Vitamin D Deficiency in the United States: How Common is it?

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Vitamin D Deficiency in the United States: How Common is it?

by

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in

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Approved:

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Abstract

**Background:** Vitamin D deficiency in the US is more prevalent than before estimated. Given the numerous roles vitamin D plays in the body, it is becoming apparent that the need for adequate intake is vital. It is generally accepted that adults require 1,000 IU per day.

**Methods:** Electronic searches were conducted using Google Scholar and Elsevier databases. Search terms included “vitamin D deficiency”, “vitamin D deficiency US”, and “vitamin D screening”. Databases were reviewed for prevalence of vitamin D deficiency in the US.

**Results:** Populations at high risk for deficiency include the elderly, adolescents, people with darker skin, those who are obese, and those with limited sun exposure. Studies have shown that a deficiency cannot only lead to bone disease, but possibly cancer, cardiovascular disease and autoimmune diseases.

**Conclusions:** Due to the increasing prevalence of vitamin D deficiency in the US, it is suggested that interventions be taken to increase intake among at-risk populations. Such interventions include increasing consumption of vitamin D rich foods, taking a vitamin D supplement, and/or increasing sun exposure to the extent needed for adequate vitamin D production.
Introduction

This topic was chosen due to the increasing awareness of vitamin D deficiency among the US population. Numerous studies have been released addressing the newly discovered roles of vitamin D in the body. It has been suggested that vitamin D deficiency may increase risk for cancer, cardiovascular disease, autoimmune diseases, and bone diseases, among others. Unfortunately, it has been proposed that the current recommendation for vitamin D intake is far too low to prevent deficiency (1). This study seeks to determine the prevalence of vitamin D deficiency among the US population. It also addresses interventions that can be made to increase vitamin D status.

Review of Literature

What is Vitamin D?

Vitamin D is a fat-soluble vitamin that is converted to a hormone within the body. By definition, hormones are considered to be chemical messengers that relay messages to cells. Hormones cause cells to express specific sequences of deoxyribonucleic acid (DNA), which is contained within the cell nucleus. When this specific sequence of DNA is expressed within a cell, the cell then responds through the process of transcription and translation and produces specific proteins, which then perform direct functions in the body (2). The active form of vitamin D, calcitriol, acts as a hormone by binding to vitamin D receptors (VDRs) both on the cell membrane as well as in the nucleus. This binding then leads to specific gene expression (3).
**Vitamin D Metabolism**

The two main sources of vitamin D are sunlight and diet. The skin synthesizes a steroid, 7-dehydrocholesterol, which is capable of absorbing specific wavelengths of light. When the skin is exposed to certain wavelengths of ultraviolet B (UVB) rays from the sun, the stored 7-dehydrocholesterol is converted to previtamin D3 or precalciferol (3, 4). In two to three days, the previtamin D3 is thermally isomerized to produce vitamin D3, cholecalciferol. Vitamin D3 then diffuses into the blood stream via a vitamin D binding protein (DBP), which then transports the vitamin to the liver. Once in the liver, it is hydroxylated and becomes 25-hydroxyvitamin D3 after which it is transported to the kidney for a second hydroxylation in which it is converted to 1,25-(OH)2 D3, which is the active form of vitamin D. Vitamin D3 is then transported throughout the body to cells where it binds to VDRs and leads to specific gene expression (3).

When dietary vitamin D is consumed, it is absorbed in a micelle along with lipids via passive diffusion. The vitamin D is incorporated into chylomicrons and is then transported to the liver (3).

Once in the liver, vitamin D is metabolized and completes its first hydroxylation after which it is released into the blood. The serum vitamin D (25-OH D3) level reflects vitamin D status and concentration within the body. If serum 25-OH-D3 becomes depleted, cholecalciferol will be released from its storage sites within the blood, muscle, and adipose tissue (3).

