

NIH Office of Dietary Supplements

Dietary Supplement Fact Sheet:

Vitamin D

[QuickFacts](#) / [Datos en español](#) / [Health Professional](#) / [Other Resources](#)

Introduction

Vitamin D is a fat-soluble vitamin that is naturally present in very few foods, added to others, and available as a dietary supplement. It is also produced endogenously when ultraviolet rays from sunlight strike the skin and trigger vitamin D synthesis. Vitamin D obtained from sun exposure, food, and supplements is biologically inert and must undergo two hydroxylations in the body for activation. The first occurs in the liver and converts vitamin D to 25-hydroxyvitamin D [25(OH)D], also known as calcidiol. The second occurs primarily in the kidney and forms the physiologically active 1,25-dihydroxyvitamin D [1,25(OH)₂D], also known as calcitriol [1].

Vitamin D promotes calcium absorption in the gut and maintains adequate serum calcium and phosphate concentrations to enable normal mineralization of bone and to prevent hypocalcemic tetany. It is also needed for bone growth and bone remodeling by osteoblasts and osteoclasts [1,2]. Without sufficient vitamin D, bones can become thin, brittle, or misshapen. Vitamin D sufficiency prevents rickets in children and osteomalacia in adults [1]. Together with calcium, vitamin D also helps protect older adults from osteoporosis.

Vitamin D has other roles in the body, including modulation of cell growth, neuromuscular and immune function, and reduction of inflammation [1,3,4]. Many genes encoding proteins that regulate cell proliferation, differentiation, and apoptosis are modulated in part by vitamin D [1]. Many cells have vitamin D receptors, and some convert 25(OH)D to 1,25(OH)₂D.

Serum concentration of 25(OH)D is the best indicator of vitamin D status. It reflects vitamin D produced cutaneously and that obtained from food and supplements [1] and has a fairly long circulating half-life of 15 days [5]. 25(OH)D functions as a biomarker of exposure, but it is not clear to what extent 25(OH)D levels also serve as a biomarker of effect (i.e., relating to health status or outcomes) [1]. Serum 25(OH)D levels do not indicate the amount of vitamin D stored in body tissues.

In contrast to 25(OH)D, circulating 1,25(OH)₂D is generally not a good indicator of vitamin D status because it has a short half-life of 15 hours and serum concentrations are closely regulated by parathyroid hormone, calcium, and phosphate [5]. Levels of 1,25(OH)₂D do not typically decrease until vitamin D deficiency is severe [2,6].

There is considerable discussion of the serum concentrations of 25(OH)D associated with deficiency (e.g., rickets), adequacy for bone health, and optimal overall health, and cut points have not been developed by a scientific consensus process. Based on its review of data of vitamin D needs, a committee of the Institute of Medicine concluded that persons are at risk of vitamin D deficiency at serum 25(OH)D concentrations <30 nmol/L (<12 ng/mL). Some are potentially at risk for inadequacy at levels ranging from 30–50 nmol/L (12–20 ng/mL). Practically all people are sufficient at levels ≥50 nmol/L (≥20 ng/mL); the committee stated that 50 nmol/L is the serum 25(OH)D level that covers the needs of 97.5% of the population. Serum concentrations >125 nmol/L (>50 ng/mL) are associated with potential adverse effects [1] (Table 1).

Table 1: Serum 25-Hydroxyvitamin D [25(OH)D] Concentrations and Health* [1]

nmol/L**	ng/mL*	Health status
<30	<12	Associated with vitamin D deficiency, leading to rickets in infants and children and osteomalacia in adults
30–50	12–20	Generally considered inadequate for bone and overall health in healthy individuals
≥50	≥20	Generally considered adequate for bone and overall health in healthy individuals
>125	>50	Emerging evidence links potential adverse effects to such high levels, particularly >150 nmol/L (>60 ng/mL)

* Serum concentrations of 25(OH)D are reported in both nanomoles per liter (nmol/L) and nanograms per milliliter (ng/mL).

** 1 nmol/L = 0.4 ng/mL

An additional complication in assessing vitamin D status is in the actual measurement of serum 25(OH)D concentrations. Considerable variability exists among the various assays available (the two most common methods being antibody based and liquid chromatography based) and among laboratories that conduct the analyses [1,7,8]. This means that compared with the actual concentration of 25(OH)D in a sample of blood serum, a falsely low or falsely high value may be obtained depending on the assay or laboratory used [9]. A standard reference material for 25(OH)D became available in July 2009 that permits standardization of values across laboratories and may improve method-related variability [1,10].

Reference Intakes

Intake reference values for vitamin D and other nutrients are provided in the Dietary Reference Intakes (DRIs) developed by the Food and Nutrition Board (FNB) at the Institute of Medicine of The National Academies (formerly National Academy of Sciences) [1]. DRI is the general term for a set of reference values used to plan and assess nutrient intakes of healthy people. These values, which vary by age and gender, include:

- Recommended Dietary Allowance (RDA): average daily level of intake sufficient to meet the nutrient requirements of nearly all

Reviewed: June 24, 2011

(97%–98%) healthy people.

- Adequate Intake (AI): established when evidence is insufficient to develop an RDA and is set at a level assumed to ensure nutritional adequacy.
- Tolerable Upper Intake Level (UL): maximum daily intake unlikely to cause adverse health effects [1].

The FNB established an RDA for vitamin D representing a daily intake that is sufficient to maintain bone health and normal calcium metabolism in healthy people. RDAs for vitamin D are listed in both International Units (IUs) and micrograms (mcg); the biological activity of 40 IU is equal to 1 mcg (Table 2). Even though sunlight may be a major source of vitamin D for some, the vitamin D RDAs are set on the basis of minimal sun exposure [1].

Table 2: Recommended Dietary Allowances (RDAs) for Vitamin D [1]

Age	Male	Female	Pregnancy	Lactation
0–12 months*	400 IU (10 mcg)	400 IU (10 mcg)		
1–13 years	600 IU (15 mcg)	600 IU (15 mcg)		
14–18 years	600 IU (15 mcg)	600 IU (15 mcg)	600 IU (15 mcg)	600 IU (15 mcg)
19–50 years	600 IU (15 mcg)	600 IU (15 mcg)	600 IU (15 mcg)	600 IU (15 mcg)
51–70 years	600 IU (15 mcg)	600 IU (15 mcg)		
>70 years	800 IU (20 mcg)	800 IU (20 mcg)		

* Adequate Intake (AI)

Sources of Vitamin D

Food

Very few foods in nature contain vitamin D. The flesh of fatty fish (such as salmon, tuna, and mackerel) and fish liver oils are among the best sources [1,11]. Small amounts of vitamin D are found in beef liver, cheese, and egg yolks. Vitamin D in these foods is primarily in the form of vitamin D₃ and its metabolite 25(OH)D₃ [12]. Some mushrooms provide vitamin D₂ in variable amounts [13,14]. Mushrooms with enhanced levels of vitamin D₂ from being exposed to ultraviolet light under controlled conditions are also available.

