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Vitamin D is a fat-soluble vitamin that is produced endogenously in the skin when ultraviolet rays from sunlight trigger its synthesis. For most people in the world about 90% of vitamin D is produced this way, whereas the remaining 10% is obtained from food and dietary supplements [1,2]. However, for people residing in high latitudes is the diet important to get enough vitamin D. Research has shown that increased skin pigment reduces the capacity of skin to synthesize vitamin D [3]. The main sources of vitamin D in the Norwegian diet are fatty fish, fortified butter and margarine [4], in addition to cod liver oil [5].

The vitamin D that is produced in the skin or obtained from the diet and supplements is biologically inert in the body, and must undergo two hydroxylations to be activated. The first hydroxylation occurs in the liver and converts vitamin D to 25-hydroxy vitamin D (25(OH)D, calcidiol). Next, the kidneys hydroxylate this metabolite to 1,25-dihydroxy vitamin D (1,25(OH)2D, calcitriol), the biologically active form of vitamin D. Many cells in other organ systems in the human body have vitamin D receptors, and some of these also convert 25(OH)D to 1,25(OH)2D [6]. The concentration of 25(OH)D measured in blood samples is a recognized measure of vitamin D status. The half-life is a few weeks [7].

Vitamin D deficiency is worldwide a prevalent health problem and has health impacts on about one billion people [8]. Deficiency or insufficiency of vitamin D is common in the US population, mainly because of inadequate dietary intake, sedentary lifestyles, and reduced sun exposure [2,9,10].

It has been estimated that  $\geq 20$  ng/mL is the serum level of 25(OH)D that covers the vitamin D needs of 97.5% of the population [11,12]. Serum levels less than this are considered as a vitamin D deficiency while levels  $< 30$  ng/mL is viewed as insufficient (Ross 2011, Wacker and Holick 2013). Some researchers have considered that 50 or 75 ng/mL is an optimal level [1,9,13].

Vitamin D promotes calcium absorption in the gut [14], maintains adequate serum calcium and phosphate concentrations, and enables a normal mineralization of bone. It is very important for bone growth, bone remodeling by osteoblasts and osteoclasts, and neuromuscular function [11,15]. A sufficient level of vitamin D in the body prevents hypocalcemic tetany [16], rickets in children, and osteomalacia in

## Short Communication

# Vitamin D Deficiency: A Global Health Problem

### Abstract

Vitamin D deficiency is a global health problem. The extent of vitamin D deficiency varies with latitude, season and sun exposure. Also the degree to which the body of religious or cultural reasons is covered, the skin color and, not least, dietary habits and the use of supplements has an impact on the vitamin D status. Women are more prone to develop vitamin D deficiency than men.

adults [17,18]. Together with calcium, vitamin D also helps to protect older adults from osteoporosis [11,19].

Vitamin D plays an important role in brain homeostasis, neurodevelopment, immunological modulation, aging, and also, importantly, in gene regulation [20,21]. It binds to more than 2700 genes and regulates the expression of more than 200 of them [22,23].

Research indicates a possible connection between vitamin D and a broad range of non-skeletal disorders, including dementia, autism, schizophrenia, depression, muscle pain, cardiovascular disease, diabetes, multiple sclerosis, cancer, and infections, and all-cause mortality [21,23-29].

Research has shown that vitamin D deficiency occurs in all parts of the world [2,9,10,30]. There is evidence that significant vitamin D deficiency (<25 nmol/L) is very common in all age groups in South Asia and the Middle East [31]. In a Canadian study it was found an average level of 24.6 nmol/L in female immigrants from the Middle East [32]. In a study from the United Arab Emirates had 84% of men and 89% of women vitamin D insufficiency in the winter period. In the same study had 30% of men and 46% of women vitamin D deficiency [33].

Gender was in a recent Jordanian study found significantly associated with vitamin D deficiency [30]. More females than males in the study had vitamin D deficiency. Other studies have also shown that women more often than men develop vitamin D deficiency [33-35]. Childbearing women and women that have given birth to many children, as well as those who of religious or cultural reasons cover the whole or parts of the body when they go out, or are mostly staying inside, are particularly vulnerable to develop vitamin D deficiency [35].

### References

1. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, et al. (2011). Evaluation, treatment, and prevention of vitamin D deficiency: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab* 96: 1911–1930.
2. Mahmoud AA, Ali AH (2014). Vitamin D receptor gene polymorphism and 25 hydroxy vitamin D levels in Egyptian patients with pulmonary tuberculosis. *Egypt J Chest Dis Tuberc* 63: 651–655.
3. Matsuoka LY, Wortsman J, Haddad JG, Kolm P, Hollis BW (1991) Racial