After the hydroxylized vitamin D enters the blood, it is primarily taken up by the kidney in order to undergo a second hydroxylation upon which it is considered to be in
the active form of the vitamin. Calcitriol, or 1,25-(OH)2 D3, is then released from the kidney and travels via DBP into the blood where it will be delivered to target tissues (3).

The following diagram is a visual representation of vitamin D metabolism (5):
**Sources of Vitamin D**

The primary sources of dietary vitamin D include dairy products, fatty fish, and fortified foods. The following table highlights the amount of vitamin D in these foods (6):

<table>
<thead>
<tr>
<th>Food</th>
<th>Serving</th>
<th>Vitamin D (IU)</th>
<th>Vitamin D (mcg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pink Salmon, canned</td>
<td>3 ounces</td>
<td>530</td>
<td>13.3</td>
</tr>
<tr>
<td>Sardines, canned</td>
<td>3 ounces</td>
<td>231</td>
<td>5.8</td>
</tr>
<tr>
<td>Mackerel, canned</td>
<td>3 ounces</td>
<td>213</td>
<td>5.3</td>
</tr>
<tr>
<td>Quaker Nutrition for Women Instant Oatmeal®</td>
<td>1 packet</td>
<td>154</td>
<td>3.9</td>
</tr>
<tr>
<td>Cow’s Milk, fortified with Vitamin D</td>
<td>8 ounces</td>
<td>98</td>
<td>2.5</td>
</tr>
<tr>
<td>Soy Milk, fortified with Vitamin D</td>
<td>8 ounces</td>
<td>100</td>
<td>2.5</td>
</tr>
<tr>
<td>Orange Juice, fortified with Vitamin D</td>
<td>8 ounces</td>
<td>100</td>
<td>2.5</td>
</tr>
<tr>
<td>Cereal, fortified</td>
<td>1 serving (usually 1 cup)</td>
<td>40-50</td>
<td>1.0-1.3</td>
</tr>
<tr>
<td>Egg Yolk</td>
<td>1 large</td>
<td>21</td>
<td>0.53</td>
</tr>
</tbody>
</table>

**Roles of Vitamin D**

Vitamin D has several roles in the body; many of these arise from its action on gene transcription and expression. Vitamin D receptors (VDRs) are located on many cells and respond to the presence of vitamin D by initiating a cascade of events that leads to transcription of specific genes (6). Currently, there are at least 200 genes known that respond to 1,25-dihydroxyvitamin D (3).

**Bone Health and Calcium Absorption**
The roles of vitamin D in the body are increasingly more understood as additional research is conducted in this area. One of its most widely known functions is its involvement in bone health. Vitamin D facilitates calcium absorption in the intestine by influencing the expression of epithelial calcium channels and thus calcium-binding proteins. This process allows calcium to be better absorbed from the foods eaten (1).

Due to the increase in absorption on calcium, parathyroid hormone (PTH) levels are better regulated. When serum calcium levels are low, the parathyroid gland secretes PTH, which leads to increased production of vitamin D3. This further increases absorption of calcium from the intestine as well as increases reabsorption of calcium by the kidneys. The third effect that increased PTH levels have on the body is that it leads to resorption of calcium from the bone in order to maintain adequate serum levels. Leaching calcium out of the matrix of bone leads to decreased bone strength. If adequate vitamin D3 is present before this occurs, PTH levels are likely to be kept low as calcium absorption is increased (6).

Another way in which vitamin D works to increase bone strength is by mediating the incorporation of calcium into the matrix of bone. This strengthens the network of fibers within the bone itself thus leading to stronger bones (4).
Cellular Differentiation

Vitamin D also plays a role in cellular differentiation. It has been shown to decrease proliferation of cells and plays a role in their maturation. This is a very important function in terms of cancer prevention. Cells that proliferate at faster rates are at increased risk for developing mutations. The active form of vitamin D, calcitriol, helps to regulate cellular proliferation by promoting cellular differentiation (6,8).