Fortified foods provide most of the vitamin D in the American diet [1,14]. For example, almost all of the U.S. milk supply is voluntarily fortified with 100 IU/cup [1]. (In Canada, milk is fortified by law with 35–40 IU/100 mL, as is margarine at ≥530 IU/100 g.) In the 1930s, a milk fortification program was implemented in the United States to combat rickets, then a major public health problem [1]. Other dairy products made from milk, such as cheese and ice cream, are generally not fortified. Ready-to-eat breakfast cereals often contain added vitamin D, as do some brands of orange juice, yogurt, margarine and other food products.

Both the United States and Canada mandate the fortification of infant formula with vitamin D: 40–100 IU/100 kcal in the United States and 40–80 IU/100 kcal in Canada [1].

Several food sources of vitamin D are listed in Table 3.

Table 3: Selected Food Sources of Vitamin D [11]

Food	IUs per serving*	Percent DV**
Cod liver oil, 1 tablespoon	1,360	340
Salmon (sockeye), cooked, 3 ounces	447	112
Mackerel, cooked, 3 ounces	388	97
Tuna fish, canned in water, drained, 3 ounces	154	39
Orange juice fortified with vitamin D, 1 cup (check product labels, as amount of added vitamin D varies)	137	34
Milk, nonfat, reduced fat, and whole, vitamin D-fortified, 1 cup	115–124	29–31
Yogurt, fortified with 20% of the DV for vitamin D, 6 ounces (more heavily fortified yogurts provide more of the DV)	88	22
Margarine, fortified, 1 tablespoon	60	15
Liver, beef, cooked, 3.5 ounces	49	12
Sardines, canned in oil, drained, 2 sardines	46	12
Egg, 1 large (vitamin D is found in yolk)	41	10
Ready-to-eat cereal, fortified with 10% of the DV for vitamin D, 0.75–1 cup (more heavily fortified cereals might provide more of the DV)	40	10
Cheese, Swiss, 1 ounce	6	2

Food	IUs per serving*	Percent DV**
------	------------------	--------------

* IUs = International Units.

** DV = Daily Value. DVs were developed by the U.S. Food and Drug Administration to help consumers compare the nutrient contents among products within the context of a total daily diet. The DV for vitamin D is currently set at 400 IU for adults and children age 4 and older. Food labels, however, are not required to list vitamin D content unless a food has been fortified with this nutrient. Foods providing 20% or more of the DV are considered to be high sources of a nutrient, but foods providing lower percentages of the DV also contribute to a healthful diet.

The U.S. Department of Agriculture's [Nutrient Database Web site](#) lists the nutrient content of many foods. It also provides a [comprehensive list of foods containing vitamin D](#). A growing number of foods are being analyzed for vitamin D content. Simpler and faster methods to measure vitamin D in foods are needed, as are food standard reference materials with certified values for vitamin D to ensure accurate measurements [15].

Sun exposure

Most people meet at least some of their vitamin D needs through exposure to sunlight [1,2]. Ultraviolet (UV) B radiation with a wavelength of 290–320 nanometers penetrates uncovered skin and converts cutaneous 7-dehydrocholesterol to previtamin D₃, which in turn becomes vitamin D₃ [1]. Season, time of day, length of day, cloud cover, smog, skin melanin content, and sunscreen are among the factors that affect UV radiation exposure and vitamin D synthesis [1]. Perhaps surprisingly, geographic latitude does not consistently predict average serum 25(OH)D levels in a population. Ample opportunities exist to form vitamin D (and store it in the liver and fat) from exposure to sunlight during the spring, summer, and fall months even in the far north latitudes [1].

Complete cloud cover reduces UV energy by 50%; shade (including that produced by severe pollution) reduces it by 60% [16]. UVB radiation does not penetrate glass, so exposure to sunshine indoors through a window does not produce vitamin D [17]. Sunscreens with a sun protection factor (SPF) of 8 or more appear to block vitamin D-producing UV rays, although in practice people generally do not apply sufficient amounts, cover all sun-exposed skin, or reapply sunscreen regularly [1,18]. Therefore, skin likely synthesizes some vitamin D even when it is protected by sunscreen as typically applied.

The factors that affect UV radiation exposure and research to date on the amount of sun exposure needed to maintain adequate vitamin D levels make it difficult to provide general guidelines. It has been suggested by some vitamin D researchers, for example, that approximately 5–30 minutes of sun exposure between 10 AM and 3 PM at least twice a week to the face, arms, legs, or back without sunscreen usually lead to sufficient vitamin D synthesis and that the moderate use of commercial tanning beds that emit 2%–6% UVB radiation is also effective [6,19]. Individuals with limited sun exposure need to include good sources of vitamin D in their diet or take a supplement to achieve recommended levels of intake.

Despite the importance of the sun for vitamin D synthesis, it is prudent to limit exposure of skin to sunlight [18] and UV radiation from tanning beds [20]. UV radiation is a carcinogen responsible for most of the estimated 1.5 million skin cancers and the 8,000 deaths due to metastatic melanoma that occur annually in the United States [18]. Lifetime cumulative UV damage to skin is also largely responsible for some age-associated dryness and other cosmetic changes. The American Academy of Dermatology advises that photoprotective measures be taken, including the use of sunscreen, whenever one is exposed to the sun [21]. Assessment of vitamin D requirements cannot address the level of sun exposure because of these public health concerns about skin cancer, and there are no studies to determine whether UVB-induced synthesis of vitamin D can occur without increased risk of skin cancer [1].

