Optimal Intakes of Vitamin D
for Persons with Intellectual Developmental Disabilities:

SUNLIGHT ISN'T ENOUGH

Almost Everyone Needs Vitamin D Supplements

BY SUNIL J. WIMALAWANSA, MD, PHD, MBA, FACE, FRCP, FACP, FRCPATH, DSC

Adequate vitamin D is essential for optimal health. In addition to the well-known effects of enhancing mineral metabolism, irrespective of the age, gender, or health status of the patient, vitamin D has beneficial effects on many other conditions, including neuromodulation, muscle strength and coordination, autoimmune disorders, cardiovascular systems, pancreas, muscles, brain, controlling infections, and certain forms of cancer. Yet, vitamin D deficiency is the most common nutritional deficiency in the world. Prevalence of vitamin D deficiency is high in children in general, and it is alarmingly high among people with intellectual developmental disabilities (PIDD or PID). Moreover, many medications commonly taken by people with ID, as well as by those with medically complex developmental disabilities (MCDD), further decrease blood vitamin D levels, requiring them to take higher amounts of vitamin D.

The best way to determine vitamin D status is to measure blood levels of 25-hydroxyvitamin D [25(OH)D]. Since there is no apparent toxicity with levels below 100 ng/mL, the American Endocrine Society's recommends a normal range 30 to100 ng/mL, with a preferred range of 40 to 60 ng/mL. Based on the currently accepted minimum levels of vitamin D in "healthy" people, approximately 80 percent of persons with MCDD are vitamin D insufficient [serum 25(OH)D levels below 30 ng/mL], and more than 50 percent are vitamin D deficient [serum 25(OH)D levels below 20 ng/mL].

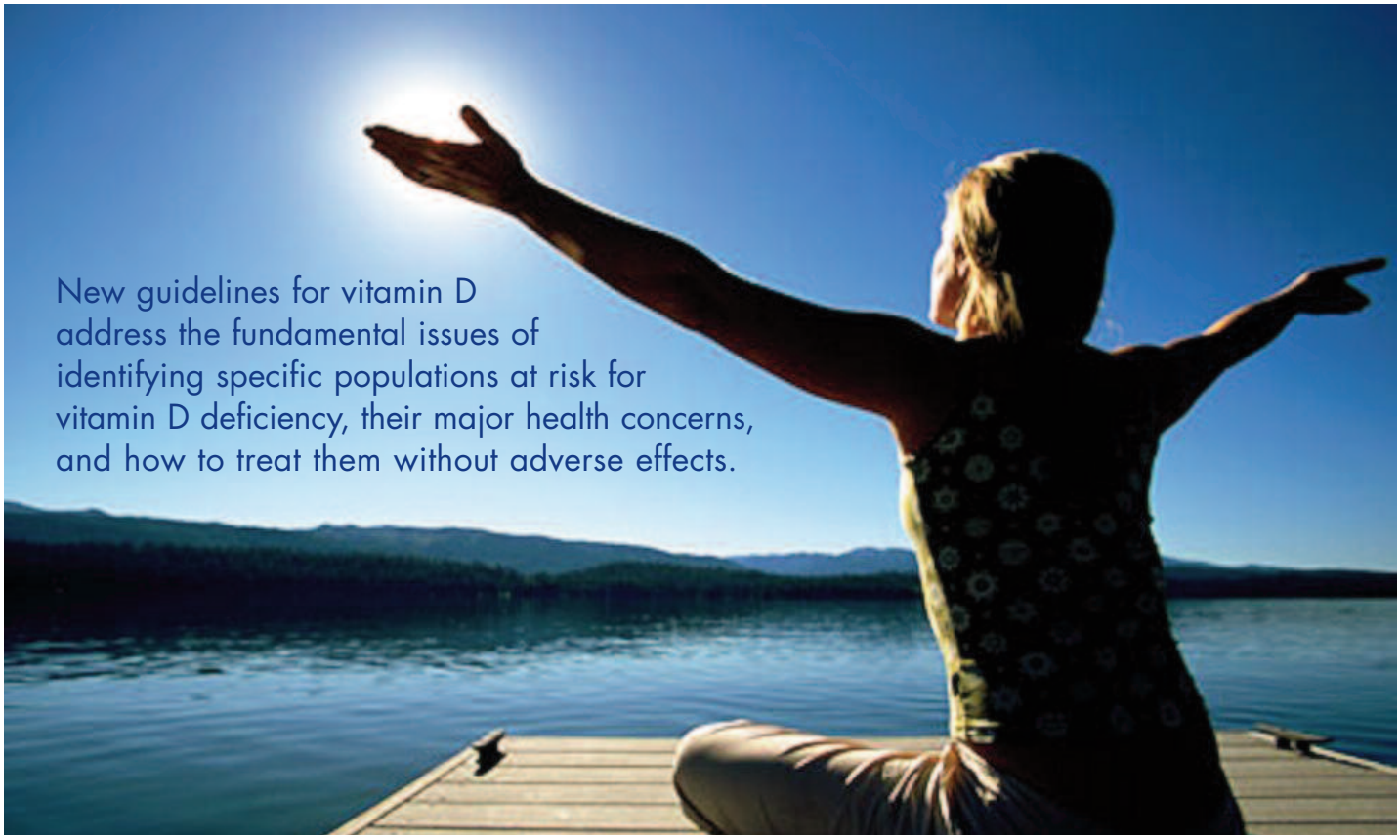
New guidelines for vitamin D address the fundamental issues of identifying specific populations at risk for vitamin D deficiency, their major health concerns, and how to treat them without adverse effects. Meanwhile, poor diets, long-term use of gastric acid-reducing agents (proton-pump inhibitors, or PPIs), and celiac disease, all reduce vitamin D status and thus calcium absorption. When calcium is deficient, active vitamin D [1,25(OH)$_2$D, which is in fact, a hormone] starts to rob calcium from bones to maintain normal blood calcium levels. Thus, both adequate calcium and vitamin D nutrition are important.