Immunomodulator

VDRs are located on activated T and B lymphocytes, monocytes, and macrophages thus showing vitamin D is also an immunomodulator. It helps to regulate the function of lymphocytes, the production of cytokines, maturation of monocytes, and
macrophage activity. Its role in the immune system may lead to the prevention of autoimmune diseases when adequate serum levels of vitamin D are maintained (1).

**Insulin Secretion**

Vitamin D and the prevention of diabetes is another recent area of study. VDRs are located on the beta cells of the pancreas. It appears that in situations in which the body requires increased amounts of insulin, vitamin D plays a role in the secretion of insulin. Recent studies have concluded that when an insufficient amount of vitamin D3 is present, glucose intolerance and impaired insulin secretion are observed in populations with type 2 diabetes (6).

**Blood Pressure**

Adequate vitamin D3 levels are also associated with a decreased risk for cardiovascular disease. VDRs are located on vascular smooth muscle, endothelium, and cardiomyocytes (9). One of the main mechanisms whereby vitamin D appears to decrease cardiovascular disease risk is its effect on hypertension through the rennin-angiotensin system. The expression of renin leads to the stimulation and production of angiotensin which then causes a series of reactions that increase blood pressure. These reactions include constriction of small arteries as well as an increase in the amount of sodium and water reabsorbed by the kidneys. Vitamin D has been shown to depress the gene expression of renin thus leading to decreased blood pressure (6).

**Serum Levels that Categorize Deficiency**

Vitamin D status is determined by measuring serum calcidiol levels, 25 (OH) D3. When calcitriol levels become low, parathyroid hormone (PTH) is secreted to increase
serum calcitriol levels. This leads to a decrease in calcidiol levels as it is hydroxylated to the active form of vitamin D. Thus, a vitamin D deficiency would be observed in people with low 25 (OH) D3 levels (3).

Prior to the influx of vitamin D research, serum levels of less than 5-7 ng/dL of calcidiol was considered to be deficient. Values around 10-12 ng/dL were considered insufficient and serum levels of 18-20 ng/dL or higher were considered normal (10). Current research has shown however, that the following cutoff values are more accurate and a better reflection of adequate vitamin D status (2):

<table>
<thead>
<tr>
<th>Recommended Vitamin D Serum Level Standards</th>
<th>ng/mL</th>
<th>nmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficient</td>
<td>&lt; 20</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Insufficient</td>
<td>21-29</td>
<td>52.5-72.5</td>
</tr>
<tr>
<td>Sufficient</td>
<td>≥30</td>
<td>&gt;75</td>
</tr>
</tbody>
</table>

**Factors Affecting Vitamin D Uptake**

There are several factors that affect vitamin D uptake and serum concentration. These factors include: aging, skin pigmentation, sunlight exposure, location, obesity, diet, metabolic abnormalities, liver dysfunction, kidney disease, and exclusively breastfeeding in infants (1,8).

**Aging**

Many elderly people remain homebound and do not receive as much sunlight exposure as those who are younger (11). When the elderly population does receive sun exposure, vitamin D production is hindered by decreased capability of the skin to utilize
the sunlight received. As a person ages, 7-dehydrocholesterol decreases in the skin, which leads to decreased absorption of UVB rays that convert precalciferol to cholecalciferol (1).

**Skin Pigmentation**

Skin pigmentation can greatly affect absorption of UVB rays. The darker the melanin in the skin, the less UV radiation is penetrated. Dark skin pigmentation can decrease the amount of UV radiation that reaches skin cells by up to 99% (4).

**Sunlight Exposure**

The amount of sunlight a person receives also makes an impact on the amount of vitamin D synthesized and that is available for cellular processes. Those whose skin is not often exposed to sunlight are likely deficient if they do not compensate by supplementing with vitamin D (4).

**Location**

The location in which a person lives can influence the amount of UVB rays absorbed. During the winter months, those that live above the 37th degree latitude are at greater risk for developing vitamin D deficiency. This is because the angle of the sun is such that it prevents UVB rays from being absorbed by the skin. Additionally, studies have shown that those who live in the northern latitudes of the United States have a greater prevalence of cancer when compared to those who reside in the southern states (1).