Dietary supplements

In supplements and fortified foods, vitamin D is available in two forms, D₂ (ergocalciferol) and D₃ (cholecalciferol) that differ chemically only in their side-chain structure. Vitamin D₂ is manufactured by the UV irradiation of ergosterol in yeast, and vitamin D₃ is manufactured by the irradiation of 7-dehydrocholesterol from lanolin and the chemical conversion of cholesterol [6]. The two forms have traditionally been regarded as equivalent based on their ability to cure rickets and, indeed, most steps involved in the metabolism and actions of vitamin D₂ and vitamin D₃ are identical. Both forms (as well as vitamin D in foods and from cutaneous synthesis) effectively raise serum 25(OH)D levels [2]. Firm conclusions about any different effects of these two forms of vitamin D cannot be drawn. However, it appears that at nutritional doses vitamins D₂ and D₃ are equivalent, but at high doses vitamin D₂ is less potent.

The American Academy of Pediatrics (AAP) recommends that exclusively and partially breastfed infants receive supplements of 400 IU/day of vitamin D shortly after birth and continue to receive these supplements until they are weaned and consume ≥1,000 mL/day of vitamin D-fortified formula or whole milk [22]. Similarly, all non-breastfed infants ingesting <1,000 mL/day of vitamin D-fortified formula or milk should receive a vitamin D supplement of 400 IU/day [22]. AAP also recommends that older children and adolescents who do not obtain 400 IU/day through vitamin D-fortified milk and foods should take a 400 IU vitamin D supplement daily. However, this latter recommendation (issued November 2008) needs to be reevaluated in light of the Food and Nutrition Board's vitamin D RDA of 600 IU/day for children and adolescents (issued November 2010 and which previously was an AI of 200 IU/day).

Vitamin D Intakes and Status

The National Health and Nutrition Examination Survey (NHANES), 2005–2006, estimated vitamin D intakes from both food and dietary supplements [4,23]. Average intake levels for males from foods alone ranged from 204 to 288 IU/day depending on life stage group; for females the range was 144 to 276 IU/day. When use of dietary supplements was considered, these mean values were substantially increased (37% of the

U.S. population used a dietary supplement containing vitamin D.) The most marked increase was among older women. For women aged 51–70 years, mean intake of vitamin D from foods alone was 156 IU/day, but 404 IU/day with supplements. For women >70 years, the corresponding figures were 180 IU/day to 400 IU/day [1].

Comparing vitamin D intake estimates from foods and dietary supplements to serum 25(OH)D concentrations is problematic. One reason is that comparisons can only be made on group means rather than on data linked to individuals. Another is the fact that sun exposure affects vitamin D status; serum 25(OH)D levels are generally higher than would be predicted on the basis of vitamin D intakes alone [1]. The NHANES 2005–2006 survey found mean 25(OH)D levels exceeding 56 nmol/L (22.4 ng/mL) for all age-gender groups in the U.S. population. (The highest mean was 71.4 nmol/L [28.6 ng/mL] for girls aged 1–3 years, and the lowest mean was 56.5 nmol/L [22.6 ng/mL] for women aged 71 and older. Generally, younger people had higher levels than older people, and males had slightly higher levels than females.) 25(OH)D levels of approximately 50 nmol/L (20 ng/mL) are consistent with an intake of vitamin D from foods and dietary supplements equivalent to the RDA [1].

Over the past 20 years, mean serum 25(OH)D concentrations in the United States have slightly declined among males but not females. This decline is likely due to simultaneous increases in body weight, reduced milk intake, and greater use of sun protection when outside [24].

Vitamin D Deficiency

Nutrient deficiencies are usually the result of dietary inadequacy, impaired absorption and use, increased requirement, or increased excretion. A vitamin D deficiency can occur when usual intake is lower than recommended levels over time, exposure to sunlight is limited, the kidneys cannot convert 25(OH)D to its active form, or absorption of vitamin D from the digestive tract is inadequate. Vitamin D-deficient diets are associated with milk allergy, lactose intolerance, ovo-vegetarianism, and veganism [1].

Rickets and osteomalacia are the classical vitamin D deficiency diseases. In children, vitamin D deficiency causes rickets, a disease characterized by a failure of bone tissue to properly mineralize, resulting in soft bones and skeletal deformities [16]. Rickets was first described in the mid-17th century by British researchers [16,25]. In the late 19th and early 20th centuries, German physicians noted that consuming 1–3 teaspoons/day of cod liver oil could reverse rickets [25]. The fortification of milk with vitamin D beginning in the 1930s has made rickets a rare disease in the United States, although it is still reported periodically, particularly among African American infants and children [3,16,21].

Prolonged exclusive breastfeeding without the AAP-recommended vitamin D supplementation is a significant cause of rickets, particularly in dark-skinned infants breastfed by mothers who are not vitamin D replete [26]. Additional causes of rickets include extensive use of sunscreens and placement of children in daycare programs, where they often have less outdoor activity and sun exposure [16,25]. Rickets is also more prevalent among immigrants from Asia, Africa, and the Middle East, possibly because of genetic differences in vitamin D metabolism and behavioral differences that lead to less sun exposure.

In adults, vitamin D deficiency can lead to osteomalacia, resulting in weak bones [1,5]. Symptoms of bone pain and muscle weakness can indicate inadequate vitamin D levels, but such symptoms can be subtle and go undetected in the initial stages.

Groups at Risk of Vitamin D Inadequacy

Obtaining sufficient vitamin D from natural food sources alone is difficult. For many people, consuming vitamin D-fortified foods and, arguably, being exposed to some sunlight are essential for maintaining a healthy vitamin D status. In some groups, dietary supplements might be required to meet the daily need for vitamin D.

Breastfed infants

Vitamin D requirements cannot ordinarily be met by human milk alone [1,27], which provides <25 IU/L to 78 IU/L [22]. (The vitamin D content of human milk is related to the mother's vitamin D status, so mothers who supplement with high doses of vitamin D may have correspondingly high levels of this nutrient in their milk [22].) A review of reports of nutritional rickets found that a majority of cases occurred among young, breastfed African Americans [28]. A survey of Canadian pediatricians found the incidence of rickets in their patients to be 2.9 per 100,000; almost all those with rickets had been breast fed [29]. While the sun is a potential source of vitamin D, the AAP advises keeping infants out of direct sunlight and having them wear protective clothing and sunscreen [30]. As noted earlier, the AAP recommends that exclusively and partially breastfed infants be supplemented with 400 IU of vitamin D per day [22], the RDA for this nutrient during infancy.

