**8 Major Minerals—**

*Calcium, Magnesium, and Phosphorus*

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I. INTRODUCTION

Calcium, phosphorus, and magnesium are elements integral to the function of the musculoskeletal system. These three elements are interrelated in the formation and transduction of energy and the maintenance of healthy bone. Thus, intakes of these nutrients that are neither too low nor excessive are needed to allow people of all ages, including those aged ~30–60 years, to participate in regular exercise and enjoy recreational physical activities.

II. CALCIUM

A. GENERAL PROPERTIES

Calcium, the fifth most abundant element in the biosphere, is an alkaline earth metal with an atomic weight of 40.08. Calcium does not exist in nature as a metal, but the divalent calcium cation (Ca^{2+}) in minerals and solutions is common. Chloride and sulfate salts of Ca^{2+} are water-soluble; most other inorganic Ca^{2+} salts are only slightly soluble in water. The properties of Ca^{2+} (ionic radius of 0.99 angstroms; forms coordination bonds with up to 12 oxygen atoms) has made it the ion of choice to fit into the folds of peptide chains for the maintenance of tertiary structure. Its ionic size and ability to bind reversibly to cell proteins have made Ca^{2+} the most common signal transmitter across the cell membrane and an activator of a number of functional proteins.

B. METABOLIC FUNCTIONS

Calcium has three major metabolic functions. In addition to the fundamental function as a second messenger, coupling intracellular responses to extracellular signals, and as an activator of some functional proteins, calcium is indispensable for skeletal function.

In the role as signaling or messenger ion, Ca^{2+} mediates vascular contraction and vasodilation, muscle contraction, nerve transmission, and hormone action. In response to a chemical, electrical, or physical stimulus, extracellular Ca^{2+} enters the cell or increases intracellularly through release from internal stores such as
endoplasmic or sarcoplasmic reticulum. Increased intracellular Ca\(^{2+}\) stimulates a specific cellular response, such as activation of a kinase to phosphorylate a protein, that results in a physiological response.

A number of enzymes, including several proteases and dehydrogenases, are activated or stabilized by bound calcium independent of changes in intracellular Ca\(^{2+}\).

Muscle contraction exemplifies the roles that Ca\(^{2+}\) plays in signaling and enzyme function. When a muscle fiber receives a nerve stimulus to contract, the initial signal transduction Ca\(^{2+}\) enters the cell from the extracellular space upon membrane depolarization. This Ca\(^{2+}\) activates intracellular release of Ca\(^{2+}\) from the internal storage sites (sarcoplasmic reticulum for muscle) that binds and activates proteins of the contraction complex. Two significant proteins that bind Ca\(^{2+}\) are troponin C, which initiates a series of steps that lead to muscle contraction, and calmodulin, which activates enzymes that break down glycogen for contraction energy. Relaxation occurs when various ionic pumps reduce cytosol Ca\(^{2+}\) by moving it to storage sites and into the extracellular space.

About 99% of total body calcium is found in bones and teeth. Bone crystals have a composition similar to hydroxyapatite \([\text{Ca}_{10}(\text{PO}_4)_6\text{OH}]_2\), which contains about 39% calcium. The crystals, which have the ability to resist compression, are arrayed in a protein matrix, which has the ability to withstand tensile loads. Alterations in either the inorganic (hydroxyapatite) or organic (protein matrix) components can result in changes in bone strength. The skeleton must undergo continuous remodeling throughout life (it is replaced every 10–12 years) to adapt its internal microstructure to changes in the mechanical and physiological environment. Additionally, bone is renewed continuously to repair microdamage to minimize the risk of fracture.

C. Body Reserves

Calcium is the most abundant mineral element in the body. Calcium accounts for 1 to 2% of body weight, or 920–1000 g in an adult female and ~1220 g in an adult male. Approximately 1% of total body calcium is found in extracellular fluids, intracellular structures, and cell membranes. Extracellular Ca\(^{2+}\) concentrations are about 10,000 times higher than intracellular Ca\(^{2+}\) concentrations (about 100 nM). Bones and teeth contain the other 99% of body calcium. The calcium in bone is a large reserve available for times of inadequate intakes to assure the maintenance of extracellular calcium concentrations. Changes in the bone calcium reserve while maintaining extracellular calcium occurs through bone turnover. Thus, a decrease in skeletal calcium reserves is equivalent to a decrease in bone mass, and an increase in reserves is equivalent to an increase in bone mass.

D. Metabolism (Absorption and Excretion)

Calcium absorption occurs through two independent routes, transcellular and paracellular. The transcellular route, localized in the proximal duodenum, is an active or saturable transfer that involves the calcium-binding protein calbindin. Biosynthesis of calbindin \(D_\text{cr}\) is totally vitamin D-dependent. Thus, transcellular absorption of
calcium is dependent upon vitamin D. Paracellular transfer occurs throughout the small intestine, and is a nonsaturable diffusion process that is a linear function of the calcium concentration in the intestinal contents. When calcium intake is moderately high, the paracellular route accounts for at least two thirds of the total calcium absorbed. About 20–25% of calcium ingested is absorbed with intakes between 600 and 1000 mg (15–25 mmol)/d.

Regardless of the source of calcium, calcium absorption efficiency decreases with increasing intake, but total calcium absorbed continues to increase. Calcium absorption is more efficient when consumed in divided doses throughout the day. Contrary to the earlier suggestion that protein decreases calcium balance, it has been shown that calcium retention is not reduced by high dietary protein from sources such as meat. Calcium absorption declines with age.

Mechanisms for calcium transport in the intestine also exist in the nephron. Most of the calcium arriving in the kidney is reabsorbed by the passive, paracellular mechanism. Active calcium reabsorption is mediated by calbindin D28k and occurs mainly in the distal convoluted tubule. Both active and passive transport systems respond to extracellular Ca\(^{2+}\) concentration that is detected by Ca\(^{2+}\)-sensing receptors, and is stimulated by parathyroid hormone and 1,25-dihydroxy vitamin D. The urinary calcium output of a 70-kg man is about 200–280 mg (5–7 mmol)/d or about 0.3% of the filtered load.

Calcium in stool comes from food and endogenous (cellular debris and body fluids) calcium entering the intestinal tract and escaping absorption. The quantity of endogenous calcium lost in stool daily is about the same as that lost in urine.

E. DIETARY AND SUPPLEMENTAL SOURCES

Milk products, the most calcium-dense foods in Western diets, contain about 300 mg (7.5 mmol) calcium per serving (e.g., 8 oz milk or yogurt, or 1.5 oz cheddar cheese). Unfortunately, milk is being replaced by sweetened soft drinks and juices that do not contain much calcium: Americans drank about 2.5 times more soft drinks than milk in 2001. Grains are not particularly rich in calcium, but when consumed in large quantities can provide a substantial portion of dietary calcium. After milk, the second most important food sources of calcium for Mexican-American and Puerto Rican adults are corn tortillas and bread, respectively.

