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Sunlight in Vitamin D Deficiency: Clinical Implications

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A B S T R A C T

Recent evidence shows that hypovitaminosis D can lead to increased morbidity and mortality. This article highlights the role of sunlight in vitamin D deficiency and its consequences for age-related diseases among older adults. The unique content of this article is the review of factors influencing vitamin D levels, primarily the lack of sun exposure whereby health providers' strategies can impact the reduction of vitamin D deficiency and its complications. Recommendations for vitamin D_3 supplementation are discussed. Clinical trials are needed to investigate specific measures to increase exposure to sunlight, mobilizing providers to take action to eliminate vitamin D deficiency worldwide.

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It is imperative to investigate lifestyle risks that result in chronic illnesses affecting older adults. Older adults are at higher risks for vitamin D deficiency related to less skin vitamin D synthesis and lifestyles, such as the engagement of indoor versus outdoor activities. Despite conflicting results of observational studies and vitamin D supplementation still being controversial, this updated review of the role of sunlight in vitamin D deficiency in older adults will be beneficial for busy primary care providers like nurse practitioners (NPs).

The purpose of this article is to encourage NPs and other health care providers to routinely recommend adequate sun exposure as a necessary option for the reduction of hypovitaminosis D, thereby mitigating risks for acquiring common age-related diseases in essentially healthy older adults. As the number of baby boomers increases, so too does the need for a more robust repertoire to maintain successful aging.

Literature Review

Because of the increased knowledge of vitamin D receptors and their influence on human gene expression, vitamin D's function has expanded.[1] Vitamin D's expanding role ranges from being that of endocrine (the most known function as the bone health regulator) to that of paracrine (e.g., acting as local hormones within monocytes for immune function), intracrine (e.g., regulator within the cells for cell growth and differentiation), and apoptosis mechanisms. Hence, vitamin D deficiency may lead to many chronic conditions of aging such as cardiometabolic, neurologic, autoimmune, osteoporosis, and cancer.[2]

Vitamin D deficiency measured by serum 25 hydroxyvitamin D is the biological indicator of vitamin D status and has been linked to several diseases. Vitamin D insufficiency levels indicate that serum 25 hydroxyvitamin D is <30 ng/mL (<75 nmol/L), which is the current acceptable level of vitamin D, whereas vitamin D deficiency is when the serum 25 hydroxyvitamin D level reaches <20 ng/mL.[1] Cutoff levels are related to serum 25 hydroxyvitamin D's inverse relationship with the parathyroid hormone (PTH) keeping serum D levels $\geq$ 30 ng/mL because it is at this level upon which PTH levels plateau (at its nadir level).[3] PTH levels must be controlled because increased PTH levels could lead to deleterious effects, such as bone calcium reabsorption, which ultimately can cause fragility fractures and other vascular problems related to increased morbidity and mortality.[1-3]

Cells and body tissues contain and can produce calcitriol (1, 25 $[OH]D_2$), the active form of vitamin D that has an impact on a large number of biological processes.[4] Calcitriol, which is tightly physiologically regulated, is not mostly affected by sun exposure compared with serum 25 hydroxyvitamin D, but it has a short half-life (< 4 hours), which does not make calcitriol a tool to measure vitamin D status.[3] To achieve a healthy vitamin D status, studies[4-6] showed evidence of the necessity for moderate sun exposure with adequate ultraviolet exposure. The most common conditions among many factors that contribute to hypovitaminosis D include low sunlight exposure, dark skin pigmentation, advancing age, and behavioral/lifestyle factors.[6,7] These risk factors have more pronounced effects among the aging (> 65 years) population.

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Factors Affecting Vitamin D Levels Causing Hypovitaminosis

Humans get most of their vitamin D from sunlight. Older (≥ 65 years) individuals tend to be less engaged in outdoor activities than their younger counterparts, especially during the winter months. Low sun exposure increases older adults' risk (e.g., seeking shade and wearing long sleeves).[8] The risk is more serious for those who live in geographic areas where there is less or no sunlight with adequate ultraviolet B (UVB) radiation most of the year. The skin production of vitamin D decreases as individuals age. Consequently, it is vital to note that the solar zenith angle plays a significant role among many environmental factors in achieving cutaneous vitamin D production.[6,7] UVB radiation with a 290- to 310-nm wavelength can be most useful in the cutaneous photosynthesis of vitamin D_3 if its photons travel in a shorter distance and are less absorbed by the stratospheric ozone layer.[7] The more direct sunlight that reaches the skin, the more UVB absorption for vitamin D synthesis there is. At latitudes approximately 33° above or below the earth's surface and during winter, there is minimal, if any, vitamin D_3 skin production from sun exposure.[6] Furthermore, other environmental factors commonly affecting the solar zenith angle and UVB that influence vitamin D production include, but are not limited to, altitude, ozone layers, clouds, air pollution, and living in urban areas.[6,7]