Older adults

Older adults are at increased risk of developing vitamin D insufficiency in part because, as they age, skin cannot synthesize vitamin D as efficiently, they are likely to spend more time indoors, and they may have inadequate intakes of the vitamin [1]. As many as half of older adults in the United States with hip fractures could have serum 25(OH)D levels <30 nmol/L (<12 ng/mL) [2].

People with limited sun exposure

Homebound individuals, women who wear long robes and head coverings for religious reasons, and people with occupations that limit sun exposure are unlikely to obtain adequate vitamin D from sunlight [31,32]. Because the extent and frequency of use of sunscreen are unknown, the significance of the role that sunscreen may play in reducing vitamin D synthesis is unclear [1]. Ingesting RDA levels of vitamin D from foods and/or supplements will provide these individuals with adequate amounts of this nutrient.

People with dark skin

Greater amounts of the pigment melanin in the epidermal layer result in darker skin and reduce the skin's ability to produce vitamin D from sunlight [1]. Various reports consistently show lower serum 25(OH)D levels in persons identified as black compared with those identified as white. It is not clear that lower levels of 25(OH)D for persons with dark skin have significant health consequences. Those of African American

ancestry, for example, have reduced rates of fracture and osteoporosis compared with Caucasians (see section below on osteoporosis). Ingesting RDA levels of vitamin D from foods and/or supplements will provide these individuals with adequate amounts of this nutrient.

People with fat malabsorption

As a fat-soluble vitamin, vitamin D requires some dietary fat in the gut for absorption. Individuals who have a reduced ability to absorb dietary fat might require vitamin D supplements [33]. Fat malabsorption is associated with a variety of medical conditions including some forms of liver disease, cystic fibrosis, and Crohn's disease [3].

People who are obese or who have undergone gastric bypass surgery

A body mass index ≥ 30 is associated with lower serum 25(OH)D levels compared with non-obese individuals; people who are obese may need larger than usual intakes of vitamin D to achieve 25(OH)D levels comparable to those of normal weight [1]. Obesity does not affect skin's capacity to synthesize vitamin D, but greater amounts of subcutaneous fat sequester more of the vitamin and alter its release into the circulation. Obese individuals who have undergone gastric bypass surgery may become vitamin D deficient over time without a sufficient intake of this nutrient from food or supplements, since part of the upper small intestine where vitamin D is absorbed is bypassed and vitamin D mobilized into the serum from fat stores may not compensate over time [34,35].

Vitamin D and Health

Optimal serum concentrations of 25(OH)D for bone and general health have not been established; they are likely to vary at each stage of life, depending on the physiological measures selected [1,2,6]. Also, as stated earlier, while serum 25(OH)D functions as a biomarker of exposure to vitamin D (from sun, food, and dietary supplements), the extent to which such levels serve as a biomarker of effect (i.e., health outcomes) is not clearly established [1].

Furthermore, while serum 25(OH)D levels increase in response to increased vitamin D intake, the relationship is non-linear for reasons that are not entirely clear [1]. The increase varies, for example, by baseline serum levels and duration of supplementation. Increasing serum 25(OH)D to >50 nmol/L requires more vitamin D than increasing levels from a baseline <50 nmol/L. There is a steeper rise in serum 25(OH)D when the dose of vitamin D is $<1,000$ IU/day; a lower, more flattened response is seen at higher daily doses. When the dose is $\geq 1,000$ IU/day, the rise in serum 25(OH)D is approximately 1 nmol/L for each 40 IU of intake. In studies with a dose ≤ 600 IU/day, the rise in serum 25(OH)D was approximately 2.3 nmol/L for each 40 IU of vitamin D consumed [1].

In March 2007, a group of vitamin D and nutrition researchers published a controversial and provocative editorial contending that the desirable concentration of 25(OH)D was ≥ 75 nmol/L (≥ 30 ng/ml) [36]. They noted that approximately 1,700 IU/day of vitamin D are needed to raise serum 25(OH)D concentrations from 50 to 80 nmol/L (20–32 ng/mL).

However, the FNB committee that established DRIs for vitamin D extensively reviewed a long list of potential health relationships on which recommendations for vitamin D intake might be based [1]. These health relationships included resistance to chronic diseases (such as cancer and cardiovascular diseases), physiological parameters (such as immune response or levels of parathyroid hormone), and functional measures (such as skeletal health and physical performance and falls). With the exception of measures related to bone health, the health relationships examined were either not supported by adequate evidence to establish cause and effect, or the conflicting nature of the available evidence could not be used to link health benefits to particular levels of intake of vitamin D or serum measures of 25(OH)D with any level of confidence.

Osteoporosis

More than 40 million adults in the United States have or are at risk of developing osteoporosis, a disease characterized by low bone mass and structural deterioration of bone tissue that increases bone fragility and significantly increases the risk of bone fractures [37]. Osteoporosis is most often associated with inadequate calcium intakes, but insufficient vitamin D contributes to osteoporosis by reducing calcium absorption [38]. Although rickets and osteomalacia are extreme examples of the effects of vitamin D deficiency, osteoporosis is an example of a long-term effect of calcium and vitamin D insufficiency. Adequate storage levels of vitamin D maintain bone strength and might help prevent osteoporosis in older adults, non-ambulatory individuals who have difficulty exercising, postmenopausal women, and individuals on chronic steroid therapy [39].

Normal bone is constantly being remodeled. During menopause, the balance between these processes changes, resulting in more bone being resorbed than rebuilt. Hormone therapy with estrogen and progesterone might be able to delay the onset of osteoporosis. However, some medical groups and professional societies recommend that postmenopausal women consider using other agents to slow or stop bone resorption because of the potential adverse health effects of hormone therapy [40,41,42].

Most supplementation trials of the effects of vitamin D on bone health also include calcium, so it is difficult to isolate the effects of each nutrient. Among postmenopausal women and older men, supplements of both vitamin D and calcium result in small increases in bone mineral density throughout the skeleton. They also help to reduce fractures in institutionalized older populations, although the benefit is inconsistent in community-dwelling individuals [1,2,43]. Vitamin D supplementation alone appears to have no effect on risk reduction for fractures nor does it appear to reduce falls among the elderly [1,2,43]; one widely-cited meta-analysis suggesting a protective benefit of supplemental vitamin D against falls [44] has been severely critiqued [1]. However, a large study of women aged ≥ 69 years followed for an average of 4.5 years found both lower (<50 nmol/L [<20 ng/mL]) and higher (≥ 75 nmol/L [≥ 30 ng/mL]) 25(OH)D levels at baseline to be associated with a greater risk of frailty [45]. Women should consult their healthcare providers about their needs for vitamin D (and calcium) as part of an overall plan to prevent or treat osteoporosis.