In addition to content, calcium sources should be evaluated based on bioavailability. About 32% of calcium in milk and dairy products is absorbed. Fractional absorption of calcium from low-oxalate vegetables such as broccoli (61%), bok choy (54%), and kale (49%) is higher than from milk. Calcium bioavailability is typically reduced by oxalate and phytate in foods, but food products from soybeans, rich in both oxalate and phytate, have relatively high calcium bioavailability.

Although the best source of calcium is food, calcium supplements are often consumed to prevent or treat bone loss that can lead to osteoporosis and fractures. Common salts used in supplements include calcium carbonate, citrate, citrate malate, lactate, gluconate, fumarate, malate fumarate, gluconate, tricalcium phosphate, dicalcium phosphate, bone meal, oyster shell, coral and algal calcium, and dolomite. Some supplements (e.g., bone meal, dolomite) may have heavy metal (e.g.,
lead) contaminants and thus should be avoided. Calcium salts, regardless of solubility, have fractional calcium absorption values similar to that of milk, with the exception of calcium citrate malate, which is slightly higher.8

F. Status Assessment

As indicated above, the skeleton is a source of calcium that assures critical cellular functions and maintains extracellular fluid concentrations. If serum calcium is more than 10% away from the population mean, disease (e.g., hypo- or hyperthyroidism) probably is the cause.3,6 Thus, there is no good biochemical indicator of calcium status for the healthy middle-aged adult. Determination of the amount of bone mineral is the best current method for assessing calcium status, but this determination may be affected by other factors such as weight, gonadal hormone status, and other dietary factors (e.g., vitamin D, magnesium levels). Total-body bone mineral can be estimated by using dual x-ray absorption, microcomputed tomography, or peripheral quantitative computed tomography.12 Numerous blood and urine tests indicate whether bone is being lost or formed after a dietary modification or a pharmacologic intervention. However, tests such as serum osteocalcin and bone-specific alkaline phosphatase for bone formation, and urinary type I collagen cross-linked N-telopeptides, type I C-telopeptide breakdown products, pyridinoline, deoxypyridinoline, and helical peptide for bone resorption, do not predict fracture risk or calcium status well.13

Many middle-aged Americans do not consume adequate intakes (AI) of calcium. Mean usual calcium intakes from food calculated for males and females, respectively, from NHANES 2001–2002 data,14 were for ages 31–50 years, 1021 and 755 mg, and for ages 51–60 years, 874 and 701 mg. Calcium intake data (four standardized 24-h dietary recalls collected 3–6 wks apart) for 4680 men and women aged 40–59 years in Japan, China, United Kingdom, and the United States indicated mean daily calcium intakes of 605, 356, 1013, and 882 mg, respectively.15

G. Toxicity

Evidence that health can be harmed in healthy adults by excessive intakes of calcium is limited. Now that the treatment of peptic ulcers with antacids plus large quantities of milk is rarely prescribed, the occurrence of a syndrome termed milk-alkali disease is rare. Symptoms of this syndrome, which causes hypocalcemia, are lax muscle tone, constipation, large urine volumes, nausea, and ultimately, confusion, coma, and death.2 A review in 1997 revealed only 26 reported cases of milk-alkali disease without renal disease associated with high calcium intakes since 1980.16

Nephrolithiasis (kidney stones) has been associated with excessive calcium intake.10 However, numerous other factors have been associated with nephrolithiasis, including high intakes of oxalate, protein, vegetable fiber, and phosphorus and low intakes of magnesium.10 As a result, it has been suggested that excess calcium intake may play only a contributing role in the development of nephrolithiasis.10

Studies of whether high calcium intakes negatively affect the metabolism of some minerals, particularly iron, magnesium and zinc, have been inconclusive. For example, although 400 mg (10 mmol) of calcium significantly decreased iron
absorption in a single meal, calcium supplementation at 1200 mg (30 mmol)/d for 6 months did not decrease iron status in 11 iron-replete adults. High calcium intakes apparently result in a reduced magnesium status in rats but not in humans. High calcium was found to decrease zinc balance in one human study, but increased milk consumption and calcium phosphate supplementation did not decrease zinc absorption in another.

H. INTERACTIONS WITH OTHER NUTRIENTS AND DRUGS

As calcium and sodium share the same transport system in the kidney proximal tubule, sodium can have a negative effect on calcium metabolism. Every 1000 mg (43 mmol) of sodium excreted by the kidney results in an additional loss of 26.3 mg calcium (26.3 mmol). This additional loss apparently is not offset by changes in calcium absorption because a high sodium intake results in bone loss. Thus, a high sodium intake has a negative effect on the calcium economy.

Dietary protein also increases urinary calcium loss, apparently through an increased urinary acid load caused by the presence of phosphoric and sulfuric acids from the breakdown of phosphorus- and sulfur-containing amino acids. However, dietary protein does not decrease calcium retention because of offsetting changes in endogenous secretion or absorption of calcium.

An over-the-counter drug that can increase calcium loss is aluminum-containing antacid. Therapeutic doses of aluminum-containing antacids can increase daily urinary excretion by 50 mg (1.25 mmol) or more.

Some non-digestible oligosaccharides (e.g., inulin) enhance calcium absorption and bone mineralization. Vitamin D has long been recognized as an effector of both absorption and excretion of calcium (see Section II.D). Vitamin D induces the formation of calcium-binding calbindins that facilitate active transport in the intestine and kidney.

A large number of drugs have been developed for the prevention or treatment of osteoporosis. These drugs act by increasing the availability of calcium from the gastrointestinal tract, decreasing the rate of bone resorption, or increasing the rate of bone formation. It is beyond the scope of this review to discuss each drug individually. Most drugs, however, are antiresorptive agents: they include estrogens, selective estrogen receptor modulators (SERMs), isoflavones (act like weak SERMs or weak estrogen agonists), bisphosphonates, and calcitonin. Some experimental anabolic agents that have promise for stimulating bone formation are parathyroid hormone, growth hormone, and insulin-like growth factor-1. Calcium ingested in supra nutritional amounts will increase the amount of calcium crossing the intestinal tract.

I. EFFECTS OF DEFICIENCY OR EXCESS ON PHYSICAL PERFORMANCE

The maintenance of extracellular Ca$^{2+}$ by the mobilization of skeletal calcium stores means that nutritional calcium deficiency almost never manifests itself as a shortage of Ca$^{2+}$ in critical cellular or physiological processes. Thus, for the physically
active healthy individual. The only concern about calcium intake is an amount that will maintain bone health. If bone renewal during remodeling or turnover is slower than bone loss, osteoporosis may occur. If bone repairing is slower than microdamage accumulation, stress fractures may occur. In a large case-controlled study of hip fracture risk in women in Europe, fracture risk declined until calcium intake rose to an estimated 500 mg (12.5 mmol)/d.\textsuperscript{27} Most studies with adults showing a positive influence of high dietary calcium in decreasing bone loss or fracture risk also had supplemental vitamin D as an experimental co-variable. Calcium supplementation alone of individuals consuming more than 500 mg (12.5 mmol) Ca/d has not been shown to decrease fracture risk.\textsuperscript{27, 30}

As indicated in the Section II.G, an excessive intake of calcium that would affect physical performance is unlikely for a healthy middle-aged individual.

