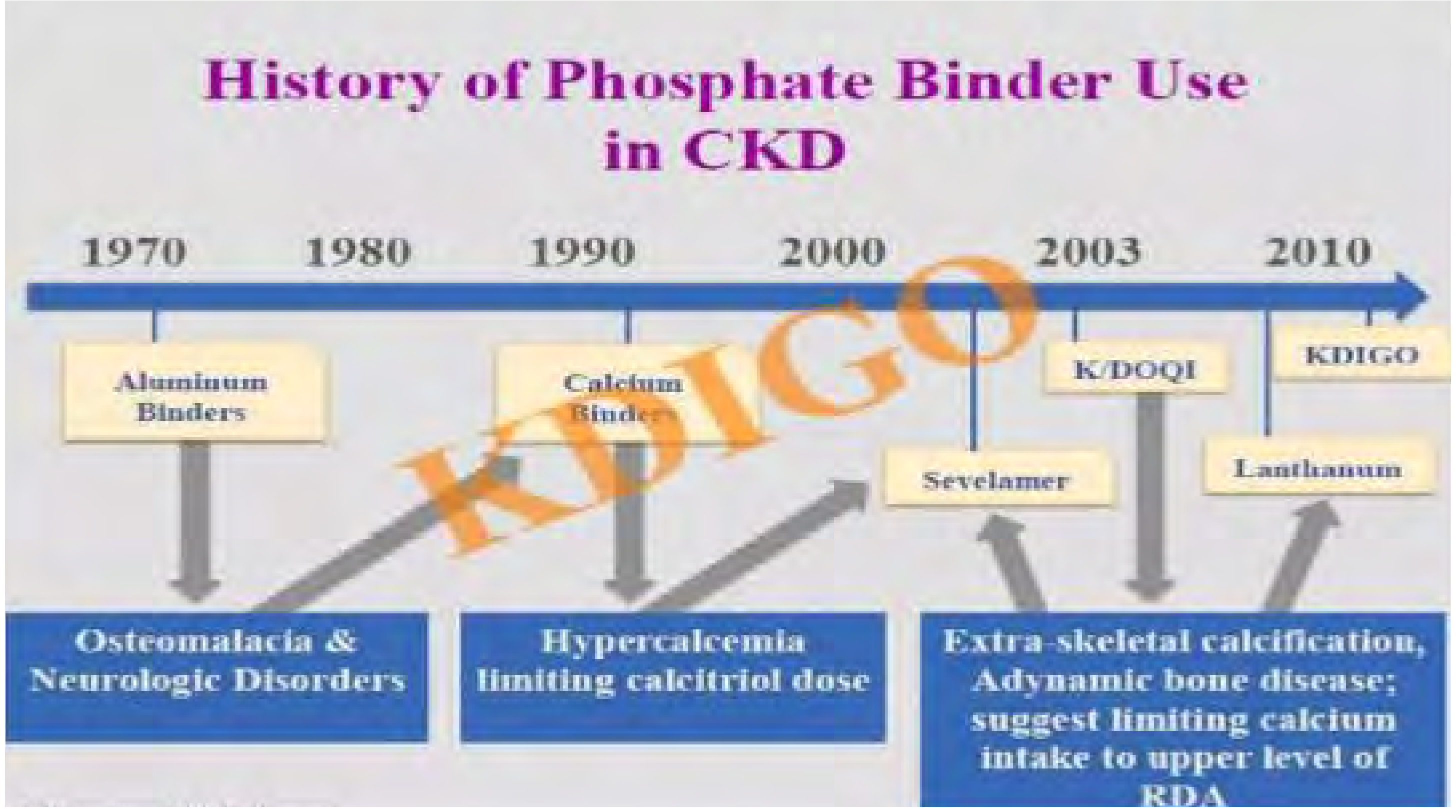
Table 2. Comparison of Common Phosphate Binding Oral Agents in Chronic Kidney Disease

