The Weston A. Price Foundation

# Vitamin D Supplementation: Panacea or Potential Problem?

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**ARTICLE SUMMARY**


- Vitamin D blood testing and oral supplementation have become an almost routine part of conventional medical care.
- Unfortunately, the results of a vitamin D blood test do not always reflect the true picture of an individual's vitamin D status, and whether or not supplementation is warranted and in what amount.
- There is a growing concern that the trend to aim for higher blood levels of vitamin D is not supported by the scientific evidence, and over time may contribute to calcification of the arteries, kidney stones and other health problems.
- Weston A. Price Foundation members will not be surprised by this. Our Foundation has always taught the critical importance of consuming all of the fat-soluble vitamins, as Dr. Price discovered and more recent research has confirmed.
- Various agency guidelines differ as to the optimum amount of dietary vitamin D. There are a number of limitations to testing, and interpretations of vitamin D levels are presented. Serum levels of 30ng/mL are adequate for preventing bone loss.
- Vitamins A, D and K2 work synergistically. Rich dietary sources of all three vitamins can enhance their health benefits while simultaneously eliminating both the need for testing and concern for potential over-supplementation.


Vitamin D has far and away been receiving the most attention of all the vitamins, both from the medical community and from health-conscious individuals. Since the new millennium, scientific research on the relationship of vitamin D to health and disease has exploded; for example, in 2015, 13% of the budget of the Office of Dietary Supplements was spent on

vitamin D research, with just 6% going to research the other twelve vitamins combined.[1] Population-wide "low-levels" have even been referred to as a "vitamin D deficiency pandemic."[2] Eighteen percent of Americans are now taking at least 1,000 International Units (IU) of vitamin D per day, up from 0.3 percent in 2000. More than three percent of adults are now taking amounts greater than 4,000 IU per day, up from 0.1% in 2007, a trend that is raising red flags in the medical community.[3,4]

Today nutritionally oriented practitioners routinely recommend vitamin D blood testing followed by supplementation, a trend that has been building over the last decade. Interest in testing surged in 2011 when the Endocrine Society published their recommendation that vitamin D levels should be no lower than 30 ng/mL.

Today even conventionally minded doctors are testing and prescribing vitamin D supplements. Medicare experienced an 83-fold increase in vitamin D blood tests from 2000 to 2010, and commercial health insurers saw a 2.5-fold increase from 2009 to 2014.[5] Having a robust vitamin D level has become akin to having a low cholesterol level, thought to be a marker of good health. Astute readers know that this modern-day quest for optimal health and longevity is fraught with risks.

As with any trend in medicine, many are now questioning the benefits of vitamin D testing. Medscape, an influential source for continuing medical education, recently advised doctors that vitamin D testing is both expensive and not fully reliable, encouraging supplements of 1,000 IU per day for adults as both safe and sufficient without the requirement for testing.[6] Health insurers are asked to cover the fifty dollar cost of the test, which they state for most cases is not medically necessary; some are now denying reimbursement, which is not surprising since the Institute of Medicine stated in their 2010 report on vitamin D, "[T]he measurements, or cut-points, of sufficiency and deficiency used by laboratories to report results have not been set based on rigorous scientific studies, and no central authority has determined which cut-points to use."[7]

**TYPICAL TESTING AND SUPPLEMENTATION PROTOCOLS**

Vitamin $D_3$ is the natural form synthesized in the skin upon exposure to UVB light, or consumed from animal-based foods; vitamin $D_2$ is the artificial or plant-derived form often

used in food fortification, supplements and pharmaceutical preparations. (See sidebar on page 32 for differences between vitamin $D_2$ and $D_3$, and problems with vitamin D supplements.) Both of these forms enter the bloodstream carried on a binding protein and then are immediately taken up by the liver for hydroxylation, or alternatively by the adipose tissue for storage. The liver hydroxylation of vitamin D yields 25(OH)D, or calcidiol, the major circulating form of vitamin D in the blood with a half-life of about three weeks; this is the form measured by the commonly ordered "vitamin D test." (Test results may be broken down by the amount of 25(OH) vitamin $D_3$ versus 25(OH) vitamin $D_2$, but more often only the sum of the two forms is reported.) Circulating 25(OH)D then undergoes a second hydroxylation either in the kidneys or in target tissues to produce the hormonally active form, 1,25(OH)2D, or calcitriol. Circulating 1,25(OH)2D can also be measured, but is most clinically useful in cases of kidney disease, or in diseases that may lead to excessive levels of vitamin D and/or calcium such as sarcoidosis or certain lymphomas.

If serum 25(OH)D comes back below or near the bottom of the laboratory reference range, doctors will typically prescribe vitamin $D_2$ at a dose of 50,000 IU per week for eight weeks. I've observed that after this initial treatment many patients will continue supplementation with an over-the-counter product containing 400 to 2,000 IU (occasionally as high as 5,000 IU) of vitamin $D_3$ per day, sometimes per the doctor's advice but more often the patient's own choosing. There does not always seem to be a clear rationale behind this practice; it may be what the patient finds at the drugstore either in a vitamin D supplement or in a multivitamin, or what the doctor generally recommends for patients. Nutritionally-oriented practitioners often recommend a daily vitamin $D_3$ supplement (usually between 2,000 and 10,000 IU per day); some are now recommending a vitamin $K_2$ supplement (100-200 mcg or more) as well. However, it is exceedingly rare for doctors to recommend food or supplements containing vitamin A in the form of retinol.

When I see a patient whose vitamin D test result is just below or near 20 ng/mL, I share with them the view that this level may not be optimal. Patients often respond, "Yes, my doctor told me that my vitamin D was really low." I assure them that levels in the 20s are not of great

concern, especially when tested in the winter. Then I advise them that supplementation to increase their levels to between 30 and 50 can be clinically appropriate, as long as we pay attention to all of the fat-soluble vitamins. This opens up a discussion about their need for vitamin A, especially when they have one or more signs of low vitamin A status: dry eyes, poor

night vision, keratinized bumps on their skin especially on the back of the upper arms, frequent respiratory infections and acne, confirmed by my review of their dietary intake. Even popular diets like the paleo diet and low-carb plans are often very poor sources of preformed vitamin A when patients don't include nourishing traditional foods.