\section{Dietary Recommendations}

Calcium intake recommendations vary widely worldwide, with the United States among the highest.\textsuperscript{10} In the United States, a Recommended Dietary Allowance (RDA) was not established for calcium because of concerns that included uncertainties in the precision and significance of balance studies, and lack of concordance between mean calcium intakes and experimentally derived values predicted as necessary for a desirable amount of calcium retention.\textsuperscript{10} As a result, only AI were established for middle-aged adults; these were 1000 mg (25 mmol)/d for ages 31–50 years, and 1200 mg (30 mmol)/d for ages 51–60 years.\textsuperscript{10} The calcium dietary reference intake in the United Kingdom for adults aged 30–60 years is a much lower 700 mg (17.5 mmol)/d.\textsuperscript{31} In India, the recommended dietary allowance for calcium is 400 mg (10 mmol)/d for adults.\textsuperscript{42} Recently, an analysis of primary calcium balance data from tightly controlled metabolic feeding studies indicated an RDA of 1035 mg (25.8 mmol)/d for men and 741 mg (18.5 mmol)/d for women.\textsuperscript{33} Several countries and organizations, including the United States and the European Community have established 2500 mg (62.4 mmol)/d as the upper limit (UL) for calcium.\textsuperscript{34}

\section{Future Research Needs}

Perhaps the most pressing need is for a marker of calcium status more determinate than the estimation of calcium reserve in the skeleton. In addition, the effect of lifestyle choices on calcium requirements needs further definition. For example, about 65\% of the U.S. population is either overweight or obese.\textsuperscript{35} Weight loss, which is recommended to prevent comorbid conditions, may cause bone loss.\textsuperscript{35} Recommendations for calcium intakes during weight loss need clarification because they apparently will vary with initial body weight, age, gender, physical activity, and conditions of dieting.\textsuperscript{35} An example of age's affecting calcium loss during weight loss is that premenopausal overweight women did not lose bone during moderate weight loss with adequate (1 g/d) or higher calcium intakes (1.8 g/d),\textsuperscript{36} but postmenopausal overweight women lost bone during moderate weight loss while consuming 1 g calcium/d.\textsuperscript{37} Consuming 1.7 g calcium/d only mitigated the bone loss in postmenopausal women.
L. SUMMARY

Calcium is a critical ion for vascular contraction and vasodilation, muscle contraction, nerve transmission, hormone action, and bone growth and maintenance in the physically active middle-aged adult. However, the skeleton maintains extracellular calcium so that a nutritional calcium deficiency almost never manifests itself as a shortage of Ca^{2+} for critical cellular physiological processes. Thus, the major calcium concern for the physically active healthy middle-aged individual is an intake that will prevent bone loss and fractures. Calcium intakes near the AI (1.0 g for ages 31–50 years and 1.2 g for ages 50–60 years) will assure bone health if there are no other health problems, lifestyle conditions, or nutrient deficiencies affecting bone turnover. Consuming greater amounts of calcium is unlikely to have any further benefit for physically active middle-aged adults.

III. MAGNESIUM

A. General Properties

Magnesium, the eighth most abundant element on earth,\textsuperscript{38} is an alkaline earth metal with an atomic weight of 24.31. Magnesium does not exist in nature as a metal, but the divalent magnesium cation (Mg^{2+}) is common in minerals and solutions. It is the second most abundant cation in seawater.\textsuperscript{38} One compound, magnesium sulfate (Epsom salts) is obtained from the wastewater (bittern) of solar salt production. Cooling diluted bittern to between −5° and −10° C precipitates up to 70% of the magnesium sulfate that is removed by filtration. Small amounts of Mg^{2+} contribute to the tartness and taste of natural waters. Mg^{2+}, although chemically similar to Ca^{2+}, does not bond as well as calcium to proteins, but still is involved in over 300 enzyme reactions through binding enzyme substrates or directly with enzymes.\textsuperscript{38} Magnesium is second to potassium as the most abundant intracellular cation. The ratio of extracellular to intracellular Mg^{2+} is 0.33, which contrasts markedly with the ratio of 10,000 for Ca^{2+}. Thus, unlike calcium, magnesium is not a common signal transmitter from the outside to the inside of cells. However, magnesium through affecting cell membrane receptors and protein phosphorylation is a critical cation for cell signaling.

B. Metabolic Functions

Magnesium is needed for enzymatic reactions vital to every metabolic pathway.\textsuperscript{10, 18, 40} These reactions include those involving DNA, RNA, protein, and adenylate cyclase synthesis, cellular energy production and storage, glycolysis, and preservation of cellular electrolyte composition. Magnesium has two functions in enzymatic reactions. It binds directly to some enzymes to alter their structure or to serve in a catalytic role (e.g., exonuclease, topoisomerase, RNA polymerase, DNA polymerase). Magnesium also binds to enzyme substrates to form complexes with which enzymes react. The predominant role of magnesium is involvement in ATP utilization. An example of this role is the reaction of kinases with MgATP to phosphorylate proteins. Magnesium exists primarily as MgATP in all cells. Magnesium at the cell membrane
level regulates intracellular calcium and potassium, and thus is a controlling factor in nerve transmission, skeletal and smooth muscle contraction, cardiac excitability, vasomotor tone, blood pressure, and bone turnover.

C. **Body Reserves**

Magnesium is the fourth most abundant cation in the body. The adult human body contains about 25 g (1028 mmol) of magnesium, which is about equally divided between bone and soft tissue. Less than 1% of the total body magnesium is in blood. Approximately one-third of skeletal magnesium is exchangeable, and acts as a pool for maintaining normal concentrations of extracellular magnesium. The other two-thirds of skeletal magnesium is an integral part of bone mineral crystals. This magnesium is not readily labile and thus is not available for metabolic needs during periods of magnesium deficiency. Normal serum magnesium concentrations, which are tightly regulated, range from 1.8 to 2.3 mg/dL (0.74–0.95 mmol/L).

D. **Metabolism (Absorption and Excretion)**

Magnesium is absorbed throughout the intestinal tract, but the greatest amount is absorbed in the distal jejunum and ileum. Between 40% and 60% of ingested magnesium is absorbed by using both passive paracellular and active transport mechanisms. Net magnesium absorption increases with increasing intake, but fractional magnesium absorption falls. About 90% of intestinal magnesium absorption is through the paracellular route when the dietary intake is adequate. A greater fractional absorption through the active transport system occurs when dietary intake is low. Thus, absorption mechanisms contribute to magnesium homeostasis.