Cancer

Laboratory and animal evidence as well as epidemiologic data suggest that vitamin D status could affect cancer risk. Strong biological and mechanistic bases indicate that vitamin D plays a role in the prevention of colon, prostate, and breast cancers. Emerging epidemiologic data

suggest that vitamin D may have a protective effect against colon cancer, but the data are not as strong for a protective effect against prostate and breast cancer, and are variable for cancers at other sites [1,46,47]. Studies do not consistently show a protective or no effect, however. One study of Finnish smokers, for example, found that subjects in the highest quintile of baseline vitamin D status had a threefold higher risk of developing pancreatic cancer [48]. A recent review found an increased risk of pancreatic cancer associated with high levels of serum 25(OH)D (≥ 100 nmol/L or ≥ 40 ng/mL) [49].

Vitamin D emerged as a protective factor in a prospective, cross-sectional study of 3,121 adults aged ≥ 50 years (96% men) who underwent a colonoscopy. The study found that 10% had at least one advanced cancerous lesion. Those with the highest vitamin D intakes (>645 IU/day) had a significantly lower risk of these lesions [50]. However, the Women's Health Initiative, in which 36,282 postmenopausal women of various races and ethnicities were randomly assigned to receive 400 IU vitamin D plus 1,000 mg calcium daily or a placebo, found no significant differences between the groups in the incidence of colorectal cancers over 7 years [51]. More recently, a clinical trial focused on bone health in 1,179 postmenopausal women residing in rural Nebraska found that subjects supplemented daily with calcium (1,400–1,500 mg) and vitamin D₃ (1,100 IU) had a significantly lower incidence of cancer over 4 years compared with women taking a placebo [52]. The small number of cancers (50) precludes generalizing about a protective effect from either or both nutrients or for cancers at different sites. This caution is supported by an analysis of 16,618 participants in NHANES III (1988–1994), in which total cancer mortality was found to be unrelated to baseline vitamin D status [53]. However, colorectal cancer mortality was inversely related to serum 25(OH)D concentrations. A large observational study with participants from 10 western European countries also found a strong inverse association between prediagnostic 25(OH)D concentrations and risk of colorectal cancer [54].

Further research is needed to determine whether vitamin D inadequacy in particular increases cancer risk, whether greater exposure to the nutrient is protective, and whether some individuals could be at increased risk of cancer because of vitamin D exposure [46,55]. Taken together, however, studies to date do not support a role for vitamin D, with or without calcium, in reducing the risk of cancer [1].

Other conditions

A growing body of research suggests that vitamin D might play some role in the prevention and treatment of type 1 [56] and type 2 diabetes [57], hypertension [58], glucose intolerance [59], multiple sclerosis [60], and other medical conditions [61,62]. However, most evidence for these roles comes from in vitro, animal, and epidemiological studies, not the randomized clinical trials considered to be more definitive [1]. Until such trials are conducted, the implications of the available evidence for public health and patient care will be debated. One meta-analysis found use of vitamin D supplements to be associated with a statistically significant reduction in overall mortality from any cause [63,64], but a reanalysis of the data found no association [43]. A systematic review of these and other health outcomes related to vitamin D and calcium intakes, both alone and in combination, was published in August 2009 [43].

Health Risks from Excessive Vitamin D

Vitamin D toxicity can cause non-specific symptoms such as anorexia, weight loss, polyuria, and heart arrhythmias. More seriously, it can also raise blood levels of calcium which leads to vascular and tissue calcification, with subsequent damage to the heart, blood vessels, and kidneys [1]. The use of supplements of both calcium (1,000 mg/day) and vitamin D (400 IU) by postmenopausal women was associated with a 17% increase in the risk of kidney stones over 7 years in the Women's Health Initiative [65]. A serum 25(OH)D concentration consistently >500 nmol/L (>200 ng/mL) is considered to be potentially toxic [5].

Excessive sun exposure does not result in vitamin D toxicity because the sustained heat on the skin is thought to photodegrade previtamin D₃ and vitamin D₃ as it is formed [6]. In addition, thermal activation of previtamin D₃ in the skin gives rise to various non-vitamin D forms that limit formation of vitamin D₃ itself. Some vitamin D₃ is also converted to nonactive forms [1]. Intakes of vitamin D from food that are high enough to cause toxicity are very unlikely. Toxicity is much more likely to occur from high intakes of dietary supplements containing vitamin D.

Long-term intakes above the UL increase the risk of adverse health effects [1] (Table 4). Most reports suggest a toxicity threshold for vitamin D of 10,000 to 40,000 IU/day and serum 25(OH)D levels of 500–600 nmol/L (200–240 ng/mL). While symptoms of toxicity are unlikely at daily intakes below 10,000 IU/day, the FNB pointed to emerging science from national survey data, observational studies, and clinical trials suggesting that even lower vitamin D intakes and serum 25(OH)D levels might have adverse health effects over time. The FNB concluded that serum 25(OH)D levels above approximately 125–150 nmol/L (50–60 ng/mL) should be avoided, as even lower serum levels (approximately 75–120 nmol/L or 30–48 ng/mL) are associated with increases in all-cause mortality, greater risk of cancer at some sites like the pancreas, greater risk of cardiovascular events, and more falls and fractures among the elderly. The FNB committee cited research which found that vitamin D intakes of 5,000 IU/day achieved serum 25(OH)D concentrations between 100–150 nmol/L (40–60 ng/mL), but no greater. Applying an uncertainty factor of 20% to this intake value gave a UL of 4,000 IU which the FNB applied to children aged 9 and older, with corresponding lower amounts for younger children.

Table 4: Tolerable Upper Intake Levels (ULs) for Vitamin D

[1]

Age	Male	Female	Pregnancy	Lactation
0–6 months	1,000 IU (25 mcg)	1,000 IU (25 mcg)		
7–12 months	1,500 IU (38 mcg)	1,500 IU (38 mcg)		
1–3 years	2,500 IU (63 mcg)	2,500 IU (63 mcg)		
4–8 years	3,000 IU (75 mcg)	3,000 IU (75 mcg)		
≥9 years	4,000 IU (100 mcg)	4,000 IU (100 mcg)	4,000 IU (100 mcg)	4,000 IU (100 mcg)

Age	Male	Female	Pregnancy	Lactation
-----	------	--------	-----------	-----------

Interactions with Medications

Vitamin D supplements have the potential to interact with several types of medications. A few examples are provided below. Individuals taking these medications on a regular basis should discuss vitamin D intakes with their healthcare providers.