The following is a visual representation of the portion of the United States that is north of the 37th parallel (12):
Inactivity and Obesity

A sedentary lifestyle leading to obesity increases the risk of vitamin D deficiency due to the fact that vitamin D is a fat-soluble vitamin and is thus stored in adipose tissue. In the case of obesity, it becomes increasingly difficult for the body to retrieve stored vitamin D because it is less bioavailable when it is imbedded in deeper adipose stores (1). Additionally, a sedentary lifestyle in which weight-bearing exercise is not used to maintain bone density leads to an increased need for vitamin D (4).

Diet and Malabsorptive Disorders

The amount of vitamin D received in the diet also has an impact on serum levels. Given the body’s ability to absorb vitamin D is not compromised, increased intake of foods rich in vitamin D, such as fatty fish and milk, will lead to increased serum levels. In regard to metabolic abnormalities due to the fact that vitamin D is fat-soluble and is best absorbed with dietary fat, if an individual has a condition in which fat absorption is compromised, such as cystic fibrosis, Crohn’s disease, or sprue, then less of the vitamin will be absorbed despite the amount of vitamin D and/or dietary fat ingested (1).

Liver Dysfunction and Kidney Disease
In cases of liver dysfunction, the hepatic enzymes that catalyze the first hydroxylation of cholecalciferol may be inhibited. Additionally, kidney disease or dysfunction leads to an inability of the kidneys to effectively convert vitamin D into its active form. Certain medications may also block the uptake of vitamin D from the gastrointestinal tract (4).

**Breastfed Infants**

Infants who are exclusively breastfed are also at risk for developing deficiency due to the absence of vitamin D in breastmilk. Women who are exclusively breastfeeding are encouraged to give their child a vitamin D supplement (8).

**Current Recommended Intakes**

The current adequate intake for vitamin D published by the Institute of Medicine is set at 200 IU per day for children and adults up to the age of 50. Those over the age of 50 are advised to consume 400-600 IU per day due to the effect of aging on vitamin D absorption and synthesis (8). Specific groups who are at risk for deficiency due to skin color, disease, lack of sun exposure, or other factors are advised to increase their intake of vitamin D rich foods or to add vitamin D supplements to their diet (13).

**Prevalence of Deficiency**

**Vitamin D Deficiency among Adolescents**

The prevalence of vitamin D deficiency is increasing among adolescents. Adequate vitamin D concentration and bone growth are vital during the young adult years not only to help prevent osteoporosis and osteopenia later in life, but also to protect
against various other diseases that vitamin D deficiency is associated with (15). A study conducted among 307 healthy, young adults in Boston revealed that 24.1% were deficient in vitamin D. The highest prevalence existed among African Americans. Additionally, a greater prevalence of deficiency was observed during the winter and spring months. These results coincide with the previously stated statistics in which darker skin pigmentation leads to a greater risk for vitamin D deficiency as well as the fact that less sunlight is available for vitamin D synthesis in the winter and spring months (14).

A relationship was noted between the foods commonly consumed and the presence of deficiency. A positive correlation existed between soda, fruit juice, and iced tea intake and deficiency. While an inverse relationship existed between milk consumption as well as cold cereal consumption and deficiency. This data is in agreement with preexisting knowledge that fortified milk is a good source of vitamin D. It was suggested that due to the decline of milk consumption in the adolescent population, vitamin D deficiency is on the rise. Milk is being replaced with other beverages including soda and juice, which provide little benefit to bone health (14).