My daughter Laura, also a registered dietitian, finds that many of her patients, like mine, are low in vitamin A. We both have seen remarkable health improvements in these individuals when a source of preformed vitamin A is provided.

Doctors' interpretations of vitamin D test results are not surprising—the lab reference ranges are typically 30-100 ng/mL, and in my experience, doctors now consider the ideal vitamin D level to be at least 50-60 ng/mL. Quest Diagnostics Labs suggests interpreting levels with this in mind: "The reference range for total 25(OH)D (20-100 ng/mL) is based on 25(OH)D correlation with physiological parameters that include parathyroid hormone [PTH] concentration and calcium absorption. The range is *not based* on the distribution of levels in an apparently healthy population" (emphasis added).[8]

With such wide laboratory reference ranges, what can we say with reasonable confidence about the level of 25(OH)D in the blood needed to optimize PTH and calcium absorption, and more importantly, to optimize overall health? Do higher levels reduce other health risks or improve problems such as cancer, autoimmune disease, heart disease or any other chronic condition? Do the benefits of higher levels outweigh any potential adverse effects? Are there potential limitations or problems with testing? Can blood levels of 25(OH)D be translated into rule-of-thumb supplementation guidelines? Is there a better way to evaluate our levels or should we even routinely test at all?

Before I offer answers to these compelling questions, we are wise to consider the practices of the healthy pre-industrial populations Dr. Price visited, populations that neither had knowledge of "vitamin D" (or any isolated vitamin) nor the "benefit" of blood testing.

**PRE-INDUSTRIAL POPULATIONS**

How did pre-industrial populations obtain vitamin D? While the discovery and isolation of vitamins in the early 20th century is considered a milestone in our understanding of nutrition

and health, humans have survived and thrived without the knowledge of which foods contained which vitamins. The populations studied by Price took great care to emphasize the consumption of foods that are now recognized as excellent sources of all the fat-soluble vitamins, including vitamin D: deep yellow butter, seafood including fish eggs, organ meats, insects and animal blood.[9] In addition, routine sun exposure during the activities of daily life resulted in significant skin vitamin D synthesis. The modern practice of hours spent "sunbathing" did not exist (see sidebar on page 33).

There are challenges in obtaining adequate amounts of vitamin D from sun exposure and diet alone. Today individuals are often genetically "mismatched" to the climate where they live. (Genetic adaptations require many successive generations.) There is a marked reduction in cutaneous vitamin D biosynthesis in northern latitudes during the winter months, and virtually none above 40 degrees.[10] Darker-pigmented individuals can require five to ten times the duration of sun exposure to produce the same amount of vitamin D as lighter-pigmented persons. With the need to cover the skin during cold weather, it is not unexpected that vitamin D levels can plummet during the winter, made worse by limited summer sun exposure, which can lead to year-round low vitamin D levels.

Contemporary habits of applying and reapplying sunscreens (SPF 8 or greater) can significantly reduce skin vitamin D synthesis. Showering shortly after outdoor activities, and swimming in chlorinated pools may also reduce the quantity of vitamin D our bodies make. Age is a factor too; adults older than seventy need about three times the duration of sun exposure to produce the same amount of vitamin D as children. Non-solar sources of vitamin D are essential in these cases, just as populations living near the poles consumed vitamin D-rich foods as part of their native diet: the organs of ocean fish especially the liver and the fish liver oils, and seal oil and whale blubber (also rich sources of vitamin A!).[11]

**HOW MUCH VITAMIN D?**

What amount of oral vitamin D is required when sun exposure is inadequate? Clearly, many individuals are less able to obtain vitamin D from sun exposure, and modern low-animal fat diets do not provide much naturally occurring vitamin D.[12] Following today's conventional approach of paying attention to only vitamin D intake, this problem appears to be fairly

simple to address: use the results of the most recent blood test, supplement accordingly, then retest and adjust the dosage as needed. Selecting a blood level of 25(OH)D to target then becomes the first decision—not always so straightforward though. For example, if the result is between 20-30 ng/mL, this is, according to the Institute of Medicine, "sufficient,"[13,14] according to the Endocrine Society "insufficient,"[15] and according to the Vitamin D Council "deficient"[16] (see chart below). Interpreting the tests depends on which health agency a practitioner or individual relies upon.[17]

The next step would be to choose a dose of vitamin D that is both safe and effective. As stated before, it is common to prescribe or recommend supplementation when a patient's levels are below 30 ng/mL, in accordance with guidelines from the Endocrine Society and the Vitamin D Council. Some practitioners may choose to be conservative with a dose of 1,000 IU per day, especially if they don't expect routine follow-up visits or testing by their patient; 2,000-4,000 IU per day might be chosen if the patient tests below 30 ng/ml and does not get adequate sun exposure; 5,000-10,000 IU per day or more for short term or if the patient will be retested in a few months; even up to 10,000 IU long term, which according to the Vitamin D Council, should not present a risk of toxicity. Rule-of-thumb supplementation has been suggested as follows: to raise serum 25(OH)D by approximately 1 ng/mL, supplement with 100 IU oral vitamin D daily. However, individual responses to standard dosages often vary considerably[18] – several caveats are discussed below.

**DESIRABLE VITAMIN D LEVELS**

Despite a lack of consensus, what appears to be a desirable vitamin D level? Vitamin D, in the activated form of 1,25(OH)2D, acts on many different cells in the body both through calcemic roles, regulating calcium and phosphate concentrations, and noncalcemic roles. Noncalcemic roles include cell differentiation and antiproliferative actions in a wide range of cell types such as the immune, muscle and gastrointestinal systems.[19] Research has shown inverse associations between vitamin D levels (regardless of sun exposure) and the incidence of several diseases, pointing to roles in the prevention of heart disease, cancer, diabetes,

autoimmune disease, neuromuscular impairment and more.[20] The evidence to support an optimum range of serum 25(OH)D for health outcomes is strongest when considering the impact of vitamin D on bone health.[13] Calcium homeostasis is achieved by the influence of 1,25(OH)2D on calcium absorption, uptake and release from bone tissue and excretion from

the kidneys. Adequate levels of 25(OH)D are needed to ensure adequate synthesis of 1,25(OH)D, important for bone mineralization and general muscle and bone health. Severe vitamin D deficiency can result in hypocalcemic seizures and weak or misshapen bones—rickets in growing children, osteomalacia and osteoporosis in adults.

