A Dermatologist’s Perspective on Vitamin D

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Abstract

Vitamin D is a fat-soluble steroid hormone that is crucial for human health and has recently generated controversy regarding its role in human health and disease. In this Special Article, we discuss our dermatologic perspective on vitamin D in a question-and-answer format. We discuss methods of obtaining vitamin D, including cutaneous photosynthesis, diet, and supplements and include the recent US Institute of Medicine recommendations. Other reviewed topics include the associations among skin pigmentation, climate, photoprotection, and vitamin D levels. We also elaborate on the popular interest in sun exposure as a method of normalizing vitamin D levels in the context of the risks of solar and artificial radiation. We also discuss groups at risk for vitamin D inadequacy, the need for testing serum vitamin D levels, and the role of phototherapy in patients with malabsorption conditions and hypervitaminosis D, with a focus on patients with sarcoidosis. Finally, we summarize our recommendations on vitamin D.

HOW DO WE OBTAIN VITAMIN D?

There are 3 sources of vitamin D: sunlight, diet, and vitamin D supplements.1-9

Sunlight

The most well-known source of vitamin D is sun exposure (Figure 1). Vitamin D is a fat-soluble prohormone steroid. Exposure to the UV-B portion of sunlight at a mean ± SD of 300 ± 5 nm, the precursor 7-dehydrocholesterol in the plasma membrane of both keratinocytes and fibroblasts is converted to previtamin D₃, the former in the basal and suprabasal layers of the epidermis of the skin (Figure 2). Previtamin D₃ is then converted to vitamin D₃ by a thermal, nonenzymatic process in the plasma membrane. Vitamin D₃ is then transported to the bloodstream.1-9,11,12

The UV-B action spectrum of vitamin D biosynthesis is the same as that responsible for the sunburn response and photocarcinogenesis. The conversion of previtamin D₃ to the inactive photoproducts lumisterol and tachysterol balances the cutaneous biosynthesis of vitamin D₃ as a feedback loop. This mechanism ensures that one cannot “overdose” on vitamin D₃ by photoexposure alone. After less than 1 minimal erythema dose (MED; ie, the amount of photoexposure required to produce faint pinkness in the skin at 24 hours after exposure), the concentration of previtamin D₃ reaches maximal levels and further UV radiation merely results in the production of inactive metabolites.

Cuttaneously synthesized vitamin D₃ is bound to vitamin D–binding protein (VBP), whereas dietary vitamin D₂ and vitamin D₃ are bound to both VBP and lipoproteins. Both vitamin D₂ and D₃ are hydroxylated by 25-hydroxylase in the liver to form 25-hydroxyvitamin D (25[OH]D).1-9,11,12

25-Hydroxyvitamin D is the major circulating form of vitamin D, and its serum levels are widely used as a reflection of total body stores of vitamin D. It is biologically inactive, however, and requires hydroxylation in the kidney to form the biologically active 1,25-dihydroxyvitamin D (1,25[OH]₂D) (Figure 1).1-9,11,12 Megalin and cubilin are endocytic receptors in the proximal tubule cells of the kidney. They are involved in reabsorption of VBP, which, in the presence of 1-α hydroxylase, results in intracellular...
conversion of 25(OH)D to 1,25(OH)2D. 13,14 1,25-dihydroxyvitamin D regulates calcium metabolism in an endocrine manner by enhancing intestinal calcium absorption, reducing renal excretion of calcium by its effects on parathyroid hormone (PTH), and mobilizing calcium from the skeleton through resorption. 1,9,11,12

In vitro studies show that 25-hydroxylase, but not 1-x hydroxylase, is present in dermal fibroblasts. There are extrarenal cells and organs that possess 1-x hydroxylase, including lung, breast, colon, prostate, keratinocytes, and monocytes. Human keratinocytes have demonstrated both an in vivo and in vitro autonomous capacity for vitamin D3 metabolism and catabolism. 1,25-dihydroxyvitamin D formed by extrarenal organs acts in an autocrine manner. 1,25-dihydroxyvitamin D levels are tightly regulated and are relatively constant, whereas serum 25(OH)D levels vary widely over time in healthy individuals. 1,9,11,12