The kidney, however, is the primary organ regulating magnesium homeostasis. About 10% of the total body magnesium is normally filtered through the glomeruli of an healthy adult, with only 5% of the filtered magnesium’s being excreted. About 90–95% of magnesium is reabsorbed through a paracellular mechanism in the proximal convoluted tubules and the loops of Henle, the other 5–10% is reabsorbed by an active transepithelial pathway in the distal convoluted tubules. Renal magnesium excretion decreases to as low as 12–24 mg (0.5–0.10 mmol)/d when dietary intakes are deficient. When body stores are normal, excess absorbed magnesium is almost entirely excreted.

E. **Dietary and Supplemental Sources**

Green leafy vegetables, whole grains, and nuts are the richest sources of magnesium. Milk (about 100 mg [4.11 mmol] Mg/L) and milk products provide moderate amounts of magnesium. A variety of magnesium salts are used as supplements, including oxide, hydroxide, citrate, chloride, gluconate, lactate, and aspartate. The fractional absorption of magnesium from these supplements depends on the solubility in the intestinal fluid. For example, highly soluble magnesium citrate is much better absorbed than poorly soluble magnesium oxide.
F. Status Assessment

Low serum magnesium is the most common method for diagnosing severe magnesium deficiency. However, plasma or serum magnesium is a poor indicator of subclinical magnesium deficiency because exchangeable skeletal magnesium and urinary responses to changes in magnesium intake maintain extracellular magnesium at a rather constant level even while tissue magnesium is decreasing. Thus, normal serum and plasma magnesium concentrations have been found in individuals with low magnesium in erythrocytes and tissues.38

Efforts to find an indicator of subclinical magnesium status (also called chronic latent magnesium deficiency38,42) have not yielded a cost-effective one that has been well validated. Among the tests evaluated as an indicator of magnesium status are serum ionized magnesium, urinary magnesium excretion, sublingual cellular magnesium, erythrocyte magnesium, and the magnesium load test.

At present, the magnesium load test is the test of choice to diagnose a total body deficit of magnesium. This test determines the percentage of magnesium retained over a given period of time after the parenteral administration of a magnesium load.38,42 Retention of a greater percentage than that (22–25%) by individuals with adequate magnesium status indicates some body magnesium depletion.38 This test is invasive, time-consuming, and expensive; requires hospitalization or close supervision for about 24 hours after magnesium infusion; and requires careful urine collection for laboratory analysis.

The ionized fraction (61%) in serum is the physiologically active form of magnesium that serves as a metabolic cofactor for many enzymatic reactions. Measurement of this fraction has been suggested as appropriate for assessing magnesium status. However, this test has the same problem as the determination of total serum magnesium in measuring subclinical magnesium deficiency. For example, the magnesium load test and serum total and ionized magnesium concentration were determined in 44 critically ill persons.43 Of the 19 subjects who were determined to be magnesium deficient by the magnesium load test, only two had serum total or ionized magnesium concentrations below the reference interval. One review found that the determination of ionized magnesium concentration had limited value in assessing magnesium status in disorders associated with chronic latent magnesium deficiency.44

A 24-hour magnesium excretion in urine that is more than 10–15% of the amount ingested suggests adequate magnesium status.38 When deficient amounts of magnesium are ingested or absorbed, there is a rapid and progressive reduction in the urinary excretion of magnesium (see Section III.D). Thus, a low urinary excretion of magnesium can occur while serum magnesium is normal and before total body reduction results in changes that become biochemically and clinically significant. Thus, urinary magnesium excretion is best used to corroborate other tests indicating a subclinical magnesium deficiency.

In experimental magnesium deficiency, a decrease in erythrocyte, or erythrocyte membrane, magnesium has been used to indicate a decrease in magnesium status.45,46 However, standard reference intervals have not been established that allow the use of this test for status assessment.

The measurement of magnesium in sublingual epithelial cells may be a method that can assess a subclinical magnesium deficiency.47 At present, this expensive test
determines cellular magnesium by using energy-dispersive X-ray analysis, thus it has been used mainly in research studies. Comparing this test with the magnesium load test would help validate the use of sub-lingual cellular magnesium as an indicator of magnesium status.

The U.S. National Health and Nutrition Examination Survey (NHANES) 2001-2002 data set indicated that the majority of adults in the survey consumed less than the Estimated Average Requirement (EAR) for magnesium. For example, 64% of women aged 51-70 years did not attain the magnesium EAR. Daily intakes of 10% adult males and females were 206 and 148 mg (8.47 and 2.39 mmol) magnesium, respectively. Thus, a significant number of middle-aged adults routinely have magnesium intakes that may result in a deficient status. However, it should be noted that a recent survey of the dietary behavior of German adults engaging in different levels of physical activity found that the median magnesium density was higher in the diets of active persons.

G. Toxicity

Severe magnesium toxicity results in high serum magnesium (hypermagnesemia). Signs of hypermagnesemia include lethargy, confusion, nausea, diarrhea, appetite loss, muscle weakness, breathing difficulty, low blood pressure, and irregular heart rhythm. Hypermagnesemia is most commonly associated with the combination of impaired renal function and high intakes of nonfood sources of magnesium such as magnesium-containing laxatives and antacids. Thus, hypermagnesemia is not an issue for the healthy middle-aged physically active adult.

The major effect of excessive magnesium intake without hypermagnesemia is diarrhea. Nausea and abdominal cramping may also occur.

H. Interactions with Other Nutrients and Drugs

Protein intake affects magnesium balance and retention. Low dietary protein (30 mg/d) resulted in negative magnesium balance in adult females when dietary magnesium was less than 180 mg (7.4 mmol)/d; a higher protein intake prevented the negative balance. Magnesium absorption by adolescent boys was lower when dietary protein was low (43 mg/d) than when high (93 mg/d). In addition, low dietary protein with a magnesium intake of 240 mg (9.87 mmol)/d resulted in negative magnesium retention, which did not occur when dietary protein was high. High protein intakes that increase renal acid load also may decrease magnesium retention through increased renal loss.

A high zinc intake decreases magnesium absorption and balance. Zinc supplementation at 142 mg (2.17 mmol)/d decreased magnesium absorption and balance in adult males. A more moderate zinc intake (53 mg [0.81 mmol]/d) decreased magnesium balance in postmenopausal women.

