

All about Phosphorous

<http://www.aakp.org/>.

Phosphorus is an essential mineral that is required by every cell in the body for normal function. The majority of the phosphorus in the body is found as phosphate (PO_4). Approximately 85% of the body's phosphorus is found in the bone.

Function

Phosphorus is a major structural component of bone in the form of a calcium phosphate salt called hydroxyapatite. Phospholipids (e.g., phosphatidylcholine) are major structural components of cell membranes. All energy production and storage are dependent on phosphorylated compounds, such as adenosine triphosphate (ATP) and creatine phosphate. Nucleic acids (DNA and RNA), which are responsible for the storage and transmission of genetic information, are long chains of phosphate-containing molecules. A number of enzymes, hormones, and cell-signaling molecules depend on phosphorylation for their activation. Phosphorus also helps to maintain normal acid-base balance (pH) by acting as one of the body's most important buffers. Additionally, the phosphorus-containing molecule 2,3-diphosphoglycerate (2,3-DPG) binds to hemoglobin in red blood cells and affects oxygen delivery to the tissues of the body.

Nutrient interactions:

Fructose

A study of 11 adult men found that a diet high in fructose (20% of total calories) resulted in increased urinary loss of phosphorus and a negative phosphorus balance (i.e., daily loss of phosphorus was higher than daily intake). This effect was more pronounced when the diet was also low in magnesium. A potential mechanism for this

effect is the lack of feedback inhibition of the conversion of fructose to fructose-1-phosphate in the liver. In other words, fructose-1-phosphate accumulates in the cell but this compound does not inhibit the enzyme that phosphorylates fructose, which consumes large amounts of phosphate. This phenomenon is known as phosphate trapping.

Calcium and vitamin D

Dietary phosphorus is readily absorbed in the small intestine, and any excess phosphorus absorbed is excreted by the kidneys. The regulation of blood calcium and phosphorus levels is interrelated through the actions of parathyroid hormone (PTH) and vitamin D. A slight drop in blood calcium levels (e.g., in the case of inadequate calcium intake) is sensed by the parathyroid glands, resulting in their increased secretion of PTH. PTH stimulates conversion of vitamin D to its active form (calcitriol) in the kidneys. Increased calcitriol levels in turn result in increased intestinal absorption of both calcium and phosphorus. Both PTH and vitamin D stimulate bone resorption, resulting in the release of bone mineral (calcium and phosphate) into the blood. Although PTH stimulation results in decreased urinary excretion of calcium, it results in increased urinary excretion of phosphorus. The increased urinary excretion of phosphorus is advantageous in bringing blood calcium levels up to normal because high blood levels of phosphate suppress the conversion of vitamin D to its active form in the kidneys.

What are the Role of Phosphorus and Vitamin D Analogs in the Pathogenesis of Vascular Calcification?

Vascular calcification is a mortality risk factor for stage 5 chronic kidney disease patients. The role of phosphorus and vitamin D analogs in the pathogenesis of vascular calcification using in vivo, ex vivo, and in vitro models was investigated. The results demonstrate that subjects receiving a hyperphosphatemia-inducing diet did not exhibit aortic calcification despite elevated levels of serum

phosphorus and calcium-phosphorus (Ca xP) product. The vitamin D analog 1 α -hydroxyvitamin-D₂ [1 α (OH)D₂] at 0.17 μ g/kg raised serum calcium, phosphorus, Ca xP product, and aortic calcification in the subjects, but 19-nor-1 α ,25(OH)₂D₂ (19-nor) at the same dose had no significant effect. At 0.67 μ g/kg, both 1 α (OH)D₂ and 19-nor had similar effects on serum calcium, phosphorus, and Ca xP product, but only 1 α (OH)D₂ induced significant aortic calcification. Only aortic rings from 1 α (OH)D₂-treated subjects exhibited a significant increase in Ca uptake ex vivo. When aortic rings from normal subjects or a primary culture of human coronary artery smooth muscle cells were treated with phosphorus or vitamin D analogs in vitro, high phosphorus induced calcium accumulation and/or Ca uptake in a dose- or time-dependent manner, whereas vitamin D analogs including 1 α (OH)D₂ up to 100 nM had no significant effect despite the presence of a functional vitamin D receptor. However, serum from 1 α (OH)D₂-treated subjects induced Ca uptake into smooth muscle cells cultured in high phosphorus. These results suggest that the regulation of vascular calcification in vivo cannot be easily replicated in the ex vivo or in vitro models, and high phosphorus and some vitamin D analogs such as 1 α (OH)D₂ exert interactive effects on modulating vascular calcification.

Is high phosphorus intake detrimental to bone health?

Some investigators are concerned about the increasing amounts of phosphates in the diet which can be attributed to phosphoric acid in soft drinks and phosphate additives in a number of commercially prepared foods. Because phosphorus is not as tightly regulated by the body as calcium, serum phosphate levels can rise slightly with a high phosphorus diet, especially after meals. High phosphate levels in the blood reduce the formation of the active form of vitamin D (calcitriol) in the kidneys, reduce blood calcium, and lead to increased PTH release by the parathyroid glands. However, high serum phosphorus levels also lead to decreased urinary calcium excretion. If sustained, elevated PTH levels could have an adverse effect on bone mineral content, but this effect has only been observed in humans on diets that were high in phosphorus and low in calcium. Moreover,

similarly elevated PTH levels have been reported in diets that were low in calcium without being high in phosphorus. Recently, a controlled trial in young women found no adverse effects of a phosphorus-rich diet (3,000 mg/day) on bone-related hormones and biochemical markers of bone resorption when dietary calcium intakes were maintained at almost 2,000 mg/day. At present, there is no convincing evidence that the dietary phosphorus levels adversely affect bone mineral density. However, the substitution of phosphate-containing soft drinks and snack foods for milk and other calcium rich foods does represent a serious risk to bone health.

