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Micronutrient Needs of the Elderly

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Abstract

Older adults are becoming a significant percentage of the world's population. A multitude of factors, from the normal aging process to the progression of chronic disease, influence the nutrition needs of this very diverse group of people. Appropriate micronutrient intake is of particular importance but is often suboptimal. Here we review the available data regarding micronutrient needs and the consequences of deficiencies in the ever growing aged population. (*Nutr Clin Pract.* 2014;29:435-444)

Keywords

geriatrics; nutritional status; aged; avitaminosis; deficiency; trace elements; vitamins

The structure of the population in the United States is positioned to change significantly over the next several decades. Specifically, those 65 years or older will experience a dramatic growth, which can be attributed to the first members of the baby boomer generation crossing into this age bracket in 2012. Between 2010 and 2050, the number of elderly Americans is projected to grow to 88.5 million, more than double its current population (Figure 1).¹ In addition, life expectancy has increased and all-cause death rates have decreased. This will result in a state where chronic disability of the aged accounts for nearly half of the health burden in the United States.²

Studies have clearly demonstrated that nutrition deficiencies play a very important role in the progression of chronic disease. Healthcare providers have an opportunity to intervene to provide nutrient-based recommendations. By proposing diet modifications, there is the potential to improve the overall health of the world as dietary risk factors account for one-tenth of the global disease burden.³ Also of importance is the contribution of nutrition to disease prevention, especially those associated with the aging process.

Geriatric nutrition is a field that presents unique challenges. The elderly represent a very diverse group of individuals. It is, however, a population particularly vulnerable to the development of nutrition deficiencies. Normal physiologic changes associated with the aging process occur and affect nutrient needs, and individuals experience these changes at different rates.⁴ Factors such as changes in the sense of taste, swallowing difficulties, medications, and appetite decreases are just some of the unique challenges that face our seniors in attainment of appropriate nutrition. Compound this by widespread financial concerns as well as social issues such as illiteracy or poverty; it is not surprising that suboptimal nutrition is common among older Americans. Taken together, these issues may lead to decreased food intake and, as a result, decreased nutrient intake. Specific dietary recommendations for the elderly

have been incorporated into the Recommended Dietary Allowances (RDA). The energy needs of the elderly are lower, but the requirement for most micronutrients is not. This underscores their need to make appropriate, nutrient-dense food choices.

Many important subgroups of the aged require special consideration. One such group is those that are hospitalized or institutionalized. The elderly have chronic health conditions and therefore find themselves in hospitals more often than their younger counterparts. Studies have demonstrated that undernutrition is particularly common in hospitalized people and has prognostic implications.⁵ Studies have shown that 66.2% of recently hospitalized older adults were classified as at risk for malnutrition or malnourished.⁶ This differs based on health status, functional status, and baseline nutrition status. This is particularly true for the very old. In this subgroup, 100% of the oldest old (>85 years) were assessed at high nutrition risk and 85.7% of those aged 75–84 years demonstrated high risk.⁷

Older women also warrant our particular attention. Due to their long life expectancy, nutrition requirements for females are of interest and therefore offer more opportunity to develop a nutrition-related ailment. Among the micronutrients, vitamin

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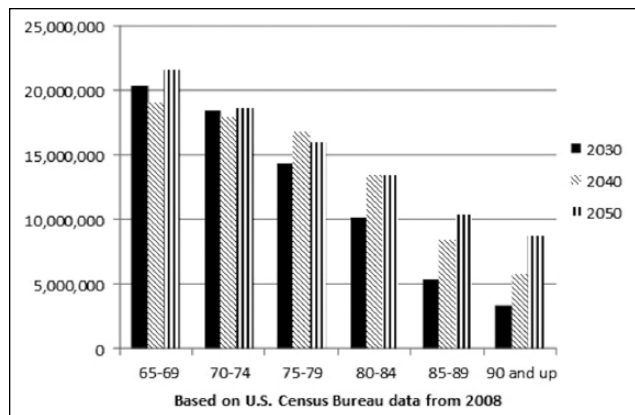


Figure 1. U.S. projected population by age, 2008.

B₁₂, vitamin A, vitamin C, vitamin D, vitamin E, iron, and zinc have been demonstrated to be of particular concern for elderly women.⁸ This risk is even higher for those women living in resource-poor settings. Specifically, these women may develop deficiencies that are below the Estimated Average Requirement (EAR).⁹ In addition, as women age, the experience of menopause affects their utilization of micronutrients. Vitamin C concentrations exhibit a gradual decrease across all stages of the menopausal transition, and this is correlated negatively to their body mass index.¹⁰ Body mass index should therefore be monitored in pre- and postmenopausal women.

A significant by-product of aging may include frailty and/or obesity. Frailty is a state of decreased physical functioning that dramatically alters the nutrition status of the aging population and for both sexes.¹¹ The amount of total body weight represented by fat increases by 50% as we age.¹² Adipose tissue has no significant metabolic activity, so this decreases a person's basal metabolic rate by approximately 2% per decade of life. The prevalence of frailty has been demonstrated to be highest among the obese (20.8%), followed by their overweight and normal-weight counterparts.¹¹ This underscores the need for targeted interventions, as specific micronutrient deficiencies can often occur in the absence of protein-calorie malnutrition.

Vitamin A

Vitamin A includes a family of compounds that are known as retinoids. Each member of this group demonstrates a biologic activity that is similar to retinol, and the 3 preformed compounds that exhibit metabolic activity are retinal, retinol, or retinoic acid. The carotenoids, most notably β -carotene, are structurally related to retinoids and are metabolized into compounds with vitamin A activity.¹³

Vitamin A is vital to vision, as rhodopsin, the eye pigment responsible for sensing low light, is composed of retinal and opsin, a protein. Tear production and debris clearance are also vitamin A-dependent processes. Vitamin A is also crucial in fighting infections. Retinoic acid acts as a promotor for T-killer

cells.¹⁴ Adequate stores of retinol are necessary for lymphocyte proliferation in the setting of antigen exposure, as rapid cellular upregulation and retinol consumption can occur.

Vitamin A deficiency (VAD) manifests most commonly as nighttime vision changes. In addition, chronic dry eye and chronic eye debris can be signs of VAD. Vitamin A toxicity, on the other hand, is most often evident by changes in skin and mucosa, including alopecia, dry lips and gums, cheilitis, and conjunctivitis. More serious complications include hepatotoxicity, bone loss, and pregnancy loss. VAD is often coincident with protein-calorie malnutrition for several reasons. Vitamin A absorption is dependent on dietary fat. Diarrheal illnesses can, therefore, result in both. In addition, vitamin A circulates bound to retinal binding protein, a compound synthesized by the liver and highly dependent on dietary intake. Because the reserves of vitamin A in the liver are adequate for months, it may take quite some time to develop the signs and symptoms of a deficient state.¹³

Dietary sources of vitamin A include animal and plant foods, such as green leafy vegetables, carrots, squash, and eggs and beef liver. Root vegetables and tubers are excellent sources of vitamin A. In the Western world, fortified foods account for a significant portion of vitamin A intake. Appropriate gastric acidification, an intact enterohepatic circulation, and fat absorption capacity are also necessary for maintaining vitamin A levels. The recommended intake of vitamin A is 700 mcg/d for females and 900 mcg/d for males.¹⁵ This reference amount is reported as a retinol activity equivalent (mcg/RAE) and can be converted to international units, which is reported on most food labeling.