For "healthy" people, the minimum level of serum 25(OH)D level required for optimal health appears to be 30 ng/mL (75 nmol/L) and the optimum range, 30 to 50 ng/mL. However, for special populations, such as institutionalized persons, PIDs, those with MCDD, and anyone receiving certain medications, such as anti-epileptic or psychotropic agents, a minimum level of 40 ng/mL (100 nmol/L) with the optimal range between 40 to 60 ng/mL is recommended. For most PIDD, this requires a daily vitamin D intake of between 2,000 and 4,000 International Units (IU) or taking 50,000 IU twice a month. The cost of this is only $10 to $15, per patient, per year.

PIDDs who reside in developmental disability centers routinely receive between 400 and 600 IU of vitamin D a day. However, at these low doses, only a very few of the residents will maintain healthy blood vitamin D levels. Suboptimal blood vitamin D status are common among certain other groups, including the obese, those with impaired gastrointestinal absorption of vitamin D, those taking anti-epileptic or psychotropic agents, and institutionalized persons, such as those in nursing homes, group homes, and developmental disability centers (less sun exposure). These groups of people require higher doses of oral vitamin D to maintain healthy serum vitamin D levels. However, a sensible and adequate sun exposure and an improved dietary and supplemental vitamin D intake prevent this global health issue.