A second study reviewed the results obtained from the National Health and Nutrition Examination Survey III study about vitamin D status among adolescents. This study examined the overall deficiency rate among adolescents categorizing a deficiency as a serum level of <11 ng/mL. The study then examined the prevalence of deficiency among the same group of adolescents categorizing a deficiency as a serum level of <20 ng/mL. It was observed that the prevalence of deficiency was 2% with the minimum acceptable serum level being 11 ng/mL. When this value was increased to 20 ng/mL and the same population statistics observed, the prevalence of deficiency increased to 14%.
These observed results are shown below in addition to the change in prevalence according to specific population characteristics (15):

![Graph showing prevalence of vitamin D deficiency across different groups]

The greatest change in the prevalence of vitamin D deficiency was observed in the Non-Hispanic Black adolescent group in which the prevalence rate has increased from 11% to 50% (15). The fact that this group had the highest prevalence rate is not surprising given that this population is at greater risk for vitamin D deficiency due to their darker complexion.

Body mass index (BMI) and gender also play a role. With regards to BMI, a 1% increase in BMI is associated with a 5% decrease in serum vitamin D levels. This observation coincides with the aforementioned data that suggests excess body fat leads to decreased bioavailability of vitamin D as vitamin D is stored deep within the subcutaneous fat. Higher intakes of vitamin D may offer little benefit in such cases. As
the prevalence of childhood obesity continues to increase, vitamin D deficiency among this population may increase as well (15).

The prevalence of deficiency was observed to be greater among females. This may impact teenage pregnancy outcomes. A prenatal deficiency of vitamin D may place the mother at greater risk for developing preeclampsia and gestational diabetes. It may also be associated with decreased bone mineralization in the fetus. A visual representation of deficiency specific to gender is shown below (15):

![Graph showing mean serum 25(OH)D levels by gender and race](image)

Although the NHANES III study consisted of a large sample that was nationally representative of the adolescent population, smaller scale studies have corroborated many of the findings and have offered insight into vitamin D deficiency. In general, it is thought that the prevalence of deficiency is much higher than the results found in the NHANES III study. For example, Gordon et al. (14) found a 42% prevalence of vitamin D deficiency (<20 ng/ml of serum vitamin D). Using the same measurement for deficiency, Harkness and Cromer (16) observed a 54% deficiency among adolescent
females alone. It should be noted that both of these studies were performed in urban areas where minority groups may have been overrepresented, which perhaps accounted for a greater prevalence than that observed in the NHANES III study. (15)

The prevalence of deficiency among adolescents must be closely observed. Due to the increasing evidence that suggests low vitamin D status is associated with the development of chronic disease, it is vital that prevention of deficiency occurs during childhood in order to prevent a greater chance of chronic disease arising later in life. Adolescents with higher BMIs who have a greater prevalence of vitamin D deficiency, are at increased risk of chronic disease related to vitamin D deficiency if interventions are not initiated. It is interesting to note that if the current standard for measuring deficiency is taken into consideration in this population and <30 ng/mL is considered vitamin D deficient, as many experts argue, 48% would be considered deficient (15).

Considering that only 50-60% of children receive the current recommended vitamin D intake of 200 IU per day from diet and/or supplements, supplementation among the adolescent population should be considered. Serum levels of vitamin D should also be monitored regularly to prevent deficiency (15).

Vitamin D Deficiency among the Elderly

The elderly population is at risk for vitamin D deficiency due to decreased dietary intake and sunlight exposure, reduced skin thickness, impaired intestinal absorption, and impaired hydroxylation of vitamin D in the liver and kidneys. Moreover, the reduction in bone density, muscle mass, and muscle strength that are associated with aging are impacted by vitamin D deficiency. The loss of muscle mass often results in functional
impairment, the need for assistance with activities of daily living, an increased risk of falling, and non-vertebral fractures (17).

It has been shown that for those over the age of 65 years, 30% will fall at least once during the year. For those over 80 years of age, the percentage of those who have fallen each year increases to 40-50%. This is alarming because falls are the primary cause of injury and mortality among the elderly. Injury sustained from falls result in 40% of all nursing home admissions. The annual health care cost for all fall-related injuries in the United States in those over the age of 65 years totaled 20.3 billion in 1994 and is projected to increase to 32.4 billion in the year 2020 (18).

