A Comprehensive Study of Phosphorus Additives in Foods and Their Effect on the American Population

by

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ABSTRACT

In an attempt to increase nutritional awareness the purpose of this study was to examine the disadvantages of phosphorus additives in foods and their potential harmful effects on the American population. This study included a comprehensive review and critical analysis of research and literature concerning the issue of present rates of consumption of phosphorus due to increased use of food additives and the physiological effects on the human body due to this increased consumption. The results of this examination indicated an increasing number of harmful side effects including numerous bone, cardiovascular, and hormonal problems. These findings implicate a need for increased awareness among health professionals, the general population and policy makers to help to decrease the adverse effects associated with the increased rate of phosphorus consumption due to increased rates of food additive consumption.

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Chapter I: Introduction

Phosphorus is an essential element of and the second most abundant mineral in the human body (Mahan & Escott-Stump, 2000). As phosphates, phosphorus is essential in numerous bodily functions. Major functions include being the base for all human ribonucleic acid (RNA) and deoxyribonucleic acid (DNA); a component of the major source of energy, adenosine triphosphate (ATP); and present in every cell membrane in the body as a component of phospholipid molecules

Modern Nutrition in Health and Disease (1999) reports that the current recommendation for daily dietary intake of phosphorus by the Food and Nutrition Board of the Institute of Medicine is 700 mg for adults between the ages of 19-70 (Shils, Olson, Shike, & Ross, 1999). Protein rich foods and cereal grains are rich sources of phosphorus. In the United States, approximately one half of dietary phosphorus comes from milk and poultry with processed meats and cheese containing more phosphorus than natural products.

As Americans continue to demand high-quality convenience foods, food processing practices have increased the use of phosphorus additives to ensure the quality and flavor that Americans have come to expect. With the surge in popularity of these foods, phosphate consumption has been an increasing trend over the past fifteen years. In 1990, phosphorus additives contributed an estimated 470 mg/dl to the American diet. Today, it is estimated that phosphate additives contribute up to 1,000 mg/dl, depending upon an individual's food choices (Murphy-Gutekunst & Barnes, 2005). Unfortunately, most nutrition educators and the general population are uninformed about these increasing amounts and the potential harmful side effects on health status.

Purpose of the Study

This purpose of this study is to examine the disadvantages of phosphorus additives in foods and their potential harmful effects on the American population. This will be achieved by means of conducting a comprehensive literature review, and analysis, and critique of the findings. Recommendations are provided for helping professionals.

Research Objectives

The study addresses three main research objectives. They are as follows:

1. Review and examine available research and literature on phosphate additives in foods.
2. Develop recommendations for nutrition educators and the general population on the effects of phosphate additives based upon the results of this study.
3. Develop recommendations focused upon potential dietary modifications to help eliminate excessive phosphate intakes based upon the results of this study.

Definition of Terms

Adenosine Monophosphate: A nucleotide found in muscle cells and important in metabolism; reversibly convertible to ADP and ATP.

Adenosine Triphosphate: A nucleotide derived from adenosine that occurs in muscle tissue; the major source of energy for cellular reactions.

Creatine Phosphate: An organic compound of creatine and phosphoric acid; found in the muscles of vertebrates where its hydrolysis releases energy for muscular contraction.

Orthophosphate: A salt of phosphoric acid.

Phosphoprotein: Any of a group of proteins, such as casein, containing chemically bound phosphoric acid.

Phospholipid Any of a group of fatty compounds, as lecithin, composed of phosphoric esters, and occurring in living cells.

Pyrophosphate: A salt or an ester of pyrophosphoric acid.

Limitations

There are two basic limitations of this study. First, due to the surge in popularity, demand, and consumption of new convenience foods, there is little professional literature written on the phosphorus additives used in the processing of these foods.

Second, the current system for estimating the nutrient content of survey foods in the National Food Consumption Survey is not capable of capturing the phosphorus intake from food additives. Therefore, determination of estimated phosphorus intakes from these additives can only be assumed.

Chapter II: Literature Review

Minerals

The minerals are a large class of micronutrients, most of which are considered essential (Mahan & Escott-Stump, 2000). They are traditionally divided into macrominerals (bulk elements) and microminerals (trace elements). Macrominerals such as calcium and phosphorus are required in amounts of 100 mg/day or more; whereas microminerals such as iron and selenium are required in less than 15 mg/day.

Macrominerals exist in the body and food chiefly in the ionic state. Sodium, potassium, and calcium form positive ions, whereas phosphorus exists as negative ions which are referred to as phosphates. Phosphorus also exists as components of organic compounds such as phosphoproteins and phospholipids

Minerals represent about 4%-5% of body weight, or 2.8 to 3.5 kg in adult women and men, respectively (Mahan & Escott-Stump, 2000). Approximately 50% of this weight is calcium, and another 25% of this weight is phosphorus.

Phosphorus

Phosphorus is derived from the greek: phôs meaning "light" and phoros meaning "bearer" and is the chemical element that has the symbol P and atomic number 15 (Wikipedia, 2007b). A multivalent nonmetal of the nitrogen group, phosphorus is commonly found in inorganic phosphate rocks.

Phosphorus is one of the most abundant elements on earth. It exists in plants and animal foods and biological fluids as phosphate ions. A maximum amount of 850 grams of elemental phosphorus is present in the adult human body at all times – 85% in the skeleton; 14% in the soft tissues; and 1% in the extracellular fluids, intracellular

structures, and cell membranes (Bowman & Russell, 2001). From infancy to adulthood, the percentage of total body phosphorus content increases from 0.5% to 0.65-1.1%. The percentage in extracellular fluids is small but this compartment is where dietary phosphorus, as phosphates ions, first enters and from which urinary inorganic phosphorus is cleared.

Absorption of Phosphorus

The relative amounts of inorganic and organic phosphorus in the diet vary with the food or supplement consumed. Regardless of the form, most phosphates are absorbed in the inorganic state (Mahan & Escott-Stump, 2000). Phosphorus from soluble products such as meat is more available than that from other sources. Milk casein contains a phosphopeptide resistant to enzymatic hydrolysis; human milk, being lower in casein, may have more phosphorus available from absorption than cow's milk. Phytic acid in cereal grains reduces bioavailability (Shils et al., 1999).