High dietary phosphorus was found to decrease magnesium absorption. The decreased absorption may have been the result of the formation of insoluble magnesium phosphate. Magnesium absorption also may be decreased through the binding with phosphate groups of phytate in high-fiber foods. The interaction between
magnesium and phosphorus may be more than an effect on absorption. Magnesium supplementation ameliorates the kidney calcification and bone loss induced by high dietary phosphorus in experimental animals. In postmenopausal women, a deficient magnesium intake (~107 mg [4.40 mmol/d]) increased urinary excretion of phosphorus, but apparent phosphorus absorption increased, so no change in phosphorus balance occurred.57

Another factor that may negatively affect magnesium requirement is vitamin B6 deficiency. Young women depleted of vitamin B6 exhibited negative magnesium balance because of increased urinary excretion.58

There is a relationship between magnesium and calcium, but this apparently is not at the intestinal level. High calcium intakes (up to 2400 mg/d) were found to have no effect on magnesium absorption or retention,59,60 and high magnesium intakes (up to 826 mg/d) were found to have no effect on calcium absorption.61 However, moderate magnesium deprivation increased calcium balance or retention in postmenopausal women, with a decreased urinary calcium excretion contributing most to the increased balance.57,62 The increased retention suggests that moderate magnesium deficiency increases intracellular calcium.

Although not a nutrient, short-chain fructo-oligosaccharides (e.g., inulin) increases the intestinal absorption of magnesium by about 25%.50,63 Vitamin D may also have a limited effect on magnesium absorption. Pharmacologic doses have been found to increase magnesium absorption in animals.64 However, the increased absorption may be counteracted by an increase in urinary magnesium excretion induced by vitamin D.

There are some drugs, particularly those used for controlling hypertension, that affect magnesium metabolism. Diuretics such as furosemide and ethacrynic acid have been shown to cause marked renal wasting of magnesium.38 On the other hand, angiotension converting enzyme (ACE) inhibitors may increase cellular and serum magnesium.66 Increased intracellular magnesium may result in heart arrhythmia.66

I. EFFECTS OF DEFICIENCY OR EXCESS ON PHYSICAL PERFORMANCE

Subclinical or chronic latent magnesium deficiency most likely affects physical performance. A subclinical magnesium deficiency impaired exercise performance in untrained postmenopausal women in a controlled metabolic unit study.45 Heart rate and oxygen consumption increased significantly during submaximal exercise when the women were fed 150 mg (6.17 mmol) compared with 320 mg (13.16 mmol) magnesium/d. Young men consuming about 250 mg (10.28 mmol) magnesium/d (most likely a deficient intake) and participating in a strength-training program showed a greater increase in peak knee-extension torque when fed a 250-mg (10.28) magnesium supplement/d than when fed a placebo.57 Moderately trained adults consuming about 250 mg (10.28 mmol) magnesium/d and taking a supplemental 250 mg (10.28 mmol) magnesium/d (instead of a placebo) had improved cardiorespiratory function during a 30-minute submaximal exercise test.68 An animal study gave similar results. Marginally magnesium-deficient rats exhibited reduced exercise capacity or endurance on a treadmill.69 Subclinical magnesium deficiency also may result in muscle spasms or cramps. For example, magnesium supplementation resolved
muscle cramps, normalized neuromuscular excitability and decreased lactate dehydrogenase and creatine kinase activities in a physically active individual determined to be magnesium-deficient. In another case, magnesium supplementation resolved muscle cramps in a tennis player with hypomagnesemia. There is a lack of reports showing that magnesium supplementation improves performance of individuals with established adequate magnesium status. Thus, differences in magnesium status may be the reason for conflicting reports about the effect of magnesium supplementation on exercise performance.

Magnesium deficiency can amplify some undesirable effects of exercise. Reactive oxygen species production or oxidative stress occurs during exercise. Magnesium deficiency has been shown to increase reactive oxygen species or oxidative stress in experimental animals. The increase apparently induces ultrastructural damage in skeletal muscle (i.e., swollen mitochondria and disorganized sarcoplasmic reticulum). In addition, dietary magnesium deficiency has been shown to induce a pro-oxidant/pro-inflammatory response to rodents characterized by enhanced free radical (lipid radicals and nitric oxide) production, accumulation of oxidation products and pro-oxidant metals, depletion of endogenous antioxidants (e.g., glutathione), and elevated inflammatory mediators (e.g., substance P). These findings suggest that a relationship may exist between oxidative stress induced by exercise and subclinical magnesium deficiency such that one may amplify the adverse effects of the other.

Controlled metabolic unit studies with postmenopausal women have shown that a subclinical magnesium deficiency may induce arrhythmias and changes in potassium metabolism that may affect heart function. Additionally, a chronic low intake of magnesium may contribute to hypertension, bone loss leading to osteoporosis, and insulin resistance and impaired insulin secretion leading to diabetes mellitus. Impaired heart function and fracture risk induced by a chronic, latent magnesium deficiency are noteworthy concerns for middle-aged physically active adults.

Severe magnesium deficiency, which is not likely to occur in the physically active middle-aged adult, usually is the result of dysfunctional states resulting in its malabsorption or excessive excretion; it results in numerous signs and symptoms. Loss of appetite, nausea, vomiting, fatigue, and weakness are early signs of magnesium deficiency. As deficiency becomes more severe, numbness, tingling, muscle contractions and cramps, seizures, personality changes, and coronary spasms (angina pectoris) occur. As indicated earlier, an excessive intake of magnesium that would affect physical performance is unlikely for a healthy middle-aged individual.

J. Dietary Recommendations

The lack of usable data has made it difficult to establish a sound recommendation for magnesium. In 1997, the U.S. Food and Nutrition Board set the magnesium RDA for men and women between ages 30 and 60 years at 420 and 320 mg (17.28 and 13.16 mmol) daily, respectively. These RDAs are consistent with the recommendation of 6 mg (0.25 mmol)/kg body weight/day suggested by Seelig and Durlach. The U.S. RDAs were based almost exclusively on findings from one poorly controlled balance study performed in 1984. In that study, subjects consumed self-selected diets in their home environment and were responsible for the collection of their urine, feces,
and duplicate diet and beverage samples used in the balance determinations. The samples were collected only 1 week each season for 1 year. There was much overlap in the magnesium intakes that resulted in negative and positive magnesium balance. For example, four of 10 women aged 35–53 years were in positive balance or equilibrium with intakes ranging from 182 to 258 mg (7.49 to 10.61 mmol)/d; the other six women were in negative balance with intakes ranging from 164 to 301 mg (6.75 to 12.38 mmol)/d. Three of seven men aged 35–53 years were in equilibrium with intakes ranging from 286 to 418 mg (11.76 to 17.19 mmol)/d; the four men were in negative balance with intakes ranging from 157 to 344 mg (6.46 to 14.15 mmol)/d.

Because of the tenuous nature of the data used, the magnesium RDAs for the United States and Canada have been appropriately questioned. For example, an expert consultation for the Food and Agriculture Organization/World Health Organization (FAO/WHO) concluded that evidence was lacking for nutritional magnesium deficiency occurring with the consumption of diets supplying a range of magnesium intakes sometimes considerably less than the RDA for the United States and Canada or the United Kingdom equivalent to the RDA called Recommended Nutrient Intake (RNI). Th
ces, the expert consultation subjectively set RNIs for magnesium at 220 and 260 mg (9.05 and 10.69 mmol)/d for women and men, respectively, including those between ages 30 and 60 years.