Phosphate Binder	Pros	Cons
Calcium-based: calcium acetate calcium carbonate calcium citrate	• Increases calcium and can correct hypocalcemia • Low cost • Moderate pill burden	• Hypercalcemia and/or positive calcium balance • Cardiovascular calcification
Sevelamer-based: sevelamer carbonate sevelamer hydrochloride	• No systemic absorption • Potentially less vascular calcification (calcium-free) • Lowers LDL cholesterol • Improvement in metabolic acidosis with carbonate variant	• Adverse GI effects • High pill burden • High cost • Binds fat-soluble vitamins • Metabolic acidosis with the hydrochloride variant
Iron-based: sucroferric oxyhydroxide	• Lower pill burden • Minimal systemic absorption, no iron overload • Greater efficacy • Increased GI motility which might be beneficial in constipated and PD patients	• High cost
Iron-based: ferric citrate	• Noninferior to sevelamer, well tolerated, beneficial effect on renal anemia	• Systemic absorption with potential for iron overload
Lanthanum carbonate	• Twice as potent as calcium and sevelamer	• High cost • Systemic absorption and potential tissue deposition/toxicity • GI intolerance, nausea • Difficult to chew

GI, gastrointestinal; LDL, low-density lipoprotein; PD, peritoneal dialysis.

Phosphate binders

Classification



Phosphate binders

WHEN?

Decisions about phosphate lowering therapies should be based on progressively or persistently increased phosphate levels.

(Not graded)

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Phosphate binders

WHICH?

In children with CKD G3a–G5D, it is reasonable to base the choice of phosphate-lowering treatment on serum calcium levels.

(Not graded)

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Phosphate binders

HOW MUCH?

They should not be used as a fixed dose, but should be adjusted to reflect the P content of a meal or snack. **(Fissell et al., Hemodial Int, 2016)**

Phosphate binders

HOW MUCH?

Approximate potential phosphate binding capacities of commonly used agents:

- Calcium carbonate 1 g binds 40 mg
- Calcium acetate 1 g binds 45 mg
- Sevelamer 1 g binds 36 mg
- Lanthanum carbonate 1 g binds 93 mg
- Aluminum hydroxide (liquid) 1 g binds 25 mg

Phosphate binders

HOW?

All binders should be taken with food to achieve maximum efficacy and avoid unwanted side effects.

(Chan et al., Aus Prescr, 2017)

Active D

It is suggested that in patients with CKD G3-5, not on dialysis, VD deficiency should be evaluated whenever iPTH levels are progressively rising or persistently above UNL. VD deficiency should be treated by native VD, with a target of circulating 25-D levels ranging between 20–30 ng/ml (2C).

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Active D

CKD G3a–G5 not on dialysis

- In children: Calcitriol and vitamin D analogues may be considered to maintain serum calcium levels in the age-appropriate normal range (Not Graded).

- In adult: We suggest that calcitriol and vitamin D analogues not be routinely used (2C). It is reasonable to reserve the use of calcitriol and vitamin D analogues for patients with CKD G4–G5 with severe and progressive hyperparathyroidism (Not Graded).

CKD G5D

- We suggest maintaining iPTH levels in the range of approximately 2 to 9 times the upper normal limit for the assay (2C).

- In patients requiring PTH-lowering therapy, we suggest calcimimetics, calcitriol, or vitamin D analogues, or a combination of calcimimetics with calcitriol or vitamin D analogues (2B)

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Dialytic management of hyperphosphatemia


HEMODIAFILTRATION
OSMOSIS.org


DIALYSIS


HAEMODIALYSIS

Table 1. A Comparison of Phosphorus Removal Between Dialysis Modalities

Modality	Frequency	Phosphorus Removal (mg/wk)
Conventional HD	3 × 4 h	1,572 ± 366
Extended HD	3 × 5 h	3,400 ± 647
Short daily HD	6 × 3 h	2,452 ± 720
Nocturnal daily HD	6 × 6-8 h	8,000 ± 2,800
CAPD	24.0 h*	2,790 ± 1,022
APD, CCPD	18.5 ± 7.3 h*	2,739 ± 1,042

- **Almost 50% of HD patients have serum P > recommended treatment targets despite the use of phosphate lowering agents and dietary recommendations.**
- **Children treated by conventional dialysis regime still have an increased risk of cardiovascular morbidity.**

Groothoff et al. Kidney Int. 2002; 61:621–629.
Foley et al. J Am Soc Nephrol. 1998; 9:S16–S23.