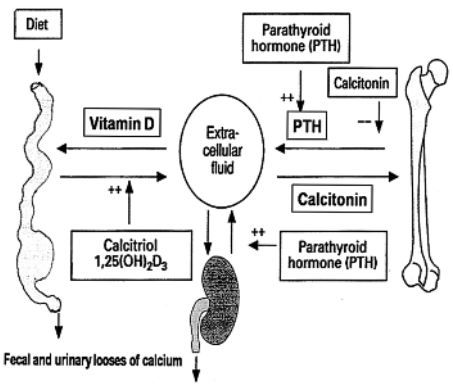
MAIN CAUSES OF VITAMIN D DEFICIENCY

There are four main causes of vitamin D deficiency: (A) lack of adequate skin exposure to sunlight (Note: less sun exposure is in part



attributable to skin color and clothing cover, concerns about skin cancer, climatic changes and atmospheric pollution, etc.), (B) less-than-adequate dietary intake (e.g., vegetarianism; other than fatty-fish, diet contain only little amounts of vitamin D), (C) enhanced catabolism of vitamin D (due to medications, especially glucocorticoids and anti-seizure medications), and (D) co-existing number of other diseases as described above. All four causes contribute to vitamin D deficiency in PIDD. In addition, lifestyle factors, obesity and changes in dietetic patterns also contribute to vitamin D deficiency. While, vitamin D deficiency aggravates a number of medical disorders, these conditions can be ameliorated by optimizing blood vitamin D status in a given person.

Inadequate vitamin D status impairs neuromuscular reflexes, leading to an increase in falls, injuries, and fractures among patients. Suboptimal vitamin D in the blood also causes poor bone calcification, leading to a softening of bones (rickets in children, osteomalacia in adults), osteoporosis, and increased fractures. In addition, vitamin D insufficiency causes worsening pain and increases the risk of viral and bacterial infections. In particular, infections such as tuberculosis and certain vial infections, including flu and most likely Ebola. In the case of the latter, treatment with high dose of vitamin D (e.g., 500,000 units orally, follow up with 50,000 IU per week for ten weeks) not only may prevent people getting this deadly infection, but also decrease the death rate from Ebola. Yet, it would only cost $10 per patient. Insufficient vitamin D is known to aggravate several aging-related morbidities, such as cardiovascular disease, cancer, and death.



CALCIUM METABOLISM DIAGRAMMED

The upper safe limit of vitamin D intake for healthy people is 10,000 IU a day, and toxicity does not develop unless serum 25(OH)D levels are far more than 100 ng/mL (which requires intake of vitamin D, more than 50,000 IU daily for long period). Therefore, there is no hazard in treating PIDD and MCDD using vitamin D dosages between 2,000 and 5,000 IU a day or 50,000 IU administered two to three times a month. The ability of the skin to produce vitamin D declines after the age 50 years in both men and women. Since the blood vitamin D levels decrease with age, the requirement for oral intake of vitamin D (food plus supplements) continues to increase after age 50.

Vitamin D facilitates the absorption of calcium from the intestine and helps to maintain bone health *(see the Calcium Metabolism diagram)*. The major biologic function of vitamin D is to maintain normal blood calcium and phosphorus levels, facilitating to maintain bodily metabolic functions. Vitamin D is essential for bone mineralization and maintenance, as well as proper neuromuscular function. Even subclinical vitamin D deficiency can precipitate or exacerbate osteoporosis through over-activation of the parathyroid glands leading to increased bone resorption.

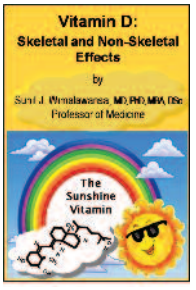
Rickets in children and osteomalacia in adults are manifestations of chronic, severe vitamin D deficiency, secondary that leads to inadequate intestinal calcium

absorption. This results in skeletal abnormalities, increased muscular weakness, falls, and fractures. Consequentially, compared with the healthy population, people with MCDD have a higher incidence of osteomalacia, falls and fractures.

Although the majority of persons with MCDD have low bone mass, this does not necessarily obligate treating all of them with potent anti-osteoporosis therapies such as bisphosphonates or denosumab, unless it is the main cause of their fractures. Most PIDD are vitamin D deficient and their bones are soft and brittle. However, when their vitamin D status is improved, the bone mineral density becomes markedly enhanced.