Calcium-Phosphorus Ratio

Understanding the Calcium-Phosphorus Ratio

Many different minerals and other nutrients have interactions between them that affect their availability or absorption in the body--
-for example, between copper and zinc, calcium and vitamin D, iron and manganese. One of the most important, and often overlooked,
of these interactions is between calcium and phosphorus. A saying is
"as goes phosphorus, so goes calcium". What this means is that for every gram of phosphorus ingested in the diet, the body must match that with another gram of calcium before the phosphorus can be absorbed through the intestinal wall into the bloodstream. If the required calcium is not available from the diet, the body will obtain it from wherever it can---such as from the storage depots in the bones. We do NOT want calcium being removed from and decreasing bone integrity.

This is where the whole idea of calcium-phosphorus ratios comes in. The purpose of calculating such a ratio is to make sure that for every gram of phosphorus you are eating, you have to eat AT LEAST an equal amount of calcium (a 1:1 ratio or better), so that calcium is not being continually mobilized from bones. Most nutritionists recommend that the ideal levels are somewhere between 1.2-2 parts

calcium to 1 part phosphorus. Any value less than 1 means the ratio is inverted, and that more phosphorus is being ingested than calcium.

Unfortunately, there are few outwardly visual signs of an inverted calcium-phosphorus ratio. What happens is that the calcium being removed from the bone is replaced by connective tissue--an attempt by the body to create stability and support when calcium-supported bone tissue is not available. Although it is going on throughout the body, it will show up most visibly in the head, with enlargements along the jawbones, especially the upper mandible.

Deficiency

Inadequate phosphorus intake results in abnormally low serum phosphate levels (hypophosphatemia). The effects of hypophosphatemia may include loss of appetite, anemia, muscle weakness, bone pain, rickets (in children), osteomalacia (in adults), increased susceptibility to infection, numbness and tingling of the extremities, and difficulty walking. Severe hypophosphatemia may result in death. Because phosphorus is so widespread in food, dietary phosphorus deficiency is usually seen only in cases of near-total starvation. Other individuals at risk of hypophosphatemia include alcoholics, diabetics recovering from an episode of diabetic ketoacidosis, and starving or anorexic patients on refeeding regimens that are high in calories but too low in phosphorus.

The Recommended Dietary Allowance (RDA)

The recommended dietary allowance (RDA) for phosphorus was based on the maintenance of normal serum phosphate levels in adults, which was believed to represent adequate phosphorus intake to meet cellular and bone formation needs.

Recommended Dietary Allowance (RDA) for Phosphorus			
Life Stage	Age	Males (mg/day)	Females

			(mg/day)
Infants	0-6 months	100 (<u>AI</u>)	100 (AI)
Infants	7-12 months	275 (AI)	275 (AI)
Children	1-3 years	460	460
Children	4-8 years	500	500
Children	9-13 years	1,250	1,250
Adolescents	14-18 years	1,250	1,250
Adults	19 years and older	700	700
Pregnancy	18 years and younger	-	1,250
Pregnancy	19 years and older	-	700
Breast-feeding	18 years and younger	-	1,250
Breast-feeding	19 years and older	-	700

Sources

Food sources

Phosphorus is found in most foods because it is a critical component of all living organisms. Dairy products, meat, and fish are particularly rich sources of phosphorus. Phosphorus is also a component of many polyphosphate food additives and is present in most soft drinks as phosphoric acid. Dietary phosphorus derived from food additives is not calculated in most food databases, so the total amount of phosphorus consumed by the average person is not entirely clear. A large survey of nutrient consumption found that the average

phosphorus intake was 1,495 mg/day in men and 1,024 mg/day in women.

The phosphorus in all plant seeds (beans, peas, cereals, and nuts) is present in a storage form of phosphate called phytic acid or phytate. Only about 50% of the phosphorus from phytate is available to humans because we lack enzymes (phytases) that liberate phosphorus from phytate. Yeasts possess phytases, so whole grains incorporated into leavened breads have more bioavailable phosphorus than whole grains incorporated into breakfast cereals or flat breads. The table below lists a number of phosphorus rich foods along with their phosphorus content in milligrams (mg)..

Food	Serving	Phosphorus (mg)
Milk, skim	8 ounces	247
Yogurt, plain nonfat	8 ounces	385
Cheese, mozzarella; part skim	1 ounce	131
Egg	1 large, cooked	104
Beef	3 ounces, cooked*	173
Chicken	3 ounces, cooked*	155
Turkey	3 ounces, cooked*	173
Fish, halibut	3 ounces, cooked*	242
Fish, salmon	3 ounces, cooked*	252
Bread, whole wheat	1 slice	57
Bread, enriched white	1 slice	25
Carbonated cola drink	12 ounces	40
Almonds [#]	1 ounce (23 nuts)	134
Peanuts [#]	1 ounce	107
Lentils [#]	1/2 cup, cooked	178

*A 3-ounce serving is about the size of a deck of cards.