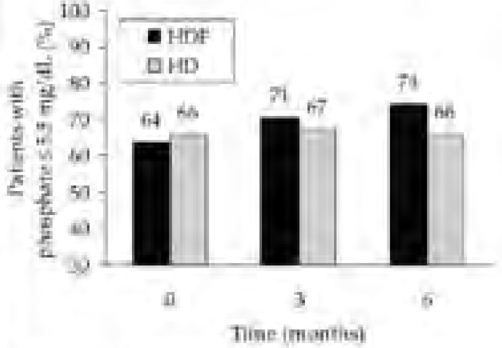

Figure 3. Proportion of patients achieving phosphate treatment targets at baseline and after 3 or 6 months of follow-up. Numbers above bars represent percentages. *$P < 0.05$ (vs baseline); †$P < 0.05$ (difference in change between groups). Abbreviations: HD, hemodialysis; HDF, hemodiafiltration.

HEMODIAFILTRATION

Table. 4. Effects of HDF and HD on Bone Mineral and Nutritional Parameters Over Time

	Baseline	3 mo	6 mo	Difference Baseline vs 3 mo	Difference Baseline vs 6 mo
Bone mineral parameters					
Phosphate (mg/dL)					
HDF	5.19 (4.99 to 5.39)	4.90 (4.70 to 5.09)*	4.87 (4.68 to 5.06)*	+0.29 (0.09 to 0.48)*	+0.31 (0.12 to 0.50)*
HD	5.10 (4.92 to 5.28)	5.08 (4.89 to 5.27)	5.03 (4.84 to 5.22)	+0.03 (−0.16 to 0.22)	+0.07 (−0.13 to 0.27)
Difference HDF vs HD	+0.09 (−0.16 to 0.35)	−0.18 (−0.45 to 0.09)	−0.17 (−0.44 to 0.11)		

Dialysis

Short-term Effects of Online Hemodiafiltration on Phosphate Control: A Result From the Randomized Controlled Convective Transport Study (CONTRAST)

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Background: Hyperphosphatemia is an independent risk factor for all-cause and cardiovascular mortality in hemodialysis (HD) patients. Phosphate control often is unsuccessful using conventional dialysis therapies.

Study Design: Short-term analysis of a secondary outcome of an ongoing randomized controlled trial.

Setting & Participants: 493 (84%) consecutive patients from 589 patients included in the Convective Transport Study (CONTRAST) by January 2009 from 26 centers in 3 countries.

Intervention: Online hemodiafiltration (HDF) versus continuation of low-flux HD.

HEMODIAFILTRATION

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The effect of dialysis modality on phosphate control: haemodialysis compared to haemodiafiltration–The Pan Thames Renal Audit

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Abstract

Background and Objectives. Hyperphosphataemia is a primary risk factor for patients with end-stage kidney failure. Phosphate clearance by traditional thrice-weekly standard haemodialysis is inadequate for patients achieving recommended dietary protein goals. We investigated whether phosphate control was improved by adding convective clearance with haemodiafiltration.

Methods. We audited pre-midweek session calcium and

This has led to the concept of non-traditional risk factors for mortality in CKD5 patients. As patients develop progressive kidney disease and lose kidney function, renal phosphate clearance declines, resulting in phosphate retention. Recently, hyperphosphataemia secondary to phosphate retention has been shown to be an independent risk factor for CKD5 dialysis patient survival [3].

If patients achieve the nutritional guidelines recommended for CKD5 patients, then their dietary phosphate