According to expert opinions, the minimum level of serum 25(OH)D needed for bone fracture prevention ranges between 20-32 ng/mL with desirable 25(OH)D concentrations between 28-32 ng/mL. It is estimated that the average older man and woman will need intakes of at least 800 to 1,000 IU per day of vitamin D3 to reach a serum 25(OH)D level of 30 ng/mL.[21] In addition, vitamin D supplementation at 600-800 IU per day has been shown in studies to reduce fracture risk when calcium is also supplemented.[22] Caution with higher levels accompanied by calcium supplementation is advised: according to the Merck Manual, "target 25(OH)D levels are > 20 to 24 ng/mL for maximal bone health; whether higher levels have other benefits remains uncertain, and higher absorption of calcium may increase risk of coronary artery disease."[23]

The efficiency of calcium absorption in the small intestine increases with higher levels of serum 25(OH)D, reaching a plateau at 32 ng/mL.[24] Some studies suggest that the lower end of the IOM reference range for 25(OH)D, 20 ng/mL, may not optimize calcium absorption, and that levels of at least 32 ng/mL may be required.[25]

Bone mass density (BMD) has been positively associated with 25(OH)D levels, as one might expect. In the Multi-Ethnic Study of Atherosclerosis involving 1,773 adult participants, this association was significant for Caucasian and Asian participants alone; in Hispanics there was a non-statistically significant association. An inverse association was actually found in Black participants: those having 25(OH)D levels less than 20 ng/mL had higher BMD than those with levels greater than 30 ng/mL. Even more surprising may be the finding that 25(OH)D levels were highest among Whites and lowest among Blacks, but BMD was highest among Blacks. The IOM states that "African-Americans present a conundrum, because, although their serum values are lower than those of white counterparts, their rates of osteoporosis and fractures are lower than Caucasians."[22] Thus it appears that when interpreting blood test results, racial differences should be considered as they may influence recommendations for vitamin D supplementation.[26]

The level of 25(OH)D required for maximum suppression of parathyroid levels is also an indicator of vitamin D requirement. Chronic elevations of serum PTH increase osteoclast (bone cell breakdown) activity, negatively affecting bone density. Besides low vitamin D levels, low dietary calcium, skeletal muscle wasting, primary or secondary hyperthyroidism, and chronic kidney disease can all be independent contributors to high serum PTH concentrations.[27] The level of 25(OH)D that maximally suppresses parathyroid hormone blood levels is somewhere around 32 ng/mL for adults.[28,29] A meta-analysis of available clinical trials indicates that vitamin D supplementation of 1000 IU per day can best suppress serum PTH levels.[27]

While many studies suggest extraskeletal benefits, overall the evidence does not strongly support the use of supplementation with vitamin D to address the myriad of diseases that have been linked to low serum 25(OH)D levels.[30] At the same time, a systematic review of studies showed that supplementation using vitamin $D_3$ alone (but not $D_2$) reduced all-cause mortality by 11 percent.[31] Being deficient in vitamin D is not good for one's health, but in the medical community the jury is out on whether levels above those needed for bone health offer clear benefits.

**MISSING PIECES OF THE PUZZLE**

Any student who has taken an introductory nutrition course learned that the fat-soluble vitamins, and especially vitamins A and D, can produce toxicity at high levels of intakes. The IOM has set the Tolerable Upper Intake Level for vitamin D at 4,000 IU per day for adults,[5] identical to the amount the Geriatric Society generally recommends to support seniors' bone and muscle strength.[8] The Vitamin D Council states that toxicity is highly unlikely until intakes are above 10,000 IU per day for several months.[32] Why do the expert opinions vary, and what risks might there be for higher-dose vitamin D supplementation?

Becoming acutely toxic from vitamin D is thought to be extremely difficult, identified by a marked elevation of 25(OH)D levels (> 150 ng/mL) in conjunction with elevated blood calcium and high normal or elevated serum phosphorus, and clinically accompanied by symptoms such as constipation, confusion, fatigue, and increased thirst and urination.[33] When levels

exceed 600 ng/mL symptoms progress to pain, anorexia, fever, chills, vomiting and weight loss, requiring complete avoidance of sun exposure and many months to resolve.[34] With chronic vitamin D excess, levels above 50-60 ng/mL rarely raise blood calcium, yet an early indicator is a substantially increased risk for kidney stones, and over time heart disease risk also increases.[35] In animal models bone loss and blood vessel calcification or "hardening" are the key pathologies. Toxicity is heightened by a rich dietary supply of calcium and phosphorus, so removal of dietary calcium is important. Conversely, toxicity is reduced by high intakes of vitamin A.[36] Moderate sun exposure is much less likely to lead to toxicity because excess vitamin $D_3$ is photodegraded into products that have no calcemic activity.[37] On the other hand, excessive sun exposure has been linked to a twenty-fold increase in kidney stones.[38]

SERUM (BLOOD) 25(OH)D TEST RESULTS

| Organization & Recommendation | < 20 ng/mL | ≥ 20 ng.mL < 30 ng/mL | ≥ 30 ng/mL < 40 ng/mL | ≥ 40 ng/mL < 50 ng/mL | > 50 ng/mL | Considered Ideal |
|---|---|---|---|---|---|---|
| Institute of Medicine | Deficient | SUFFICIENT | | | Toxicity Possible | |
| Endocrine Society | Deficient | Insufficient | SUFFICIENT | | | 40-60 |
| Vitamin D Council | Deficient | Deficient | Deficient | Insufficient | SUFFICIENT | 50-80 |

Dr. Price's detailed observations give us solid clues as to why the results of modern-day studies do not support the practice of supplementation of high doses of vitamin D alone. The trio of fat-soluble vitamins A, D and $K_2$ (Price's x-factor) are best derived from nourishing foods where they occur in natural balance, along with sensible sun exposure. The dietary wisdom of native peoples far surpasses that of modern medicine. Most doctors, dietitians and nutritionists are unfortunately still unaware that vitamins A, D and $K_2$ work together to produce and activate proteins that direct calcium to the bones and away from soft tissues, one of their many vital and varied roles in the body.