Some recent reports suggest that the RNIs set by the FAO/WHO consultation may be valid. Based on balance data and findings of heart rhythm changes and impaired physiologic function in postmenopausal women fed slightly less than 200 mg (8.23 mmol)/magnesium/d under controlled metabolic unit conditions, consistent intakes less than the RNIs set by the FAO/WHO probably would result in chronic latent magnesium deficiency. Balance data from 27 different tightly controlled metabolic unit studies revealed that neutral magnesium balance, without considering surface losses, occurred at an intake of 165 mg (6.79 mmol)/d with a 95% prediction interval of 113 and 213 mg (4.65 and 8.76 mmol)/d. These recent findings suggest that middle-aged physically active adults should strive for dietary magnesium intakes of over 220 mg (9.05 mmol)/d.

The U.S. Food and Nutrition Board determined that magnesium ingested as a naturally occurring substance in foods would not exert any adverse effects. Because the primary initial manifestation of excessive magnesium intake from nonfood sources is diarrhea, the Board used diarrhea as sensitive hazard to set the UL for magnesium. For middle-aged adults, the UL was set at 350 mg (14.6 mmol) of supplementary magnesium.

K. Future Research Needs

The most pressing research need is for biochemical indicators that provide an accurate and specific assessment of magnesium status. These status assessment indicators then can be used to determine the extent of subclinical or chronic latent magnesium deficiency in apparently healthy populations, including middle-aged physically active adults. There also is a need to assess the relationships between dietary magnesium intakes, indicators of magnesium status, and possible adverse health outcomes such impaired physical performance, bone loss and arrhythmias. Findings from
the assessment of the relationships would help determine intervention strategies to improve magnesium status and determine their impact on specific health outcomes.

L. SUMMARY

Magnesium is needed for enzymatic reactions vital to every metabolic pathway including cellular energy production and storage, glycolysis, and preservation of cellular electrolyte composition. Magnesium also is a controlling factor in nerve transmission, skeletal and smooth muscle contraction, cardiac excitability, blood pressure, and bone turnover. Based on dietary surveys, subclinical or chronic latent magnesium deficiency may occur in significant numbers in middle-aged adults. Subclinical magnesium deficiency has been found to impair energy utilization and exercise performance in adults. Additionally, subclinical magnesium deficiency may amplify some undesirable effects of exercise including oxidative stress; it may impair heart function and result in bone loss. Unfortunately, there is no practical biochemical indicator that provides an accurate and specific assessment of subclinical magnesium status. However, balance studies indicate that intakes of more than 220 mg (9.05 mmol) magnesium/d are needed to prevent adverse effects of chronic latent magnesium deficiency. An intake near the RDA set by the U.S. Food and Nutrition Board (420 and 320 mg [17.28 and 13.16 mmol/d for men and women, respectively) should assure adequate magnesium status for almost all physically active middle-aged adults. Consuming greater amounts of magnesium is unlikely to have any further benefit, thus there is no reason to exceed the U.S. UL of 350 mg (14.40 mmol) supplementary magnesium/d.

IV. PHOSPHORUS

A. GENERAL PROPERTIES

Phosphorus (atomic number 15 and atomic weight of 30.97) is too reactive to exist in nature in its elemental form. About 0.12% of the earth's crust is phosphorus, which often is found combined with oxygen as inorganic or organic phosphates. Inorganic phosphates have the same basic orthophosphate tetrahedron structure, which is 1 phosphorus atom surrounded by 4 oxygen atoms with either a monovalent or a divalent anionic charge. The predominant species of inorganic phosphate in all biological fluids and tissues is the divalent anion, HPO$_4^{2-}$. At normal blood pH, the ratio of HPO$_4^{2-}$ ions to H$_2$PO$_4^{-}$ is 4 to 1. Phosphate ions in blood serve as a buffer of blood pH and as a regulator of whole body acid–base balance through facilitating the renal excretion hydrogen ions by shifting from HPO$_4^{2-}$ to H$_2$PO$_4^{-}$. Phosphate ions contribute about 50% to daily urinary hydrogen ion excretion, or titratable acidity.

B. METABOLIC FUNCTIONS

Phosphorus is involved in virtually every aspect of metabolism. It is an integral part of structural molecules including phospholipids and phosphoproteins. Membranes that surround all cells and separate intracellular organelles from
cytoplasm are primarily a bilayer of phospholipids. Glucose, the ultimate energy source for most cellular activities, must be phosphorylated before entering into the glycolytic pathway. Energy storage and use is in the form of phosphorus-containing compounds ATP and creatine phosphate. Cyclic AMP and cyclic GMP are intracellular second messengers regulating many biochemical processes including the actions of many hormones. DNA and RNA contain phosphate groups linking deoxyribose and ribose along the backbone of these molecules, respectively. Phosphorus is a critical component of virtually all enzyme reactions, often in the form of a co-enzyme such MgATP and nicotinamide adenine dinucleotide, and the addition or removal of phosphate moieties changes the catalytic activity of many enzymes. In extracellular fluids, about 30% of phosphorus exists as inorganic ions that help maintain osmotic pressure and acid-base balance. The highly anionic organic phosphate, 2,3-diphosphoglycerate binds to hemoglobin to facilitate the release of oxygen to tissues. In bones and teeth, phosphorus is a component of crystalline hydroxyapatite \([Ca_{10}(PO_4)_6(OH)_2]\). Over 50% of bone mineral mass is the phosphate ion.

C. **Body Reserves**

The adult human body contains about 850 g (27.45 mol) of elemental phosphorus (about 1.1% of total body weight) with about 85% in the skeleton, 14% in the soft tissues, and 1% in extracellular fluids, intracellular structures, and cell membranes. Phosphate is the most abundant anion in the cell. The hydroxyapatite in bone crystals has a constant calcium-phosphate ratio of about 2:1. Bone acts as a reservoir for exchangeable phosphate ions. The flux between plasma and bone phosphate ion is very high, about 5 g (1614 mmol)/d. Bone mineral phosphate ion efflux and influx occurs through ionic exchange and active bone resorption. Bone usually has a slow turnover rate so dynamic ion exchange is the primary bone mechanism for maintaining phosphate ion concentrations in plasma and extracellular fluids. The kidney (see below) is the other major regulator of phosphate balance in the body. The kidney and bone keep serum phosphate concentration, which has circadian rhythm, between 2.5 to 4.5 mg/dL (0.87 to 1.45 mmol/L).