Population studies have demonstrated that vitamin A is usually not deficient, with only 3.9% of subjects with inadequate serum retinol concentrations.¹⁶ Despite this fact, some elderly people are at risk of deficiency, which may put them at higher risk for various disease states. Due to its effect on inflammation, vitamin A may play a role in the progression of several disease states such as cancer and heart disease. Specifically, some studies have shown that the risk of deaths related to cardiovascular disease rose when plasma retinol levels fell to low levels.¹⁷ Studies also suggest that elderly patients who are in the highest quartile of dietary vitamin A intake had a 47% reduced risk of having moderate or greater hearing loss.¹⁸ Moreover, interest in studies related to serum retinol and the risk of prostate cancer is ongoing. There have been hypotheses that exposure to retinol may prevent prostate cancer, but studies to date have shown mixed results since there is equal evidence that under some conditions, it could promote cell growth and de-differentiation.¹⁹ However, monitoring specific antioxidant levels may be helpful in the early detection of prostate cancer.²⁰ Also of importance, vitamin A has been clinically shown to slow the progression of dementia and inhibit the formation of β -amyloid fibrils, which is a feature of Alzheimer disease.²¹ Thus, vitamin A may be a key therapeutic option in the prevention and therapy of this debilitating disease. Further research is needed to address this question.

Table 1. Dietary Recommendations for Various Micronutrients.

Micronutrient	Men		Women	
	Age 51–70 y	Age >70 y	Age 51–70 y	Age >70 y
Vitamin A, µg/d, RDA	900	900	700	700
Vitamin C, mg/d, RDA	90	90	75	75
Vitamin D, µg/d, AI	15	20	15	20
Vitamin E, mg/d, RDA	15	15	15	15
Vitamin K, µg/d, AI	120	120	90	90
Thiamin, mg/d, RDA	1.2	1.2	1.1	1.1
Riboflavin, mg/d, RDA	1.3	1.3	1.1	1.1
Niacin, mg/d, RDA	16	16	14	14
Vitamin B ₆ , mg/d, RDA	1.7	1.7	1.5	1.5
Folate, µg/d, RDA	400	400	400	400
Vitamin B ₁₂ , µg/d, RDA	2.4	2.4	2.4	2.4
Magnesium, mg/d, RDA	420	420	320	320
Zinc, mg/d, RDA	11	11	8	8
Iron, mg/d, RDA	8	8	8	8
Calcium, mg/d, AI	1200	1200	1200	1200

AI, Adequate Intake; RDA, Recommended Daily Allowances.

Vitamin B₁/Thiamine

Vitamin B₁, or thiamine, is a water-soluble compound with a half-life of approximately 18 days. It exists as both free thiamine and thiamine pyrophosphate. Absorption occurs primarily in the duodenum and proximal jejunum, and phosphorylation by the liver soon follows. Free thiamine circulates bound to serum albumin, but most thiamine circulates in red blood cells as thiamine pyrophosphate.

Thiamine's key functions lie in the energy production pathways. The production of nicotinamide adenine dinucleotide phosphate (NADPH) and pentoses via transketolase and the pentose phosphate pathway are dependent on the presence of thiamine. Oxidative decarboxylation of pyruvate, the link between glycolysis and the Krebs cycle, is also dependent on thiamine. Thiamine is required for the synthesis of acetylcholine and γ -aminobutyric acid (GABA) as well as the production of myelin for nerve conduction.¹³

Plants, bacteria, and fungi have the ability to produce B₁, but it is an essential nutrient for mammals. It is found in high concentrations in yeast, legumes, and whole grains. Other sources include eggs, cauliflower, and kale. Although whole grains are a better source of thiamine, cereals and processed grain-based foods are very often fortified with thiamine. The RDA for thiamin is 1.1–1.2 mg/d.

Thiamine deficiency is commonly referred to as beriberi. It is traditionally considered to have 2 forms, wet and dry beriberi. Dry beriberi is characterized by symmetric peripheral neuropathy and muscle tenderness. In wet beriberi, in addition to peripheral neuropathy, patients will exhibit confusion, ataxia, edema, tachycardia, and even coma. Wernicke encephalopathy, another manifestation of thiamine deficiency most

encountered in people who misuse alcohol, consists of gait disturbance, confusion, and paralysis of extraocular movements. This is the most common manifestation of B₁ deficiency in the United States. Thiamine deficiency and Wernicke encephalopathy are believed to be an underrecognized problem in the United States.

Vitamin B₁₂/Cobalamin

Vitamin B₁₂ is a group of closely related compounds that structurally consist of a corrin ring with the mineral cobalt incorporated into the center. The only reliable source of this vitamin is animal products, but microorganisms are the ultimate source of all naturally occurring B₁₂.¹³ The 2 active coenzyme forms of vitamin B₁₂ are deoxyadenosylcobalamin and methylcobalamin. These enzymes are required for the synthesis of succinyl coenzyme A, which is essential in the metabolism of lipid and carbohydrate as well as the synthesis of methionine.

Meat is the most important contributor to the intake of the B vitamins throughout European countries as well as in the United States.²² Other dietary sources of vitamin B₁₂ include fish, poultry, eggs, and fortified cereals. Strict vegetarians are at higher risk for the development of this deficiency compared with the general population. The recommended dietary intake of vitamin B₁₂ is shown in Table 1. Epidemiological studies have demonstrated a prevalence of cobalamin deficiency of around 20% in the general population in industrialized countries.²³ Most deficiencies arise from a loss of intestinal absorption of this vitamin due to disease states such as pernicious anemia, pancreatic insufficiency, atrophic gastritis, and ileal disease. In deficient states, patients have megaloblastic anemia and/or demyelinating neurologic disease. Altered mentation,

depression, and psychosis have been reported. Studies report significant associations with vitamin B₁₂ deficiency and risk of dementia or global cognitive decline.^{24,25} Metabolic evidence of B₁₂ deficiency has also been associated with diseases such as vascular dementia and multiple sclerosis but is felt to possibly be associated with a compromised vitamin B₁₂ metabolism in those disease states due to stress.²⁶ Because of the potential for irreversible neurological abnormalities associated with vitamin B₁₂ deficiency, it is imperative to identify the signs and symptoms that alert the clinician to the possibility of this deficiency. Newer research has suggested that using biomarkers of B₁₂ deficiency that include homocysteine or methylmalonic acid might be appropriate.^{27,28}

Folic Acid

Folic acid is a group of related pterin compounds with the metabolically active forms having reduced pteridine rings with up to 11 glutamic acids attached. More than 35 forms of the vitamin are found naturally, but the fully oxidized form (folic acid) is the pharmacologic form of the vitamin. All functions of folate relate to its ability to transfer 1 carbon group and is essential in the synthesis of nucleotides and in amino acid metabolism.¹³