Additional clues surfaced around the middle of the 20th century when researchers demonstrated that vitamin A and vitamin D when administered together were substantially more effective at protecting against infections like colds than when given alone,[39] and that even very high doses of both over a course of three years did not result in toxic effects when

administered together. Furthermore, even modest amounts of vitamin D have been shown to deplete vitamin A, whether provided by sunlight or injection.[40]

In a 2006 paper published in *Medical Hypothesis*,[41] Chris Masterjohn, PhD, explains how it is likely that vitamin D will exert toxic effects when vitamin K and vitamin A are in short supply. It is known that higher intakes of vitamin D lead to "hypervitaminosis D," causing lethargy, growth retardation, bone resorption and soft tissue calcification, as observed in animal studies. While elevated levels of blood calcium are considered the hallmark cause of the toxic effects of too much vitamin D,[42] Masterjohn points out that these adverse effects can occur in the absence of elevated blood calcium. He proposes that vitamin D's toxic effect is primarily the result of higher levels leading to a deficiency of vitamin K and postulates that patients can be given higher doses of vitamin D, potentially offering greater therapeutic value, by administering vitamins A, D and K simultaneously.[41] It is hoped that future studies will evaluate their interactions, in order to better treat patients, and support public health efforts, as recently proposed by the Weston A. Price Foundation to the National Institutes of Health[43] as the agency considers the Strategic Nutrition Research Agenda.

As stated before, more up-to-date practitioners are now giving patients vitamin $K_2$ along with vitamin D, and several supplement manufacturers have developed formulations that combine both D and $K_2$, but no A. One company, Allergy Research Corporation, does make a supplement containing vitamins A, D and $K_2$. Time-tested cod liver oil containing natural vitamins along with nourishing traditional foods is still ideal, due to the known presence of other naturally-occurring nutrients, along with those that we have yet to understand.

**MANAGING VITAMIN D**

Do we need to closely manage serum 25(OH)D levels? How should we supplement if we don't test? As stated earlier, ordering vitamin D blood testing has become fairly routine. In my experience, follow-up testing may also be done after vitamin D supplementation is prescribed. But are these tests needed, and if so, how often?

I am actually in favor of doing baseline vitamin D testing for many patients in an effort to confirm suspicions that they could be grossly deficient in vitamin D; or conversely, taking too much supplemental vitamin D, which is not uncommon. After a moderate course of supplementation with 1,000-2,000 IU per day, or the complete discontinuance of vitamin D in

some cases, retesting after six to twelve months may determine whether the patient is on target, with blood levels of 25(OH)D somewhere between 30 and 50 ng/ml, lower in winter, higher in summer. Tests are not without limitations (see sidebar, page 36)—if we supplement too aggressively we may "overshoot" ideal levels of vitamin D or unknowingly cause adipose stores to rise to excessive levels. As we have seen, blood levels may not necessarily reflect the body's total vitamin D stores or status.

Dr. Alan Gaby, author of "Nutritional Medicine"[52] warns that the safety and efficacy of long term vitamin D supplementation with more than 2,000 IU per day for the purpose of achieving a target 25(OH)D level has not been established. Dr. Pamela Lutsey, lead researcher for a recent study on vitamin D supplementation, concurs that higher intakes can be dangerous leading to the overabsorption of calcium and subsequent deposition in soft tissue such as the heart and kidneys.[3,4] In the shorter term, amounts over 2,000 appear to be reasonable. If a practitioner decides to forgo vitamin D testing, a safe and effective daily dose of vitamin D is likely between 800 and 1,200 IU. Individuals who are supplementing on their own are cautioned not to exceed that, especially if sun exposure is habitual or if fortified foods are consumed.

These approaches represent a medicalized approach to nutrient sufficiency, applicable in some cases. However, a more safe, effective and sustainable approach would be one that considers the full trio of fat-soluble vitamins and how they work synergistically together, both to support each other's functions and protect from the risk of toxicity. Eating an array of delicious, nourishing foods that lead to a natural balance of vitamin D with its partners, vitamins A and $K_2$, and getting outdoors on a regular basis are good for our mental and physical health. Our diets should include a range of pastured animal foods and wild seafoods: egg yolks, butter and cheese, organ meats, whole fish and shellfish, and animal fats such as lard; like us, animals obtain vitamin D from the sun and store it in their bodies and in their fat. The addition of a high-vitamin cod liver oil is highly recommended too. This will remove the need for repeated vitamin D testing and eliminate all worry of potential toxicity from too much vitamin D. By following the wisdom of our ancestors, our quest for optimal health can be risk-free.

**SIDEBARS**

**MY PERSONAL EXPERIENCE**

My introduction to the importance of vitamin D came at my first Wise Traditions conference in 2001 where I learned about Dr. Weston A. Price's research, which clearly demonstrates the unsurpassed value of nourishing traditional diets for reproduction, growth and health at all stages of life. The conference focused on the vital importance of all of the fat-soluble vitamins, not just vitamin D alone, but in conjunction with vitamin A and Dr. Price's "x-factor," subsequently identified in 2008 by Chris Masterjohn as vitamin $K_2$.[1] One of Price's key findings strongly resonated with me: the quantity of fat-soluble vitamins in traditional diets was ten times higher than that of typical American diets. I knew this was a missing piece in what I thought was my "healthy" semi-vegetarian diet.