Inorganic phosphorus, derived from an inorganic form in foods or after digestive release from organic molecules, is absorbed by epithelial cells of the small intestine, primarily by facilitated diffusion rather than an active process. A high efficiency of absorption exists – in the range of 60-70% in adults over a wide range of intakes (Bowman & Russell, 2001). The linear relationship between absorption and intake holds across the broad range of intakes, and the rise in inorganic phosphate concentrations in blood follows within one hour after ingestion. The ability of the gut to absorb inorganic phosphorus by an active transport process is called into action only when phosphorus intake is low or the demand for it is greatly increased. The hormonal form of vitamin D

may enhance the intestinal absorption of phosphate ions when intakes of phosphorus have been low, but evidence for this action remains limited.

Excretion and Reabsorption of Phosphorus

The whole-body balance of phosphorus is maintained through the actions of the two major input and output components –small intestine and the kidneys (Mahan & Escott-Stump, 2000). To maintain homeostasis, renal phosphorus excretion normally matches the amount of daily gastrointestinal absorption. Excretion occurs in the proximal tubule and is largely dependent on the filtered phosphorus load. As the filtered load increases, a higher fraction of excreted phosphorus is reabsorbed. Major determinants of urinary phosphorus loss are increased intake of phosphate, an increase in phosphate absorption, and the plasma phosphorus concentration.

Functions of Phosphorus

As phosphates, phosphorus is involved in numerous essential physiological functions as it is distributed to every cell in the body, found in extracellular fluid, and a major component of all bones. Organic phosphates are so widely used in key roles in cellular biochemistry that life on earth would not exist without them (Bowman & Russell, 2001). At a cellular level, the membranes that surround all cells and separate the intercellular organelles from cytoplasm consist primarily of a bilayer of phospholipids. The genetic material of the cell, both ribonucleic acid (RNA) and deoxyribonucleic acid (DNA) contain phosphate groups linking the deoxyribose and ribose sugars along the backbone of the molecules. Phosphates are components of the intercellular structure and also function in the metabolic reactions that occur within the cells.

Glucose, the energy source for most cellular activities, must be phosphorlated inside the cell before entering the glycolytic pathway (Bowman & Russell, 2001). The energy derived from both anaerobic and aerobic glycolysis is stored in the form of high-energy pyrophosphate bonds in adenosine triphosphate (ATP). In muscle cells, some energy storage also involves creatine phosphate. ATP can also be converted to cyclic adenosine monophosphate (cAMP) which is a messenger through which many hormones signal changes in cellular activity.

Phosphorus is also a critical element of bones where it exists as hydroxyapatite crystals that contain a constant ratio of calcium to phosphate at 2:1 (Bowman & Russell, 2001). Inorganic phosphorus moves in and out of bone mineral by two processes: ionic exchange and active bone reabsorption. Bone typically has a slow rate of turnover in adults, but its dynamic quality permits the maintenance of inorganic phosphorus concentrations as well as that of calcium in blood serum and extracellular fluids.

Human blood contains two fractions of phosphate: organic (70%) and inorganic (30%; Shils et al., 1999). Within the blood compartment, phosphate ions are both very effective buffers of blood pH and regulators of whole-body acid-base balance, both metabolic and respiratory, by aiding in the renal excretion of hydrogen ions.

The amount and rate at which other phosphate is available for absorption depends on their enzymic hydrolysis to orthophosphates (International Program on Chemical Safety, 2007). The level of inorganic phosphate in the blood is stabilized by exchange with the mineral deposit in the skeleton through the action of parathyroid hormone. This hormone inhibits tubular reabsorption of phosphates by the kidney and brings about demineralization of bone tissue through the action of osteoclasts. The amount of

parathyroid hormone that enters the circulation is probably regulated by the calcium level of the blood. Intestinal absorption depends on requirements and is therefore limited. Excretion takes place mainly in the feces as calcium phosphate so that the continuous use of excessive amounts of sodium phosphate and phosphoric acid may cause a loss of calcium. As a result of physiological regulating mechanisms, man and animals can tolerate large variations in phosphate intake without the balance being upset.

Dietary Recommendations of Phosphorus

According to *Present Knowledge in Nutrition* (Bowman & Russell, 2001), the most recent estimates of the recommended daily allowance for phosphorus are shown in Tables 1-4.

Table 1

Daily Phosphate Allowance for Infants and Children

Life Stage	Phosphorus
0-6 months	100
7-12 months	275
1-3 years	460
4-8 years	500

Table 2

Daily Phosphate Allowance for Males

Life Stage	Phosphorus
9-13 years	1250
14-18 years	1250
19-30 years	700
31-50 years	700
51-70 years	700
70+ years	700

Table 3

Daily Phosphate Allowance for Females

Life Stage	Phosphorus
9-13 years	1250
14-18 years	1250
19-30 years	700
31-50 years	700
51-70 years	700
70+ years	700

Table 4

Daily Phosphate Allowance for Pregnant and Lactating Women

	Life Stage	Phosphorus
	18 years	1250
Pregnancy	19-30 years	700
	31-50 years	700
	18 years	1250
Lactation	19-30 years	700
	31-50 years	700

The reported estimated average intake of phosphorus for adults between the ages of 19-50 is 580 milligrams per day with the tolerable upper intake level being 4000 milligrams per day (Bowman & Russell, 2001).

According to the Food and Nutrition Board of the Institute of Medicine (1997), a large survey of nutrient consumption in the United States found the average phosphorus intake in men to be 1,485 mg/day and the average phosphorus intake in women to be 1,024 mg/day. In order to avoid the adverse effects of hyperphosphatemia, the Food and Nutrition Board set an upper level of oral phosphorus intake for generally healthy individuals at 4.0 grams/day for men and women 19-70 years old and 3.0 grams/day for men and women over 70 years of age. The lower upper limit for individuals over 70 years of age reflects the increased likelihood of impaired kidney function above age 70.

Kidney Function and Phosphorus

A major function of the kidneys is to remove waste products and excess fluid from the body (HealthCentralNetwork, Inc., 2001). These waste products and excess fluid are removed through urine. The production of urine involves highly complex steps of excretion and reabsorption. This process is necessary to maintain a stable balance of body chemicals.