The dietary intake recommendation for folate is 400 mcg/d for older men and women. Sources of folate in the diet include dark leafy vegetables, fruits, nuts, beans, peas, dairy products, eggs, seafood, poultry, and meat. Since 1998, the Food and Drug Administration (FDA) has required manufacturers to add folate to a variety of grain products such as bread, cereal, pasta, and rice. The classic deficiency syndrome of folate deficiency is megaloblastic anemia and diarrhea. In this disorder, the hematopoietic cells in the bone marrow become enlarged and exhibit immature nuclei as a result of the ineffective DNA synthesis. A study that assessed the inadequacy of folate intake across 8 European countries found that up to 25% of adult women were deficient in this nutrient, which is concerning.²⁹

The relationship between serum homocysteine levels and various disease states has garnered increased attention in the nutrition literature. Elevated serum homocysteine levels have recently been implicated as a risk factor for osteoporosis, and the use of folic acid and vitamin B₁₂ supplementation showed significant reductions in plasma homocysteine levels.³⁰ Moreover, higher serum folate levels are associated with lower homocysteine levels in older adults.³¹ Also concerning, increased homocysteine levels demonstrate a significantly negative association with physical function decline in older adults, as demonstrated by balance and gait assessments.³² Also, there may be a potential benefit of folic acid supplementation in stroke prevention as a result of its association with lowering of homocysteine levels and its effect in cardiovascular disease.³³

Vitamin C

Vitamin C, including ascorbic acid and dehydroascorbic acid, is a water-soluble vitamin that must be obtained from the diet. It serves as an antioxidant and reacts with superoxide and hydroxyl radicals in aqueous environments. In vivo, vitamin C has a multitude of roles, including biosynthesis of collagen, bile acids, carnitine, and the transmitter norepinephrine. The proper functioning of the hepatic mixed-function oxidase system is also dependent on the presence of vitamin C, as well as the intestinal absorption of iron.¹³

Deficiency of this vitamin in its least severe form causes fatigue, muscle pain, and increased susceptibility to infection. The classic deficiency syndrome is called scurvy and is characterized by widespread abnormalities in connective tissues, which leads to petechial hemorrhages, bleeding gums, anemia, joint effusions, inflamed gingivae, impaired wound healing, and even death.

Vitamin C can easily be obtained from consuming a diet rich in fruits and vegetables such as citrus fruits, broccoli, strawberries, green peppers, cantaloupe, and tomatoes. Also playing a role are foods and beverages that are fortified with vitamin C. The recommended amount of vitamin C for older adults ranges from 75–90 mg/d, with elderly women requiring the upper range.³⁴

Vitamin C deficiency in the United States was examined in the National Health and Nutrition Survey (NHANES). The overall prevalence of vitamin C deficiency was 7.1%, which has improved over the recent past.³⁵ However, in Europe, vitamin C had a higher risk of inadequate intake, especially in the elderly.³⁶ However unlikely, scurvy is still diagnosed in our hospitals today.^{37,38} Patients who smoke are at particular risk of a deficiency of this vitamin as smoking decreases tissue levels of vitamin C and therefore requires an increase in the intake of vitamin C by 35 mg/d.

Research abounds regarding the potential benefits of vitamin C to overall health. Studies have suggested that a beneficial relationship exists between plasma vitamin C concentration and physical performance.³⁹ Remaining active in older life improves the quality of life as we age, and a possible link with nutrient intake is promising. Also to be considered, factors influencing total and vitamin C have shown to have a negative association with homocysteine levels.⁴⁰

Vitamin D

Vitamin D represents a group of sterol compounds and refers to both ergocalciferol (vitamin D₂) and cholecalciferol (vitamin D₃). This vitamin is either consumed in the diet or formed in the skin after exposure to solar or artificial ultraviolet light. The vitamin then must undergo sequential hydroxylation in the liver and the kidney to become biologically active.¹³ The function of vitamin D in the body is to maintain appropriate

calcium and phosphorus levels to support cellular processes and has recently been shown to participate in the growth and differentiation of hematopoietic and immune cell lines.

Inadequacy of vitamin D levels is common worldwide, especially in the aged and institutionalized elderly.⁴¹ Deficiency of vitamin D has also been demonstrated in certain groups, including dark-skinned older adults as well as those who have limited sun exposure as the vitamin D precursor that is found in the skin decreases with advanced age. This finding has been incorporated into the adequate intake recommendations for the elderly and increases from 15–20 mcg/d after age 70 years.⁴²

Very few foods naturally contain vitamin D, and sunlight is the major source of this vitamin, but some dietary sources include fatty fish such as salmon, tuna, and mackerel. Fortified foods such as milk provide a significant amount of the vitamin D intake, and up to 43.7% of the dietary vitamin D intake is provided by fortified milk and milk products in some populations.⁴³

Inadequacy of vitamin D levels is common worldwide, especially in the aged and institutionalized elderly.⁴¹ In older adults, low levels of this vitamin reduce mobility, add to the risk for falls and fractures, and are associated with increased risk of death by cardiovascular means.⁴⁴ Vitamin D₃ has been even studied for a potential effect of decreasing mortality in elderly people and has shown some positive outcomes.⁴⁵ Normal levels of vitamin D have also been implicated in the favorable reduction of the prostaglandin cascade associated with cancer.⁴⁶ The greatest risk for cancer, infections, and metabolic diseases is associated with levels below 20 ng/mL.⁴⁷

A balanced calcium and vitamin D metabolism seems to be of paramount importance for stress fracture prevention in elderly patients. Vitamin D deficiency has been demonstrated to increase both the initiation and propagation of cracks in the bone by 22%–31%.⁴⁸ In addition, circulating levels of parathyroid hormone (PTH) and bone turnover decline in the presence of vitamin D.⁴⁹ In a group of individuals with stress fractures, studies support that as high as 83.8% of the group exhibits vitamin D insufficiency, and this number may be even more significant in those who are overweight.⁵⁰ However, meta-analysis of the benefit of widespread use of vitamin D for osteoporosis prevention illustrated that it is mainly beneficial in the femoral neck without significant effect at other sites.⁵¹ Also of note, in women with vitamin D deficiency, the risk of osteoporosis was even higher if there were elevated serum retinol levels.⁵² Evidence suggests that a simple and cost-effective strategy to reduce fractures in institutionalized individuals is to consider taking 800 IU of vitamin D together with 1000 mg of calcium, which represents a simple and cost-effective way that may reduce fractures by 30%.⁵³

Studies are ongoing regarding the role of vitamin D in the improvement of muscle strength and physical performance in the elderly, and it appears that vitamin D status is associated with functional limitations cross-sectionally and longitudinally in aged individuals.⁵⁴ Results are mixed, but data have shown that there is an inverse correlation between vitamin D and body

fat, which suggests that higher supplementation may be needed as an individual's weight increases. Also of importance, the test subjects who seem to demonstrate the most improvement in their physical performance testing with supplementation are those with the lowest baseline functioning.⁵⁵ Women are also more likely to demonstrate a strong inverse correlation between levels of vitamin D and loss of muscle mass and function compared with men.⁵⁶ However, any relationship between improvement in their performance and vitamin D levels has been obviated in the group of the oldest old, persons 80 years and older.⁵⁷

Lower serum vitamin D status is also associated with poorer cognitive function in the elderly.⁵⁸ In addition, lower vitamin D level has been associated with depression. Patients with severe vitamin D deficiency are twice as likely to have depression than those without a deficiency.⁵⁹ In the elderly, this problem cannot be overemphasized.