I immediately began to add cod liver oil to my and my family's diet, along with full-fat raw dairy, pastured eggs and liver pâté, excellent sources of all four fat-soluble vitamins, A, D and $K_2$. Having always enjoyed beef liver and onions as a child, this was a welcome addition to my own diet. I feel immensely grateful that I did not just start supplementing with vitamin D alone, as I had recently learned that I am genetically a very poor converter of carotenes to true vitamin A, not an uncommon trait in people of my ancestry. In a short period of time, we noticed improvements in our health: freedom from colds and infections like strep throat, clearer skin for my teenagers, and for me, resolution of my chronically dry eyes. Through membership in the Weston A. Price Foundation, I continued to learn much more about the roles of the fat-soluble vitamins, information not available through educational materials directed to registered dietitians or functional medical practitioners. In 2016, concerned about the widespread problem of inadequate intakes among women, I wrote on the necessity of vitamin A before, during and after pregnancy.[2]

My own health care providers now routinely order vitamin D testing. My two most recent results were exactly the same, 37 ng/mL, yet the two nurse practitioners who had ordered them had very different interpretations. One said "your vitamin D is way too low;" the second made no comment, which generally indicates that the results are "unremarkable," or not of

concern. Interpretation of vitamin D test results is still quite variable among practitioners. Never have I had a doctor or nurse ask about vitamin A or $K_2$ intake or levels, not atypical in medical practice.

My vitamin D levels over the past several years have ranged between the mid-30s and mid-50s, lower in winter, higher at the end of the summer. The results of a baseline bone mass density scan I had in 2007 were well above the mean—my doctor ordered it because she thought I would be at higher risk for osteoporosis since I am small-boned, relatively thin and Caucasian. I feel good about what I have been doing for the past seventeen-plus years: reasonable sun exposure, foods rich in vitamins A, D and $K_2$, raw and fermented dairy to ensure I am getting easily-absorbed sources of calcium on a daily basis, bone broth for collagen and minerals, and tapering off of supplements containing vitamin D as summer approaches.

1. https://www.westonaprice.org/health-topics/abcs-of-nutrition/on-the-trail-of-the-elusive-x-factor-a-sixty-two-year-old-mystery-finally-solved/

2. Vitamin A: The Scarlet Nutrient. The Unfair Stigmatization of Vitamin A during Pregnancy. https://www.westonaprice.org/health-topics/abcs-of-nutrition/vitamin-a-the-scarlet-nutrient/.

**$D_2$ vs. $D_3$?**

Some doctors prescribe vitamin $D_2$, others recommend an over-the-counter supplement that contains vitamin $D_3$. What is the difference? Vitamin $D_2$ is the plant form, ergosterol, found in foods like mushrooms. Vitamin $D_3$ is the animal form, cholecalciferol, made in our skin but also available from animal fats. The pharmacokinetic properties of vitamins $D_2$ and $D_3$ differ, with more consistent and higher serum concentrations of 25(OH)D after vitamin $D_3$ supplementation. Clinicians are advised to recommend vitamin $D_3$ supplementation at intervals of four months or less. Longer intervals between doses of vitamin $D_2$ will result in large fluctuations of serum 25(OH)D concentrations (due to more rapid metabolic degradation via 24-hydroxlation and a lesser affinity to the vitamin D-binding protein[1]), therefore the dosing interval with vitamin $D_2$ is should not exceed fourteen days. The term “calciferol” on a preparation refers to vitamin $D_2$ and “cholecalciferol” to vitamin $D_3$.

While the use of vitamin $D_3$ is generally preferred, a 2013 *JAMA* study found that many of the over-the-counter vitamin $D_3$ supplements on the market do not contain the amount the label indicates, with the authors more concerned about the fact that they provided too little rather than too much $D_3$. In any case, only trusted sources should be used if vitamin D is supplemented in isolation.

If your choice for a vitamin D source is a fermented cod liver oil, keep in mind that all natural cod liver oils contain an array of different metabolites derived from vitamin $D_3$, with a diverse array of biological activities. While magnetic resonance analysis seems to indicate the presence of vitamin $D_2$, it is likely a mixture of these metabolites. Furthermore, the benefits of natural cod liver oil are often realized without a marked rise in serum 25(OH)D. Masterjohn cautions that this blood test is not only being overused as an indicator of vitamin D nutritional status, but is "being used in an overly simplistic manner."[4]

1. Zerwekh JE. Blood biomarkers of vitamin D status. Am J Clin Nutr. 2008;87:1087S-91S.

2. American Geriatrics Society Workgroup on Vitamin D Supplementation for Older Adults. Am Geriatr 2014; 62:147–152.

3. http://www.webmd.com/vitamins-and-supplements/news/20130211/vitamin-d-supplements-is-what-you-see-what-you-get#1

4. https://www.westonaprice.org/health-topics/cod-liver-oil/vitamin-d-in-cod-liver-oil/.

**VITAMIN D FROM SUNLIGHT**

Masterjohn has described the practices of healthy pre-industrial societies regarding sun exposure including seeking shade in the midday and using a variety of methods to protect the skin when in the sun, such as primitive forms of sunscreen like coconut oil, body paint and

protective clothing. He has also discussed findings which indicate that genetic adaptations direct an increased synthesis of vitamin D in northern indigenous populations, and conversely, an increased degradation of vitamin D in southerly populations.

Indigenous populations are innately adapted to their native climate, allowing their bodies to better control vitamin D production in accordance with their needs. Furthermore, his analysis of the evidence indicates that it does not support the conclusion that levels of 40-60 ng/mL for vitamin D are “natural;” people outside of tropical and subtropical regions may not be genetically adapted to having vitamin D levels so high.

SOURCE: http://www.westonaprice.org/our-blogs/cmasterjohn/vitamin-d-problems-with-the-latitude-hypothesis/.

**VITAMIN D DAILY INTAKE GUIDELINES**

INSTITUTE OF MEDICINE: Recommended Dietary Allowance[1,2,3]
(meets needs of 97.5% population, assumes minimal or no sun exposure)
Adult 600 – 800 IU (age-dependent); Tolerable Upper Intake Level: 4,000 IU
Child 400 – 600 IU (age-dependent); Tolerable Upper Intake Level: 1,000 – 4,000 IU

The 4,000 Upper Level still includes a large safety margin, at least based on acute toxicity symptoms of hypercalcemia.[4]

Based on IOM guidelines, the CDC estimates 8 percent of Americans are deficient in vitamin D, and another 24 percent have inadequate vitamin D levels.[2]

ENDOCRINE SOCIETY: Daily dose (minimum) needed to raise above baseline of 30 ng/mL[5]
Adult 1,500-2,000 IU
Child 1,000 IU

AMERICAN GERIATRIC SOCIETY[6]
Adult at least 1,000 IU, majority of older adults require 4,000 IU (diet and oral)

NATIONAL OSTEOPOROSIS FOUNDATION[7]
Adult 18-49 years 400-800 IU
Adult 50 years and older 800-1,000 IU

VITAMIN D COUNCIL: Daily dose needed to raise from 25 to 50 ng/mL[8]
Adult (150 lbs.) 3,700 IU
Toxicity possible if taking more than 10,000 IU per day for 3 months or longer.