Autocrine-acting 1,25(OH)2D acts as a steroid hormone and links a steroid hormone receptor to a vitamin D receptor. This complex then binds to a retinoic acid X receptor, which then binds to vitamin D response elements in the genome and subsequently modifies gene transcription. At least 60 human cell types express the vitamin D receptor, with an estimated 200 genes that are responsive to vitamin D. These genes are involved in processes such as cell proliferation, differentiation, apoptosis, and production of bactericidal proteins. 1,9,11,12

Interestingly, there are also noncalcemic analogues of vitamin D that have been shown to have prodifferentiation and apoptotic effects. A recent study demonstrated that 20-hydroxvitamin D2 inhibited DNA synthesis in epidermal keratinocytes and melanoma cells; these effects were facilitated by interactions with the vitamin D receptor. 15 Furthermore, although the products of vitamin D3 metabolism and 7-dehydrocholesterol metabolism, such as 1-x 20-dihydroxyvitamin D3, 20-hydroxyvitamin D3, and 7-dehydropregnenolone, have little to moderate calcemic activity when compared with 1,25(OH)2D, they have been shown to have antiproliferative and prodifferentiation effects on human and mouse leukemia cells. 16,17

Dietary Sources
There are only a few natural sources of vitamin D. These sources include cod liver oil, cheese, egg yolks, mackerel, salmon, tuna fish, and beef liver (Table 1). 3,9,18 Because it is not easy for many individuals to obtain adequate vitamin D intake from natural dietary sources alone, many countries, including the United States, fortify foods such as orange juice, milk, yogurt, and cereal with vitamin D. For the past few years, vitamin D3 has been the most common type of vitamin D used for fortification. 3,9,18

Vitamin D Supplements
Many inexpensive supplemental vitamin D forms are readily available over the counter in both vitamin D3 and vitamin D2 forms and with or without calcium.

ARE THERE DIFFERENCES BETWEEN VITAMIN D2 AND D3?
There are 2 forms of vitamin D: vitamin D2 and vitamin D3. Vitamin D2 is naturally found in sun-dried mushrooms, such as shiitake mushrooms, and is also formed from UV irradiation of the yeast sterol ergosterol. Vitamin D3 is synthesized in human skin and is present in vitamin D–rich food (Table 1). 3,9,16 The biological activity of vitamin D2 vs vitamin D3 is likely equivalent but is an active and controversial area of research. 1,10-25 Most vitamin D–fortified foods, vitamin D supplements, and multivitamins in the United States contain vitamin D3. The prescription form of vitamin D in the United States, however, is vitamin D2 (eg, as 50,000-IU capsules). 1,3,9,18-25

WHAT IS THE EVIDENCE THAT VITAMIN D HAS AN EFFECT ON HEALTH AND DISEASE?
This topic has been extensively reviewed elsewhere. 3,26-28 The relative strength of evidence for the effect of vitamin D on various diseases, which we have rated subjectively, is listed in Table 2. 3 Suffice it to state that in the recent report from the Institute of Medicine (IOM), it was concluded that the strongest evidence was only for the beneficial effect of
vitamin D on skeletal health, whereas evidence for the effect of vitamin D on extraskeletal outcomes was considered "inconsistent, inconclusive, and insufficient to inform nutritional requirements."10

**DOES SKIN PIGMENTATION AFFECT VITAMIN D LEVELS?**

Variation in skin color is an immediately noticeable human polymorphism. The causes of human skin pigmentation have been discussed from the time of Hippocrates in the fifth century. Aristotle and his followers developed a "climactic theory," which related human features to their surroundings, including the association of darker skin pigmentation with warm environments.29

Differences in human skin pigmentation are due to varying epidermal melanin contents. Because epidermal melanin is a large polymer that absorbs photons across the entire visible light and UV range, its content determines the number of photons that can eventually result in the cutaneous synthesis of vitamin D₃. There are 2 levels of natural selection hypothesized to play a role in human skin pigmentation. The first is the need for photoprotection against high levels of equatorial UV-A and UV-B, which resulted in darker skin pigmentation. The second is to promote seasonal UV-B photosynthesis of vitamin D₃ in areas proximal to the poles, resulting in lighter skin pigmentation.1-8,20-32 Vitamin D may even have a role in melanogenesis because melanocytes in situ express the vitamin D receptor.32