#Phosphorus from nuts, seeds, and grains is about 50% less bioavailable than phosphorus from other sources.

Supplements

Sodium phosphate and potassium phosphate salts are used for the treatment of hypophosphatemia, and their use requires medical supervision. Calcium phosphate salts are sometimes used as calcium supplements.

Toxicity

The most serious adverse effect of abnormally elevated blood levels of phosphate (hyperphosphatemia) is the calcification of non-skeletal tissues, most commonly the kidneys. Such calcium phosphate deposition can lead to organ damage, especially kidney damage. Because the kidneys are very efficient at eliminating excess phosphate from the circulation, hyperphosphatemia from dietary causes is usually only a problem in people with kidney failure (end-stage renal disease) or hypoparathyroidism. When kidney function is only 20% of normal, even typical levels of dietary phosphorus may lead to hyperphosphatemia. Pronounced hyperphosphatemia has also occurred due to increased intestinal absorption of phosphate salts taken by mouth as well as due to colonic absorption of the phosphate salts in enemas. To avoid the adverse effects of hyperphosphatemia, the Food and Nutrition Board set a tolerable upper intake level (UL) for oral phosphorus intake in generally healthy individuals. The lower UL for individuals over 70 years of age reflects the increased likelihood of impaired kidney function in elderly individuals. The UL does not apply to individuals with significantly impaired kidney function or other health conditions known to increase the risk of hyperphosphatemia.

Tolerable Upper Intake Level (UL) for Phosphorus
--

Age Group	UL (mg/day)
Infants 0-12 months	Not possible to establish*
Children 1-3 years	3,000 (3.0 g)
Children 4-8 years	3,000 (3.0 g)
Children 9-13 years	4,000 (4.0 g)
Adolescents 14-18 years	4,000 (4.0 g)
Adults 19-70 years	4,000 (4.0 g)
Adults 70 years and older	3,000 (3.0 g)
Pregnancy	3,500 (3.5 g)
Breast-feeding	4,000 (4.0 g)

*Source of intake should be from food and formula only.

Drug Interactions

Aluminum-containing antacids reduce the absorption of dietary phosphorus by forming aluminum phosphate, which is unabsorbable. When consumed in high doses, aluminum-containing antacids can produce abnormally low blood phosphate levels (hypophosphatemia) as well as aggravate phosphate deficiency due to other causes. As little as one ounce of aluminum hydroxide gel three times a day for several weeks can diminish serum phosphate levels and lead to increased urinary calcium loss. Excessively high doses of calcitriol, the active form of vitamin D, or its analogs may result in hyperphosphatemia.

Potassium supplements or potassium-sparing diuretics taken together with a phosphate may result in high blood levels of potassium (hyperkalemia). Hyperkalemia can be a serious problem, resulting in life threatening heart rhythm abnormalities (arrhythmias). People taking such a combination must inform their health care provider and have their serum potassium levels checked regularly.

Linus Pauling Institute Recommendation

The Linus Pauling Institute supports the RDA for phosphorus (700 mg/day for adults). Although few multivitamin/multimineral supplements contain more than 15% of the current RDA for phosphorus, a varied diet should easily provide adequate phosphorus for most people.

Older adults (65 years and older)

At present, there is no evidence that the phosphorus requirements of older adults differ from that of younger adults (700 mg/day). Although few multivitamin/multimineral supplements contain more than 15% of the current RDA for phosphorus, a varied diet should easily provide adequate phosphorus for most people.

Causes of high phosphorus: Youth; exercise; dehydration and hypovolemia; high phosphorus content enema; acromegaly; hypoparathyroidism; pseudohypoparathyroidism; bone metastases; hypervitaminosis D; sarcoidosis; milk-alkali syndrome; liver disease, such as portal cirrhosis; catastrophic events such as cardiac resuscitation, pulmonary embolism, renal failure; diabetes mellitus with ketosis; serum artifact - sample not refrigerated; overheated, hemolyzed sample, or serum allowed to remain too long on the clot.

Although phosphate accumulation occurs as renal disease progresses, hyperphosphatemia is not a feature of early renal failure; it does not usually develop before renal function has diminished to about 25% of normal. Osteitis fibrosa in uremic subjects, from excessive bone turnover, relates to hyperphosphatemia. The role of hyperphosphatemia in promotion of such secondary hyperparathyroidism is well established. A relationship to osteomalacia in hemodialysis patients exists.

Causes of low phosphorus: (Hypophosphatemia may occur with

or without phosphate depletion. Serum levels vary as much as 2.0 mg/dL during the day.)

Very severely malnourished subjects may have low phosphate levels, but even in starvation, phosphorus levels usually are normal.

Antacids, diuretics, and long term steroids are among the common agents bearing a relationship to severe hypophosphatemia. Recent carbohydrate ingestion decreases phosphorus, as does intravenous glucose administration; cases of hypophosphatemia relate to I.V. carbohydrate, dialysis, hyperalimentation, prolonged intravenous administration of phosphate-free fluids, metabolic states involving glucose, potassium, and pH. Depletion of phosphate occurs in diabetic ketoacidosis. Like potassium, phosphorus returns to the cell with therapy of diabetic ketoacidosis and serum levels may diminish significantly during treatment. Osmotic diuresis induced by glycosuria in poorly controlled diabetes may lead to urinary phosphate losses with negative phosphorus balance. PO₄ levels may prove useful in initiation of insulin therapy, in diabetic ketoacidosis and other situations of insulin lack; with hyperglucagonemia, corticosteroid and epinephrine use, and in respiratory alkalosis. Association of hypophosphatemia with impaired glucose metabolism is thought to reflect decreased tissue sensitivity to insulin.