1. Dietary Reference Intakes for Calcium and Vitamin D. Available at: http://www.nationalacademies.org/hmd/~/media/Files/Report%20Files/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D/Vitamin%20D%20and%20Calcium%202010%20Report%20Brief.pdf

2. In 2011, the IOM updated their vitamin D guidelines based on “nearly 1,000 published studies as well as testimony from scientists and stakeholders.” Noting the existence of many studies on vitamin D’s benefits, “such as protection against cancer, heart disease, autoimmune diseases, and diabetes,” they judged that only the evidence on bone health was strong enough to base vitamin D recommendations on. http://www.livescience.com/42481-vitamin-d-supplement-facts.html

3. Due to “emerging concerns about elevated 25(OH)D, the IOM has shifted the paradigm from thinking about ‘more is better’ to a more risk-averse approach. Because adverse effects of vitamin D supplementation may take decades to be realized, clinicians (mindful of the medical ethics precept ‘First, do no harm’) should err on the side of caution; follow the IOM guideline and wait for the results of long-term vitamin D studies.” Mangin M, Sinha R, Fincher K. Inflammation and vitamin D: the infection connection. Inflamm Res. 2014; 63(10): 803–819.

4. Jones, G. Vitamin D. In Ross AC et al editors. Modern Nutrition in Health and Disease, 11th ed. Philadelphia: Lippincott, Williams & Wilkins; 2014.

5. Holick B et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2011;96:1911-1930.

6. American Geriatrics Society Workgroup on Vitamin D Supplementation for Older Adults. J Am Geriatr Soc. 2014;62:147–152.

7. Cosman F et al. Clinician’s Guide to Prevention and Treatment of Osteoporosis. Osteoporos

Int. 2014;25(10):2359-81.

8. www.vitamindcouncil.org/about-vitamin-d/?-am-i-getting-too-much-vitamin-d/

**ARE VITAMIN D TESTS RELIABLE?**

Can we be confident that our 25(OH)D test results represent what is going on in the body?

In this article we have made a broad assumption, namely that we can and should rely on the 25(OH)D blood test to accurately reflect the body's level of circulating vitamin D, then use this to make a personal or clinical decision on how much vitamin D to supplement, or whether to increase the consumption of vitamin-D rich foods or time spent in the sun. In many cases, this assumption is likely a good one. However, exceptions have been found in individuals who have obesity, certain genetic variations, and/or acute or chronic inflammatory disease, raising questions on the broad applicability of the 25(OH)D test. In addition, some of the laboratory protocols used to test vitamin D have inherent limitations.

The blood test for 25(OH)D measures only the hydroxylated forms of vitamin $D_3$ and $D_2$ that circulate in the bloodstream. While considered the best test for vitamin D, is it a reliable indicator of whole body vitamin D status? Unfortunately, not always. It does not measure the amount of vitamin D itself (in the non-hydroxylated form) that is stored in the body fat, which for some people may be considerable. Obese persons have lower blood levels of vitamin D, yet at the same time can have considerable quantities stored in their body fat. For example, one study found after taking a supplement of 20,000 IU of vitamin $D_3$ once per week for 3-5 years, serum 25(OH)D was 39.6 ng/mL versus 24.8 ng/mL in the placebo group, but abdominal fat contained 209 ng/g of vitamin $D_3$ versus 32 ng/g in placebo group. Assuming that the 209 ng/g concentration was the same in all body fat, the supplemented group had an average of 264,000 IU stored vitamin D in their bodies.[44] Whether this presents a problem is unknown, but there is the potential for stored vitamin D to be released during even modest weight loss.[22]

GENETIC VARIATION

Serum 25(OH)D levels are influenced by a number of genes that govern the vitamin D binding and receptor proteins and the enzymes that hydroxylate vitamin D. Increases in serum

25(OH)D with vitamin D supplementation vary according to common differences (polymorphisms) in these genes, with potential implications for vitamin D-related health outcomes.[45] The differences in 25(OH)D levels attributed to polymorphisms has been estimated to range from 23 to 77 percent, rivaling differences due to sun avoidance, and may be related to the season of the year.[46] This is an emerging area of research, but it appears that in those affected, serum 25(OH)D may actually "overestimate" actual vitamin D intakes from food or sunlight.[10]

## INFLAMMATION AND INFECTION

Multiple observational studies have reported a correlation of vitamin D deficiency with inflammation and inflammatory diseases, however cause and effect has yet to be conclusively demonstrated for the majority of these relationships. For example, a review of several clinical trials of vitamin D supplementation with overweight or obese individuals found no overall improvement in inflammatory markers.[47] Evidence has emerged to suggest another hypothesis—that low 25(OH)D is a result of the chronic disease process, provoked by a chronic bacterial infection.[48]

## LABORATORY TEST VARIABILITY

Between different laboratories, substantial variations in results for serum 25(OH)D have been reported.[49] The Office of Dietary Supplements of the National Institutes of Health has instituted the Vitamin D Standardization Program (VDSP), a collaborative venture of several governmental and non-governmental organizations, to address this problem.[50] It has been suggested that the preferred assay is mass spectrometry for a number of reasons, but costs and high levels of expertise required are barriers to its availability. In the future, the CDC plans to post on their website a list of laboratories that meet the VDSP certification standards.