A study in Ireland[9] showed that older community dwellers who live in the northern higher latitude of 51° who are usually at risk for hypovitaminosis D but engage in sun enjoyment activities were found to have lower risks for low serum D levels versus those who do not. In sunny Australia, researchers[10] found an increased prevalence of low serum D levels among older men (≥ 70 years). During winter, these men's serum D levels were shown to be at their lowest, effects consistent with other studies.[5,11]

Individuals with low serum D levels commonly have dark skin pigmentation. More blacks than whites are known to be vitamin D deficient.[12] Webb et al[13] conducted a study that showed that individuals from the United Kingdom with type V (brown) skin needed ~25 minutes of noontime sunlight daily to have adequate serum D levels, which was more time than those with lighter skin. Furthermore, Holick[5] emphasized that in addition to having limited or no sunshine exposure, having poor dietary intake and having no vitamin D supplementation magnify the occurrence of vitamin D deficiency among hyperpigmented individuals.

As individuals age, the amount of 7-dehydrocholesterol found in the skin decreases, leading to less capacity for skin production of vitamin D_3.[5] A study[14] noted that vitamin D synthesis is reduced linearly with age starting at 10 years old and reaching a reduction of 50% at age 70. This study comparing young (20-30 years) versus older adults (62-80 years) who were subjected to UVB radiation using tanning beds found that seniors have the worst scenario of not making enough vitamin D because they could hardly attain the minimum amount of vitamin D_3 (600-800 IU/d) compared with outdoor-working adults.[14] However, Wacker and Holick[6] claimed that a 75-year-old man who has adequate UVB tanning exposure (3× per week for 7 weeks) has a comparable capacity of increasing serum D levels with that of a healthy young adult.

Lifestyles related to vitamin D deficiency as reported in a study of older Australian adults (60-84 years) showed that less time spent outdoors, less physical activities, poor vitamin D intake, and low exposure to ambient ultraviolet radiation were positively linked with deficient serum D levels.[15] Hence, these researchers suggested that the simplest preventive strategy to reduce these modifiable risk factors among the elderly is increasing vitamin D intake by supplementation if adequate sun exposure is not feasible. Similarly, Chen et al[16] found that older Chinese people residing in places with low ambient UVB levels needed to be encouraged to take vitamin D supplementation with sensible sun exposure. Chinese women believe that having a pale whitish skin color is prettier.[7]

The tendency to be less engaged in outside activities makes older adults prone to becoming sedentary, especially during winter months. Laird et al[17] found that the determinants of vitamin deficiency in older Irish adults in addition to wintertime include those with no supplementation, obese adults, and those who are sedentary. For those who are nonambulatory or homebound and have no adequate sunlight, the use of sunbeds with UVB radiation has been suggested as a feasible strategy.[18] These studies reinforce the need to have sufficient sunlight to prevent the clinical consequences described later.

Clinical Implications of Vitamin D Deficiency

Hypovitaminosis D's influence on increasing risks for cardiovascular and metabolic diseases includes, but is not limited to, hypertension,[19] metabolic syndrome,[20] and type 2 diabetes,[21] although the direction of these associations remains unclear.[22] These known associative factors not only could increase myocardial infarction by as much as 50% in vitamin D–deficient individuals but also increases their mortality risk to 100% during a myocardial infarction.[6] In contrast, a recent study[23] reported that those who regularly engage in outdoor activities had a lower cardiovascular disease mortality rate, regardless of their serum D levels. Sunlight has protective effects dependent and independent of vitamin D synthesis because of other non-UVB factors like nitric oxide production (a vasodilator).[22] Moreover, sunlight is known to generate beta-endorphins from the skin, which might explain the feeling of wellness when one is outdoors.[22]

Studies[24,25] have looked into vitamin D deficiency's associations with cancer. Such studies emphasized that individuals living in higher latitudes with less exposure to sunlight, which already puts them at higher risk for vitamin D deficiency, are at a higher risk for cancer as well. Garland and Gorham[24] established that there is a positive relationship between living in high latitudes and cancer death rates. Furthermore, these authors' meta-analysis showed that individuals with low levels of serum D have higher risks of colorectal cancer.