Alcoholism and other hepatic disorders are found very frequently among patients with low PO₄. Alcoholic ketosis and alcohol withdrawal are among causes of hypophosphatemia. There is a slight decrease in serum phosphorus in the last trimester of pregnancy. Primary hyperparathyroidism and other causes of calcium elevation, including ectopic hyperparathyroidism (pseudohyperparathyroidism). Patients with sepsis, including Legionnaires' disease and other respiratory infections. Twenty-two percent of instances of respiratory infections had serum phosphorous ≤ 2.4 . Halevy and Bulvik report gram-negative septicemia as a common cause of severe hypophosphatemia among 55,000 chemistry profiles of hospitalized patients they studied. (Hypophosphatemia impairs bactericidal

activity).

Vitamin D deficiency; osteomalacia, inherited and sporadic forms of hypophosphatemic rickets. In work-up for osteomalacia, look for decreased calcium and phosphorus and increased alkaline phosphatase. Biopsy, however, can be abnormal even when these biochemical parameters are within normal limits.

Renal tubular disorders (Fanconi syndrome, renal tubular acidosis); use of antacids that bind phosphorus (look for hypercalciuria, low urinary phosphorus, high alkaline phosphatase); dialysis, vomiting; saline or lactate I.V.; steatorrhea, malabsorption, severe diarrhea, nasogastric suction; hypokalemia; negative nitrogen balance; decreased dietary PO_4 intake; recovery from severe burn injury; salicylate poisoning; acute gout; tumor-related: described as including hemangiopericytomas (uncommon pathologic entities) and neurofibromatosis; transfusion of blood; arteriography.

The signs and symptoms of phosphate depletion may include neuromuscular, neuropsychiatric, gastrointestinal, skeletal, and cardiopulmonary systems. Manifestations usually are accompanied by serum levels <1.0 mg/dL.

Severe hypophosphatemia is most common in elderly patients and is often found in postoperative subjects.

Complications of hypophosphatemia: Effect on RBC 2,3-diphosphoglycerate and oxygen dissociation. Depression of myocardial function (contractibility), decreased cardiac output; respiratory failure and respiratory muscle weakness; increased incidence of sepsis, impairment of bactericidal activities. CNS consequences: polyradiculopathy, paresthesias, tremor, ataxia, weakness, slurred speech, stupor, coma, seizure; joint stiffness; myopathy; renal stones, hypercalciuria secondary to renal phosphate leak; insulin resistance, glucose intolerance. Rhabdomyolysis may

complicate marked hypophosphatemia. A mortality rate of 20% is described in patients whose phosphorus concentration was 1.1-1.5 mg/dL.

What Is Hyperphosphatemia?

Simply put, hyperphosphatemia is a high level of phosphorus in your blood. When your kidneys are not working properly, phosphorus can build up to dangerous levels without proper treatment.

Unfortunately, dialysis alone cannot maintain normal levels of phosphorus in your blood. You can help treat your phosphorus levels with changes to your diet, but many patients also need medicine, as well, in order to keep their phosphorus at an acceptable level.

Controlling phosphorus, calcium, and parathyroid hormone (PTH) is important because high levels of each can cause serious problems, including bone disease and the formation of mineral deposits in certain areas of your body (for example, kidneys, heart and blood vessels, lungs, eyes, joints, and skin).

Untreated, hyperphosphatemia may lead to serious complications.

Causes of hyperphosphatemia

Renal Failure

Dehydration

Addison's Disease

Hypervitaminosis D

HypoParathyroid

Magnesium deficiency

Milk-Alkali Syndrome

Transfusions

Hemolysis

Sarcoidosis

Bone metastases

Myelogenous Leukemia

1. Evaluation: Renal Failure
 1. Chronic Renal Failure
 1. Increased phosphate when GFR falls below 20 ml/min
 2. Usually Phosphate in 5-10 mg/dl range
 2. Acute Renal Failure
 1. Especially in oliguric or hyper catabolic forms
 2. Highest in:
 1. Myoglobinuric Acute Tubular Necrosis
 2. Surgery
 3. Trauma

2. Evaluation: Normal Renal Function
 1. Check Urinary phosphate excretion (Urine phosphate)
 2. Increased Urine phosphate: over 1500 mg/dl
 1. Endogenous source
 1. Cytotoxins
 2. Rhabdomyolysis
 3. Malignant hyperpyrexia (Fever)
 2. Exogenous source
 1. Laxative Abuse
 2. Enemas
 3. Phosphate (Oral, IV, or poisoning)

 3. Normal Urine phosphate: Under 1500mg/dl
 1. Increased reabsorption
 1. Hyperparathyroidism
 2. Menopause
 3. Extra cellular Fluid Volume contraction
 4. Tumoral calcinosis

3. Management
 1. Antacid binding gel: Aluminum hydroxide (AlternaGel)

1. Dose 600 mg/5 ml, 5 ml qid with meals and at bedtime.
2. Low Phosphate diet.

High Phosphorus Levels Tied To Cardiovascular Disease

Increased serum phosphorus levels may be associated with an increased cardiovascular disease risk in a community setting.