The critical regulation of the body's salt, potassium, phosphorus and acid content is performed by the kidneys. The kidneys also produce hormones and vitamins that affect the function of other organs. For example, a hormone produced by the kidneys stimulates red blood cell production. In addition, other hormones produced by the kidneys help regulate blood pressure and others help control calcium metabolism.

There are two kidneys, each about the size of a fist, located on either side of the spine at the lowest level of the rib cage. Each kidney contains about one million functioning units, called nephrons.

A nephron consists of a filtering unit of tiny blood vessels, called a glomerulus, attached to a tubule. When blood enters the glomerulus, it is filtered and the remaining fluid passes along the tubule. In the tubule, chemicals and water are either added to or removed from this filtered fluid, according to the body's needs, with the final product being the urine excreted.

According to the American Kidney Fund (2007), more than 10 million Americans have kidney problems. Some of these problems include infections, kidney stones, kidney cancer and Polycystic Kidney Disease. Many people also have chronic kidney disease. When a person has chronic kidney disease, his or her kidneys do not work as

well as they should. Chronic kidney disease can lead to kidney failure. Kidney failure can only be treated with dialysis or a kidney transplant. Present facts about kidney disease include the following:

1. About 1 in 12 people in America has a kidney disease.
2. Over 20 million adults over age 20 have chronic kidney disease.
3. Diabetes is the number one cause of kidney failure. High blood pressure is number two.
4. Over 80,000 people with kidney failure die each year. Kidney disease is America's ninth leading cause of death.
5. There are 450,000 people being kept alive through dialysis or kidney transplants.
6. Over 65,000 patients are on the waiting list for a kidney transplant.

Chronic kidney disease is a common and serious medical problem (eMedicineHealth, 2005). It can be caused by a primary kidney disease or result from a disease elsewhere in the body that injures the kidneys or prevents them from working. Chronic kidney disease can greatly impact life as it progresses (WebMD, 2005). At first, the kidneys are still able to regulate the balance of fluids, salts, phosphorus, and waste products in your body. But as kidney function decreases, complications begin to develop. The number and severity of complications caused by chronic kidney disease increase as kidney function gets worse.

If a person is unable to control the disease, his or her kidney function will continue to get worse. When kidney function falls below a certain point, it is called kidney failure. Kidney failure has harmful effects throughout the entire body. It can

cause serious heart, bone, and brain problems and make a person feel very ill. Once kidney failure is developed, it will be necessary either to have dialysis or have a kidney transplant.

According to Mayo Clinic Staff (2007), factors that lead to chronic kidney disease are related to age and your genetic makeup. People may be able to control other things that increase your risk, such as dietary habits and exercise.

The main uncontrollable risk factors for the development of chronic kidney disease are as follows:

- Age. The kidney begins to get smaller at about age 35. By age 80, most people have lost about 30% of their kidney mass.
- Race. African-Americans and Native Americans are more likely to develop chronic kidney disease.
- Gender. Men have a higher risk of developing chronic kidney disease than women.
- Family history. Family history is a factor in the development of both diabetes and high blood pressure, the major causes of chronic kidney disease. Polycystic kidney disease is one of several inherited diseases that cause kidney failure

The major uncontrollable risk factors for development of chronic kidney disease are as follows:

- Diabetes. Diabetes mellitus is a leading cause of chronic kidney failure in the United States. Chronic kidney failure is related to both type 1 and type 2 diabetes.

- High blood pressure (hypertension). Untreated or inadequately treated high blood pressure is another common cause of chronic kidney failure in the United States. The added force of elevated blood pressure exerted on the glomeruli can cause damage and scarring. When this happens, the nephrons containing the damaged glomeruli eventually lose their ability to filter waste from your blood.
- Obstructive nephropathy. This occurs when urine outflow is blocked over time by an enlarged prostate, kidney stones or tumors, or by vesicoureteral reflux, a condition that results from urine backing up into your kidneys from your bladder. The backflow pressure in your kidneys reduces their function.
- Kidney diseases. These include clusters of cysts in the kidneys (polycystic kidney disease), kidney infection (pyelonephritis) and inflammation of the glomeruli (glomerulonephritis), a condition that causes your kidneys to leak protein into your urine and damages nephrons.
- Kidney (renal) artery stenosis. This is a narrowing or blockage of the kidney artery before it enters your kidney. In older adults, blockages often result when fatty deposits accumulate under the lining of the artery walls (atherosclerosis). Kidney artery stenosis can also affect young women in the form of a condition known as fibromuscular dysplasia, which causes the walls of the arteries to become thicker. Both conditions are often associated with high blood pressure.
- Toxins. Ongoing exposure to fuels and solvents, such as carbon tetrachloride, and lead — in lead-based paint, lead pipes, soldering materials, jewelry and even alcohol distilled in old car radiators — can lead to chronic kidney failure.

Failing kidneys may not be able to remove phosphorus from your blood (National Kidney Foundation, 2007). This causes the level of phosphorus in blood to become too high. Because unhealthy kidneys are no longer able to remove phosphorus from the blood and get rid of the excess in urine, high levels of phosphorus referred to as hyperphosphatemia is a problem for people with stage 4 and 5 kidney disease, especially stage 5 (also known as end stage renal disease or ESRD).

Hyperphosphatemia is defined as a serum phosphorus level >5 mg/dL (1.6 mmol/L), usually in the form of inorganic phosphorus (Shire US Inc., 2007). The most common causes of this condition are acute and chronic renal failure. Hyperphosphatemia can be a consequence of decreased filtration rate, increased tubular resorption of PO_4, or increased phosphorus load. The pathophysiology of hyperphosphatemia involves decreased Ca^{++} due to increased serum phosphorus, increased parathyroid hormone secretion, and associated sequelae.

Hyperphosphatemia causes hypocalcemia by precipitating calcium, decreasing vitamin D production, and interfering with parathyroid hormone-mediated bone resorption (Patterson, 2007). Severe life-threatening hypocalcemia may result. Signs and symptoms of acute hyperphosphatemia are due to the effects of hypocalcemia.

Prolonged hyperphosphatemia promotes metastatic calcification, an abnormal deposition of calcium phosphate in previously healthy connective tissues such as cardiac valves and in solid organs such as muscles. The calcium-phosphate product predicts the risk of metastatic calcification.