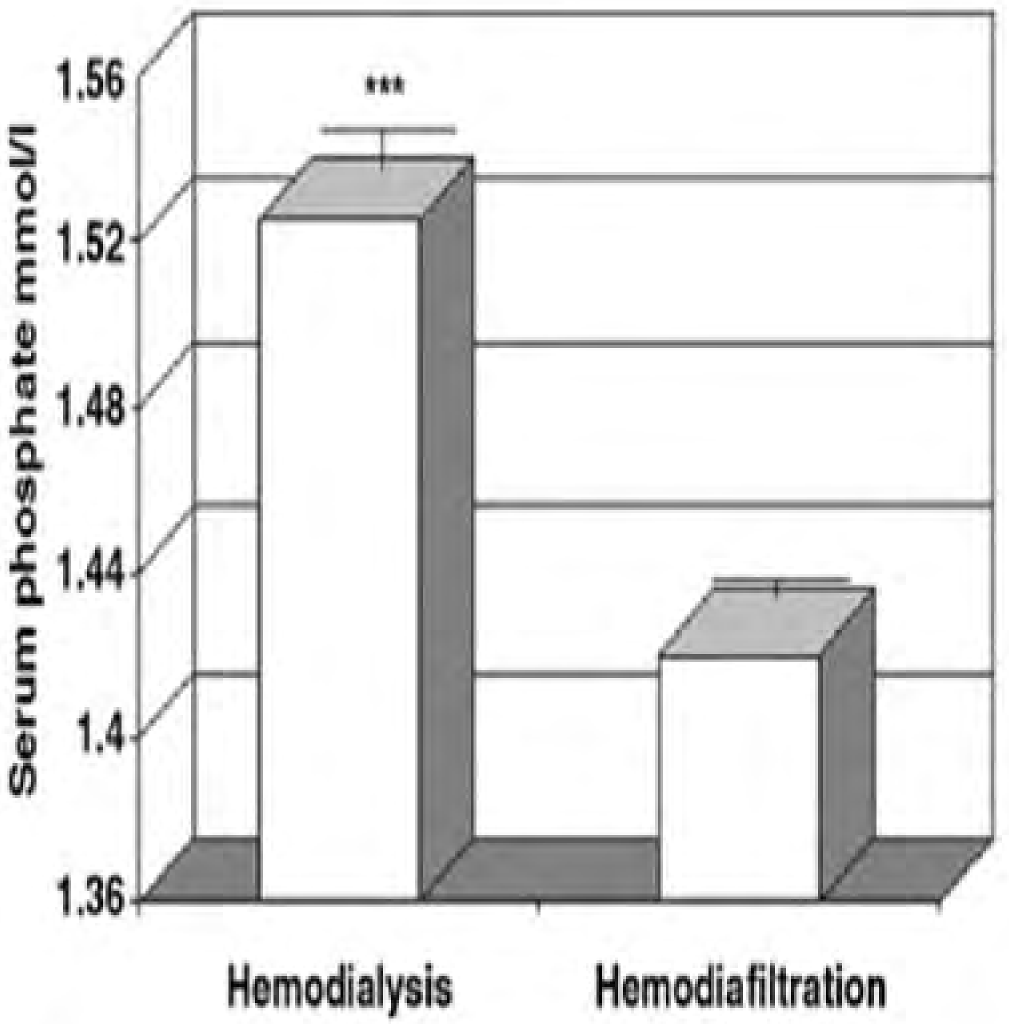