Falls often result in fractures, which contribute greatly to morbidity and mortality in the elderly. It has been shown that by the age of 90 years, one-third of women and one-sixth of men will have sustained a fracture. Bischoff-Ferrari et al. (19) looked specifically at hip fractures. The consequences of hip fractures among the elderly were observed as follows: 50% will develop a functional disability, 15-25% will require long-term nursing home care, and 10-20% will die within one year.

As the population continues to age, the incidence of hip fractures are expected to increase worldwide. The best intervention is prevention. One such intervention is vitamin D supplementation. A meta-analysis of twelve randomized controlled trials on vitamin D supplementation and its effect on hip and non-vertebral fractures revealed that doses of 700-800 IU per day of vitamin D (with or without calcium supplementation) reduced the risk for hip fracture by 26%. It was also shown that vitamin D supplementation reduced the risk for non-vertebral fractures by 23%. Additionally, those
that received 400 IUs of vitamin D daily did not have a decreased risk for hip or non-vertebral fractures (19).

There was a correlation between higher serum levels of 25-hydroxyvitamin D and a decreased risk of fracture. This correlation was explained by the physiological roles of vitamin D. First, vitamin D decreases bone loss in the elderly. Second, vitamin D has been associated with increased muscle strength and balance. Thirdly, it has been shown that there is a 22% decreased risk of falling in those older persons who are not deficient in vitamin D (19).

Such findings are consistent with other studies. The National US Survey for adults over 50 years of age found that as the serum level of vitamin D increased up to 80 nmol/L, so did bone mineral density. Additionally, it was observed that 800 IU of vitamin D per day decreased fall risk by 35% (19).

Vitamin D deficiency has also been associated with muscle weakness, which is most commonly observed in the proximal muscle groups and is recognized by feelings of heaviness in the legs, tiring easily, and difficulty mounting stairs as well as rising from chairs. Vitamin D deficiency most often affects the weight-bearing muscles of the lower limbs that are required for balance and walking (17).

Vitamin D receptors (VDRs) have been identified in the skeletal muscle that binds 1,25 (OH) D3. It is involved in the synthesis of the final transcription complex via transportation to the nucleus and modulation by biochemical processes and transcription factors. Janssen et al. (17) stated, “this pathway was found to influence muscle cell membrane and phospholipid metabolism and mediate cell proliferation and subsequently differentiation into mature muscle fibers” (17).
Each of the symptoms experienced are reversible with adequate supplementation of vitamin D. It has been observed that improved muscle strength, walking distance, functional ability, and body sway are all related to vitamin D supplementation. Vitamin D supplementation can preserve muscle strength and functional ability in the elderly. However, muscle weakness is multifaceted and vitamin D supplementation alone may not improve outcomes. Muscle weakness is often related to immobility or other comorbidities regardless of vitamin D status. Regardless, it should be noted that muscle tissue is a direct target site for vitamin D and the elderly population in general may benefit from adequate intake of vitamin D (17). The amount of dietary vitamin D is debatable but there is much evidence that suggests 700-800 IU per day may decrease the risk of falls and fractures (19).

**Vitamin D Screening and Supplementation**

Given the benefits of adequate vitamin D in the body, it is becoming increasingly important to screen for deficiency as well as reevaluate the current recommendations for vitamin D intake in all populations. As mentioned throughout, a high percentage of the US population is deficient in vitamin D. Tangpricha et al. (20) conducted a study in Boston, Massachusetts in which they measured the serum 25 (OH)D levels of 142 subjects at the end of summer and 165 subjects at the end of winter. Sixty-percent of the sample population was Caucasian. The table below represents the percentage of deficiency among those whose serum levels were tested (20):

<table>
<thead>
<tr>
<th>Age Groups</th>
<th>After Winter</th>
<th>After Summer</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The average percentage of vitamin D insufficiency after the winter was 30% while the average after the summer was 11%. One possible explanation for this is that those greater than 50 years are more likely to consume a multivitamin containing vitamin D on a routine basis (20).