D. **Metabolism (Absorption and Excretion)**

Most phosphorus absorption occurs as the inorganic phosphate ion because intestinal phosphatases hydrolyze most organic phosphorus in foods to this form. Phosphorus is highly bioavailable (55–70%) from most food sources. Foods containing phosphorus as phytate, the storage form of phosphorus in plant seeds (e.g., beans, peas, nuts, cereals), is an exception. The hydrolysis of phytate depends on exogenous phytase provided by yeasts, colonic bacteria, and foods. For example, leavening with yeast that produces phytase increases phosphorus bioavailability from breads. Because a number of factors influence the presence of phytase, phosphorus bioavailability from food phytate is quite variable. Milk casein contains a phosphopeptide that also is resistant to enzymatic hydrolysis. Absorption of phosphate ions occurs throughout the small intestine, primarily by facilitated diffusion. Active transport of phosphate
becomes important only when phosphorus intake is low or the demand for phosphorus is greatly increased.\textsuperscript{82}

The kidney is the primary organ regulating phosphorus homeostasis. Humoral phosphaturic factors (phosphatonin), parathyroid hormone, and 1,25-hydroxy vitamin D influence the reabsorption of filtered phosphate ions.\textsuperscript{82} The kidneys reabsorb about 80% of filtered phosphate ions, with 60% reabsorbed in the proximal convoluted tubule, 15–20% in the proximal straight tubule, and less than 10% in the distal segments of the nephron.\textsuperscript{82}

An uncompensated change in absorption, excretion, or exchange with bone mineral will result in either hypophosphatemia or hyperphosphatemia.\textsuperscript{82} Hypophosphatemia also can induced by exercise or changes in arterial blood acid–base balance, which redistribute phosphate from extracellular fluids to intracellular sites.\textsuperscript{82}

E. Dietary and Supplemental Sources

Phosphorus is found in nearly all foods, where it occurs as a mixture of inorganic phosphate and various organic phosphorus compounds. Foods high in protein are generally high in phosphorus. About 15 mg (0.48 mmol) of phosphorus is consumed with every gram of protein. Thus, meat and dairy products are major contributors to the daily intake of phosphorus.

The phosphorus content of the U.S. food supply has increased in recent years because phosphate salts are added to processed foods for non-nutrient functions (moisture retention, smoothness, and binding), and cola soft drinks are acidulated with phosphoric acid.\textsuperscript{82} As a result, the median phosphorus intakes by females and males in the United States exceed the RDA (700 mg [22.6 mmol]/d for middle-aged adults) by 300 and 800 mg (9.69 and 25.83 mmol)/d.\textsuperscript{10}

Phosphorus in supplement form for most people usually occurs as an anion component of another nutrient (e.g., calcium phosphate) in supplements. However, there are reports that supplemental phosphorus may have some ergogenic properties.\textsuperscript{84} As a result, supplemental phosphorus in the forms of high-energy bars and shakes, creatine monophosphate, and sodium phosphate may be consumed to enhance athletic performance and build muscle mass. If these supplements are consumed, the daily intake of phosphorus can easily exceed the UL for phosphorus (4000 mg [129.2 mmol]/d). For example, consumption of some high-energy bars and shakes, or creatine monophosphate supplements at the manufacturer’s recommended daily dose alone, can provide up to 3000 mg (96.9 mmol) phosphorus/d.\textsuperscript{82}

F. Status Assessment

Serum phosphorus concentration is generally used as an indicator of phosphorus status. Normal concentrations for adults are 2.5–4.5 mg/dL (0.87–1.45 mmol/L). However, the concentration of phosphorus in serum can be falsely elevated or depressed, which results in concentrations that appear normal when body stores are low, or concentrations that appear low when body stores are adequate. For example, consuming laxatives containing high amounts of sodium phosphate and phosphate loading for ergogenic purposes elevate serum
phosphate concentrations. Exercise, which results in the redistribution of phosphate from extracellular fluids to intracellular sites, decreases serum phosphate concentrations. Other factors that decrease serum phosphorus concentrations include respiratory alkalosis, various disease states, and changes in hormonal status. For example, individuals with insulin-dependent diabetes mellitus have fluctuating serum phosphorus concentrations because insulin decreases serum phosphorus concentrations. Thus, assessing phosphorus status by using serum phosphorus concentrations requires awareness of possible factors, especially the large number that cause hypophosphatemia without phosphorus deficiency, affecting values obtained.

.G. Toxicity

Excessive phosphorus intake from any source is expressed as hyperphosphatemia. Essentially all phosphorus toxicity effects are caused by elevated inorganic phosphate in the extracellular fluid.

A potential problem caused by excessive phosphorus intake is nonskeletal tissue calcification (ectopic or metastatic calcification), particularly of the kidney. This calcification occurs when calcium and phosphorus concentrations of the extracellular fluid exceed limits of calcium phosphate solubility. Metastatic calcification of the kidney is not known to occur through dietary means alone in persons with adequate renal function, but occurs often in patients with end-stage renal disease and is associated with increased all-cause and cardiovascular mortality and vascular calcification.

Hyperphosphatemia also can induce changes in the hormonal regulation of calcium metabolism and utilization. Hyperphosphatemia induced by phosphate loading results in decreased extracellular fluid-ionized calcium, and increased circulating parathyroid hormone and 1,25-dihydroxy vitamin D concentrations. These changes in calcium-regulating hormones can cause impairment of adaptive mechanisms for adequate calcium absorption and the removal of calcium and phosphorus from bone. If these changes continue for an extended period, bone loss may occur. Clinical evidence indicating parathyroid hormone-induced bone loss in individuals with healthy kidneys consuming high amounts of phosphorus is lacking. However, support is supplied by findings from epidemiological and animal studies. In a cross-sectional study with perimenopausal women, a significant positive relationship between bone mineral density and dietary calcium:phosphorus ratio was found. Experimental animals fed diets with a low dietary calcium:phosphorus ratio when calcium intake was adequate or deficient exhibited secondary hyperparathyroidism and bone resorption. In humans, however, phosphorus supplements have been found to decrease bone turnover markers. Thus, the long-term consequences of a low calcium:phosphorus ratio or a high phosphorus intake are unclear. Although calcium intakes are often low, the U.S. Food and Nutrition Board concluded that current phosphorus intakes experienced by the U.S. population are unlikely to adversely affect bone health.
H. **INTERACTIONS WITH OTHER NUTRIENTS AND DRUGS**

In the United States, phosphorus intake is consistently higher than calcium intake in the absence of calcium supplementation; as indicated in the toxicity section, the significance of this fact is controversial. The intestinal absorption of phosphate ions may be enhanced by 1,25-dihydroxy vitamin D when phosphorus intakes are low, but the evidence supporting this interaction comes only from limited animal studies. The magnesium interaction section above describes the interaction between phosphorus and magnesium, which results in high dietary phosphorus decreasing magnesium absorption, and magnesium supplementation ameliorating kidney calcification and bone loss induced by high dietary phosphorus in experimental animals.

Drugs that can cause hypophosphatemia include corticosteroids, diuretics, and phosphate-binding antacids. A deficient phosphorus intake while ingesting diuretics or a phosphate-binding drug could have pathological consequences. For example, dietary phosphorus deprivation while consuming the phosphate-binding aluminum hydroxide gel can result in severe and potentially fatal phosphorus deficiency.