McCullough et al[25] recently conducted a cutting-edge study on colorectal cancer risk determinations. Results showed that those with 25(OH)D above sufficiency levels (ie, higher than the current recommendations[26] [30-34 ng/mL or 75 to < 87.5 nmol/L and 35-40 ng/mL or 87.5-100 nmol/L]) had 19% (relative ratio [RR] = 0.81; 95% confidence interval [CI], 0.67-0.99) and 27% (RR = 0.73; 95% CI, 0.59-0.91) lesser risk of colorectal cancer, respectively. In contrast, those within the lower range of the Institute of Medicine (IOM)-suggested sufficient levels (20-25 ng/mL or 50-62.5 nmol/L) showed higher risks for colorectal cancer (RR = 1.31; 95% CI, 1.05-1.62). Researchers in this study concluded that the optimal serum vitamin D levels for the reduction of cancers of the colon and rectum must be 75 to 100 nmol/L (30-40 ng/mL), which is higher than what the IOM currently recommends.

Higher serum D levels with chronic sun exposure have a risk-reducing effect on colorectal and breast cancers.[4] Aungst and Rainer[27] noted that despite being at higher risks for vitamin D deficiency, postmenopausal women who are at risk for breast cancers[28] are not routinely screened for serum D levels. There are no screening guidelines nor are there definitive supplementation recommendations for protection against cardiovascular disease or breast cancer.

Accumulating evidence shows a linkage between hypovitaminosis D and the risk for depression,[29] Alzheimer disease,[30] schizophrenia,[31] and neurocognitive decline.[32] Bogers et al[31] found that low serum D levels could be related to schizophrenia, but therapy-resistant schizophrenics showed no improvement of their low serum D levels with daylight exposures. The latest conference of

international experts reported agreement among these experts that vitamin D deficiency could possibly be linked to cognitive decline and dementia in older adults.[33]

Implications/Recommendations

Assessing possible causes of vitamin D deficiency is vital and should include routine assessment of the individual's sunlight exposure and vitamin D dietary sources. The effects of vitamin D deficiency are more pronounced among older adults because of the presence of comorbidities worsening with the aging process such as hypertension, type 2 diabetes, and abdominal obesity.[28] This also means asking specific questions regarding sunlight exposure such as the time of day, duration and use of protective clothing or sunblock, and if vitamin D serum levels have been measured. Providers must inquire about the following physical conditions that limit sunlight exposure: malabsorption syndrome, bowel surgeries, kidney, liver problems, and bone disorders that inhibit vitamin D synthesis. It is equally essential for practitioners to inform individuals that supplementation is needed when sun exposure is nil because there is minimal vitamin D from dietary sources even with fortified dairy products. Thus, food sources are not enough. For essentially healthy older adults, the target of serum D is between 40 and 60 ng/mL (100-150 nmol/L) to attain not only for bone health but also for other nonskeletal benefits.

To meet required vitamin D levels, sun exposure means considering the best timing of the day, season, latitude, weather, and the individual's skin color. Melanin acts a barrier in which UVB photons cannot reach skin's 7-dehydrocholesterol.[9,12] Skin types are based on Fitzpatrick's skin typing (I [fair]-VI [always tan]).[13] Typically, a fair-skinned white person might need approximately 5 to 15 minutes of sunlight or when the individual gets the minimal erythemal dose (MED) after 2 to 3 times of sun exposure in a week.[5] One MED means exposure of the limbs/extremities, abdomen, and back if possible that would cause mild stinging/sunburn feeling.[5,7] Sensible sun exposure entails exposure that is about 50% the amount of what it takes to incur a mild pinkish skin. Holick[5] and Grant[7] explained that an individual could get a UVB dose of 0.5 MED using a bathing suit exposing most body parts, which equates to taking 7,000 to 10,000 IU vitamin D_2 orally.