It is important to note that a vitamin D serum level of less than 20 ng/mL was considered insufficient. As previously mentioned, there is some evidence that serum values below 30 ng/mL should be considered insufficient while levels below 20 ng/mL are deficient (8). The following table represents values that many studies deem to be more appropriate (3).

<table>
<thead>
<tr>
<th>Age Group</th>
<th>% of Vitamin D insufficiency</th>
<th>% of Insufficiency</th>
<th>% of Deficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-29</td>
<td>32</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>30-39</td>
<td>25</td>
<td>18</td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td>30</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>≥50</td>
<td>16</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Recommended Vitamin D Serum Level Standards</th>
</tr>
</thead>
<tbody>
<tr>
<td>ng/mL</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>Deficient</td>
</tr>
<tr>
<td>Insufficient</td>
</tr>
<tr>
<td>Sufficient</td>
</tr>
</tbody>
</table>

Holick (8) has suggested that to attain maximum bone health and prevention of chronic disease, serum vitamin D concentration should be at least 30-40 ng/mL. It was
also suggested that screening of serum levels should take place on an annual basis and interventions taken place to ensure adequate vitamin D status.

If vitamin D deficiency is identified, it is often treated by high supplementation of vitamin D for a short period of time. For instance, one dose of 50,000 IU of vitamin D3 may be given per week for a period of 8 weeks to help increase serum levels. Another regimen is to provide 50,000 IU of vitamin D3 per day for seven days and then follow-up by supplementing 400-800 IU of D3 per day. Periodic screening is then conducted thereafter to determine the effectiveness of the treatment. The individual may then be retreated if warranted (21).

Such treatments have often been given to those considered to be severely deficient (below 8 ng/mL). However, the level that classifies deficiency is in question and it is possible that these treatments may become more common among the general population due to the high prevalence of vitamin D deficiency among the general population. Current national guidelines developed in 1997 no longer appear to be adequate. Many experts agree upon changing the current recommendations to those displayed in the table below (21).

<table>
<thead>
<tr>
<th>Guidelines for Vitamin D Intake</th>
<th>1997 Adequate Intake</th>
<th>2003 Informal Recommendations</th>
<th>Safe Upper Limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1 y</td>
<td>200 IU</td>
<td>--</td>
<td>1000 IU</td>
</tr>
<tr>
<td>1-50 y</td>
<td>200 IU</td>
<td>1000 IU</td>
<td>2000 IU</td>
</tr>
<tr>
<td>Pregnant and Lactating women</td>
<td>200 IU</td>
<td>1000 IU</td>
<td>2000 IU</td>
</tr>
<tr>
<td>51-70 y</td>
<td>400 IU</td>
<td>1000 IU</td>
<td>2000 IU</td>
</tr>
</tbody>
</table>
Different populations have different recommended interventions to ensure adequate intake of vitamin D. The elderly for example, may not comply with vitamin D supplementation. This may be due in part to polypharmacy and increased pill burden (21).

In addition, lactose intolerance is common, especially among minority groups, and increasing milk intake may not be advisable. Thus, vitamin D intake may be decreased in such cases (21).

As a result, some studies are being conducted to test the effectiveness of consuming 100,000 IU of vitamin D3 every four months. Positive results have been reported, which may be a better alternative for the populations described above (21).

For those who are hospitalized, studies have observed a deficiency of over 50% among this population. It has been suggested that those admitted to the hospital are screened upon admission and their vitamin D status assessed. Appropriate treatment should then follow, which may include counseling from the dietitian or routine supplementation (21).