Excess free serum phosphorus is taken up into vascular smooth muscle via a sodium-phosphate cotransporter. The increased cellular phosphate activates a gene, *cbfa-*

1, which promotes calcium deposition in the vascular cell, making smooth muscle cells engage in osteogenesis. Vascular walls become calcified and arteriosclerotic, leading to increased systolic blood pressure, widened pulse pressure, and subsequent left ventricular hypertrophy.

Hyperphosphatemia is an independent risk factor contributing to the increased incidence of aortic and mitral stenosis and other cardiovascular disease among dialysis-dependent patients. A peripheral form known as calcific uremic arteriolopathy (calciphylaxis) can induce necrotic ulceration and gangrene in affected extremities. Also, hyperphosphatemia-induced resistance to parathyroid hormone contributes to secondary hyperparathyroidism and renal osteodystrophy.

Dietary Sources of Phosphorus

As a vital constituent of cell membranes, plant cell walls, and structural and soluble cellular proteins, virtually all the foods we eat contain some amount of phosphorus. Phosphorus, primarily in the form of phosphates, is found in three major dietary sources: foods containing natural phosphorus, foods processed with phosphorus, and dietary supplements containing phosphorus (Bowman & Russell, 2001).

In general, good sources of protein are also good sources of phosphorus. Meat, poultry, fish, and eggs are excellent sources. Milk and milk products are good sources, as are nuts and legumes, cereals and grains. (Phosphorus is bound to a few amino acids, especially serine, threonine, and tyrosine, in food proteins). In the outer coating of cereal grains, particularly wheat, phosphorus exists in the form of phytic acid, which can form a complex structure with some minerals to create insoluble compounds.

Most phosphorus (about 60%) comes from milk, meat, poultry, fish, and eggs (Mahan & Escott-Stump, 2000). Cereals and legumes provide another 20%, and less than 10% is derived from fruits and their juices. Other dietary sources such as tea, coffee, vegetable oils, and spices supply only small amounts of phosphorus. The estimated amount provided by food additives is almost 10%.

The phosphorus content of food is also influenced by the amount of processing involved in manufacturing the food product. Processed meats and cheese contain more phosphorus than natural products (Shils et al., 1999).

Food Additives Containing Phosphorus

According to the United States Department of Agriculture (USDA) Food Safety and Inspection Service (FSIS; 2001) food additives are defined by the Food and Drug Administration (FDA) as any substance used to provide a technical effect in foods. The use of food additives has become more prominent in recent years, due to the increased production of prepared, processed, and convenience foods. Additives are used for flavor and appeal, food preparation and processing, freshness, and safety. At the same time, consumers and scientists have raised questions about the necessity and safety of these substances.

Before any substance can be added to food, its safety must be assessed in a stringent approval process. The FSIS and USDA share responsibility with the FDA for the safety of food additives used in meat, poultry, and egg products. All additives are initially evaluated for safety by the FDA.

When an additive is proposed for use in a meat, poultry, or egg product, its safety, technical function, and conditions of use must also be evaluated by the Labeling and

Consumer Protection Staff of FSIS, as provided in the Federal Meat Inspection Act, the Poultry Products Inspection Act, the Egg Products Inspection Act, and related regulations. Although the FDA has overriding authority regarding additive safety, the FSIS may apply even stricter standards that take into account the unique characteristics of meat, poultry, and egg products. Several years ago, for instance, permission was sought to use sorbic acid in meat salads. Although sorbic acid was an approved food additive, permission for use in meat salad was denied because such usage could mask spoilage caused by organisms that cause food borne illness.

Additives are never given permanent approval. The FDA and FSIS continually review the safety of approved additives, based on the best scientific knowledge, to determine if approvals should be modified or withdrawn.

The first efforts to pass laws to govern foods were state laws (1850 and beyond). These laws were difficult to enforce. The first major federal law governing food was the 1906 Federal Food and Drug Act. It set the framework for the regulation of foods and stated that it was illegal to sell misbranded or adulterated foods and drugs in interstate commerce. It listed chemicals that were illegal to add to foods, such as borax or formaldehyde. The law was weak in that there was no method of enforcement and no punishment.

In 1938, the Federal Food and Drug Act was revised to account for changes in medical science and food technology, and was renamed the Federal Food, Drug, and Cosmetic Act. Among the many provisions of the law was a requirement for truthful labeling of additives.

The 1958 Food Additives Amendment to the Federal Food, Drug, and Cosmetic Act provided for the first specific regulations of food additives. Approval of new food additives was required before they could be marketed, and the responsibility for proving their safety was placed on the manufacturer.

To use or market a substance to be used as a food additive, a company must first file a petition with the FDA outlining the tests that prove the substance to be safe under the proposed conditions of use. If it is approved as safe under the proposed conditions of use, FDA prescribes in its regulations, the types of foods it may be used in, and how it may be used.

The Food Additives Amendment exempted two groups of food additives from the FDA's testing and approval process. One is the list of substances known as "generally recognized as safe" (GRAS). This group includes a variety of substances, from commonly used flavorings and spices to phosphates. These substances are considered harmless under prescribed conditions of use. Past extensive use of these substances has produced no known harmful effects (United States Department of Agriculture, 2001).

As the population continues to demand high-quality convenience food, food processing practices have stepped up the use of phosphorus additives (Murphy-Gutekunst, 2005). Though the reported estimated average intake for phosphorus for adults between the ages of 19-50 is 580 to 1405 milligrams per day, an important point that should not be overlooked is that the surveys used to collect this data may grossly underestimate actual phosphorus intakes because the nutrient databases used do not account for contributions from phosphorus additives used in the foods consumed (Bowman & Russell, 2001). Regardless of contributions from phosphorus additives not

being accounted for at the 50th percentile intake, teen and adolescent girls consume slightly more than the estimated average intake requirements for phosphorus whereas boys exceed this intake recommendation. Both young and older adult men and women greatly exceed their phosphorus estimated average intake of 580 milligrams per day at the 50th percentile.