Vitamin E

Vitamin E includes 8 compounds that have been found to have biologic activity. Four tocopherols (α , β , γ , and δ) and 4 tocotrienols (α , β , γ , and δ). The most active form is α -tocopherol, which acts as a free radical scavenger in lipophilic environments, most notably in cell membranes and functions to maintain their integrity.¹³ With a deficiency of vitamin E, red blood cell fragility can occur and produces a hemolytic anemia. Degeneration of nerve cells can lead to peripheral neuropathies, destruction of posterior columns of the spinal cord, and ophthalmoplegia. This condition is irreversible if the deficiency is not recognized and corrected appropriately.

Many foods contain vitamin E, including nuts, seeds, green vegetables, and vegetable oils. Deficiency due to dietary intake is rare, and the RDA for vitamin E is 15 mg/d.³⁴ In developing countries, the prevalence of factors such as malaria and human immunodeficiency virus (HIV) infection predisposes these populations to develop vitamin E deficiency due to fat malabsorption.⁶⁰

There is a renewed interest in the antioxidant properties of vitamin E, and studies have been done to evaluate any association between this vitamin and various disease states. Higher intake of vitamin E at baseline was associated with a lower long-term risk of dementia in some studies but not in others.^{61,62} Also of note, elderly persons exhibited a slower rate of global cognitive decline if they belonged in the highest quartile of intake of vitamin C, vitamin E, and carotenes.⁶³ To date, clinical studies have not provided the appropriate answer to whether antioxidants can improve cognitive performance, despite our efforts. Also of concern, increasing the level of a vitamin could have detrimental effects that may not be well understood. According to some investigations, dietary supplementation with vitamin E may increase the risk of prostate cancer in healthy men.⁶⁴ Yet some evidence suggests that intake of vitamin E above recommended levels may enhance T-cell

function in aged animals and humans.⁶⁵ Interestingly, vitamin E-supplemented study participants exhibited a 6-month longer life expectancy in the older age group that was studied.⁶⁶ Other potential benefits of vitamin E to elderly populations have been studied, and there is accumulating evidence that supports the effects of the antioxidant vitamins such as vitamin E (as well as vitamin C) in eye health for slowing the progression of age-related cataracts and delaying macular degeneration.⁶⁷⁻⁶⁹

Vitamin K

Vitamin K is a trio of compounds with distinct sources but identical functions. K₁ (phyloquinone) is found in plants, whereas K₂ (menaquinone) is found in fish and meats. In addition, K₂ is synthesized by certain bacteria, some of which are found in the human gastrointestinal tract. K₂ or menadion is a synthetic form of the vitamin; this is readily converted to K₂ by gut flora. Vitamin K, like all fat-soluble vitamins, is absorbed in a fat-dependent process. Impairment of lipid intake, absorption, or processing can all result in vitamin K deficiency. Mineral oil can also interfere with uptake of vitamin K. The body does not have any storage capacity for vitamin K.

Posttranslational carboxylation of clotting factors is the primary job of vitamin K. In addition, there is some evidence to suggest that vitamin K plays a role in bone health.¹³ Vitamin D, vitamin K, and calcium may all have a codependent function in regulating the bone mineralization process. The clinical implications of this are unclear, but there is some evidence to suggest that vitamin K can reduce the rate of fracture in postmenopausal women.⁷⁰ Also of importance, evidence is accumulating that vitamin K may have a role in cognition, and studies demonstrate that higher serum levels are associated with better memory performances.⁷¹

The adequate intake for vitamin K ranges from 90–120 mcg/d, and therefore deficiency in vitamin K is rare, but it can result in varying degrees of bleeding if present. This is potentiated by the presence of warfarin. Bleeding complications can range from bruising to life-threatening hemorrhage. Parenteral administration of vitamin K fat emulsion can be used to treat bleeding associated with warfarin use and vitamin K deficiency.

Iron

Iron is needed for numerous essential functions, but most notably it is known for its role in oxygen transport to tissues through hemoglobin and myoglobin. Iron is also involved in immune, cognitive, and muscle function. The RDA for iron is 8 mg/d in both men and women older than 50 years.¹⁵ The requirement for iron in women decreases with age as postmenopausal women no longer require extra iron to account for iron lost during menses.

Dietary iron exists in 2 forms, heme and nonheme iron. Heme iron is more readily absorbed in the gut and is found in beef, fish, poultry, and pork. Nonheme iron from plant sources such as beans, dried fruit, enriched grains, and fortified cereals must be changed into a soluble form before it can be absorbed.

Atrophic gastritis, which affects about 20% of older adults, may decrease absorption of iron, especially in those who diets consist primarily of nonheme iron.⁷²

Iron deficiency is the most common nutrition cause of anemia, which is prevalent among hospitalized, institutionalized, or chronically ill patients. Deficiency can also lead to decreased immune function and increased susceptibility to infections, which is problematic in the elderly population as they already have a compromised immune system.^{73,74} Symptoms of iron deficiency include hypochromic anemia, fatigue, weakness, paleness, spoon-shaped nails, cheilosis, glossitis, headaches, and tachycardia. The recommended treatment for iron deficiency is supplementation of iron sulfate (325 mg 3 times per day).⁷⁵ Taking iron with a meal including vitamin C or a meat source has been noted to improve absorption.⁷⁶

Zinc

Zinc is essential in cellular metabolism, and more than 300 different enzymes are zinc dependent.⁷⁷ Zinc is also involved in cell structure and regulatory functions. Zinc plays an important role in the immune response, growth and development, neurological function, and reproduction. Requirements for zinc, 11 mg/d for men and 8 mg/d for women, do not change as adults get older. However, adults older than 70 years are more likely to have inadequate intakes of zinc according to the NHANES data.⁷⁸ Good sources of zinc include beef, poultry, pork, fish, legumes, nuts, fortified cereals, and dairy products.

Zinc deficiency can be especially deleterious in the elderly since is required for the synthesis of immune regulatory proteins and for maintaining normal immune function, and even a mild zinc deficiency can reduce immune function.⁷⁹ Furthermore, elderly patients with pressure ulcers are likely to have low zinc levels, and supplementation of zinc may improve wound healing, but the remains research unclear.^{80,81} Low zinc levels have recently been associated with Alzheimer disease, possible due to alterations in homeostasis that occur with aging.^{72,82} Zinc deficiency can also cause dermatitis, diarrhea, depression, decreased appetite, and impaired taste.