However, senior investigator Dr. Ramachandran S. Vasan says "since our report is the first one to evaluate relations of serum phosphorus to cardiovascular risk in the community -- in a sample with a low prevalence of renal disease -- we should await confirmation of these findings by other investigators in similar community-based samples." It was noted that higher levels of serum phosphorus and the calcium-phosphorus product are associated with increased mortality from cardiovascular disease in patients with chronic kidney disease or prior heart disease.

To evaluate whether serum phosphorus levels influence vascular risk in individuals without such problems, the researchers prospectively followed 3368 Framingham Offspring study participants.

After a mean follow-up of more than 16 years, there were 524 incident cardiovascular disease events. After adjustment, a higher level of serum phosphorus was associated, in a continuous fashion, with an increased risk.

Compared to those with the lowest levels, those with the highest had a 1.55 times greater risk. These findings remained robust after adjustments updating risk and allowing for renal function.

However, without confirmation, continued Dr. Vasan, "the main message for doctors is to stay tuned for more research. It is premature to make any clinical recommendations based on our study results."

The findings "should fuel more research to better understand what is a 'normal' serum phosphorus level" and how "higher levels promote cardiovascular disease risk," he said.

In an accompanying editorial, Dr. Robert N. Foley of the Minneapolis Medical Research Foundation, Minnesota notes that if the connection is indeed confirmed, "unlike many novel

cardiovascular risk factors discovered in community-based studies, several potential interventions suitable for controlled trials may already exist."

Phosphorus: Blood

Other drugs -- Drugs called angiotensin-converting enzyme (ACE) inhibitors, normally used for high blood pressure, may decrease phosphorus levels. They include benazepril (Lotensin), captopril (Capoten), enalapril (Vasotec), fosinopril (Monopril), lisinopril (Zestril, Prinivil), quinapril (Accupril), or ramipril (Altace). Other drugs include cyclosporine (used to decrease the immune system), cardiac glycosides (digoxin or Lanoxin), heparins (blood thinning drugs), anti-inflammatory drugs (such as ibuprofen or Motrin). Salt substitutes contain high levels of potassium which may lower phosphorus levels if used long-term (chronically).

In a healthy individual, the kidney is responsible for maintaining a constant blood phosphorus level. As the dietary intake of phosphorus increases, the kidney will excrete phosphorus in the urine bringing the blood level back to its initial concentration. This balance is under the influence of a major hormone, parathyroid hormone (PTH). Impaired kidney function eventually results in elevated phosphorus levels (hyperphosphatemia), which causes a decrease in blood-calcium levels and can lead to bone loss. For this reason, people with kidney disease often need to limit dietary phosphorus. High phosphorus levels cause blood calcium levels to drop. When phosphorus levels are high a message is sent to the bones (by a hormone) telling them to release calcium and "bind" the phosphorus, that is, removes the phosphorus from the blood. Bones become brittle due to the loss of the calcium

High doses of insulin may decrease blood levels of phosphorus in people with diabetic ketoacidosis (a condition caused by severe insulin insufficiency). This serious condition is treated in a hospital

and replacement of phosphorous takes place under the direction of a doctor.

FAQs

What is phosphorus?

Phosphorus is a mineral found in your bones. Along with calcium, phosphorus is needed for building healthy strong bones, as well as keeping other parts of your body healthy. The majority of the phosphorus in the body is found as phosphate (PO_4). Phosphorus the second most abundant element (after calcium) present in our bodies, makes up about 1 percent of our total body weight. It is present in every cell, but 85 percent of the phosphorus is found in the bones and teeth. In the bones, phosphorus is present in the phosphate form as the bone salt calcium phosphate in an amount about half that of the total calcium. Both these important minerals are in constant turnover, even in the bone structure.

The body uses a variety of mechanisms to control the calcium-phosphorus ratio and metabolism. The ratio of these two elements in the diet has been the subject of much recent interest. The typical diet provides too much phosphorus and not enough calcium, leading to reduced body storage of calcium; thus, many of the problems of calcium deficiency discussed earlier may develop. Phosphorus and calcium can compete for absorption in the intestines. High consumption of meats or soft drinks increases phosphorus intake and may contribute to this imbalance. The ideal ratio of calcium to phosphorus in the diet is 1:1. Phosphorus is absorbed more efficiently than calcium. Nearly 70 percent of phosphorus is absorbed from the intestines, although the rate depends somewhat on the levels of calcium and vitamin D and the activity of parathyroid hormone (PTH), which regulates the metabolism of phosphorus and calcium. Most phosphorus is deposited in the bones, a little goes to the teeth, and the rest is contained in the cells and other tissues. Much is found in the red blood cells. The plasma phosphorus measures about 3.5 mg. (3.5 mg. of phosphorus per 100 ml. of

plasma), while the total blood phosphorus is 30-40 mg.. The body level of this mineral is regulated by the kidneys, which are also influenced by PTH. Phosphorus absorption may be decreased by antacids, iron, aluminum, or magnesium, which may all form insoluble phosphates and be eliminated in the feces. Caffeine causes increased phosphorus excretion by the kidneys.

What are its Functions?