A study published in 1991 investigated the relationship between skin pigmentation and vitamin D₃ formation.31 The investigators found that after a fixed dose of UV-B radiation, serum vitamin D₃ levels were significantly higher in white and Asian groups than in African American and East Indian groups. Interestingly, they also found that serum 1,25(OH)₂D levels and VBP levels were similar in all groups, regardless of skin pigmentation.31

A study in 2004 detailed the 25(OH)D levels in different groups in the United States. White individuals had a mean serum level of 32 ng/mL. (to convert

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**FIGURE 2.** Structure of human skin. 7-Dehydrocholesterol is converted to previtamin D₃ in the basal (stratum basale) and suprabasal layers of the epidermis of the skin.1,9,11,12
to nmol/L, multiply by 2.496), Mexican Americans had a level of 24 ng/mL, and African Americans had a level of 20 ng/mL.33 Furthermore, among individuals older than 60 years, more than 67% of whites and 88% of African Americans had serum levels lower than 32 ng/mL.33 A study published in 2010 evaluated the seasonal variations of vitamin D levels at high latitudes in fall vs winter with consideration of other factors, including skin pigmentation, sun exposure, and dietary vitamin D intake. They reported that South Asians and East Asians had substantially lower 25(OH)D levels than Europeans.34 Another 2010 study assessed the association among skin pigmentation, sun exposure, skin reflectance, vitamin D intake, and body surface area exposed to the sun.35 The authors concluded that to maintain serum 25(OH)D levels of greater than 30 ng/mL, individuals of European ancestry with high sun exposure need a supplemental dietary intake of 1300 IU/d of vitamin D, whereas individuals of African ancestry with low sun exposure need 2100 to 3100 IU/d year round.35

A recent study demonstrated that after broadband UV-B (290-320 nm) exposure in a laboratory setting, the increase in serum 25(OH)D levels was independent of the constitutive skin pigmentation of the studied individuals.36 This finding suggests that the innate vitamin D biosynthesis abilities of the skin of individuals of different pigmentation are similar. This study was performed during the winter season in Denmark, when melanin content was lower and expected to reside in the deeper basal layer of the epidermis, therefore playing a relatively less protective role.36 In summary, most available evidence indicates that there is an inverse association between skin pigmentation and serum 25(OH)D levels.1-8,29-36

DO PEOPLE RESIDING IN AREAS WITH SUNNY CLIMATES HAVE ADEQUATE SERUM VITAMIN D LEVELS?

Authors of a study published in 2007 recruited 93 healthy, young, clinically tanned adults from the University of Hawaii and a Honolulu skateboard shop.37 These individuals had a mean of 29 hours of sun exposure per week during the 3 months preceding the study, and 40% of them reported never using sunscreen. The group’s mean 25(OH)D concentration was 32 ng/mL, and 51% of the study’s participants had serum 25(OH)D levels below 30 ng/mL, levels considered inadequate at the time of the study.37

Similarly, studies from Santiago (Chile), Kashmir (India), and East Asia reported that more than 50% of individuals living in areas with sunny climates had inadequate serum vitamin D levels (<30 ng/mL).38-41 Possible explanations for these findings include photoprotection practices (due to lifestyles or cultural beliefs), older age and obesity (which are associated with lower serum 25(OH)D levels), and inadequate dietary intake.1-9,42

A 2010 review of published serum 25(OH)D levels in the northern hemisphere, however, reported mean levels in the winter of 48.3 nmol/L (ranging from a low of 29.0 nmol/L in Helsinki to a high of 62.3 nmol/L in Miami) and in the summer of 70.0 nmol/L (ranging from a low of 57.0 nmol/L in Kalamazoo to a high of 85.4 nmol/L in Boston).43 Considering the November 2010 IOM recommendation that adequate levels of 25(OH)D are 50 nmol/L or higher,10 this report strongly indicates that in the northern hemisphere inadequate vitamin D status in a population was most likely to occur only in those residing in high latitudes in the winter.43