Diagnosing zinc deficiency can be difficult since serum zinc levels are not always reflective of dietary intake and can be affected by other factors, including inflammation. Low-dose supplementation of zinc (40–50 mg/d) is recommended for deficiency unless copper status is being regularly monitored.⁸³ Copper absorption can be inhibited by high doses of zinc, and extended supplementation can cause copper deficiency. Other side effects of excessive zinc include gastric irritation, reduced immune function, and decreased levels of high-density cholesterol.⁸⁴

Calcium

Calcium is vital in many physiological processes, including muscle contraction, hormone secretion, and nerve impulse transmission, and functions as an essential cofactor for multiple

enzymes.⁸⁵ However, it is calcium's role in bone health and prevention of osteoporosis that is of primary importance in the elderly, since the prevalence of osteoporosis and osteoporosis-related fractures continues to increase. More than 2 million osteoporosis-related fractures occurred in 2005, and this number is expected to increase to over 3 million by 2025, resulting in over \$25 billion in healthcare costs.⁸⁶

Requirements for calcium increase with age for many reasons, including decreased intestinal absorption of calcium and vitamin D, increased excretion of urinary calcium, decreased circulation of gonadal hormones, and decreased physical activity. In addition, bone mineral density (BMD) begins to decline after age 40 years at a rate of 0.5%–1% per year, especially in women after menopause.⁸⁵ To meet these increased needs, the RDA for men older than 70 years and women older than 50 years increases to 1200 mg/d compared with 1000 mg/d in younger adults.⁴² Unfortunately, NHANES data show that calcium intake is inadequate in 62% of men and 56% of women older than 70 years.⁸⁷

Milk, yogurt, and cheese provide the greatest source of dietary calcium, with 200–300 mg per serving. Smaller quantities of calcium can be found in dried beans, kale, spinach, and tofu. In addition, an increasing number of fruit juices, milk alternatives, and other foods are being fortified with calcium. Dietary counseling on calcium-rich foods has been shown to significantly increase calcium intake.⁸⁸

Calcium and vitamin D supplementation is often recommended as part of the baseline treatment for osteoporosis and prevention of fractures in addition to antiosteoporotic drugs.⁸⁹ However, the routine use of calcium supplementation has been put into question in light of recent research and meta-analysis showing calcium supplementation increased the risk of cardiovascular events.^{90,91} The American Society for Bone and Mineral Research continues to recommend combination supplementation of calcium and vitamin D but recognizes that increased dietary calcium is preferred over supplements. Although the controversy remains unsettled and continued research is needed, calcium supplementation should be considered in the institutionalized elderly and those who are unable able to achieve the recommended level of intake through dietary means alone.⁹²

Supplements and Fortification

Adding nutrients to foods by enrichment or fortification enhances the nutrient content of the food that we eat, considering that naturally nutrient-dense foods such as fruits, vegetables, whole grains, and lean proteins are sometimes not eaten in appropriate quantities. In the United States, substantial percentages of the population have intakes of vital nutrients below the EAR when only naturally occurring sources are considered, but those percentages fall dramatically with the addition of micronutrients from enriched and fortified foods.⁹³ Supplement use is also widespread, and nearly half of U.S. adults report taking at least 1 dietary supplement within the past month.⁹⁴ However,

some elderly populations still remain undernourished despite supplementation.⁹⁵ Dietary consumption was inadequate for vitamin D, vitamin E, calcium, and magnesium as part of a study conducted in seniors at senior centers. Supplementation in this group most improved intakes of vitamin B₆, vitamin D, vitamin E, folate, and calcium.⁹⁶ Micronutrient supplementation in those with mild to moderate cognitive decline raised blood levels of vitamin A, the B vitamins, folate, and the carotenoids and was associated with an improved self-perception of general health status in elderly test participants.⁹⁷ Our role is to guide the appropriate use of these supplements and to formulate a specific dietary plan with each individual.

Conclusion

The nutrition status of the elderly is complex and requires our individualized attention as adequate nutrition intake can affect health. As discussed, a considerable proportion of successfully aging elderly are deficient in several essential vitamins despite a total energy intake that could be expected to cover the recommendations for nutrients.⁹⁸ However, a lack of valid studies due to heterogeneity of the elderly population complicates making appropriate recommendations. The Dietary Reference Intakes (DRI) have incorporated age-specific recommendations in an attempt to address these concerns.

Consuming a varied diet that is rich in fruits and vegetables, lean meats, cheese, nuts, whole grains, and other dairy products should be appropriate for most older adults. Due to the nutrient richness of dairy products, these foods are essential components in the diet of elderly people.⁹⁹ These foods, specifically milk and cheese, can contribute substantially to the intake of calcium, selenium, zinc, and vitamin B₁₂.¹⁰⁰

As unique as each elderly person's individual health characteristics may be, their nutrition plan needs to be unique as well. Medications, social and financial issues, and ongoing disease processes can affect their nutrition status in a multitude of ways. Nutrition education cannot be underemphasized in this age group since it has shown benefit. After a short-term educational study to address the nutrition deficiencies in one group of the elderly, the test participants' intake of several nutrients was increased significantly, including 8 micronutrients.¹⁰¹ Therefore, healthcare professionals who are knowledgeable in the micronutrient needs of the elderly are essential for implementing interventions to optimize the nutrition status of older adults to promote health and wellness and to prevent and manage chronic disease.

Interest in investigating the role that nutrition plays in the progression and/or prevention of many disease states that are common in the elderly is ongoing. These are only a few examples of how future investigations may affect how we care for our patients, and we are interested in their results.

- Heart failure is a common condition among the elderly, and the incidence is expected to increase in the coming

years. Nutrition status in heart failure is important, and studies have suggested that patients with this disease have a lower status of a number of micronutrients, with the exception of copper, which tends to be elevated.¹⁰² A higher plasma level of vitamin C has been associated with a reduced risk of heart failure in older men and is of interest for future studies to evaluate this effect.¹⁰³

- Nutrition compounds have been studied regarding their role in the pathophysiological processes that are affected in Alzheimer disease. Of note, significantly lower plasma levels of folate, vitamin A, vitamin B₁₂, vitamin C, and vitamin E were demonstrated.¹⁰⁴ Given this potential role of nutrients in this process, the role of nutrition may offer unknown potential in the management of this disease.
- Hearing loss is a particularly important problem associated with aging and is known to affect a large number of elderly patients. In a sample of U.S. adults, studies have suggested that there is a dose-responsive relationship between improved hearing thresholds and intakes of antioxidant vitamins and magnesium.¹⁰⁵
- Pancreatic cancer is a devastating disease that has a significant impact on the lives of those diagnosed with it and their families. Oxidative stress has been shown to cause damage to the human cells, and much interest in this as a mechanism of carcinogenesis in pancreatic cancer has developed. Studies regarding the role of nutrition in the possible prevention of this dreaded disease have suggested that there is a significant inverse relationship between pancreatic cancer and nutrients such as magnesium, potassium, selenium, and other antioxidant vitamins.¹⁰⁶ Moreover, dietary selenium intake was inversely associated with the risk of pancreatic cancer, and the observed association was attenuated with supplementation with selenium.¹⁰⁷

As healthcare providers to the elderly, this review demonstrates that it is imperative that we are well educated in this area and strive to understand the special needs of this diverse and soon to be very populous group of people. It behooves us to educate ourselves and our patients about the tremendous role that appropriate nutrition plays in health and disease. Decisions that we make today have long-lasting effects on our patients' health, and we anxiously await further needed research into this important area of nutrition.