Steroids

Corticosteroid medications such as prednisone, often prescribed to reduce inflammation, can reduce calcium absorption [66,67,68] and impair vitamin D metabolism. These effects can further contribute to the loss of bone and the development of osteoporosis associated with their long-term use [67,68].

Other medications

Both the weight-loss drug orlistat (brand names Xenical® and alli™) and the cholesterol-lowering drug cholestyramine (brand names Questran®, LoCholest®, and Prevalite®) can reduce the absorption of vitamin D and other fat-soluble vitamins [69,70]. Both phenobarbital and phenytoin (brand name Dilantin®), used to prevent and control epileptic seizures, increase the hepatic metabolism of vitamin D to inactive compounds and reduce calcium absorption [71].

Vitamin D and Healthful Diets

The federal government's 2010 *Dietary Guidelines for Americans* notes that "nutrients should come primarily from foods. Foods in nutrient-dense, mostly intact forms contain not only the essential vitamins and minerals that are often contained in nutrient supplements, but also dietary fiber and other naturally occurring substances that may have positive health effects. ...Dietary supplements...may be advantageous in specific situations to increase intake of a specific vitamin or mineral."

For more information about building a healthful diet, refer to the *Dietary Guidelines for Americans* and the U.S. Department of Agriculture's food guidance system, [MyPlate](#).

The *Dietary Guidelines for Americans* describes a healthy diet as one that:

- Emphasizes a variety of fruits, vegetables, whole grains, and fat-free or low-fat milk and milk products.
Milk is fortified with vitamin D, as are many ready-to-eat cereals and some brands of yogurt and orange juice. Cheese naturally contains small amounts of vitamin D.
- Includes lean meats, poultry, fish, beans, eggs, and nuts.
Fatty fish such as salmon, tuna, and mackerel are very good sources of vitamin D. Small amounts of vitamin D are also found in beef liver and egg yolks.
- Is low in saturated fats, trans fats, cholesterol, salt (sodium), and added sugars.
Vitamin D is added to some margarines.
- Stays within your daily calorie needs.

References

1. Institute of Medicine, Food and Nutrition Board. *Dietary Reference Intakes for Calcium and Vitamin D*. Washington, DC: National Academy Press, 2010.
2. Cranney C, Horsely T, O'Donnell S, Weiler H, Ooi D, Atkinson S, et al. Effectiveness and safety of vitamin D. Evidence Report/Technology Assessment No. 158 prepared by the University of Ottawa Evidence-based Practice Center under Contract No. 290-02.0021. AHRQ Publication No. 07-E013. Rockville, MD: Agency for Healthcare Research and Quality, 2007. [[PubMed abstract](#)]
3. Holick MF. Vitamin D. In: Shils ME, Shike M, Ross AC, Caballero B, Cousins RJ, eds. *Modern Nutrition in Health and Disease*, 10th ed. Philadelphia: Lippincott Williams & Wilkins, 2006.
4. Norman AW, Henry HH. Vitamin D. In: Bowman BA, Russell RM, eds. *Present Knowledge in Nutrition*, 9th ed. Washington DC: ILSI Press, 2006.
5. Jones G. Pharmacokinetics of vitamin D toxicity. *Am J Clin Nutr* 2008;88:582S-6S. [[PubMed abstract](#)]
6. Holick MF. Vitamin D deficiency. *N Engl J Med* 2007;357:266-81. [[PubMed abstract](#)]
7. Carter GD. 25-hydroxyvitamin D assays: the quest for accuracy. *Clin Chem* 2009;55:1300-02.
8. Hollis BW. Editorial: the determination of circulating 25-hydroxyvitamin D: no easy task. *J. Clin Endocrinol Metab* 2004;89:3149-3151.
9. Binkley N, Krueger D, Cowgill CS, Plum L, Lake E, Hansen KE, et al. Assay variation confounds the diagnosis of hypovitaminosis D: a call for standardization. *J Clin Endocrinol Metab* 2004;89:3152-57. [[PubMed abstract](#)]
10. National Institute of Standards and Technology. [NIST releases vitamin D standard reference material](#), 2009.
11. U.S. Department of Agriculture, Agricultural Research Service. 2010. [USDA National Nutrient Database for Standard Reference, Release 23](#).
12. Ovesen L, Brot C, Jakobsen J. Food contents and biological activity of 25-hydroxyvitamin D: a vitamin D metabolite to be reckoned with? *Ann Nutr Metab* 2003;47:107-13. [[PubMed abstract](#)]
13. Mattila PH, Piironen VI, Uusi-Rauva EJ, Koivisto PE. Vitamin D contents in edible mushrooms. *J Agric Food Chem* 1994;42:2449-53.
14. Calvo MS, Whiting SJ, Barton CN. Vitamin D fortification in the United States and Canada: current status and data needs. *Am J Clin Nutr* 2004;80:1710S-6S. [[PubMed abstract](#)]