### TABLE 1. Vitamin D and Dietary Intake

<table>
<thead>
<tr>
<th>Food</th>
<th>Vitamin D, IU per serving</th>
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<tbody>
<tr>
<td>1 Tbsp of cod liver oil</td>
<td>1360</td>
</tr>
<tr>
<td>Sockeye salmon, cooked, 3 oz</td>
<td>447</td>
</tr>
<tr>
<td>Mackerel, cooked, 3 oz</td>
<td>388</td>
</tr>
<tr>
<td>Tuna fish, canned in water, drained, 3 oz</td>
<td>154</td>
</tr>
<tr>
<td>Orange juice, vitamin D fortified, 1 c</td>
<td>137 (amount varies by product)</td>
</tr>
<tr>
<td>Milk, vitamin D fortified, 1 c (nonfat, reduced fat, and whole)</td>
<td>115-124</td>
</tr>
<tr>
<td>Yogurt, fortified with 20% of the daily value for vitamin D, 6 oz</td>
<td>88</td>
</tr>
<tr>
<td>Beef liver, cooked, 3.5 oz</td>
<td>49</td>
</tr>
<tr>
<td>Ready-to-eat cereal, 10% of the daily value of vitamin D, ¾-1 c</td>
<td>40 (amount varies by product)</td>
</tr>
<tr>
<td>Egg, 1 large (vitamin D is in yolk)</td>
<td>41</td>
</tr>
<tr>
<td>Swiss cheese, 1 oz</td>
<td>6</td>
</tr>
</tbody>
</table>

Data from the National Institutes of Health Office of Dietary Supplements.18

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TABLE 2. Vitamin D and Human Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skeletal health</td>
<td>++</td>
</tr>
<tr>
<td>Cancer</td>
<td>+</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>+</td>
</tr>
<tr>
<td>Macular degeneration</td>
<td>+</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>+</td>
</tr>
<tr>
<td>Melanoma</td>
<td>+</td>
</tr>
<tr>
<td>Hypertension</td>
<td>+/-</td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td>+/-</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>-</td>
</tr>
<tr>
<td>Diabetes mellitus type 2</td>
<td>-</td>
</tr>
<tr>
<td>Rarer cancers</td>
<td>-</td>
</tr>
<tr>
<td>Depression</td>
<td>-</td>
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</tbody>
</table>

*Evidence is rated by the authors from strongest (+++ to no beneficial association (–)).
b Rarer cancers are endometrial, esophageal, gastric, kidney, ovarian, and pancreatic cancers, and non-Hodgkin lymphoma.

**WHAT SHOULD WE ADVISE PATIENTS ON PHOTOPROTECTION PRACTICES AND VITAMIN D LEVELS?**

In laboratory settings, it has been shown that adequate application of a sunscreen with a sun protection factor (SPF) of 8 significantly suppressed the increase in 25(OH)D after one MED of simulated sunlight.44 A review of available evidence published in 2009, however, concluded that although sunscreens can reduce significantly the cutaneous synthesis of vitamin D under very strictly controlled conditions, their normal use by the general population does not generally result in vitamin D insufficiency.45 A possible explanation for this conclusion is the well-documented fact that in actual use most individuals apply sunscreens at much lower concentrations than the concentration mandated for sunscreen SPF testing (0.5-0.8 mg/cm² in actual use vs 2 mg/cm² required for SPF determination), hence resulting in a significantly lower in-use SPF compared with the labeled SPF.46-48

It has been shown that full body exposure of fair-skinned individuals to 10 to 15 minutes of midday summer sun is equivalent to one MED and resulted in the synthesis of approximately 15,000 IU/d of vitamin D.48 This is the well-documented fact that in actual use most individuals apply sunscreens at much lower concentrations than the concentration mandated for sunscreen SPF testing (0.5-0.8 mg/cm² in actual use vs 2 mg/cm² required for SPF determination), hence resulting in a significantly lower in-use SPF compared with the labeled SPF.46-48

**WHAT IS THE RELATIONSHIP BETWEEN THE USE OF TANNING BEDS AND VITAMIN D LEVELS?**

Indoor tanning is a burgeoning and increasingly popular industry that has become more accessible to the general public during the last 30 years. Some estimates in recent years state that the indoor tanning business earns $5 billion per year, with more than 28 million customers annually and more than 50,000 tanning facilities in the United States. The major emission spectra of tanning lamps is in the UV-A range (320-400 nm, which causes tanning) rather than in the UV-B range, the action spectrum of vitamin D biosynthesis.56

Although the use of tanning booths can cause increases in serum 25(OH)D levels, this may be confounded by higher overall duration of sun exposure in users.56 In recent years, strong evidence has indicated that the use of tanning beds is addictive and associated with the development of skin cancers, including melanoma.52,56-64 As previously mentioned, in 2007, the International Agency for Research on Cancer published a special report on human carcinogens and raised the classification of UV-emitting tanning devices to group 1 (carcinogenic to humans).52 The World Health Organization has recommended a complete ban on indoor tanning for minors younger than 18 years.52 On the basis of this evidence, there is no indication for the use of tanning beds to obtain adequate vitamin D status.