References

1. Vincent GK, Velkoff VA; U.S. Census Bureau. *The Next Four Decades: The Older Population in the United States: 2010 to 2050*. Washington, DC: U.S. Department of Commerce, Economics and Statistics Administration, U.S. Census Bureau; 2010.
2. The state of US health, 1990-2010: burden of diseases, injuries, and risk factors. *JAMA*. 2013;310(6):591-608.
3. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012;380(9859):2224-2260.
4. Chernoff R. Issues in geriatric nutrition. *Nutr Clin Pract*. 2009;24(2):176-178.
5. Marian M, Sacks G. Micronutrients and older adults. *Nutr Clin Pract*. 2009;24(2):179-195.
6. de Oliveira MR, Leandro-Merhi VA. Food intake and nutritional status of hospitalised older people. *Int J Older People Nurs*. 2011;6(3):196-200.
7. Esmayel EM, Eldarawy MM, Hassan MM, Hassanin HM, Reda Ashour WM, Mahmoud W. Nutritional and functional assessment of hospitalized elderly: impact of sociodemographic variables. *J Aging Res*. 2013;2013:101725.
8. Chernoff R. Micronutrient requirements in older women. *Am J Clin Nutr*. 2005;81(5):1240S-1245S.
9. Torheim LE, Ferguson EL, Penrose K, Arimond M. Women in resource-poor settings are at risk of inadequate intakes of multiple micronutrients. *J Nutr*. 2010;140(11):2051S-2058S.
10. Wiacek M, Zubrzycki IZ, Bojke O, Kim HJ. Menopause and age-driven changes in blood level of fat- and water-soluble vitamins. *Climacteric*. 2013;16(6):689-699.
11. Smit E, Winters-Stone KM, Loprinzi PD, Tang AM, Crespo CJ. Lower nutritional status and higher food insufficiency in frail older US adults. *Br J Nutr*. 2013;110(1):172-178.
12. Elmadfa I, Meyer AL. Body composition, changing physiological functions and nutrient requirements of the elderly. *Ann Nutr Metab*. 2008;52(suppl 1):2-5.
13. Gottschlich MM, DeLegge MH, Mattox T, Mueller C, Worthington P. *The A.S.P.E.N. Nutrition Support Core Curriculum: A Case-Based Approach*. Silver Spring, MD: American Society for Parenteral and Enteral Nutrition; 2007.
14. Wardwell L, Chapman-Novakofski K, Herrel S, Woods J. Nutrient intake and immune function of elderly subjects. *J Am Diet Assoc*. 2008;108(12):2005-2012.
15. Institute of Medicine, Panel on Micronutrients. *DRI: Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc: A Report of the Panel on Micronutrients . . . and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine*. Washington, DC: National Academies Press; 2001.
16. Carmem-Costa-do-Nascimento C, Cristhine-Pordeus-de-Lima R, Rios-Asciutti LS, et al. The importance of habitual vitamin A dietary intake on the serum retinol concentration in the elderly: a population-based study. *Rev Invest Clin*. 2011;63(5):450-460.
17. Brazionis L, Walker KZ, Itsiopoulos C, O'Dea K. Plasma retinol: a novel marker for cardiovascular disease mortality in Australian adults. *Nutr Metab Cardiovasc Dis*. 2012;22(10):914-920.
18. Gopinath B, Flood VM, McMahon CM, et al. Dietary antioxidant intake is associated with the prevalence but not incidence of age-related hearing loss. *J Nutr Health Aging*. 2011;15(10):896-900.
19. Mondul AM, Watters JL, Mannisto S, et al. Serum retinol and risk of prostate cancer. *Am J Epidemiol*. 2011;173(7):813-821.
20. Beydoun HA, Shroff MR, Mohan R, Beydoun MA. Associations of serum vitamin A and carotenoid levels with markers of prostate cancer detection among US men. *Cancer Causes Control*. 2011;22(11):1483-1495.
21. Ono K, Yamada M. Vitamin A and Alzheimer's disease. *Geriatr Gerontol Int*. 2012;12(2):180-188.
22. Olsen A, Halkjaer J, van Gils CH, et al. Dietary intake of the water-soluble vitamins B1, B2, B6, B12 and C in 10 countries in the European Prospective Investigation into Cancer and Nutrition. *Eur J Clin Nutr*. 2009;63(suppl 4):S122-S149.
23. Andres E, Loukili NH, Noel E, et al. Vitamin B12 (cobalamin) deficiency in elderly patients. *CMAJ*. 2004;171(3):251-259.
24. Doets EL, van Wijngaarden JP, Szczecinska A, et al. Vitamin B12 intake and status and cognitive function in elderly people [published online December 5, 2012]. *Epidemiol Rev*.