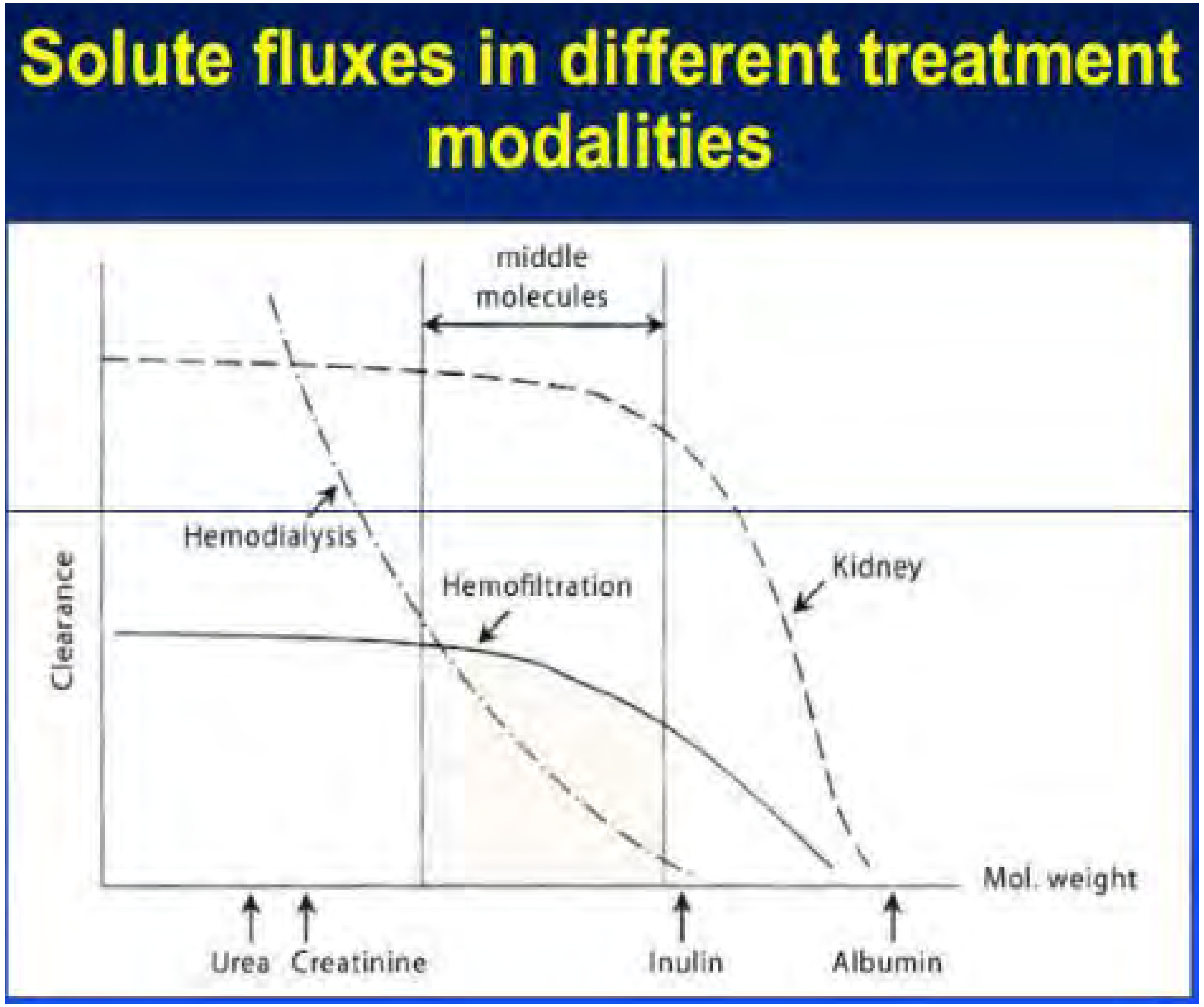