Phosphorus is involved in many functions besides forming bones and teeth. Like calcium, it is found in all cells and is involved in some way in most biochemical reactions. Phosphorus is vital to energy production and exchange in a variety of ways. It provides the phosphate in adenosine triphosphate (ATP), which is the high-energy carrier molecule in the body's primary metabolic cycles. Phosphorus is important to the utilization of carbohydrates and fats for energy production and also in protein synthesis for the growth, maintenance, and repair of all tissues and cells. As inorganic phosphate in ATP. It is needed in protein synthesis and in the production of the nucleic acids in DNA and RNA, which carry the genetic code for all cells.

Phosphorus is a major structural component of bone in the form of a calcium phosphate salt called hydroxyapatite. Phospholipids (e.g., phosphatidylcholine) are major structural components of cell membranes. All energy production and storage are dependent on phosphorylated compounds, such as adenosine triphosphate (ATP) and creatine phosphate. Nucleic acids (DNA and RNA), which are responsible for the storage and transmission of genetic information, are long chains of phosphate-containing molecules. A number of enzymes, hormones, and cell signaling molecules depend on phosphorylation for their activation. Phosphorus also helps to maintain normal acid-base balance (pH) in its role as one of the body's most important buffers. The phosphorus-containing molecule

2,3-diphosphoglycerate (2,3-DPG) binds to hemoglobin in red blood cells and affects oxygen delivery to the tissues of the body.

Why is phosphorus important to you?

Normal working kidneys can remove extra phosphorus in your blood. When you have Chronic Kidney Disease (CKD) your kidneys cannot remove phosphorus very well. High phosphorus levels can cause damage to your body. Extra phosphorus causes body changes that pull calcium out of your bones, making them weak. High phosphorus and calcium levels also lead to dangerous calcium deposits in blood vessels, lungs, eyes, and heart. Phosphorus and calcium control is very important for your overall health.

What is a safe blood level of phosphorus?

A normal phosphorus level is 3.5 to 5.5 mg/dL.

How can I control my phosphorus level?

You can keep your phosphorus level normal by understanding your diet and medications for phosphorus control.

What are the Nutrient interactions?

Calcium and vitamin D: Dietary phosphorus is readily absorbed in the small intestine, and any excess phosphorus absorbed is excreted into urine by the kidneys. The regulation of blood calcium and phosphorus levels are interrelated through the actions of parathyroid hormone (PTH) and vitamin D. A slight drop in blood calcium levels (e.g., in the case of inadequate calcium intake) is sensed by the parathyroid glands resulting in their increased secretion of PTH. PTH stimulates increased conversion of vitamin D to its active form (calcitriol) in the kidneys. Increased calcitriol levels result in increased intestinal absorption of both calcium and phosphorus. Both PTH and vitamin D stimulate bone resorption, resulting in the release of bone mineral (calcium and phosphate) into the blood. Though PTH stimulation results in decreased urinary excretion of calcium, it results in increased urinary excretion of phosphorus. The increased urinary

excretion of phosphorus is advantageous in bringing blood calcium levels up to normal because high blood levels of phosphate suppress the conversion of vitamin D to its active form in the kidneys.

Is high phosphorus intake detrimental to bone health? Some investigators are concerned about the increasing amounts of phosphates in the diet which can be attributed to phosphoric acid in soft drinks and phosphate additives in a number of commercially prepared foods. Because phosphorus is not as tightly regulated by the body as calcium, blood phosphate levels can rise slightly with a high phosphorous diet, especially after meals. High blood phosphate levels reduce the formation of the active form of vitamin D (calcitriol) in the kidneys, reduce blood calcium, and lead to increased PTH release by the parathyroid glands. However, high blood phosphorus levels also lead to decreased urinary calcium excretion. If sustained, elevated PTH levels could have an adverse effect on bone mineral content, but this effect has only been observed in humans on diets that were high in phosphorus and low in calcium. Moreover, similarly elevated PTH levels have been reported in diets that were low in calcium without being high in phosphorus. At present there is no convincing evidence that the dietary phosphorus levels experienced adversely affect bone mineral density in humans. However, the substitution of phosphate containing soft drinks and snack foods for milk and other calcium rich foods does represent a serious risk to bone health.

Fructose: A recent study of 11 adult men found that a diet high in fructose (20% of total calories) resulted in increased urinary loss of phosphorus and a negative phosphorus balance (i.e., daily loss of phosphorus was higher than daily intake). This effect was more pronounced if the diet was also low in magnesium. A potential mechanism for this effect is the lack of feed back inhibition of the conversion of fructose to fructose-1-phosphate in the liver. In other words, increased accumulation of fructose-1-phosphate in the cell does not inhibit the enzyme that phosphorylates fructose, using up large amounts of phosphate. This phenomenon is known as phosphate trapping. This finding is relevant because fructose

consumption has been increasing rapidly, while magnesium intake has decreased over the past century.

How about Deficiency?

Inadequate phosphorus intake results in abnormally low blood phosphate levels (hypophosphatemia). The effects of hypophosphatemia may include loss of appetite, anemia, muscle weakness, bone pain, rickets (in children), osteomalacia (in adults), increased susceptibility to infection, numbness and tingling of the extremities, and difficulty walking. Severe hypophosphatemia may result in death. Because phosphorus is so widespread in food, dietary phosphorus deficiency is usually seen only in cases of near total starvation. Other individuals at risk of hypophosphatemia include alcoholics, diabetics recovering from an episode of diabetic ketoacidosis, and starving or anorexic patients on refeeding regimens that are high in calories but too low in phosphorus.