As reported in the Journal of Renal Nutrition (2005), phosphorus additives contributed and estimated 470 milligrams per day to the American diet in 1990 (Murphy-Gutekunst & Uribarri). With the surge in popularity, demand, and consumption of these new foods, additives could now be contributing up to 1,000 milligrams or more of phosphorus per day to individuals' diets depending on the food choices.

Bowman and Russell (2001) estimated that nationally representative nutrient intake of the U.S. population is usually derived from national food consumption surveys such as the Continuing Survey of Food Intakes by Individuals conducted by the U.S. Department of Agriculture. The current system for estimating the nutrient content of survey foods in the National Food Consumption Survey is not capable of capturing the phosphorus intake from food additives. Among the 45 or more direct food additives containing phosphorus that are listed in the Code of Federal Regulations, phosphoric acid and the polyphosphate compounds are the most widely used with their use doubling between 1980 and 1990.

In addition to the unknown estimated consumption of phosphorus additives by the U.S population, is the fact that phosphorus additives are highly absorbable (Murphy-Gutekunst, 2005). In a typical mixed diet of grains, meat, and dairy, only 60% of the

dietary phosphorus is absorbed, whereas phosphoric acid and various polyphosphates and pyrophosphates are almost 100% absorbed.

Identifying these new higher-phosphorus foods can be challenging (Murphy-Gutekunst, 2005). Manufacturers are no longer required to list the phosphorus content on the food label. If the manufacturer does analyze the product for phosphorus, it is sometimes classified as proprietary information. Often the analysis is not readily accessible to consumer service representatives, who must submit the request for information to one or two different departments and even then there is no guarantee that the company can locate the information.

Another challenge is the practice of products being affiliated with one company and manufactured, packaged, and distributed by another company (Murphy-Gutekunst, 2005). For example, Country Time Lemonade is considered a Kraft-brand food, but some Country Time Lemonade products are manufactured and distributed through Dr. Pepper/7-Up. Finally, each company formulates its products differently, and within each product, the individual delivery packages may be formulated differently. For instance, a bottled iced tea may be different than the same brand of canned iced tea.

Label Monitoring of Phosphorus

According to the USDA (2001), the statutes and regulations to enforce the statutes require certain information on labels of meat and poultry products so consumers will have complete information about a product. In all cases, ingredients must be listed on the product label, in the ingredients statement in order by weight, from the greatest amount to the least.

Substances such as spices and spice extractives may be declared as "natural flavors," "flavors," or "natural flavoring" on meat and poultry labels without naming each one. This is because they are used primarily for their flavor contribution and not their nutritional contribution (United States Department of Agriculture, 2001).

Regular monitoring of in-store food nutrition labels is the best way to keep abreast of the nutrition and phosphorus content in foods (Murphy-Gutekunst & Barnes, 2005). However, it was also reported that there are problems with obtaining accurate analyses from widely used resources available to dietitians and patients. In some instances, it was reported that three different analyses for the same product all found different results.

Foods manufacturers change formulations throughout the year, but are only required to report nutritional content of their products to the USDA once per year. As a result, nutrition analyses found in the USDA National Food Database and nutrition analysis programs that use the USDA for their databases may be obsolete.

Also reported by Murphy-Gutenkunst and Barnes (2005) was that nutrition fact labels listed on a company's website are not always accurate. On visiting a website, the researchers reported finding a product believed to be low in phosphorus with a picture of the product's nutrition label that listed the phosphorus content as 0% daily value. However, in a different area of the website, which provided a detailed description of the product, the phosphorus content was reported to be 230 mg per serving.

Phosphorus Enhanced Meat

Enhanced meat is fresh meat that has been injected with solution containing water and other ingredients including phosphate salts (Murphy-Gutekunst & Uribarri, 2005).

The other ingredients consist of a mixture of sodium salts, phosphate salts, potassium salts, antioxidants, and/or flavoring. In appearance, enhanced meat resembles fresh meat, however, it is differs in nutritional value. Phosphate salts enhance meat color, reduce purge, help to retain moisture and reduce rancidity from oxidation caused by the metals within the meat.

Consumer convenience is the first reason that meat is enhanced (Murphy-Gutekunst & Uribarri, 2005). Enhanced meat products provide a quick and easy solution to everyday meal planning. In appearance, enhanced meat resembles fresh meat, however, it differs in nutritional value. Another major reason meat is enhanced is to increase profitability. As discussed above, enhanced meats have less purge, a longer shelf life, and longer-lasting color, resulting in more products being sold than wasted. Additionally, enhanced meats require little, if any, additional labor to bring them from processor to market. Many enhanced meats are package-ready for display, whereas others require repackaging into individual sales units. Because no traditional butchering of the meat is required, labor costs are lower.

Despite federal guidelines requiring manufacturers to include a notification statement of enhancement and a nutrition label, most consumers are not aware that they are purchasing an altered product (Murphy-Gutekunst & Uribarri, 2005). The notification statement is usually written in small print often not noticed by the purchaser. Also if the product has been repackaged into individual selling portions, the store is responsible for affixing the provided nutrition label on each individual packet, a step that often may be missed.

Employee and meat managers may not be certain whether the product they are selling is enhanced (Murphy-Gutekunst & Uribarri, 2005). Some rely on central purchasing for their fresh meat inventory. One week they may receive meat that is not enhanced, and another week they may receive enhanced meat. If the label is not on the individual selling unit, neither the public nor the store knows for certain.

Enhanced meats have hidden sources of phosphorus with each salt having multiple names, making it harder to identify added phosphate ingredients on the nutrition labels when they are present (Murphy-Gutekunst & Uribarri, 2005; see Table 5). Currently, there is not a practical way to know how much additional phosphate enhanced meats have. The nutrition label no longer requires the inclusion of phosphorus, and specifics about how much phosphate salt is used in considered proprietary information by the food manufacturers.