To treat vitamin D deficiency (and to fill up depleted vitamin D stores), 50,000 IU vitamin D_2 or D_3 should be taken once a week for 8 weeks (which equals taking ~6,600 IU/d).[1,7,34] For sufficient 25(OH)D levels, an individual has to take 50,000 IU D_2 or D_3 once every 2 weeks indefinitely. If an individual is essentially healthy, taking 4,000 to 5,000 IU daily will meet the maintenance goal of 40 to 60 ng/mL.[1] Individuals with a body mass index $\geq$ 30 may need 3 to 5 times more dosage both for treatment and prevention of vitamin D deficiency. Those with granulomatous disorders (ie, sarcoidosis) need to have reduced dosages (400 IU/d) to prevent hypersensitivity that could lead to hypercalciuria and hypercalcemia.[3,5,7] The need to monitor toxicity is equally crucial; monitoring of serum 25 hydroxyvitamin D 3 months after the start of treatment[27] and at least once a year when there are no health or medication changes is needed. Older adults with uncontrolled comorbidities and polypharmacy need closer monitoring of their serum 25 hydroxyvitamin D levels.

Vitamin D toxicity in the form of hypercalcemia is very rare and usually happens accidentally or when individuals intentionally decide to take extremely high doses reaching >200 ng/mL (500 nmol/L) serum vitamin D levels for several months.[1,5,7,34] Of note, sun exposure is known to cause no vitamin D intoxication because the human body is capable of converting excess previtamin D hormone to other isoforms that have noncalcemic photoproducts and are readily excreted.[5] If toxicity does occur, management consists of hydration and stopping all sources of vitamin D, which is sufficient to reverse hypercalcemia and usually without known consequences.[1,7,34]

Significance

Despite the current lack of consensus on vitamin D supplementation, as health care providers, the big goal is not merely disease prevention but also nutritional repletion,[35] especially for the elderly in whom poorer health confounds hypovitaminosis D. There ought to be baseline screening for those > 65 years old if we are to give supplementation safely beyond the current IOM recommendations. However, the United States Preventive Task Force recommends no screening needed for primary prevention in the asymptomatic elderly (category "I").[36,37] The task force's primary source was from the Women's Health Initiative calcium plus vitamin D supplementation trial[38] that had been criticized for limitations including using low-dose cholecalciferol (400-IU dosage) and less controlled laboratory reagents, among other flaws. Likewise, the US Preventive Task Force has not recommended supplementation for the primary prevention of fractures[39] despite data showing a 20% fracture reduction with higher dosage.[40] The task force did recommend physical activities but no supplementation for primary fall prevention as interventions for community dwellers not known to have osteoporosis or vitamin D deficiency.[41] This latest guideline is questioned by noted researchers,[35,42] as has been the conclusion of this author. Because older adults are prone to falls and lifestyles with less sun exposure, the likelihood of having hypovitaminosis D is high, and it is reasonable to follow the recommendations of the US Endocrine Society,[5] the National Osteoporosis Foundation,[34] and the American Society of Geriatrics[43] to supplement with 800 to 1000 IU vitamin D.[40]

Secondary prevention strategies for vitamin D deficiency are even less defined. Welles et al's prospective study[44] of those with stable coronary heart disease hypothesized that supplementation might be ineffective as a primary or secondary preventative measure if hypovitaminosis D is merely a marker of poor health. Al Mheid and Quyyumi's review[39] noted the possibility that the relationship between hypovitaminosis D and cardiovascular diseases could be an epiphenomenon. These authors further explained that most of the current studies were from tertiary prevention trials and that thousands of participants would be needed to decipher vitamin D's protective effects. Clinical practitioners are called to individualize treatments, and the development of a risk assessment tool has been suggested.[37]

The following future large population randomized controlled trials that are about to report results could improve the understanding of current inconsistencies: US VITAL (VITamin D and Omega-3 triaL), Europe's DO-HEALTH, Finland's FIND, and Australia's D-Health.[42]

Conclusion

The role of sunlight in preventing or mitigating the effects of vitamin D deficiency is well-documented. Despite the uncertain status of supplementation and institutional differences in the cutoff category levels of serum D sufficiency, adequate and appropriate sunlight exposure is unequivocal. Sunlight exposure as 1 viable option for vitamin D deficiency management remains low, possibly because of recommendations for sun avoidance. Patient-provider conversations call for an intentional review of sensible sun exposure activities (ie, leisure and socialization behaviors to motivate individuals to purely "embrace sunlight" as an integral part of an older adult's lifestyle practices).

Supplementation is safe, and diet fortification is needed to be instituted at times when there is limited or no sunshine exposure.

Older adults who have limited sunlight or are unable to access sunlight could benefit from UVB tanning beds and engage in active or passive mobility exercises. Further clinical research and prospective studies are required to examine specific strategies that incorporate sunlight as a treatment or preventive measure against vitamin D deficiency that could ultimately reduce its effects on age-related chronic diseases worldwide.

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