Fig. 1. Serum phosphate in hemodialysis and hemodiafiltration cohorts. Data expressed as mean (SEM). ***$P < 0.001$.

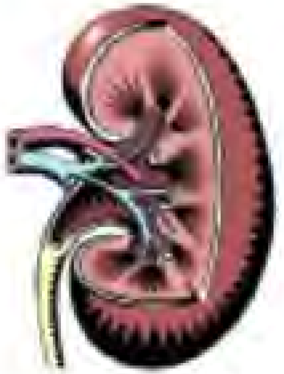

HD
HDF


Solute fluxes in different treatment modalities
middle molecules
Hemodialysis
Hemofiltration
Kidney
Clearance
Mol. weight
Urea
Creatinine
Inulin
Albumin

Non-dietary management

Source of High Phosphorus

Treatments and Limitations

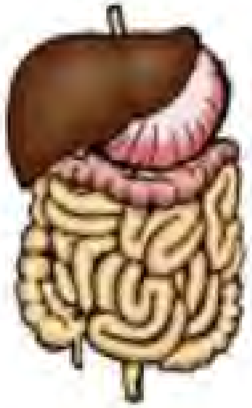


KIDNEY

Loss of kidney function and impaired renal excretion of phosphorus

Regular dialysis:
Dialyzer removes phosphorus from the blood

- Dialysis removal not sufficient to reach target range



GUT

Dietary phosphorus absorption

Dietary changes:
Reduce intake of phosphorus and phosphate additives

- Increased protein requirement necessitates dietary phosphorus

Phosphate binders:
Reduce phosphorus absorption

- High pill burden and adverse GI effects

BONE

Bone resorption releases stored phosphorus

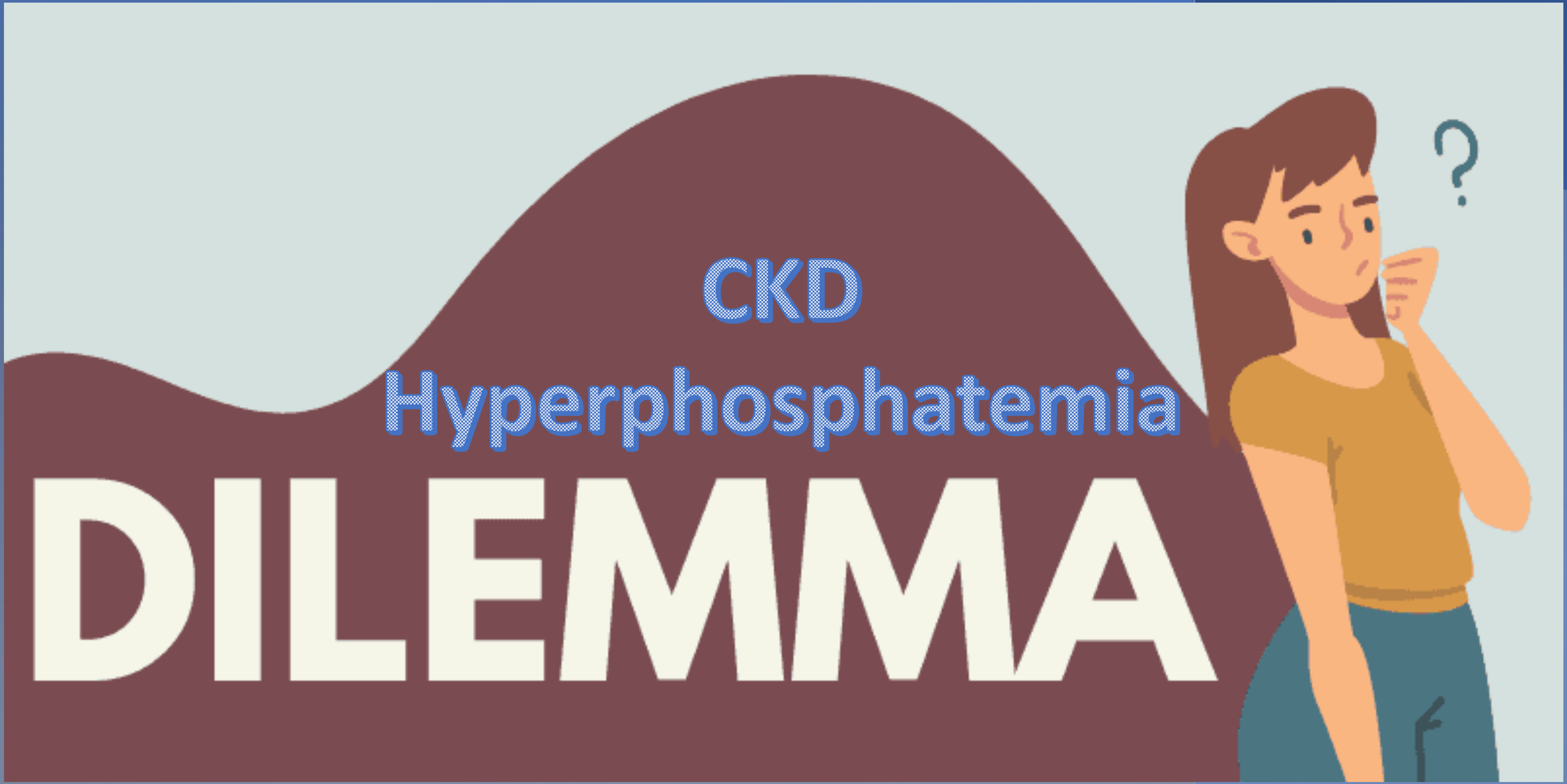
Vitamin D:
Increases calcium and suppresses PTH