Table 5

Most Common Phosphate Salts Used

Phosphate Salt	Alternative Names	Purpose
Disodium phosphate	Sodium phosphate, dibasic; DSP/A; disodium monohydrogen orthophosphate; disodium monophosphate	Texturizer
Monosodium phosphate	Monosodium dihydrogen orthophosphate; sodium phosphate dibasic	Emulsifier
Potassium tripolyphosphate	Pentapostassium triphosphate, KTPP	Moisture retention
Sodium acid pyrophosphate	SAPP; disodium dihydrogen pyrophosphate; acid sodium pyrophosphate	Color
Sodium hexametaphosphate	HMP; sodium polyphosphate; graham's salt	Reduce purge, emulsifier
Sodium tripolyphosphate	Sodium triphosphate; pentasodium triphosphate; STPP, STP	Flavor Enhancer
Tetrasodium pyrophosphate	Sodium pyrophosphate; tetrasodium diphosphate; sodium diphosphate, TSPP	Moisture retention
Trisodium triphosphate	Sodium phosphate; tribasic; TSP; TSPA; trisodium monophosphate	Antimicrobial

Phosphoric Acid

Phosphoric acid, also known as orthophosphoric acid or phosphoric(V) acid, is a $_3PO_4$ (Wikipedia, 2007a). Alternatively, orthophosphoric acid molecules can combine with themselves to form a variety of compounds referred to as phosphoric acids in a more general way. The term "phosphoric acid" can also refer to a chemical or reagent consisting of phosphoric acids, usually mostly orthophosphoric acid. Most people and even chemists simply refer to orthophosphoric acid as "phosphoric acid." Orthophosphoric acid is a non-toxic, inorganic, rather weak triprotic acid which, when pure, is a solid at room temperature and pressure.

Phosphoric acid is manufactured commercially from phosphate rock mined principally in North Africa and North America ("Acidulants," n. d.). Phosphoric acid is the acidulant acid used in the second largest amount by the food industry. This is because of its use in one single product that is produced in massive amounts: cola drinks.

Cola drinks are the best selling flavored soft drinks in the world. The acid used in these drinks is exclusively phosphoric acid. This has a harsh, biting taste which complements the cola flavor.

Phosphorus in Soda

The greatest contributor to increases in phosphorus consumption among the population is due to soda. The National Soft Drink Association (as cited in Valentine, 2006) indicated that consumption of soft drinks is now over 600 12-ounce servings per person per year. Since 1978, soda consumption is the United States has tripled for boys

and doubled in girls. Young males ages 12-29 are the biggest consumers at over 160 gallons per year or almost two quarts per day.

Over the last 30 years, a virtual tome of information has been published linking soft drink consumption to a rise in osteoporosis and bone fractures. New evidence has shown an alarming rise in deficiencies of calcium and other minerals and resulting bone fractures in young girls. A 1994 report published in the Journal of Adolescent Health (Frisch & Wyshak) summarized a small study (76 girls and 51 boys) and pointed toward an increasing and "strong association between cola beverage consumption and bone fractures in girls" (p. 210). High calcium intake offered some protection. For boys, only low total caloric intake was associated with a higher risk of bone fractures. The study concluded with the following: "The high consumption of carbonated beverages and the declining consumption of milk are of great public health significance for girls and women because of their proneness to osteoporosis in later life" (p. 210).

A larger, cross sectional retrospective study of 460 high school girls was published in Pediatrics and Adolescent Medicine in June 2000 (as cited in Valentine, 2006). The study indicated that cola beverages were "highly associated with bone fractures" (p. 211). In their conclusion the authors warned that, ". . . national concern and alarm about the health impact of carbonated beverage consumption on teenaged girls is supported by the findings of this study" (p. 211).

As reported in *Present Knowledge in Nutrition* (Bowman & Russell, 2001), phosphorus intake in individuals who consume soda may range from 120-360 mg/day depending on the amount consumed. The phosphoric acid in soda has been associated with adverse health effects. Studies in men who had experienced an incident of renal

stones showed that restricted consumption of phosphorus-containing soft drinks significantly reduced the three-year recurrence rate of stones compared to unrestricted controls. A case study in Mexican children (as cited in Bowman & Russell, 2001) assessed whether the intake of soft drinks containing phosphoric acid was associated with hypocalcemia. A similar study (as cited in Bowman & Russell, 2001) examined the relationship between phosphoric acid-containing soft drinks and hypocalcemia. Both found that cola consumption was significantly associated with hypocalcemia. High soft drink consumption has also been linked to higher incidence of bone fracture in adolescence.

Now that soft drinks are sold in almost all public and private schools, dentists are noticing a condition in teenagers that used to be found only in the elderly-a complete loss of enamel on the teeth, resulting in yellow teeth (Valentine, 2006). The culprit is phosphoric acid in soft drinks, which causes tooth rot as well as digestive problems and bone loss. Dentists are reporting complete loss of the enamel on the front teeth in teenaged boys and girls who habitually drink sodas.

Normally the saliva is slightly alkaline, with a pH of about 7.4 (Valentine, 2006). When sodas are sipped throughout the day, as is often the case with teenagers, the phosphoric acid lowers the pH of the saliva to acidic levels. In order to buffer this acidic saliva, and bring the pH level above 7 again, the body pulls calcium ions from the teeth. The result is a very rapid depletion of the enamel coating on the teeth. When dentists do cosmetic bonding, they first roughen up the enamel with a chemical compound - phosphoric acid! Young people who must have all their yellowed front teeth cosmetically

bonded have already done part of the dentist's job, by roughening up the tooth surface with phosphoric acid.

However, Fitzpatrick (2002) reported the use of phosphoric acid in cola beverages in most of the past studies have been flawed because of inaccuracies on actual nutritional intake. When a person drinks too much soda, it is at the expense of not drinking milk, a beverage that contains much more calcium (and phosphate). If the body is nutritionally replete with calcium, then substances such as phosphoric acid (or caffeine) do not affect bone metabolism. Thus, it is the substitution of cola beverages for a healthier beverage such as milk that is associated with the bone loss.

A study conducted by the Center for Population Studies (Frisch & Wyshak, 1994) explored the association between carbonated beverage consumption, as well as other nutritional intake, and the occurrence of bone fractures in girls (mean +/- SD) 14.3 yr +/- 1.8 and boys 14.6 yr +/- 1.6. Food-frequency questionnaires and medical histories were obtained from 76 girls and 51 boys. Subjects were recruited from a swimming club and physicians' offices; their physical characteristics are representative of the normal adolescent population. The data show a strong association between cola beverage consumption and bone fractures in girls [the adjusted odds ratio (OR) = 3.59; 95% confidence interval (CI) 1.21, 10.75; p = 0.022]. High intake of dietary calcium was protective (adjusted OR = 0.284; 95% CI 0.087, 0.920; p = 0.036). The high consumption of carbonated beverages and the declining consumption of milk are of great public health significance for girls and women because of their proneness to osteoporosis in later life.