Most important of all, vitamin D deficiency must be fully rectified before treating them with any potent anti-osteoporosis pharmaceutical agent, including bisphosphonates or denosumab. These agents strongly inhibit bone resorbing osteoclast cells. If treated in the presence of vitamin D deficiency, these drugs can harm PIDD; leading to further softening of their bones (development of osteomalacia), as well as precipitating adverse clinical symptoms due to low bold calcium, and increasing fractures.

Having strong bones and good neuromuscular control are the keys to minimizing falls and fractures. Fall risks increase when other disorders are present, such as taking various medications (e.g., psychotropic drugs, diuretics, etc.), and in the presence of visual impairment, arthritis, orthopedic disabilities, nerve dysfunction, poor-coordination and muscle weakness. The severity of these can be decreased by correcting vitamin deficiency. Because of the relatively shorter half-life of vitamin D2, vitamin D3 is the preferred agent to give. However, to obtain its full benefits, it should be administered no less frequently than once a month.



Vitamin D: Everything You Need to Know
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Most relationships reported to date between serum 25(OH)D levels and non-skeletal disorders are associations and not proven yet to have a cause-effect relationships. In many patients, low serum 25(OH)D levels are a marker of poor nutrition, frailty, comorbidities, and ill health. Unfortunately, many conditions that worsen due to low vitamin D are labeled as "age-related" morbidities. Examples include high incidence of falls, muscle weakness, swallowing difficulty, overactive bladder, poor oral hygiene, decreased lung function, decline of cognitive function, and cancer. Vitamin D also reduces the risks and the severity of dental caries and periodontal disease, autism, and attention deficit-hyperactivity disorders.

Living far from the equator and inadequate exposure to sunlight leads to lower vitamin D status. It is associated with an increased incidence and severity of autoimmune disorders, such as multiple sclerosis and rheumatoid arthritis and heart attacks—as well as Gaucher's disease, Williams syndrome, Down syndrome, infectious diseases, insulin resistance, type 2 diabetes mellitus, cardiovascular disease, dementia, autism, depression, chronic fatigue syndrome, chronic pain syndrome, fibromyalgia, and anxiety and depression.

D DEFICIENCY: Suboptimal vitamin D in the blood causes poor bone calcification, leading to a softening of bones and rickets in children.

Overall, the current evidence confirms the benefit from calcium and adequate vitamin D supplementation with regard to osteoporosis and prevention of falls. In spite of some controversies, there is no cardiovascular or other harm, when the calcium intake does not exceed a total of 1.4 g a day (i.e., diet plus supplements; usually one calcium tablet per day is enough for most people) and the vitamin D supplement does not exceed 10,000 IU per day. However, giving vitamin D supplements at intervals of greater than once a month must be avoided.

CONCLUSION

In conclusion, considering all available data, we recommend a minimum serum/blood 25(OH) vitamin D level of 40 ng/mL (100 pmol/L) and the optimal range of 40 to 60 ng/mL, for PIDD/MCDD population. Recently, the vitamin D task-force of the American Association of Developmental Medicine and Dentistry (AADMD) also recommended 25(OH)D concentration between 40 to 60 ng/mL for the MCDD population.

Moreover, it has been estimated that having year-around serum 25(OH)D level between 40 to 60 ng/mL (100–150 nmol/L) would help prevent approximately 58,000 new cases of breast cancer and 49,000 new cases of colorectal cancer, each year in the United States. There is no downside to taking vitamin D3 equal to or less than 5,000 IU per day, so as increasing the population blood vitamin D status to greater than 40 ng/mL. Considering that most people including PIDD/MCDD are vitamin D deficient, the relatively high laboratory expenses and difficulties of obtaining blood from some PIDD, and the considerable safety of using vitamin D, the routine measurement of serum 25(OH)D levels is not necessary in most PIDD once an optimal stable serum vitamin D of 40 to 60 ng/mL is established. Vitamin D is one of the safest and the most cost-effective agent that physicians could prescribe. •

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