- Can increase phosphorus absorption from gut

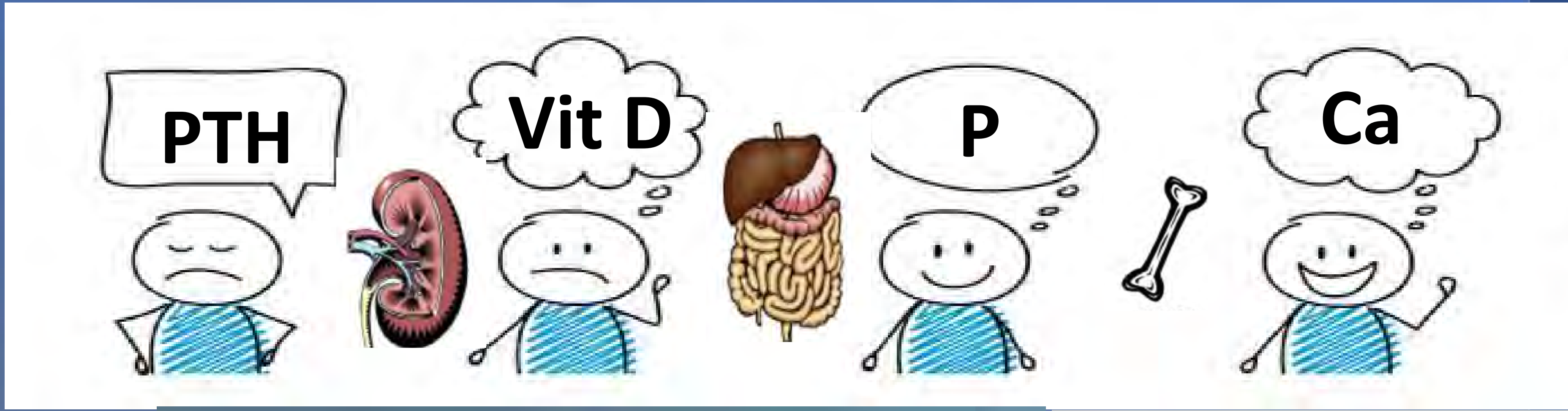
Calcimimetics:
Suppress PTH-induced bone turnover and phosphorus release