Adverse Effects of High Phosphorus Diets

According to the Linus Pauling Institute (Higdon, 2003), 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 and their effects on the body.

Phosphorus toxicity can result in diarrhea, nausea, vomiting, twitching, jerking, and convulsions (Obikoya, 2006). However, the major problem from metabolism of high consumption of phosphorus is accompanied by inadequate intake of calcium (Mahan & Escott-Stump, 2000). When phosphorus intakes are too great, insufficient bone accretion and direct stimulation of parathyroid hormone secretion results. This process leads to hypocalcemia, increasing calcium release from bones leaving them brittle and weak. The development of peak bone mass requires adequate amounts of calcium, phosphorus and other nutrients. Adequate intakes of these minerals have a significant impact on peak bone mass development and maintenance. At almost anytime throughout the lifecycle when intake balances are not at recommended levels, parathyroid hormone concentrations in the blood increase. A persistent elevation may contribute to low bone mass.

The relative content of calcium against phosphorus is recognized as a dietary regulator to maintain mineral homeostasis and bone metabolism (Bowman & Russell, 2001). The normal adult range of calcium-phosphorus ratio is 2.3-3.3 with the optimal adult range of 2.8. The normal child range for calcium-phosphorus ratio is 1.3-3.3 with the optimal child range of 2.3. Epidemiologically, high phosphorus intake compared to calcium intake is associated with reduced bone mineral density.

Young adults who are still accruing peak bone mass and ingesting a chronic high phosphorus intake may impair the adaptive mechanism needed for adequate calcium absorption and optimal bone accretion.

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 (Higdon, 2003). 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 parathyroid hormone release by the parathyroid glands. However, high blood phosphorus levels also lead to decreased urinary calcium excretion. If sustained, elevated parathyroid hormone 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 parathyroid hormone levels have been reported in diets that were low in calcium without being high in phosphorus.

Though the amount of research is still limited, there are a number of research journals beginning to publish articles on the effects of high phosphorus diets. More important is the fact that professional health organizations are also beginning to recognize high phosphorus diets as a potential health issue and making statements accordingly.

According to the British Journal of Nutrition (Kempi, Karkkainen & Lamberg-Allardt, 2006), too ample a phosphorus intake, typical of Western diets, could be deleterious to bone through the increase of parathyroid hormone secretion. Few controlled dose-response studies are available on the effects of high phosphorus intake in men. However, a study examining the short-term effects of four phosphorus doses on calcium and bone metabolism in 14 healthy women, 20-28 years of age, who were

randomized to four controlled study days was conducted. Phosphorus supplement doses of 0 (placebo), 250, 750, 1500 mg were taken, divided into three doses during the study day. The meals served were exactly the same during each study day and provided 495 mg phosphorus and 250 mg calcium. The phosphorus doses affected the serum parathyroid hormone in a dose dependent manner. There was a decrease in serum calcium concentration only in the highest dose. The marker of bone formation, bone specific alkaline phosphates, decreased and the bone resorption marker increased with phosphorus doses. The controlled dose response study showed that phosphorus has a dose dependent effect on parathyroid hormone and increases parathyroid hormone secretion significantly when calcium intake is low. High phosphorus intake adversely affects bone metabolism by decreasing bone formation and increasing bone resorption, as indicated by the bone metabolism markers.

Experimental studies of phosphorus loading and investigations using diets reflecting typical consumption patterns have induced hormonal changes that are not conducive to the development or even maintenance of peak bone mass in young adults. Most clinical studies of acute and longer exposures to phosphorus loading in the presence or absence of adequate levels of calcium show an increase in parathyroid hormone levels. The acute rise in parathyroid hormone is considered to be secondary to a decrease in serum ionized calcium concentrations, as demonstrated by abrupt increases in serum phosphorus after an oral load. Several days of high dietary phosphorus intake result in increases in the circadian peak height of serum phosphorus and an elevation in serum Parathyroid hormone levels over the entire circadian pattern.

No prospective study in humans has been conducted to demonstrate bone loss with consumption of a diet with low calcium-phosphorus ratio (Bowman & Russell, 2001). However, in the absence of no long-term clinical studies examining effects on bone, animal models can be used to help resolve whether adverse health effects result from low calcium intake alone or from the prevalent dietary pattern of combined low-calcium, high-phosphorus intake. Secondary hyperparathyroidism and increased bone resorption have been demonstrated in several animal models in response to a low dietary calcium-phosphorus ratio when calcium intake was adequate or deficient. To date, only one study has attempted to discriminate between the effects of a low-calcium diet and those of a low-calcium, high-phosphorus diet. Pettifor, Marie, and Sly (1984) fed diets with various calcium phosphorus contents for 16 months to young vitamin D-replete baboons. The phosphorus content of the four experimental diets was adequate and constant in all but one, and the calcium content was high, medium, or low. After 16 months, the baboons fed the low-calcium, normal phosphorus diet showed histological evidence of increased bone resorption and lower femoral ash content whereas those fed the low-calcium, low-phosphorus diet showed only histological features of osteomalacia. In these growing primates, the diet with the lower calcium-phosphorus ratio appeared to be more harmful to bone that calcium inadequacy alone.

According to the Journal of Nutrition (Calvo & Park, 1996), the dietary intake of phosphorus in the United States is high relative to calcium. Intake estimates from the 1989-1991 Continuing Surveys of Food Intakes by Individuals conducted by the USDA show that for both men and women, median calcium intakes do not meet the Recommended Dietary Allowances (RDAs) for most age groups over 10 years of age,

whereas phosphorus intakes exceed the RDAs for most age groups. The use of phosphorus-containing food additives in the processing of foods contributes substantially to the daily phosphorus intake, and their use is increasing. Because much of the phosphorus through food additive use is not reflected in the estimates of phosphorus intakes derived from national food consumption surveys, these estimates underestimate true dietary intakes of phosphorus. High phosphorus intake has been shown to cause secondary hyperparathyroidism and bone loss in several animal models. High phosphorus, low calcium consumption consistent with current observed intake levels resulted in changes in calcium-regulating hormones that were not conducive to optimizing peak bone mass in young women. Evidence that such high phosphorus intakes may impair synthesis of the active metabolite of vitamin D and disrupt calcium homeostasis particularly in older women has also been shown.

