

# Vitamin D status and consequences of vitamin D deficiency

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**Abstract-** Serum 25-hydroxyvitamin D3 [25(OH)D3] concentrations are presently diagnosed because the useful reputed indicator for diet D. Evidence is reviewed that suggests that serum 25(OH)D3 concentrations of < eighty nmol/L are related to decreased calcium absorption, osteoporosis, and accelerated fracture risk. For popular older individuals, supplemental oral intakes of ~1300 IU/d are required to obtain the lower end of the best range. Evidence of considerable issues in recurring medical size of serum 25(OH)D3 concentrations amongst sufferers is cited. There is extremely good want for standardization and advanced reproducibility and sensitivity of measurements of serum 25(OH)D3 concentrations.

**Key words:** Vitamin D, osteoporosis, calcium absorption, fractures, serum 25-hydroxyvitamin D3.



Published in IJIRMP (E-ISSN: 2349-7300), Volume 2, Issue 5, Sep-Oct 2014

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## 1. INTRODUCTION

In its recent review of recommended bone-related nutrient intakes, the Food and Nutrition Board (FNB) identified serum 25-hydroxyvitamin D3 [25(OH)D3] concentrations as an appropriate functional index of vitamin D status [1]. However, based on the evidence available at the time, the FNB Calcium and Related Nutrients Group was unable to link specific serum concentrations of 25(OH)D3 with health conditions and different illnesses. Furthermore, due to the lack of necessary information, FNB again used the absence of rickets and osteomalacia as a de facto indicator of adequate vitamin D intake. Other health or disease-related consequences have not been considered in recommendations for vitamin D intake. Although much work remains to be done, enough information has been collected over the past 8 years to cover some of the information gaps that the Calcium and Related Nutrients Council encountered during its discussions in the mid-1990s. This brief overview highlights some aspects of this new information.

## 2. VITAMIN D AND DISEASE

Although rickets (in children) and osteomalacia (in adults) have long been considered manifestations of vitamin D deficiency, it is increasingly believed that milder deficiencies can also occur, causing bone disorders. The primary function of vitamin D is to facilitate the active transport component of intestinal calcium absorption, and there has never been evidence that absorption is optimal at adequate vitamin D concentrations. To prevent rickets or osteomalacia. In 1990, based on his extensive experience in histomorphometric analysis of adult bone specimens, Parfitt [2] introduced an important diagnostic reconceptualization of bone diseases caused by vitamin D, from which he coined the term vitamin D deficiency osteoporosis. He recognized 3 tiers of the ailment related to growing stages of vitamin D deficiency. In stage 1, the only detectable pathophysiological change is a decrease in intestinal calcium absorption, due to this reduces the amount of calcium stored in the bones and causes osteoporosis. On

biopsy, stage 1 bone showed no signs of osteomalacia. In stage 2 of vitamin D deficiency, as in stage 1, there is a decrease in intestinal calcium absorption and a decrease in bone mass, but early osteomalacia can be determined by biopsy, i.e. increased cover the bone of the bone and reduce the rate of mineral appointment. Patients with degree 2 sickness don't have any medical or laboratory symptoms and symptoms of osteomalacia. Its only clinical manifestation is decreased bone mass, i.e. osteoporosis. In stage 3 vitamin D deficiency, there is persistent calcium absorption and clinically evident osteomalacia. The importance of this redecorate is that the conventional index sickness for nutrition D deficiency has been certainly delineated as representing best the maximum intense diploma of deficiency out of . 25(OH)D3 of the patients who provided biopsy samples for analysis, Parfitt [2] modified into now no longer capable of quantitatively relate histhree ranges to unique values for what the FNB might later seek advice from as practical indicator . Parfitt's art work made it smooth that the then recommended each day dose for adults (200 IU/day), which modified into barely enough to save you medical osteomalacia, turned into inadequate to save you degree 1 or 2 nutrition D deficiency osteopathy to protect. Only now's it possible, as a minimum tentatively, to assign particular serum 25(OH)D3 concentrations to the boundary among sickness stage1 and the regular state, and to estimate the nutrition D consumption required to attain such concentrations on this delineation of regular and poor concentrations, it must be stated that a developing frame of proof summarized in different reviews of this symposium factors to A position for nutrition D now no longer handiest in calcium metabolism however additionally in a number of muscle and/or muscle and/or nutrition D ranges are indicative of neuromuscular features and withinside the manage of mobileular proliferation and differentiation (with implications for oncogenesis). [3], in a latest evaluation of facts from the National Health and Nutrition Examination Survey, confirmed that decrease extremity muscle feature stepped forward with improved serum concentrations of 25(OH)D3 , as a minimum at degrees withinside the 80-one hundred nmol/L range . Estimates of optimal serum 25(OH)D3 concentrations for such health outcomes may soon be available.

### **3. DEFINITION OF CRITICAL VALUES FOR SERUM 25(OH)D3**

Serum 25(OH)D3 refers to the measurement of the circulating level of 25-hydroxyvitamin D3 in the blood, which is the primary circulating form of vitamin D. It is an important marker used to assess an individual's vitamin D status. The critical values for serum 25(OH)D3 typically refer to specific cutoff points that indicate different levels of vitamin D deficiency or sufficiency. These values can vary slightly depending on the laboratory and the reference range used, but the following general ranges are commonly used:

**Deficient:** A serum 25(OH)D3 level below 20 ng/mL (nanograms per milliliter) is often considered deficient. This indicates a severe deficiency of vitamin D and may be associated with an increased risk of developing conditions such as rickets in children or osteomalacia in adults.