## INTERFERING COMPOUNDS

Many of the commonly available 25(OH)D laboratory tests are unable to distinguish true 25(OH)D from related forms called epimers and isobars. In some individuals, especially those with autoimmune diseases, there can be relatively high amounts of these forms leading to a high degree of uncertainty on what is actually being measured by the blood test.[51]

**REFERENCES**


1. http://dcift.org/wp-content/uploads/2017/02/NIH-Initiatives-in-Nutrition-Roadmap-Strategic-Plan%E2%80%A6.pdf (http://dcift.org/wp-content/uploads/2017/02/NIH-Initiatives-in-Nutrition-Roadmap-Strategic-Plan%E2%80%A6.pdf)

2. Hossein-nezhad A, Holick M. Vitamin D for heath: a global perspective. Mayo Clin Proc. 2013;88(7):720-755.

3. Rooney MR et al. Trends in use of high-dose vitamin D supplements exceeding 1000 or 4000 International Units daily. J*AMA*. 017;317(23):2448-2450.

4. https://www.reuters.com/article/us-health-vitamind-supplements-idUSKBN19B2F0 (https://www.reuters.com/article/us-health-vitamind-supplements-idUSKBN19B2F0)

5. Kolata G. "Why are So Many People Popping Vitamin D?" New York Times, 4/10/2017. Available at: https://www.nytimes.com/2017/04/10/health/vitamin-d-deficiency-supplements.html?_r=0 (https://www.nytimes.com/2017/04/10/health/vitamin-d-deficiency-supplements.html?_r=0). Accessed 5/20/2017.

6. "Low Vitamin D Concerns Lead to Overtesting and Overtreatment," http://www.medscape.com/viewarticle/876655 (http://www.medscape.com/viewarticle/876655).

7. Dietary Reference Intakes for Calcium and Vitamin D. Available at: http://www.nationalacademies.org/hmd/~/media/Files/Report%20Files/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D/Vitamin%20D%20and%20Calcium%202010%20Report%20Brief.pdf

(http://www.nationalacademies.org/hmd/~/media/Files/Report%20Files/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D/Vitamin%20D%20and%20Calcium%202010%20Report%20Brief.pdf)

8. http://www.questdiagnostics.com/home/physicians/testing-services/by-test-name/vitamind (http://www.questdiagnostics.com/home/physicians/testing-services/by-test-name/vitamind)

9. Price WA. *Nutrition and Physical Degeneration*. 6th ed. Los Angeles CA. Keats Publishing; 1998.

10. Combs GF. *The Vitamins*. 4th ed. London UK. Academic Press; 2012. This however may be less of a factor than commonly believed: http://www.westonaprice.org/our-blogs/cmasterjohn/vitamin-d-problems-with-the-latitude-hypothesis/ (http://www.westonaprice.org/our-blogs/cmasterjohn/vitamin-d-problems-with-the-latitude-hypothesis/).

11. Anderson S et al. Vitamin D status in Greenland is influenced by diet and ethnicity: a population-based survey in an Arctic society in transition. *Br J Nutr*. 2013;109(5):928-935.

12. Fatty fish such as wild salmon, are a good source of vitamin D, milk must be fortified, and other foods such as orange juice can be fortified with D2 or D3. The FDA will require all food and beverage labels to list vitamin D content as of July 26, 2018. This is expected to increase the number of foods fortified with vitamin D.

13. The RDA as set by the Institute of Medicine is based on the amount of vitamin D needed by 97.5% of the population to reach a threshold for 25(OH)D of 20 ng/mL for normalization of bone health. Jones, G. Vitamin D. In Ross AC et al editors. *Modern Nutrition in Health and Disease*, 11th ed. Philadelphia: Lippincott, Williams & Wilkins; 2014.

14. The Institute of Medicine is the most highly regarded authority of these and critically reviewed the widest breadth of studies, coming to the most conservative opinion on the evidence. The IOM disagreed with the Endocrine Society's conclusions on three major points, stating: 1. 25(OH)D levels of 30 or higher do not provide health benefits over 20 ng/mL; 2. All persons are not deficient at levels below 20; and 3. Large subgroups characterized by the Society as at risk are not actually at risk. They concluded that "it would be difficult to justify billions in health care dollars that would be required as well as the administration of high doses of vitamin D in the absence of data to demonstrate safety." Rosen CJ et al. IOM Committee Members Respond to Endocrine Society Guideline. *J Clin Endocrinol Metab.*

2012;97(4):1146-1152.

15. Holick B et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2011;96:1911-1930.

16. www.vitamindcouncil.or/i-tested-my-vitamin-d-level-what-do-my-my-results-mean/ (http://www.vitamindcouncil.or/i-tested-my-vitamin-d-level-what-do-my-my-results-mean/)

17. "The most definitive way to determine the ideal 25(OH)D level would be to conduct a randomized, controlled trial with different levels of vitamin D supplementation targeted at reaching specific blood levels of 25(OH)D and to test the effects of the different levels of supplementation on clinical outcomes, such as bone mineral density, fracture rate, insulin resistance, glucose tolerance, cancer, or heart disease. We do not yet have this type of data. We do, however, have some strong support for raising 25(OH)D levels to at least 35 ng/mL.." https://www.westonaprice.org/our-blogs/are-some-people-pushing-their-vitamin-d-levels-too-high/ (https://www.westonaprice.org/our-blogs/are-some-people-pushing-their-vitamin-d-levels-too-high/)

18. Cronin SC. The Dual Vitamin D Pathways: Considerations for adequate supplementation. *Nephr Nurs J.* 2010:37(1):19-28.

19. Jones, G. Vitamin D. In Ross AC et al editors. *Modern Nutrition in Health and Disease,* 11th ed. Philadelphia: Lippincott, Williams & Wilkins; 2014.

20. Combs GF. *The Vitamins*. 4th ed. London UK. Academic Press; 2012.

21. Dawson-Hughes B et al. Estimates of optimal vitamin D status. *Osteoporos Int*. 2005;16(7):713-716.

22. Rosen CJ et al. IOM Committee Members Respond to Endocrine Society Guideline. *J Clin Endocrinol Metab*. 2012;97(4):1146-1152.