Calcium and phosphorus homeostatic regulation is affected rather early in the process of renal failure, however, clinical signs do not manifest until later in the development of the disease (Bowman & Russell, 2001). As glomarular filtration rate declines, the serum concentration of calcium falls whereas the phosphorus levels rise because the kidneys can neither excrete phosphorus nor efficiently reabsorb calcium. Lower serum ionized calcium initially signals the parathyroid gland to secrete parathyroid hormone, which acts on the renal proximal tubules to decrease the rate of phosphorus resorption. To restore serum calcium concentration to its set level, parathyroid hormone stimulates calcium and phosphorus release from bone as well as vitamin D synthesis by the kidney; the newly synthesized vitamin D facilitates the active absorption of calcium by the intestine. As renal failure progresses vitamin D synthesis is

impaired and low serum calcium levels resulting from poor intestinal absorption stimulate sustained secretions of parathyroid hormone and promote parathyroid gland hyperplasia.

Several studies have demonstrated that dietary phosphorus restriction, independent of serum levels of calcium and vitamin D, attenuates parathyroid hyperplasia and prevents secondary hyperparathyroidism in individuals with chronic renal failure and experimental animal models of renal failure (Bowman & Russell, 2001). Phosphorus-restricted diets are usually prescribed for individuals with chronic renal failure for which the goal is to limit phosphorus intake to 800-1000 mg/day. Because all food contains phosphorus, this dietary goal is difficult to achieve. Most often the diet is unpalatable because high-protein foods and processed foods should be avoided.

Chapter III: Summary

Introduction

This chapter contains a summary of the information obtained during the Review of Literature chapter, as well as an analysis of the findings, limitations and recommendations to this study.

Summary

This purpose of this study was to examine the disadvantages of phosphorus additives in foods and their potential harmful effects on the American population. This was done by reviewing and examining available research and literature on phopshorus, phosphate additives, absorption of phosphorus, the bodily excretion and reabsorption of phosphorus, dietary functions of phosphorus, dietary recommendations of phosphorus, kidney function relating to phosphorus, dietary sources of phosphorus, food additives containing phosphorus, lable monitoring of phosphorus, meat enhancement using phosphorus, phosphoric acid, phosphorus in soda and the adverse effects of high phosphorus diets.

Analysis of Findings

Phosphorus is one of the most abundant elements on earth and is an essential element to human life. It ranks second to calcium in abundance within human tissue as it is present in every cell in the body with functions associated with DNA, RNA, ATP, and the formation of all cell structures.

In relation to food, phosphorus exists in all animal and plant items. In general, good sources of protein are good sources of phosphorus including milk, fish, and eggs. However, the general public is demanding an increasing variety of convenience foods. In order to meet these demands food manufactures have increased their reliance on

phosphorus containing additives to enhance shelf life, decrease cooking time or preparation, enhance food texture, and improve flavor acceptability. Due to this increase phosphorus consumption has been increasing over the past decade and a half. Presently, the government requires processed foods to be labeled with specific nutrients. Unfortunately, phosphorus content is not required to be on labels. Compounding this problem is the fact that phosphorus consumed through food additive use is not reflected in daily phosphorus intake averages derived from national food consumption surveys.

The harmful effects resulting from the increase in phosphorus consumption due to increased food additive intake is great. Research now indicates that increased consumption of phosphorus is linked to rises in rates of osteoporosis, bone fractures, depletion of the enamel coating on the teeth, weak and brittle bones, decrease in optimal bone accretion in youth, reduced levels of Vitamin D in blood, increase of parathyroid hormone secretion, secondary hyperparathyroidism, and increased risk of abnormal deposition of calcium phosphate in previously healthy connective tissues such as cardiac valves and in solid organs such as muscles.

Limitations

While there is an abundant amount of literature regarding phosphorus and how it functions in the body, there is only a minimal amount of information on the actual amounts of phosphorus individuals are now consuming with the increased consumption of convenience foods. Also, the current system for estimating the nutrient content of survey foods in the National Food Consumption Survey is not capable of capturing the phosphorus intake from food additives. Therefore, determination of estimated phosphorus intakes from these additives can only be assumed. In addition, there are also

are limited amounts of longitudinal studies designed to study the effects of high phosphorus diets on animal or human subjects.

Recommendations

In order to aid nutritionists and the general population, the following recommendations are made regarding phosphorus consumption:

For Nutritionists:

1. It is recommended that nutritionists become aware of the basic functions of phosphorus in the human body.
2. It is recommended that nutritionists become aware of food sources that contain high levels of phosphorus and phosphorus additives.
3. It is recommended that nutritionists become aware of the terms used under the ingredients list that indicate food may contain high amounts of phosphorus.
4. It is recommended that nutritionists become familiar with the treatment of individuals who have high phosphorus levels.
5. It is recommended that nutritionists develop alternative food plans for individuals who are consuming high phosphorus diets.
6. It is recommended that nutritionists contact organizations such as the National Kidney Foundation to request additional information on high phosphorus diets.
7. It is recommended that nutritionists inform local media about the potential dangers of high phosphorus diets.
8. It is recommended that nutritionists express to their grocers that they do not want to purchase enhanced meat products.

9. It is recommended that nutritionists contact their Congressional leaders asking that the Food and Drug Administration look at the long-term effects of phosphate overexposure.

For General Consumers:

1. It is recommended that general consumers consult a nutritionist to help educate them more about the adverse affects of high phosphorus diets and what foods contain high amounts of phosphorus and phosphorus additives
2. It is recommended that general consumers consider their portion sizes when eating all foods, especially those that are processed and likely high in phosphorus and phosphorus additives.
3. It is recommended that general consumers become aware of the terms used under the ingredients list that indicate food may contain high amounts of phosphorus.
4. It is recommended that general consumers express to their grocers that they do not want to purchase enhanced meat products.
5. It is recommended that general consumers contact their Congressional leaders asking that the Food and Drug Administration look at the long-term effects of phosphate overexposure.

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