**WHAT ARE THE CURRENT RECOMMENDATIONS ON VITAMIN D INTAKE AND SERUM LEVELS?**

In November 2010, after a 2-year study, the IOM released its recommendations on vitamin D.10 The recommendations were made based on data on skeletal health only, on data on extraskeletal outcomes, including cancer, cardiovascular diseases, diabetes, and such factors as age, sex, race, and body weight.10 The recommendations were: for the general public, a daily intake of 600 IU of vitamin D for those 1-70 years of age (1500 IU for those 71 years and older) and a serum 25(OH)D level of 20-30 ng/mL.
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mellitus, and autoimmune disorders, were consid-
ered “inconsistent, inconclusive as to causality, and
insufficient to inform nutritional requirements.”1-10
In recognition of the wide variability of vitamin D
synthesis secondary to sun exposure (due to factors
such as cloud cover, season, time of day, and skin
pigmentation) and the risks of skin cancer, the rec-
ommendations were made with an assumption of
minimal or no sun exposure.1-12,57-64

In November 2010, the IOM established recom-
dended dietary allowances (RDAs) for adults and
allowable intake (AI) levels for infants (0-12 months
of age) for vitamin D; RDA is defined as intake that
meets the needs of 97.5% or more of the population,
and AI is defined as a level of intake assumed to
ensure adequate nutrition when there is lack of ev-
dence to set an RDA.10,18 The AIs and RDAs are as
follow: 0 to 12 months of age, 400 IU/d; between 1
and 70 years of age, 600 IU/d; and older than 70
years, 800 IU/d. The RDA for pregnant and lactating
women was also set at 600 IU/d. Furthermore, the
IOM stated that serum 25(OH)D levels of 20 ng/mL
cover the requirements for 97.5% of the population.
The IOM also commented that although there are
sparse data on upper serum levels of 25(OH)D, lev-
els higher than 50 ng/mL “should raise concerns
among clinicians about potential adverse effects.”10
Upper intake levels (the highest daily intake likely to
pose no risk) were as follows: for 1 to 3 years of age,
2500 IU/d; for 4 to 8 years of age, 3000 IU/d; and for
9 years and older, 4000 IU/d.10

SHOULD TESTING FOR SERUM VITAMIN D
LEVELS BE RECOMMENDED FOR ALL
INDIVIDUALS?

In the United States, the cost of a serum 25(OH)D
test is approximately $100. Therefore, if testing
were performed on all individuals, there would be
an added economic burden to society.3,26 As stated
earlier, a recent review of serum 25(OH)D levels in
individuals residing in the northern hemisphere
showed adequate levels of greater than 20 ng/mL,
with the exception of some regions in colder cli-
mates during the winter.33,40 Therefore, we re-
commend testing for serum 25(OH)D levels in the ap-
propriate clinical context only in individuals known
to be at risk for vitamin D inadequacy (Table 3).
Those at risk for vitamin D inadequacy include el-
derly individuals (≥70 years old) because aged skin
has decreased capacity to synthesize vitamin D, in-
dividuals with limited sun exposure, obese individ-
uals, those with darker skin (Fitzpatrick skin pho-
totypes IV-VI), and individuals with malabsorption
syndromes because vitamin D is a fat-soluble vita-
m.1-9,42,51,65-71 Finally, because human breast
milk contains only 25 IU/L of vitamin D in contrast
to vitamin D–enriched infant formula (100 IU of
vitamin D₃ per 8 oz), vitamin D drop supplemen-
tation should be given to breastfed infants.1-9,67-69

SHOULD UV-B PHOTOTHERAPY BE
ADMINISTERED TO PATIENTS WITH
MALABSORPTION DISORDERS TO ACHIEVE
ADEQUATE VITAMIN D STATUS?