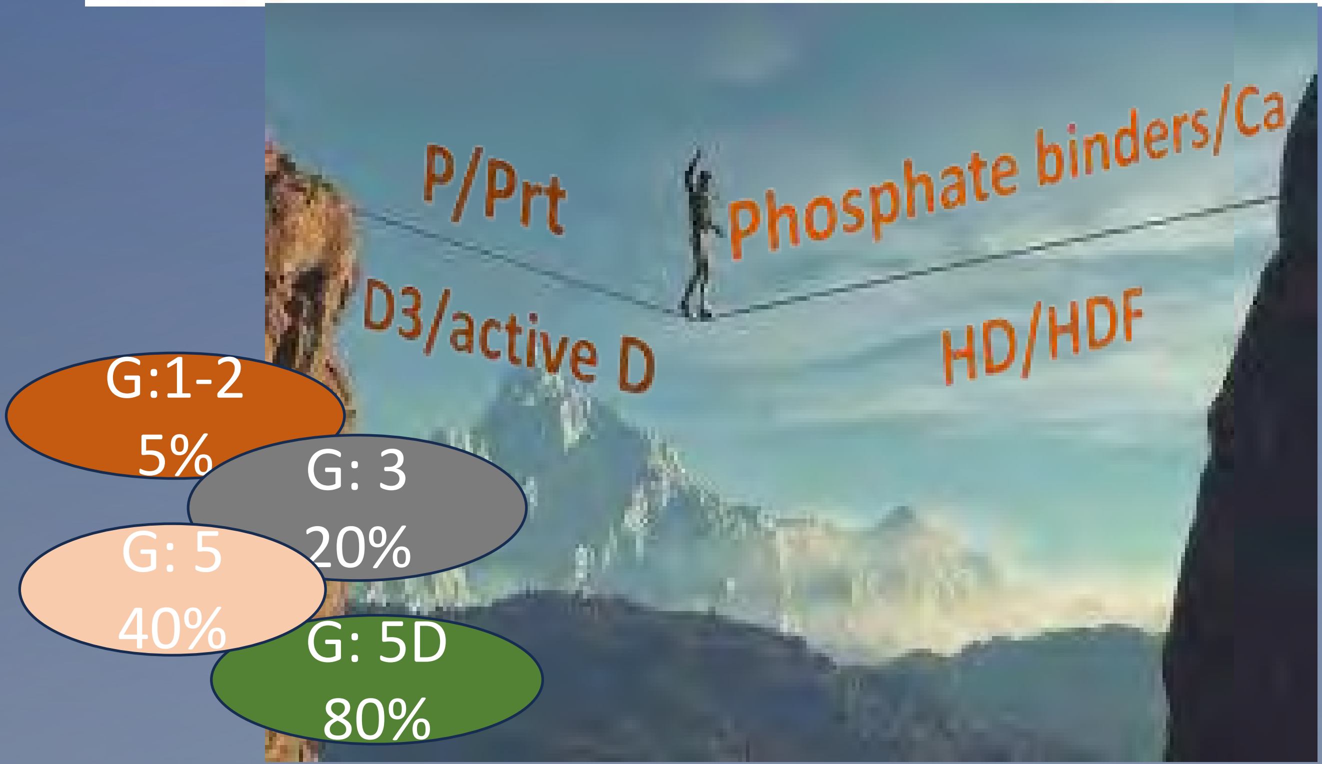
- Possible hypocalcemia and GI symptoms

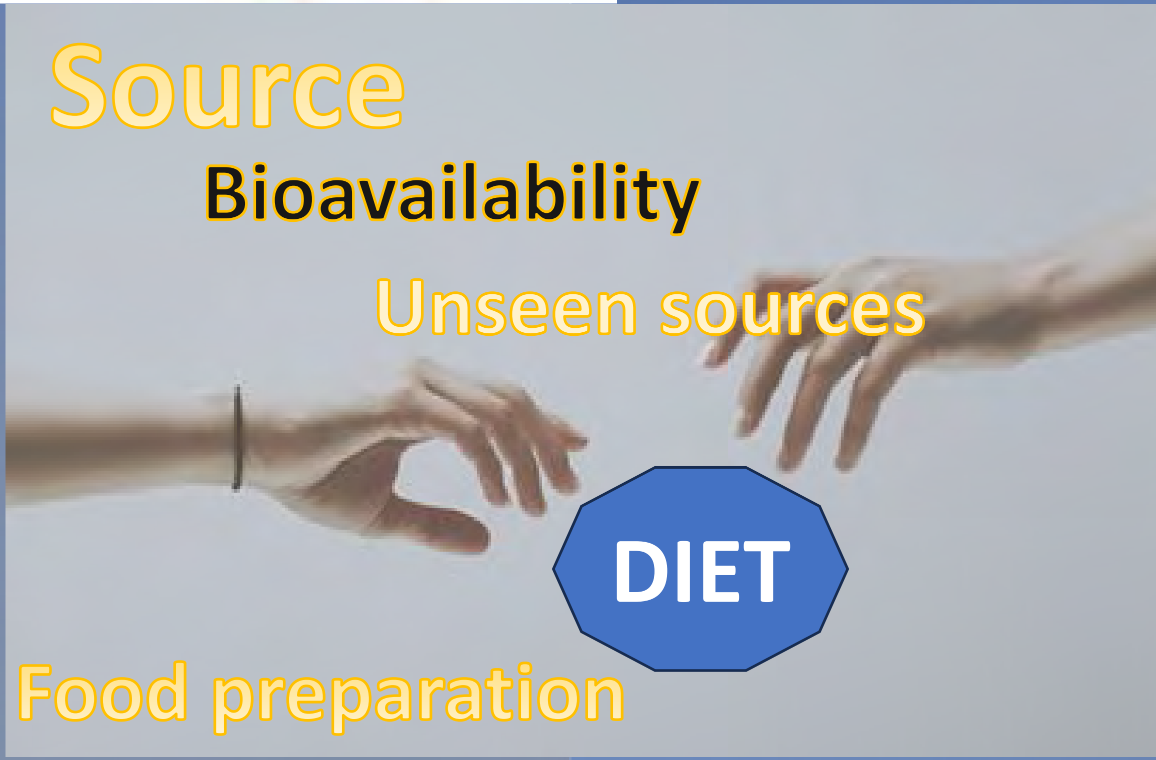
CKD Hyperphosphatemia



PLAYERS







Education

CHALENGES

INFLUENTIALS


Thank you

Hyperphosphatemia in children with CKD:

a. It starts earlier than in adults with CKD.
b. It occurs secondary to Hyperparathyroidism only.
c. It is efficiently managed by conventional hemodialysis.
d. None of the above.

In children with advanced CKD and hyperphosphatemia, it is important to:

a. Limit dietary P intake to within the SDI.
b. Preferer food with high protein/phosphate.
c. Recommend animal origin than plant origin protein.
d. None of the above.

In the pharmacologic treatment of hyperphosphatemia in children with CKD:

a. P binders should be started early.
b. Sevelamer HCL is preferred than ca carbonate.
c. Active D is routinely given in CKD-stage 4.
d. None of the above.