What is the RDA:

The recommended dietary allowance (RDA) for phosphorus was based on the maintenance of normal blood phosphate levels in adults, which was felt to represent adequate phosphorus intake to meet cellular and bone formation needs.

Adult men and women ages 19 through 50 years: 700 milligrams (mg) phosphorus/day

Adult men and women ages 51 years and over: 580 mg phosphorus/day.

What are the Food Sources?

Since phosphorus is part of all cells, it is readily found in food, especially animal tissues. Most protein foods are high in phosphorus. Meats, fish, chicken, turkey, milk, cheese, and eggs all contain substantial amounts. Most red meats and poultry have much more phosphorus than calcium-between 10 and 20 times as much-whereas fish generally has about 2 or 3 times as much phosphorus as calcium. The dairy foods have a more balanced calcium-phosphorus ratio. Seeds and nuts also contain good levels of phosphorus (although they

have less calcium) as do the whole grains, brewer's yeast, and wheat germ and bran. Most fruits and vegetables contain some phosphorus and help to balance the ratio of phosphorus to calcium in a wholesome diet.

In recent years, the increased consumption of soft drinks, which are buffered with phosphates, has been a concern. There may be up to 500 mg. of phosphorus per serving of a soft drink, with essentially no calcium. In 1970, the average per capita intake of soft drinks was 23 gallons, whereas in 1981, 39 gallons a year were consumed by the "average" person. Since some people do not drink any of these "beverages," quite a number of people are drinking even more than the average amount of soda pops, and thus consuming a lot of phosphorus.

Dietary phosphorus derived from food additives is not calculated in most food databases, so the total amount of phosphorus consumed by the average person is not entirely clear. A large survey of nutrient consumption in the U.S. found the average phosphorus intake in men to be 1,495 mg daily and the average phosphorus intake in women to be 1,024 mg/day. The Food and Nutrition Board estimates phosphorus consumption in the U.S. has increased 10% to 15% over the past 20 years.

The phosphorus in all plant seeds (beans, peas, cereals, and nuts) is present in a storage form of phosphate called phytic acid or phytate. Only about 50% of the phosphorus from phytate is available to humans because we lack enzymes (phytases) that liberate it from phytate. Yeasts possess phytases, so whole grains incorporated into leavened breads have more bioavailable phosphorus than whole grains incorporated into breakfast cereals or flat breads. The table below lists a number of phosphorus rich foods along with their phosphorus content in milligrams (mg).

Food	Serving	Phosphorus (mg)
Milk, skim	8 ounces	247

Yogurt, plain nonfat	8 ounces	383
Cheese, mozzarella; part skim	1 ounce	131
Egg	1 large, cooked	104
Beef	3 ounces, cooked*	173
Chicken	3 ounces, cooked*	155
Turkey	3 ounces, cooked*	173
Fish, halibut	3 ounces, cooked*	242
Fish, salmon	3 ounces, cooked*	252
Bread, whole wheat	1 slice	64
Bread, enriched white	1 slice	24
Carbonated cola drink	12 ounces	44
Almonds [#]	1 ounce	139
Peanuts [#]	1 ounce	101
Lentils [#]	1/2 cup, cooked	356

*A 3-ounce serving is about the size of a deck of cards.

[#]Phosphorus from nuts, seeds, and grains is about 50% less bioavailable than phosphorus from other sources.

High Phosphorus Food to limit or avoid

Beverages

ale

beer

	chocolate drinks	cocoa
	drinks made with milk	dark colas
	canned iced teas	
Dairy Products	cheese	cottage cheese
	custard	ice cream
	milk	pudding
	cream soups	yogurt
Protein	carp	crayfish
	beef liver	chicken liver
	fish roe	organ meats
	oysters	sardines
Vegetables	<i>dried beans and peas:</i>	
	baked beans	black beans
	chick peas	garbanzo beans
	kidney beans	lentils
	limas	northern beans
	pork ' n beans	split peas
	soy beans	
Other foods	bran cereals	brewer's yeast
	caramels	nuts
	seeds	wheat germ
	whole grain products	

What are the Safety precautions?

Toxicity: The most serious adverse effect of abnormally elevated blood levels of phosphate (hyperphosphatemia) is the calcification of non-skeletal tissues, most commonly the kidneys. Such calcium phosphate deposition can lead to organ damage, especially kidney damage. Because the kidneys are very efficient at eliminating excess phosphate from the circulation, hyperphosphatemia from dietary causes is a problem mainly in people with severe kidney failure (end-stage renal disease) or hypoparathyroidism. When kidney function is only 20% of normal, even typical levels of dietary phosphorus may lead to hyperphosphatemia. Pronounced hyperphosphatemia has also occurred due to increased intestinal absorption of phosphate salts taken by mouth, as well as due to colonic absorption of the phosphate salts in enemas. In order to avoid the adverse effects of hyperphosphatemia, the Food and Nutrition Board set an upper level (UL) of oral phosphorus intake for generally healthy individuals:

Men and women 19 through 70 years: 4.0 grams (4000 milligrams) of phosphorus/day

Men and women over 70 years: 3.0 grams (3000 milligrams) of phosphorus/day

The lower UL for individuals over 70 years of age reflects the increased likelihood of impaired kidney function above age 70. The UL does not apply to individuals with significantly impaired kidney function or other health conditions known to increase the risk of hyperphosphatemia.

Will dialysis help with phosphorus control?