15. Byrdwell WC, DeVries J, Exler J, Harnly JM, Holden JM, Holick MF, et al. Analyzing vitamin D in foods and supplements: methodologic challenges. *Am J Clin Nutr* 2008;88:554S-7S. [[PubMed abstract](#)]
16. Wharton B, Bishop N, Ricketts. *Lancet* 2003;362:1389-400. [[PubMed abstract](#)]
17. Holick MF. Photobiology of vitamin D. In: Feldman D, Pike JW, Glorieux FH, eds. *Vitamin D, Second Edition, Volume I*. Burlington, MA: Elsevier, 2005.
18. Wolpowitz D, Gilchrist BA. The vitamin D questions: how much do you need and how should you get it? *J Am Acad Dermatol* 2006;54:301-17. [[PubMed abstract](#)]
19. Holick MF. Vitamin D: the underappreciated D-lightful hormone that is important for skeletal and cellular health. *Curr Opin Endocrinol Diabetes* 2002;9:87-98.
20. International Agency for Research on Cancer Working Group on ultraviolet (UV) light and skin cancer. The association of use of sunbeds with cutaneous malignant melanoma and other skin cancers: a systematic review. *Int J Cancer* 2006;120:1116-22. [[PubMed abstract](#)]
21. American Academy of Dermatology. [Position statement on vitamin D](#). November 1, 2008.
22. Wagner CL, Greer FR; American Academy of Pediatrics Section on Breastfeeding; American Academy of Pediatrics Committee on Nutrition. [Prevention of rickets and vitamin D deficiency in infants, children, and adolescents](#). *Pediatrics* 2008;122:1142-1152. [[PubMed abstract](#)]
23. Bailey RL, Dodd KW, Goldman JA, Gahche JJ, Dwyer JT, Moshfegh AJ, et al. Estimation of total usual calcium and vitamin D intakes in the United States. *J Nutr* 2010;140:817-822. [[PubMed abstract](#)]
24. Looker AC, Pfeiffer CM, Lacher DA, Schleicher RL, Picciano MF, Yetley EA. Serum 25-hydroxyvitamin D status of the US population: 1988-1994 compared with 2000-2004. *Am J Clin Nutr* 2008;88:1519-27. [[PubMed abstract](#)]
25. Chesney R. Rickets: an old form for a new century. *Pediatr Int* 2003;45: 509-11. [[PubMed abstract](#)]
26. Goldring SR, Krane S, Avioli LV. Disorders of calcification: osteomalacia and rickets. In: DeGroot LJ, Besser M, Burger HG, Jameson JL, Loriaux DL, Marshall JC, et al., eds. *Endocrinology*. 3rd ed. Philadelphia: WB Saunders, 1995:1204-27.
27. Picciano MF. Nutrient composition of human milk. *Pediatr Clin North Am* 2001;48:53-67. [[PubMed abstract](#)]
28. Weisberg P, Scanlon KS, Li R, Cogswell ME. Nutritional rickets among children in the United States: review of cases reported between 1986 and 2003. *Am J Clin Nutr* 2004;80:1697S-705S. [[PubMed abstract](#)]
29. Ward LM, Gaboury I, Ladhani M, Zlotkin S. Vitamin D-deficiency rickets among children in Canada. *CMAJ* 2007;177:161-166. [[PubMed abstract](#)]
30. American Academy of Pediatrics Committee on Environmental Health. Ultraviolet light: a hazard to children. *Pediatrics* 1999;104:328-33. [[PubMed abstract](#)]
31. Webb AR, Kline L, Holick MF. Influence of season and latitude on the cutaneous synthesis of vitamin D3: Exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin. *J Clin Endocrinol Metab* 1988;67:373-8. [[PubMed abstract](#)]
32. Webb AR, Pilbeam C, Hanafin N, Holick MF. An evaluation of the relative contributions of exposure to sunlight and of diet to the circulating concentrations of 25-hydroxyvitamin D in an elderly nursing home population in Boston. *Am J Clin Nutr* 1990;51:1075-81. [[PubMed abstract](#)]
33. Lo CW, Paris PW, Clemens TL, Nolan J, Holick MF. Vitamin D absorption in healthy subjects and in patients with intestinal malabsorption syndromes. *Am J Clin Nutr* 1985;42:644-49. [[PubMed abstract](#)]
34. Malone M. Recommended nutritional supplements for bariatric surgery patients. *Ann Pharmacother* 2008;42:1851-8. [[PubMed abstract](#)]
35. Compber CW, Badellino KO, Boullata JI. Vitamin D and the bariatric surgical patient: a review. *Obes Surg* 2008;18:220-4. [[PubMed abstract](#)]
36. Vieth R, Bischoff-Ferrari H, Boucher BJ, Dawson-Hughes B, Garland CF, Heaney RP, et al. The urgent need to recommend an intake of vitamin D that is effective. *Am J Clin Nutr* 2007;85:649-50. [[PubMed abstract](#)]
37. National Institutes of Health Osteoporosis and Related Bone Diseases National Research Center. [Osteoporosis overview](#). October 2010.
38. Heaney RP. Long-latency deficiency disease: insights from calcium and vitamin D. *Am J Clin Nutr* 2003;78:912-9. [[PubMed abstract](#)]
39. LeBoff MS, Kohlmeier L, Hurwitz S, Franklin J, Wright J, Glowacki J. Occult vitamin D deficiency in postmenopausal US women with acute hip fracture. *JAMA* 1999;281:1505-11. [[PubMed abstract](#)]
40. Kirschstein R. Menopausal hormone therapy: summary of a scientific workshop. *Ann Intern Med* 2003;138:361-4. [[PubMed abstract](#)]
41. American College of Obstetricians and Gynecologists. [Frequently Asked Questions About Hormone Therapy. New Recommendations Based on ACOG's Task Force Report on Hormone Therapy](#).
42. North American Menopause Society. Role of progestogen in hormone therapy for postmenopausal women: position statement of The North American Menopause Society. *Menopause* 2003;10:113-32. [[PubMed abstract](#)]
43. Chung M, Balk EM, Brendel M, Ip S, Lau J, Lee J, et al. [Vitamin D and calcium: a systematic review of health outcomes](#). Evidence Report/Technology Assessment No. 183 prepared by the Tufts Evidence-based Practice Center under Contract No. 290-2007-10055-I. AHRQ Publication No. 09-E015. Rockville, MD: Agency for Healthcare Research and Quality, 2009.
44. Bischoff-Ferrari HA, Dawson-Hughes B, Staehelin HB, Orav JE, Stuck AE, Theiler R, et al. Fall prevention with supplemental and active forms of vitamin D: a meta-analysis of randomised controlled trials. *BMJ* 2009;339:b3692. [[PubMed abstract](#)]
45. Ensrud KE, Ewing SK, Fredman L, Hochberg MC, Cauley JA, Hillier TA, et al. Circulating 25-hydroxyvitamin D levels and frailty status in older women. *J Clin Endocrinol Metab* 2010;95:5266-5273. [[PubMed abstract](#)]
46. Davis CD. Vitamin D and cancer: current dilemmas and future research needs. *Am J Clin Nutr* 2008;88:565S-9S. [[PubMed abstract](#)]
47. Davis CD, Hartmuller V, Freedman M, Hartge P, Picciano MF, Swanson CA, Milner JA. Vitamin D and cancer: current dilemmas and future needs. *Nutr Rev* 2007;65:S71-S74. [[PubMed abstract](#)]
48. Stolzenberg-Solomon RZ, Vieth R, Azad A, Pietinen P, Taylor PR, Virtamo J, et al. A prospective nested case-control study of vitamin D status and pancreatic cancer risk in male smokers. *Cancer Res* 2006;66:10213-9. [[PubMed abstract](#)]
49. Kathy J. Helzlsouer for the VDPP Steering Committee. Overview of the Cohort Consortium Vitamin D Pooling Project of Rarer