**Insufficient:** Serum 25(OH)D3 levels between 20-30 ng/mL are often considered insufficient. While not as severe as a deficiency, this range suggests that an individual may have inadequate vitamin D levels and could benefit from increasing their intake or exposure to sunlight.

**Sufficient:** Serum 25(OH)D3 levels between 30-50 ng/mL are generally considered sufficient for most individuals. This range is associated with optimal bone health and overall well-being.

**Optimal:** Some experts argue that an optimal range for serum 25(OH)D3 levels is between 40-60 ng/mL or even higher. However, there is ongoing debate and scientific discussion regarding what constitutes the ideal level of vitamin D.

It's important to note that these critical values are not universally agreed upon, and different organizations or medical professionals may use slightly different cutoffs. Additionally, individual requirements for vitamin D may vary based on factors such as age, health status, and geographical location.

The publication of a large British vitamin D intervention study in 2003 handed the important substantiation demanded. Serum 25(OH)D3 attention were measured for a group of the cohort and equaled 53 nmol/L for the placebo group and 74 nmol/L for the vitamin D-treated group. Fracture threat bones were reduced by 22

of them. The group reviewed had a 33 reduction in those treated with the supplement and typical osteoporotic fractures. studies were within the reference range; In fact, the two studies included nearly the same range of values( 50 and 53) nmol/ L( for undressed subjects in the 2 studies and 74 and 86 nmol/ L for undressed subjects). subject being treated). These lately published studies easily demonstrate calcium malabsorption and increased fracture threat at serum 25( OH) D3 attention below 80 nmol/ L 1 and 2 and give quantitative references that Parfitt didn't have when he proposed his bracket system for vitamin D- related conditions. also, these results punctuate the counteraccusations concern of increased PTH situations at 25( OH) D3, 80nmol/ L. The primary cause of bone conditions in grown-ups is serum 25( OH) D3 situations shown in Figure 1.

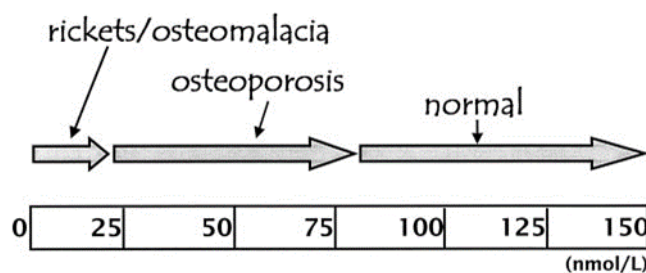


FIGURE 1. Suggested mapping of the principal vitamin D-related bone diseases onto the serum 25(OH)D3 concentration continuum. (To convert values to nanograms per milliliter, divide values by 2.5.)

## 1.REPLACEMENT OF MEASURED VITAMIN D DEFICIENCIES

General experience in the field shows that administration of vitamin D in amounts within the current appropriate intake range (defined by FNB as 200 to 600 IU/day) does not produce a significant increase in measured intake. Okay. . D3 concentration, indicates that the effectiveness of the preparations used is not sufficiently effective or that the need is greater than that implied by the concept of adequate absorption. Therefore, my colleagues and I [18] attempted to quantify both daily vitamin D intake and the amount required to achieve the desired increase in serum 25(OH)D3 concentration. Vitamin D approaches 4000 IU (100 µg) and at steady state, serum 25(OH)D3 concentrations increase by 0.7 nmol/L for every 1 µg (40 IU) of vitamin D3 taken orally as a normal dose. usually daily. Several other studies have provided data to calculate this rate of increase; they usually give similar slope values, i.e. between 0.6 and 1.2 nmol/L for 1 µg/day [17, 19, 20]. Trivedi et al. [17] showed an increase of almost exactly 1 nmol/L per 1 µg/day. By taking a value in the middle of the range of observed slopes (e.g.g. 0.9 nmol/L for 1 µg/day), it can be calculated that the recommended daily dose for adults 50 to 70 years of age (400 IU) will only increase serum 25(OH)D3 concentration by 9 nmol /l (3.6 ng/ ml). Since this increase is erroneous with most laboratory methods, it is now clear why the use of such doses does not produce a significant increase in 25(OH)D3 concentrations in the blood. serous. There is reason to believe that the rate of increase may be much faster in more fatigued people than in participants in our study or the British study, and my colleagues and I [20] have reviewed several published studies and showed that the response to a given dose may be an inverse function of the initial 25(OH)D3 concentration. However, when a moderate level of vitamin D replacement is achieved, a gradient within the range just mentioned is applied and determines the final amount of vitamin D that must be administered to achieve the desired level.

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µg/d [17, 19, 20]. Le Trivedi et al. [17] performed an antifracture test that showed an increase of almost exactly 1 nmol/L per 1 µg/day. By taking a value in the middle of the observed slope range (e.g.g. 0.9 nmol/l for 1 µg/day), it can be calculated that the recommended daily dose for adults aged 50 to 70 years should be achieved at age (400 IU) increases serum 25(OH)D<sub>3</sub> is only 9 nmol/L (3.6 ng/mL). Since this increase is a failure of most laboratory methods, it is now clear why the use of such doses does not cause a significant increase in 25(OH)D<sub>3</sub> concentrations in serum. There is reason to believe that the rate of increase in subjects with severe fatigue may be much faster than in participants in our study or the British study, and my colleagues and I [20] have previously published reviews of several studies showing that the response to a given dose of may be an inverse function of the initial concentration of 25(OH)D<sub>3</sub>. However, when a moderate level of vitamin D replacement is achieved, a gradient within the range just mentioned appears to apply and govern the amount of vitamin D that must ultimately be administered to achieve the desired level. values.

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