Yes. Dialysis can remove some phosphorus from your blood. It is important for you to understand how to limit build up of phosphorus between your dialysis treatments.

What are medications for phosphorus control?

A medicine called a phosphate binder taken with meals and snacks. This medicine will help control the amount of phosphorus your body absorbs from the foods you eat. There are many different kinds of

phosphate binders. Pills, chewable tablets, and powders are available. Some types also contain calcium, while others do not.

What do I do if my phosphorus level is too high?

When your phosphorus level is too high, think about your diet and substitute lower phosphorus foods for a while, making changes in your diet and use phosphate binders.

INSTEAD OF HIGH PHOSPHORUS FOODS

TRY LOW PHOSPHORUS FOODS

	Phosphorus (mg)		Phosphorus (mg)
8 ounce milk	230	8 ounce nondairy creamer or 4 ounce milk	100 115
8 ounce cream soup made with milk	275	8 ounce cream soup made with water	90
1 ounce hard cheese	145	1 ounce cream cheese	30
½ cup ice cream	80	½ cup sherbet or 1 popsicle	0
12-ounce can cola	55	12 ounce can of Ginger Ale or lemon soda	3
½ cup lima or pinto beans	100	½ cup mixed vegetables or green beans	35
½ cup custard or pudding	150	½ cup pudding or custard made with	50

made with milk		nondairy creamer	
2 ounce peanuts	200	1 ½ cup light salt/low fat popcorn	35
1 ½ ounce chocolate bar	125	1 ½ ounce hard candy, fruit flavors or jelly beans	3
2/3 cup oatmeal	130	2/3 cup cream of wheat or grits	40
½ cup bran cereal	140-260	½ cup non-bran cereal, shredded wheat, rice cereals, or corn flakes	50-100

More than 20 million Americans—one in nine adults—have chronic kidney disease and most don’t even know it. More than 20 million others are at increased risk.

Drug Interactions:

Aluminum-containing antacids reduce the absorption of dietary phosphorus by forming aluminum phosphate, which is unabsorbable. When consumed in high doses, aluminum containing antacids can produce abnormally low blood phosphate levels (hypophosphatemia), as well as aggravate phosphate deficiency due to other causes. As little as 1 ounce of aluminum hydroxide gel three times a day for several weeks can diminish blood phosphate levels and lead to increased urinary calcium loss.

Excessively high doses of calcitriol, the active form of vitamin D, or its analogs may result in hyperphosphatemia.

Potassium supplements or potassium-sparing diuretics taken together with a phosphate may result in high blood levels of potassium

(hyperkalemia). Hyperkalemia can be a serious problem, resulting in life threatening heart rhythm abnormalities (arrhythmias). People on such a combination need to be sure their health care provider is aware of it, and have their blood potassium levels checked regularly.

Is drinking soda bad for my bones?

In general, the negative impact of soda on bone health is not due to the phosphorus content of the soda. Instead the detrimental effect of soda on bones occurs because calcium-containing beverages are often omitted in favor of soda. Soda is a nutrient-poor beverage that often replaces other potentially nutrient-rich beverages such as milk, fortified soy milk or calcium-fortified juices. The low calcium intake is what's bad for your bones.

It is concerning that soda consumption is on the rise among our nation's youth and this is occurring during the years (age 9-18) when calcium needs for building bone are the highest. In addition, the extra calories contained in (non-diet) sodas may contribute to the problem of obesity among youth that is at an all-time high. Many schools have initiated campaigns to replace soda machines in schools with alternative, nutritious beverages.

Can I consume too much phosphorus in my diet?

The maximum safe intake of phosphorus for adults is 4000mg per day. This amount is very rarely consumed in the U.S. population and could only be reached by using excessive amounts of phosphorous supplements in addition to a typical diet.

Is it true that too much phosphorous can interfere with calcium absorption?

When calcium intake is adequate, a high phosphorus intake is not detrimental to calcium absorption. The ratio of calcium to phosphorus is probably more important than the amount of phosphorus in the diet alone. Milk and dairy products have a

balanced ratio of calcium to phosphorus that results in good absorption of both nutrients and a favorable effect on bone health.

Is phosphorus deficiency common?

Phosphorus is so plentiful in the food supply that deficiency does not occur in the healthy population. Phosphorus deficiency is rare and usually occurs secondary to other conditions rather than dietary intake. For example, phosphorus deficiency can result from poorly managed diabetes (diabetic ketoacidosis), during recovery from alcoholism, from high dose antacid use, and in other conditions associated with malnutrition. Phosphorus deficiency is easily diagnosed by a medical professional and is important to treat. Untreated phosphorus deficiency could lead to an increased risk for fracture.

Does Phosphorus restriction prevent parathyroid gland growth?

Phosphorus restriction prevents parathyroid gland growth. High phosphorus directly stimulates PTH secretion in vitro. Dietary phosphorus (P) restriction is known to ameliorate secondary hyperparathyroidism in renal failure patients. In early renal failure, this effect may be mediated by an increase in 1,25-(OH)₂D₃, whereas in advanced renal failure, P restriction can act independent of changes in 1,25-(OH)₂D₃ and serum ionized calcium (ICa). The effect of P is posttranscriptional, affecting PTH at a translational or posttranslational step. Collectively, the results of in vivo and in vitro experimental results demonstrate a direct action of P on PTG function that is independent of ICa and 1,25-(OH)₂D₃.