For those who are pregnant, it is in question whether 400 IU of vitamin D, which is the amount contained in prenatal vitamins, is sufficient. Given current evidence, it would seem that this amount is insufficient. A more appropriate amount of supplementation may be 1000 IU. Screening for vitamin D deficiency in this population may be vital due to the effects that inadequate serum vitamin D levels may have on the developing fetus. Pregnant women should be screened at follow-up appointments that should be scheduled throughout pregnancy. It is advised that those at high-risk for
vitamin D deficiency, such as those with dark skin or who are often heavily clothed, be evaluated for deficiency more often (21).

In regards to the general population, many are at decreased risk for developing a severe vitamin D deficiency; however, they are often at increased risk for vitamin D insufficiency. Routine supplementation of 1000 IU per day of vitamin D may be sufficient to prevent insufficiency. This population should likely be screened for deficiency by their primary care physician annually at a minimum. If a deficiency is detected, the physician may advise higher supplementation of vitamin D or refer to a registered dietitian for nutritional advice (21).

Conclusion

In conclusion, it is my recommendation to increase current dietary vitamin D recommendations among all populations. From this literature review, it is apparent that vitamin D deficiency has a high prevalence among the US population and that interventions must be taken to ensure Americans receive adequate amounts in order to promote optimal health. Due to the lack of vitamin D containing foods, supplementation in general should be recommended. As has been suggested by Hickey and Gordon (21), 1,000 IU per day may likely be the best amount.
References


What is Vitamin D?

Vitamin D is a fat soluble vitamin that is converted into a hormone inside the body. Hormones relay messages to other cells in the body and tell them what to do. Without enough vitamin D, our risk of cancer, diabetes, heart disease, and bone problems increases.

Am I Deficient?

It is best to talk with your doctor to find out what your level of vitamin D is in your blood. If you do not have enough vitamin D, you may be given recommendations for increasing your vitamin D in your blood. The following chart will help you understand what your vitamin D number means.

<table>
<thead>
<tr>
<th>Vitamin D Blood Level</th>
<th>ng/mL</th>
<th>nmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficient</td>
<td>&lt; 20</td>
<td>&lt;50</td>
</tr>
<tr>
<td>Insufficient</td>
<td>21-29</td>
<td>52.5-72.5</td>
</tr>
<tr>
<td>Sufficient</td>
<td>≥30</td>
<td>&gt;75</td>
</tr>
</tbody>
</table>

Where Can I Get More?

Vitamin D is found in milk, fatty fish such as salmon and tuna, and some fortified grains and orange juices. Vitamin D supplements are also a great source. Studies suggest that the average adult needs about 1,000 IU per day from food or supplements.
Vitamin D is a fat soluble vitamin that is converted into a hormone inside the body. Hormones relay messages to other cells in the body and tell them what to do. Without enough vitamin D, our risk for cancer, diabetes, heart disease, and bone problems increases.

What is Vitamin D?

Aim for 1,000 IU each day of vitamin D from foods and/or supplements.

What Affects My Vitamin D Level?

- **Aging**—the older we get, the harder it is for our bodies to make vitamin D from sunlight and absorb vitamin D from our foods.
- **Skin Pigmentation**—People with darker skin absorb less UVB rays and thus produce less vitamin D from sunlight.
- **Sunlight Exposure**—if we aren’t in the sun much, our bodies cannot make as much vitamin D from sunlight.
- **Location**—where we live may decrease the amount of UVB rays needed to make vitamin D from the sun.
- **Inactivity and Obesity**—vitamin D is stored in your fat; in cases of obesity, it is hard for the body to retrieve stored vitamin D.
- **Diet**—increased intake of vitamin D rich foods like milk and fatty fish increase vitamin D levels.
- **Malabsorptive Disorders** (Cystic Fibrosis, Crohn’s disease, etc)—if dietary fat isn’t absorbed well in the body, vitamin D will not be absorbed well either.
- **Liver Dysfunction and Kidney Disease**—Both disorders make it harder to use vitamin D in the body
- **Exclusively Breastfeeding**—Infants who are exclusively breastfed need vitamin D supplementation because breastmilk has little vitamin D.