I. **EFFECTS OF DEFICIENCY OR EXCESS ON PHYSICAL PERFORMANCE**

When inorganic phosphorus concentrations in extracellular fluid are low, cellular dysfunction occurs. The consequences of hypophosphatemia include anorexia, anemia, muscle weakness, bone pain, osteomalacia, increased susceptibility to infection, paresthesias, ataxia, confusion, and even death. The muscle weakness, which particularly involves proximal muscle when prolonged or severe, can result in muscle fiber degeneration. A deficient phosphorus intake also becomes potentially limiting for bone growth. However, the typical abundance of phosphorus in the diet minimizes the risk of hypophosphatemia and its consequences in healthy adults. Near total starvation is required to produce phosphorus deficiency by only dietary deprivation that manifests as hypophosphatemia.

As indicated in the toxicology section, excessive phosphorus intakes induce an increase in circulating parathyroid hormone that may impair the adaptive mechanism needed for adequate calcium absorption and optimal bone accretion. However, based on the determination by the U.S. Food and Nutrition Board, bone health is unlikely to be adversely affected by usual phosphorus intakes of middle-aged healthy, physically active adults in the United States.

One form of high phosphorus intake called phosphate loading has received much attention as an ergogenic aid. Phosphate loading is the consumption of 4 to 10 g/d of sodium or calcium phosphate supplements for 3 to 4 days prior to engaging in athletic events. Phosphate loading increases serum phosphate about 10% and the increase apparently can enhance the synthesis of ATP and creatine phosphate, which are depleted rapidly with high-intensity exercise, and the synthesis of 2,3-diphosphoglycerate to facilitate oxygen release to tissues. An extensive review found reports showing that phosphate loading also elevated intracellular phosphate concentrations that promoted glycolysis, attenuated anaerobic threshold, enhanced cardiovascular efficiency or performance, increased peripheral extraction of oxygen and maximal oxygen uptake, and improved endurance exercise performance or efficiency. The
review noted that not all studies found ergogenic benefits with phosphate loading, and suggested that the reason for this may have been differences in the type and amount of phosphate ingested, the experimental design and procedures used, and the type of exercise evaluated. It also was stated that single-dose acute (e.g., 1.5 g sodium phosphate 2.5 hours before athletic competition) and chronic calcium phosphate supplementation (e.g., 3.7 g sodium phosphate for 6 or more days) provided little ergogenic benefits, and phosphate loading was of some benefit mainly to athletes performing high-intensity or endurance exercise.

J. Dietary Recommendations

The U.S. Food and Nutrition Board established DRIs for phosphorus based on the amount needed to maintain serum inorganic phosphate at the bottom end of the normal range (2.5 to 2.8 mg/dL or 0.8 to 0.9 mmol/L). The RDA set for men and women aged 30–60 years was 700 mg/d. The UL was set at 4.0 g (130 mmol)/d based on the finding that the upper boundary of adult normal serum phosphate concentration (4.5 mg/dL or 1.45 mmol/L) is reached at a daily phosphorus intake of 3.5 g (113 mmol).

K. Future Research Needs

For the middle-aged physically active adult, how bone mineral mass is affected by different dietary phosphorus intakes needs further clarification. This clarification should include the determination of high phosphorus intakes on mineral elements needed for bone maintenance, including magnesium, iron, copper, and zinc. In addition, the effects of phosphate loading in moderately or untrained adults under varying exercise conditions should be determined.

L. Summary

Phosphorus is involved in virtually every aspect of metabolism. It is a component of the bone mineral hydroxyapatite; cell membranes in the form of phospholipids; molecules involved in energy storage and production; cellular messenger mechanisms; and virtually all enzyme reactions. Bone, which acts as reservoir for exchangeable phosphate ions, and the kidney, through regulating urinary excretion, loosely maintain serum phosphate concentrations between 2.5–4.5 mg/dL (0.87–1.4 mmol/L). Serum phosphorus concentration is generally used as an indicator of phosphorus status, but with caution because other factors can cause falsely high or low values. These factors include exercise, respiratory alkalosis, various disease states, and hormonal status. Low serum phosphorus (hypophosphatemia) may cause muscle weakness, and a low dietary phosphorus intake may potentially limit bone growth. However, the typical abundance of phosphorus in the diet (median intakes 300–800 mg [9.69–25.83 mmol]/d greater than RDA of 700 mg [22.6 mmol]/d) minimizes the risk of hypophosphatemia. Near total starvation is required to produce phosphorus deficiency by dietary deprivation alone. High serum phosphorus (hyperphosphatemia) can cause nonskeletal tissue calcification, but is not known to occur through dietary means in persons with adequate renal function. A high dietary phosphorus-calcium
ratio induces changes in hormonal regulation of calcium metabolism and utilization that may cause bone loss. However, clinical evidence for bone loss in individual with healthy kidneys is lacking. Thus, current phosphorus intakes by healthy physically active adults are unlikely to have adverse effects. Phosphate loading (4-10 g/d of sodium phosphate for 3-6 days prior athletic events) may have some ergogenic benefit for endurance or intensely exercising athletes, but is unlikely to benefit adults participating in moderate physical activity. Thus, phosphorus nutrition is not a concern for the physically active middle-aged adult with normal renal function, and the consumption of bars or supplements in an attempt to increase energy in the form of creatine phosphate or ATP is unlikely to enhance exercise performance.

V. CONCLUSIONS

Calcium, magnesium and phosphorus are essential elements critically important for the function of the musculoskeletal system, including the formation and transduction of energy and the maintenance of healthy bone. The major calcium concern for physically active healthy middle-aged adults is to consume enough calcium to prevent bone loss and fractures; an intake at the AI level (1.0-1.2 g [25-30 mmol/d]) should accomplish this. Phosphorus intakes in the United States indicate that neither phosphorus deficiency nor excess is a nutritional concern for healthy middle-aged adults. Based on dietary surveys, subclinical or chronic latent magnesium deficiency, which impairs energy utilization and exercise performance, may occur in significant numbers of middle-aged adults. Subclinical magnesium deficiency may also impair heart function, result in bone loss, and amplify oxidative stress induced by exercise. Balance studies indicate that intakes of more than 220 mg (9.05 mmol) magnesium/d are needed to prevent adverse effects of chronic latent magnesium deficiency. Consuming amounts greater than the RDA for magnesium (420 mg and 320 mg [17.28 and 13.16 mmol/d for men and women, respectively) is unlikely to provide any exercise performance benefits for healthy physically active middle-aged adults.

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Nutrition and Exercise Concerns of Middle Age

Edited by Judy A. Driskell
Nutrition and exercise concerns of middle age / editor, Judy A. Driskell.
p. cm.
Includes bibliographical references and index.
1. Middle-aged persons--Nutrition. 2. Exercise for middle-aged persons. I.
Driskell, Judy A. (Judy Anne)

Library of Congress Cataloging-in-Publication Data

TX361.M47N88 2008
613.2'0844--dc22 2009001261

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Section IV

Minerals