23. http://www.merckmanuals.com/professional/nutritional-disorders/vitamin-deficiency,-

dependency,-and-toxicity/vitamin-d (http://www.merckmanuals.com/professional/nutritional-disorders/vitamin-deficiency,-dependency,-and-toxicity/vitamin-d)

24. Heany R. Vitamin D and calcium interactions: functional outcomes. *Am J Clin Nutr*. 2008; 88(2): 541S-544S.

25. Heaney RP, Dowell MS, Hale CA, Bendich A. Calcium absorption varies within the reference range for serum 25-hydroxyvitamin D. *J Am Coll Nutr*. 2003;22(2):142-146.

26. Van Ballegooijen AJ et al. Vitamin D metabolites and bone mineral density: The multi-ethnic study of atherosclerosis. *Bone*. 2015;78:186-193.

27. Lotito A et al. Serum Parathyroid Hormone Responses to Vitamin D Supplementation in Overweight/Obese Adults: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. Nutrients. 2017; 9:241.

28. Heaney RP, Dowell MS, Hale CA, Bendich A. Calcium absorption varies within the reference range for serum 25-hydroxyvitamin D. *J Am Coll Nutr*. 2003;22(2):142-146.

29. The IOM states that “available evidence shows that PTH values decline to a plateau at different levels of serum 25(OH)D, ranging between 15 and 50 ml. This varies widely among individuals and appears to be dependent on age, race, ethnicity, body composition, renal function, and geographic locations.”

30. Pfotenhauer KM, Shubrook JH. Vitamin D deficiency, its role in health and disease, and current supplementation recommendations. *J Am Osteopath Assoc*. 2017;177:301-305.

31. Chowdhury R et al. Vitamin D and risk of cause specific death: systematic review and meta-analysis of observational cohort and randomised intervention studies. *BMJ*. 2014;348.

32. https://www.vitamindcouncil.org/about-vitamin-d/am-i-getting-too-much-vitamin-d/ (https://www.vitamindcouncil.org/about-vitamin-d/am-i-getting-too-much-vitamin-d/).

33. Holick MF. Vitamin D. In Shils ME et al editors. *Modern Nutrition in Health and Disease*.

10th ed. Philadelphia: Lippincott, Williams & Wilkins; 2006.

34. Hathcock JN, Shao A, Vieth R, Heaney R. Risk Assessment for Vitamin D. *Am J Clin Nutr*. 2007;85:6-18.

35. https://www.westonaprice.org/our-blogs/vitamin-d-problems-with-the-latitude-hypothesis/ (https://www.westonaprice.org/our-blogs/vitamin-d-problems-with-the-latitude-hypothesis/)

36. Horst RL, Reinhardt TA, Reddy SG. In Pike JW, Glorieux FH, Feldman D editors. *Vitamin D,* 2nd edition, Academic Press, pp.15-36. Available at https://www.researchgate.net/publication/235939182_Vitamin_D_Metabolism (https://www.researchgate.net/publication/235939182_Vitamin_D_Metabolism).

37. Wacker M, Holick MF. Sunlight and vitamin D, a global perspective for health. *Dermatoendocrinol*. 2013; 5(1):51-108.

38. https://www.westonaprice.org/our-blogs/an-ancestral-perspective-on-vitamin-d-status-part-1-problems-with-the-naked-ape-hypothesis-of-optimal-serum-25ohd/ (https://www.westonaprice.org/our-blogs/an-ancestral-perspective-on-vitamin-d-status-part-1-problems-with-the-naked-ape-hypothesis-of-optimal-serum-25ohd/)

39. Spiesman IG. Massive doses of vitamins A and D in the prevention of the common cold. *Arch Otolaryngol.*1941;34(4):787-791. Courtesy Chris Masterjohn, PhD.

40. Masterjohn C. From Soil Health to Human Health. Wise Traditions 2016. Montgomery AL.

41. Masterjohn C. Vitamin D toxicity redefined: Vitamin K and the molecular mechanism. *Med Hypothesis*. 2007;68:1026-1034.

42. Jones G. Pharmacokinetics of vitamin D toxicity. *American Journal of Clinical Nutrition*. 2008;88(2):582S–6S.

43. https://www.nih.gov/news-events/news-releases/nih-task-force-formed-develop-first-

nutrition-strategic-plan (https://www.nih.gov/news-events/news-releases/nih-task-force-formed-develop-first-nutrition-strategic-plan). The Weston A. Price Foundation submitted a request to research the requirements for fat-soluble vitamins during pregnancy and breastfeeding stages of life. Evidence shows that many women are deficient in vitamin A at delivery and may indeed be supplementing with excessive amounts of vitamin D.

44. Didriksen A. Vitamin D3 increases in abdominal subcutaneous fat tissue after supplementation with vitamin D3. *Eur J Endocrinol* 2015;172:235-241.

45. Zhang M. SNP rs11185644 of RXRA gene is identified for dose-response variability to vitamin D3 supplementation: a randomized clinical trial. *Sci Rep*. 2017;7:40593.

46. Engelmen CD et al. Vitamin D Intake and Season Modify the Effects of the GC and CYP2R1 Genes on 25-Hydroxyvitamin D Concentrations. *J Nutr*. 2013;143(1)17-26.

47. Zuk A, Fitzpatrick T, Rosella LC. Effect of Vitamin D3 Supplementation on Inflammatory Markers and Glycemic Measures among Overweight or Obese Adults: A Systematic Review of Randomized Controlled Trials. *PLoS One*. 2016;11(4).

48. Mangin M, Sihha R, Fincher. Inflammation and vitamin D: the infection connection. *Inflamm Res*. 2014;63(10):803-819.

49. Binkley N et al. Assay variation confounds the diagnosis of hypovitaminosis D: a call for standardization. *J Clin Endocrinol Metab.* 2004 Jul;89(7):3152-7.

50. Binkley N, Sempos CT. Standardizing Vitamin D Assays: The Way Forward. *J Bone Min Res*. 2014;29(8):1709-1714.

51. Shah I et al. Misleading measures in Vitamin D analysis: A novel LC-MS/MS assay to account for epimers and isobars. *Nutr J*. 2011; 10:46.

52. https://doctorgaby.com/the-book/ (https://doctorgaby.com/the-book/)

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