Most vitamin D is absorbed in the distal duodenum
and proximal jejunum; therefore, patients with mal-
absorption disorders are at risk for inadequate vita-
m D status.1-9,51,63,66 It is well established that
narrowband UV-B (311-313 nm) phototherapy re-
results in increases in serum 25(OH)D levels.72-74
Narrowband UV-B units are widely available in der-
matology centers and offices and are also available as
home units with a physician’s prescription.73,75

A study published in 2008 that evaluated more
than 4600 patients who received narrowband UV-B
phototherapy from 1985 to 2002 found no increase
in skin cancer development.75 The cost of pho-
totherapy in the United States is approximately $50
per treatment, whereas the cost of a home unit is
approximately $2500. At this time, there are no data
on the safety and efficacy of long-term, potentially
lifelong phototherapy in patients with malabsorp-
tion issues. This is the reason that we recommend
referring these patients to our endocrinology col-
leagues for management with careful monitoring of
serum 25(OH)D levels.3,26,72-74 The recent Endo-
crine Society Practice Guidelines on vitamin D rec-
ommended at least 6000 to 10,000 IU/d of vitamin
D for patients with malabsorption disorders until a
serum 25(OH)D level of 30 ng/mL is reached, with
maintenance dosing of 3000 to 6000 IU/d.9

SHOULD WE BE CONCERNED ABOUT
HYPERVITAMINOSIS D?

In healthy adult patients, vitamin D has a good
safety profile. Although the 2011 IOM recommen-

<table>
<thead>
<tr>
<th>TABLE 3. Individuals at Risk for Vitamin D Deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Individuals with low sun exposure (due to geography, photoprotection practices, or clothing worn because of cultural beliefs)</td>
</tr>
<tr>
<td>Exclusively breastfed infants</td>
</tr>
<tr>
<td>Elderly individuals, especially those with a history of or risk for falls and fractures</td>
</tr>
<tr>
<td>Obese individuals</td>
</tr>
<tr>
<td>Individuals with darker skin types</td>
</tr>
<tr>
<td>Patients with malabsorption conditions, such as inflammatory bowel disease</td>
</tr>
<tr>
<td>Patients with cystic fibrosis</td>
</tr>
<tr>
<td>Patients taking anticonvulsant and antifungal (eg, ketoconazole) medications</td>
</tr>
<tr>
<td>Patients taking medications for an autoimmune deficiency syndrome (eg, AIDS)</td>
</tr>
</tbody>
</table>

Data from references 1-9, 42,51,65-71.
skele
total health are well established; however, more research is needed to unravel its complicated ties to other human conditions. On the basis of currently available data, it is clear that dietary or supplemental vitamin D should be the preferred modern-day method of maintaining normal serum levels. Because of the known deleterious effects of solar and artificial UV radiation, UV exposure is not an appropriate way to achieve adequate vitamin D levels.

Abbreviations and Acronyms. 25(OH)D = 25-Hydroxyvitamin D; 1,25(OH)2D = 1,25-Dihydroxyvitamin D; AI = allowable intake; IOM = Institute of Medicine; IU = international units; MED = minimal erythema dose; ng/mL = nanograms/milliliter; nm = nanometer; nmol/L = nanomoles/Liter; SPF = sun protection factor; RDA = recommended dietary allowance; UV = ultraviolet; UV-A = ultraviolet A; UV-B = ultraviolet B; VBP = vitamin D binding protein

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REFERENCES

CONCLUSION
Many aspects of vitamin D are relevant to dermatology and the broader field of medicine. Its potential benefits on skeletal health are well established; however, more research is needed to unravel its complicated ties to other human conditions. On the basis of currently available data, it is clear that dietary or supplemental vitamin D should be the preferred modern-day method of maintaining normal serum levels. Because of the known deleterious effects of solar and artificial UV radiation, UV exposure is not an appropriate way to achieve adequate vitamin D levels.


42. Harris SS, Dawson-Hughes B. Reduced sun exposure does not explain the inverse association of 25-hydroxyvitamin D with percent body fat in older adults. J Clin Endocrinol Metab. 2007;92(8):3155-3157.