25. O'Leary F, Allman-Farinelli M, Samman S. Vitamin B(1)(2) status, cognitive decline and dementia: a systematic review of prospective cohort studies. *Br J Nutr*. 2012;108(11):1948-1961.
26. McCaddon A. Vitamin B12 in neurology and ageing; clinical and genetic aspects. *Biochimie*. 2013;95(5):1066-1076.
27. Oberlin BS, Tangney CC, Gustashaw KA, Rasmussen HE. Vitamin B12 deficiency in relation to functional disabilities. *Nutrients*. 2013;5(11):4462-4475.
28. Stabler SP. Clinical practice: vitamin B12 deficiency. *N Engl J Med*. 2013;368(2):149-160.
29. Tabacchi G, Wijnhoven TM, Branca F, et al. How is the adequacy of micronutrient intake assessed across Europe? A systematic literature review. *Br J Nutr*. 2009;101(suppl 2):S29-S36.
30. Keser I, Ilich JZ, Vrkic N, Giljevic Z, Colic Baric I. Folic acid and vitamin B(12) supplementation lowers plasma homocysteine but has no effect on serum bone turnover markers in elderly women: a randomized, double-blind, placebo-controlled trial. *Nutr Res*. 2013;33(3):211-219.
31. Solomon LR. Advanced age as a risk factor for folate-associated functional cobalamin deficiency. *J Am Geriatr Soc*. 2013;61(4):577-582.
32. Ng TP, Aung KC, Feng L, Scherer SC, Yap KB. Homocysteine, folate, vitamin B-12, and physical function in older adults: cross-sectional findings from the Singapore Longitudinal Ageing Study. *Am J Clin Nutr*. 2012;96(6):1362-1368.
33. Yang HT, Lee M, Hong KS, Ovbiagele B, Saver JL. Efficacy of folic acid supplementation in cardiovascular disease prevention: an updated meta-analysis of randomized controlled trials. *Eur J Intern Med*. 2012;23(8):745-754.
34. Institute of Medicine, Panel on Dietary Antioxidants and Related Compounds. *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids: A Report of the Panel on Dietary Antioxidants and Related Compounds, Subcommittees on Upper Reference Levels of Nutrients and of Interpretation and Use of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine*. Washington, DC: National Academies Press; 2000.
35. Schleicher RL, Carroll MD, Ford ES, Lacher DA. Serum vitamin C and the prevalence of vitamin C deficiency in the United States: 2003-2004 National Health and Nutrition Examination Survey (NHANES). *Am J Clin Nutr*. 2009;90(5):1252-1263.
36. Roman Vinas B, Ribas Barba L, Ngo J, et al. Projected prevalence of inadequate nutrient intakes in Europe. *Ann Nutr Metab*. 2011;59(2-4):84-95.
37. Swanson AM, Hughey LC. Acute inpatient presentation of scurvy. *Cutis*. 2010;86(4):205-207.
38. Raynaud-Simon A, Cohen-Bittan J, Gouronnec A, et al. Scurvy in hospitalized elderly patients. *J Nutr Health Aging*. 2010;14(6):407-410.
39. Saito K, Yokoyama T, Yoshida H, et al. A significant relationship between plasma vitamin C concentration and physical performance among Japanese elderly women. *J Gerontol A Biol Sci Med Sci*. 2012;67(3):295-301.
40. Breilmann J, Pons-Kuhnemann J, Brunner C, Richter M, Neuhauser-Berthold M. Effect of antioxidant vitamins on the plasma homocysteine level in a free-living elderly population. *Ann Nutr Metab*. 2010;57(3-4):177-182.
41. Hilger J, Friedel A, Herr R, et al. A systematic review of vitamin D status in populations worldwide. *Br J Nutr*. 2014;111(1):23-45.
42. Institute of Medicine, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. *Dietary Reference Intakes: For Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride*. Washington, DC: National Academies Press; 1997.
43. Moore CE, Radcliffe JD, Liu Y. Vitamin D intakes of adults differ by income, gender and race/ethnicity in the USA, 2007 to 2010. *Public Health Nutr*. 2014;17(4):756-763.
44. Boucher BJ. The problems of vitamin d insufficiency in older people. *Aging Dis*. 2012;3(4):313-329.
45. Bjelakovic G, Gluud LL, Nikolova D, et al. Vitamin D supplementation for prevention of mortality in adults. *Cochrane Database Syst Rev*. 2014;(1):CD007470.
46. Qin W, Smith C, Jensen M, Holick MF, Sauter ER. Vitamin D favorably alters the cancer promoting prostaglandin cascade. *Anticancer Res*. 2013;33(9):3861-3866.
47. Bouillon R, Van Schoor NM, Gielen E, et al. Optimal vitamin D status: a critical analysis on the basis of evidence-based medicine. *J Clin Endocrinol Metab*. 2013;98(8):E1283-E1304.
48. Busse B, Bale HA, Zimmermann EA, et al. Vitamin D deficiency induces early signs of aging in human bone, increasing the risk of fracture. *Sci Transl Med*. 2013;5(193):193ra188.
49. Aloia JF, Dhaliwal R, Shieh A, Mikhail M, Islam S, Yeh JK. Calcium and vitamin D supplementation in postmenopausal women. *J Clin Endocrinol Metab*. 2013;98(11):E1702-E1709.
50. Breer S, Krause M, Marshall RP, Oheim R, Amling M, Barvencik F. Stress fractures in elderly patients. *Int Orthop*. 2012;36(12):2581-2587.
51. Reid IR, Bolland MJ, Grey A. Effects of vitamin D supplements on bone mineral density: a systematic review and meta-analysis. *Lancet*. 2014;383(9912):146-155.
52. Mata-Granados JM, Cuenca-Acevedo JR, Luque de Castro MD, Holick MF, Quesada-Gomez JM. Vitamin D insufficiency together with high serum levels of vitamin A increases the risk for osteoporosis in postmenopausal women. *Arch Osteoporos*. 2013;8(1-2):124.
53. Gallagher JC. Vitamin D and aging. *Endocrinol Metab Clin North Am*. 2013;42(2):319-332.
54. Sohl E, van Schoor NM, de Jongh RT, Visser M, Deeg DJ, Lips P. Vitamin D status is associated with functional limitations and functional decline in older individuals. *J Clin Endocrinol Metab*. 2013;98(9):E1483-E1490.
55. Lagari V, Gomez-Marin O, Levis S. The role of vitamin D in improving physical performance in the elderly. *J Bone Miner Res*. 2013;28(10):2194-2201.
56. Park S, Ham JO, Lee BK. A positive association of vitamin D deficiency and sarcopenia in 50 year old women, but not men [published online October 6, 2013]. *Clin Nutr*.
57. Mathei C, Van Pottelbergh G, Vaes B, Adriaensen W, Gruson D, Degryse JM. No relation between vitamin D status and physical performance in the oldest old: results from the Belfrail study. *Age Ageing*. 2013;42(2):186-190.
58. Gschwind YJ, Bischoff-Ferrari HA, Bridenbaugh SA, Hardi I, Kressig RW. Association between serum vitamin D status and functional mobility in memory clinic patients aged 65 years and older. *Gerontology*. 2014;60(2):123-129.
59. Lapid MI, Cha SS, Takahashi PY. Vitamin D and depression in geriatric primary care patients. *Clin Interv Aging*. 2013;8:509-514.
60. Dror DK, Allen LH. Vitamin E deficiency in developing countries. *Food Nutr Bull*. 2011;32(2):124-143.
61. Devore EE, Grodstein F, van Rooij FJ, et al. Dietary antioxidants and long-term risk of dementia. *Arch Neurol*. 2010;67(7):819-825.
62. Alavi Naeini AM, Elmadfa I, Djazayeri A, et al. The effect of antioxidant vitamins E and C on cognitive performance of the elderly with mild cognitive impairment in Isfahan, Iran: a double-blind, randomized, placebo-controlled trial [published online December 11, 2013]. *Eur J Nutr*.
63. Rafnsson SB, Dilis V, Trichopoulou A. Antioxidant nutrients and age-related cognitive decline: a systematic review of population-based cohort studies. *Eur J Nutr*. 2013;52(6):1553-1567.
64. Klein EA, Thompson IM Jr, Tangen CM, et al. Vitamin E and the risk of prostate cancer: the Selenium and Vitamin E Cancer Prevention Trial (SELECT). *JAMA*. 2011;306(14):1549-1556.
65. Pae M, Meydani SN, Wu D. The role of nutrition in enhancing immunity in aging. *Aging Dis*. 2012;3(1):91-129.
66. Hemila H, Kaprio J. Vitamin E may affect the life expectancy of men, depending on dietary vitamin C intake and smoking. *Age Ageing*. 2011;40(2):215-220.
67. Mathew MC, Ervin AM, Tao J, Davis RM. Antioxidant vitamin supplementation for preventing and slowing the progression of age-related cataract. *Cochrane Database Syst Rev*. 2012;6:CD004567.
68. Evans JR, Lawrenson JG. Antioxidant vitamin and mineral supplements for slowing the progression of age-related macular degeneration. *Cochrane Database Syst Rev*. 2012;11:CD000254.