- Cancers. *Am J Epidemiol* 2010;172:4-9. [[PubMed abstract](#)]
50. Lieberman DA, Prindiville S, Weiss DG, Willett W. Risk factors for advanced colonic neoplasia and hyperplastic polyps in asymptomatic individuals. *JAMA* 2003;290:2959-67. [[PubMed abstract](#)]
 51. Wactawski-Wende J, Kotchen JM, Anderson GL, Assaf AR, Brunner RL, O'Sullivan MJ, et al. Calcium plus vitamin D supplementation and the risk of colorectal cancer. *N Engl J Med* 2006;354:684-96. [[PubMed abstract](#)]
 52. Parfitt AM. Osteomalacia and related disorders. In: Avioli LV, Krane SM, eds. *Metabolic bone disease and clinically related disorders*. 2nd ed. Philadelphia: WB Saunders, 1990:329-96.
 53. Freedman DM, Looker AC, Chang S-C, Graubard BI. Prospective study of serum vitamin D and cancer mortality in the United States. *J Natl Cancer Inst* 2007;99:1594-602. [[PubMed abstract](#)]
 54. Jenab M, Bueno-de-Mesquita HB, Ferrari P, van Duijnhoven FJB, Norat T, Pischon T, et al. Association between pre-diagnostic circulating vitamin D concentration and risk of colorectal cancer in European populations: a nested case-control study. *BMJ* 2010;340:b5500. [[PubMed abstract](#)]
 55. Davis CD, Dwyer JT. The 'sunshine vitamin': benefits beyond bone? *J Natl Cancer Inst* 2007;99:1563-5. [[PubMed abstract](#)]
 56. Hyppönen E, Läärä E, Reunanen A, Järvelin MR, Virtanen SM. Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. *Lancet* 2001;358:1500-3. [[PubMed abstract](#)]
 57. Pittas AG, Dawson-Hughes B, Li T, Van Dam RM, Willett WC, Manson JE, et al. Vitamin D and calcium intake in relation to type 2 diabetes in women. *Diabetes Care* 2006;29:650-6. [[PubMed abstract](#)]
 58. Krause R, Bühring M, Hopfenmüller W, Holick MF, Sharma AM. Ultraviolet B and blood pressure. *Lancet* 1998;352:709-10. [[PubMed abstract](#)]
 59. Chiu KC, Chu A, Go VL, Saad MF. Hypovitaminosis D is associated with insulin resistance and beta cell dysfunction. *Am J Clin Nutr* 2004;79:820-5. [[PubMed abstract](#)]
 60. Munger KL, Levin LI, Hollis BW, Howard NS, Ascherio A. Serum 25-hydroxyvitamin D levels and risk of multiple sclerosis. *JAMA* 2006;296:2832-8. [[PubMed abstract](#)]
 61. Merlino LA, Curtis J, Mikuls TR, Cerhan JR, Criswell LA, Saag K. Vitamin D intake is inversely associated with rheumatoid arthritis: results from the Iowa Women's Health Study. *Arthritis Rheum* 2004;50:72-7. [[PubMed abstract](#)]
 62. Schleithoff SS, Zittermann A, Tenderich G, Berthold HK, Stehle P, Koerfer R. Vitamin D supplementation improves cytokine profiles in patients with congestive heart failure: a double-blind, randomized, placebo-controlled trial. *Am J Clin Nutr* 2006;83:754-9. [[PubMed abstract](#)]
 63. Autier P, Gandini S. Vitamin D supplementation and total mortality: a meta-analysis of randomized controlled trials. *Arch Intern Med* 2007;167:1730-7. [[PubMed abstract](#)]
 64. Giovannucci E. Can vitamin D reduce total mortality? *Arch Intern Med* 2007;167:1709-10. [[PubMed abstract](#)]
 65. Jackson RD, LaCroix AZ, Gass M, Wallace RB, Robbins J, Lewis CE, et al. Calcium plus vitamin D supplementation and the risk of fractures. *N Engl J Med* 2006;354:669-83. [[PubMed abstract](#)]
 66. Buckley LM, Leib ES, Cartularo KS, Vacek PM, Cooper SM. Calcium and vitamin D3 supplementation prevents bone loss in the spine secondary to low-dose corticosteroids in patients with rheumatoid arthritis. A randomized, double-blind, placebo-controlled trial. *Ann Intern Med* 1996;125:961-8. [[PubMed abstract](#)]
 67. Lukert BP, Raisz LG. Glucocorticoid-induced osteoporosis: pathogenesis and management. *Ann Intern Med* 1990;112:352-64. [[PubMed abstract](#)]
 68. de Sevaux RGL, Hoitsma AJ, Corstens FHM, Wetzels JFM. Treatment with vitamin D and calcium reduces bone loss after renal transplantation: a randomized study. *J Am Soc Nephrol* 2002;13:1608-14. [[PubMed abstract](#)]
 69. McDuffie JR, Calis KA, Booth SL, Uwaifo GI, Yanovski JA. Effects of orlistat on fat-soluble vitamins in obese adolescents. *Pharmacotherapy* 2002;22:814-22. [[PubMed abstract](#)]
 70. Compston JE, Horton LW. Oral 25-hydroxyvitamin D3 in treatment of osteomalacia associated with ileal resection and cholestyramine therapy. *Gastroenterology* 1978;74:900-2. [[PubMed abstract](#)]
 71. Gough H, Goggin T, Bissessar A, Baker M, Crowley M, Callaghan N. A comparative study of the relative influence of different anticonvulsant drugs, UV exposure and diet on vitamin D and calcium metabolism in outpatients with epilepsy. *Q J Med* 1986;59:569-77. [[PubMed abstract](#)]

Disclaimer

This fact sheet by the Office of Dietary Supplements provides information that should not take the place of medical advice. We encourage you to talk to your health care providers (doctor, registered dietitian, pharmacist, etc.) about your interest in, questions about, or use of dietary supplements and what may be best for your overall health. Any mention in this publication of a specific brand name is not an endorsement of the product.