69. Traber MG, Manor D. Vitamin E. *Adv Nutr*. 2012;3(3):330-331.
70. Cheung AM, Tile L, Lee Y, et al. Vitamin K supplementation in postmenopausal women with osteopenia (ECKO trial): a randomized controlled trial. *PLoS Med*. 2008;5(10):e196.
71. Presse N, Belleville S, Gaudreau P, et al. Vitamin K status and cognitive function in healthy older adults. *Neurobiol Aging*. 2013;34(12):2777-2783.
72. Russell RM. Factors in aging that effect the bioavailability of nutrients. *J Nutr*. 2001;131(4)(suppl):1359S-1361S.
73. Ahluwalia N, Sun J, Krause D, Mastro A, Handte G. Immune function is impaired in iron-deficient, homebound, older women. *Am J Clin Nutr*. 2004;79(3):516-521.
74. Goodwin JS. Decreased immunity and increased morbidity in the elderly. *Nutr Rev*. 1995;53(4, pt 2):S41-S46.
75. Thomas DR. Anemia and quality of life: unrecognized and undertreated. *J Gerontol A Biol Sci Med Sci*. 2004;59(3):238-241.
76. Hallberg L, Hulthen L. Prediction of dietary iron absorption: an algorithm for calculating absorption and bioavailability of dietary iron. *Am J Clin Nutr*. 2000;71(5):1147-1160.
77. McCall KA, Huang C, Fierke CA. Function and mechanism of zinc metalloenzymes. *J Nutr*. 2000;130(5)(suppl):1437S-1446S.
78. Briefel RR, Bialostosky K, Kennedy-Stephenson J, McDowell MA, Ervin RB, Wright JD. Zinc intake of the U.S. population: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *J Nutr*. 2000;130(5)(suppl):1367S-1373S.
79. Bogden JD. Influence of zinc on immunity in the elderly. *J Nutr Health Aging*. 2004;8(1):48-54.
80. Gengenbacher M, Stahelin HB, Scholer A, Seiler WO. Low biochemical nutritional parameters in acutely ill hospitalized elderly patients with and without stage III to IV pressure ulcers. *Aging Clin Exp Res*. 2002;14(5):420-423.
81. Sernekos LA. Nutritional treatment of pressure ulcers: what is the evidence? *J Am Assoc Nurse Pract*. 2013;25(6):281-288.
82. Nuttall JR, Oteiza PI. Zinc and the aging brain. *Genes Nutr*. 2014;9(1):379.
83. Failla ML. Considerations for determining 'optimal nutrition' for copper, zinc, manganese and molybdenum. *Proc Nutr Soc*. 1999;58(2):497-505.
84. Fosmire GJ. Zinc toxicity. *Am J Clin Nutr*. 1990;51(2):225-227.
85. Shils ME, Shike M. *Modern Nutrition in Health and Disease*. 10th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
86. Ross AC, Taylor CL, Yantine AL, Del Valle HB. *Dietary reference intakes—calcium and vitamin D*. Institute of Medicine; 2011. <http://www.iom.edu/Activities/Nutrition/SummaryDRIs/DRI-Tables.aspx>.
87. Lichtenstein AH, Rasmussen H, Yu WW, Epstein SR, Russell RM. Modified MyPyramid for older adults. *J Nutr*. 2008;138(1):5-11.
88. Wong SY, Lau EM, Lau WW, Lynn HS. Is dietary counselling effective in increasing dietary calcium, protein and energy intake in patients with osteoporotic fractures? A randomized controlled clinical trial. *J Hum Nutr Diet*. 2004;17(4):359-364.
89. Rachner TD, Khosla S, Hofbauer LC. Osteoporosis: now and the future. *Lancet*. 2011;377(9773):1276-1287.
90. Bolland MJ, Grey A, Avenell A, Gamble GD, Reid IR. Calcium supplements with or without vitamin D and risk of cardiovascular events: reanalysis of the Women's Health Initiative limited access dataset and meta-analysis. *BMJ*. 2011;342:d2040.
91. Xiao Q, Murphy RA, Houston DK, Harris TB, Chow WH, Park Y. Dietary and supplemental calcium intake and cardiovascular disease mortality: the National Institutes of Health—AARP diet and health study. *JAMA Intern Med*. 2013;173(8):639-646.
92. Bauer DC. The calcium supplement controversy: now what? *J Bone Miner Res*. 2014;29(3):534-541.
93. Fulgoni VL III, Keast DR, Bailey RL, Dwyer J. Foods, fortificants, and supplements: where do Americans get their nutrients? *J Nutr*. 2011;141(10):1847-1854.
94. Kennedy ET, Luo H, Houser RF. Dietary supplement use pattern of U.S. adult population in the 2007-2008 National Health and Nutrition Examination Survey (NHANES). *Ecol Food Nutr*. 2013;52(1):76-84.
95. Anderson JJ, Suchindran CM, Roggenkamp KJ. Micronutrient intakes in two US populations of older adults: lipid research clinics program prevalence study findings. *J Nutr Health Aging*. 2009;13(7):595-600.
96. Weeden A, Remig V, Holcomb CA, Herald TJ, Baybutt RC. Vitamin and mineral supplements have a nutritionally significant impact on micronutrient intakes of older adults attending senior centers. *J Nutr Elderly*. 2010;29(2):241-254.
97. von Arnim CA, Dismar S, Ott-Renzer CS, Noeth N, Ludolph AC, Biesalski HK. Micronutrients supplementation and nutritional status in cognitively impaired elderly persons: a two-month open label pilot study. *Nutr J*. 2013;12(1):148.
98. Toffanello ED, Inelmen EM, Minicuci N, et al. Ten-year trends in vitamin intake in free-living healthy elderly people: the risk of subclinical malnutrition. *J Nutr Health Aging*. 2011;15(2):99-103.
99. van Staveren WA, de Groot LC. Evidence-based dietary guidance and the role of dairy products for appropriate nutrition in the elderly. *J Am Coll Nutr*. 2011;30(5)(suppl 1):429S-437S.
100. Vissers PA, Streppel MT, Feskens EJ, de Groot LC. The contribution of dairy products to micronutrient intake in the Netherlands. *J Am Coll Nutr*. 2011;30(5)(suppl 1):415S-421S.
101. Kim BH, Kim MJ, Lee Y. The effect of a nutritional education program on the nutritional status of elderly patients in a long-term care hospital in Jeollanamdo province: health behavior, dietary behavior, nutrition risk level and nutrient intake. *Nutr Res Pract*. 2012;6(1):35-44.
102. McKeag NA, McKinley MC, Woodside JV, Harbinson MT, McKeown PP. The role of micronutrients in heart failure. *J Acad Nutr Diet*. 2012;112(6):870-886.
103. Wannamethee SG, Bruckdorfer KR, Shaper AG, Papacosta O, Lennon L, Whincup PH. Plasma vitamin C, but not vitamin E, is associated with reduced risk of heart failure in older men. *Circ Heart Fail*. 2013;6(4):647-654.
104. Lopes da Silva S, Vellas B, Elemans S, et al. Plasma nutrient status of patients with Alzheimer's disease: systematic review and meta-analysis [published online October 18, 2013]. *Alzheimers Dement*.
105. Choi YH, Miller JM, Tucker KL, Hu H, Park SK. Antioxidant vitamins and magnesium and the risk of hearing loss in the US general population. *Am J Clin Nutr*. 2014;99(1):148-155.
106. Jansen RJ, Robinson DP, Stolzenberg-Solomon RZ, et al. Nutrients from fruit and vegetable consumption reduce the risk of pancreatic cancer. *J Gastrointest Cancer*. 2013;44(2):152-161.
107. Han X, Li J, Brasky TM, et al. Antioxidant intake and pancreatic cancer risk: the Vitamins and Lifestyle (VITAL) Study. *Cancer*. 2013;119(7):1314-1320.