- pigmentation and the cutaneous synthesis of vitamin D. *Arch Dermatol* 127: 536-538.
4. Løken-Amsrud KI, Holmøy T, Bakke SJ, Beiske AG, Bjerve KS, et al. (2012) Vitamin D and disease activity in multiple sclerosis before and during interferon- $\beta$  treatment. *Neurology* 79: 267-273.
  5. Saltyté Benth J, Myhr KM, Løken-Amsrud KI, Beiske AG, Bjerve KS, et al. (2012) Modelling and prediction of 25-hydroxyvitamin D levels in Norwegian relapsing-remitting multiple sclerosis patients. *Neuroepidemiology* 39: 84-93.
  6. Holick MF (2003) Vitamin D: A millenium perspective. *J Cell Biochem* 88: 296-307.
  7. Jones G (2008) Pharmacokinetics of vitamin D toxicity. *Am J Clin Nutr* 88: 582S-586S.
  8. Holick MF (2010). The vitamin D deficiency pandemic: a forgotten hormone important for health. *Public Health Rev* 32: 267-283.
  9. Weng FL, Shults J, Leonard MB, Stallings VA, Zemel BS (2007) Risk factors for low serum 25-hydroxyvitamin D concentrations in otherwise healthy children and adolescents. *Am J Clin Nutr* 86: 150-158.
  10. Wacker M, Holick MF (2013) Vitamin D - effects on skeletal and extraskeletal health and the need for supplementation. *Nutrients* 5: 111-148.
  11. Ross AC (2011) The 2011 report on dietary reference intakes for calcium and vitamin D. *Public Health Nutr* 14: 938-939.
  12. Letter to Veugelers, P.J. and Ekwuru, J.P., Heaney R, Garland C, Baggerly C, et al. (2014) A statistical error in the estimation of the recommended dietary allowance for vitamin D. *Nutrients* 6: 4472-4475.
  13. Lips P (2007) Relative value of 25(OH)D and 1,25(OH)2D measurements. *J Bone Miner Res* 22: 1668-1671.
  14. Gawlik A, Gepstein V, Rozen N, Dahan A, Ben-Yosef D, et al. (2015) Duodenal expression of 25 hydroxyvitamin D3-1 $\alpha$ -hydroxylase is higher in adolescents than in children and adults. *J Clin Endocrinol Metab*. 100: 3668-3675.
  15. Cranney A, Horsley T, O'Donnell S, Weiler H, Pui L, et al. (2007) Effectiveness and safety of vitamin D in relation to bone health. *Evid Rep Technol Assess (Full Rep)* 158: 1-235.
  16. Lu YY, Wu JF, Ni YH, Peng SS, Shun CT, et al. (2009) Hypocalcemia and tetany caused by vitamin D deficiency in a child with intestinal lymphangiectasia. *J Formos Med Assoc* 108: 814-818.
  17. Goldring SR, Krane S, Avioli LV (1995) Disorders of calcification: osteomalacia and rickets. In: DeGroot LJ, Besser M, Burger HG, Jameson JL, Loriaux DL, Marshall JC, et al., eds. *Endocrinology*. 3rd ed. WB Saunders, Philadelphia. P. 1204-1227.
  18. Reid IR (2015) What diseases are causally linked to vitamin D deficiency? *Arch Dis Child*. 2015 Jul 22. pii: archdischild-2014-307961.
  19. Drake MT, Clarke BL, Lewiecki EM (2015) The pathophysiology and treatment of osteoporosis. *Clin Ther* 37: 1837-1850.
  20. Yang S, Smith C, Prahl JM, Luo X, DeLuca HF (1993) Vitamin D deficiency suppresses cell-mediated immunity in vivo. *Arch Biochem Biophys* 303: 98-106.
  21. Saad K, Abdel-Rahman AA, Elserogy YM, Al-Atram AA, Cannell JJ, et al. (2015) Vitamin D status in autism spectrum disorders and the efficacy of vitamin D supplementation in autistic children. *Nutr Neurosci*. 2015 Apr 15. [Epub ahead of print].
  22. Ramagopalan SV, Heger A, Berlanga AJ, Maugeri NJ, Lincoln MR, et al. (2010) A ChIP-seq defined genome-wide map of vitamin D receptor binding: associations with disease and evolution. *Genome Res* 20: 1352-1360.
  23. Kočovská E, Fernell E, Billstedt E, Minnis H, Gillberg C (2012) Vitamin D and autism: clinical review. *Res Dev Disabil* 33: 1541-1550.
  24. Norman AW, Henry HH (2006) Vitamin D. In: Bowman BA, Russell RM, eds. *Present Knowledge in Nutrition*, 9th ed. ILSI Press, Washington, DC.
  25. Villamor E (2006) A potential role for vitamin D on HIV infection? *Nutr Rev* 64: 226-233.
  26. Lamberg-Allardt C, Brustad M, Meyer HE, Steingrimsdottir L (2013) Vitamin D - a systematic literature review for the 5th edition of the Nordic Nutrition Recommendations. *Food Nutr Res* 57.
  27. Autier P, Boniol M, Pizot C, Mullie P (2014). Vitamin D status and ill health: a systematic 413 review. *Lancet Diabetes Endocrinol* 2: 76-89.
  28. Shrivakumar V, Kalmady SV, Amaresha AC, Jose D, Narayanaswamy JC, et al. (2015) Serum vitamin D and hippocampal gray matter volume in schizophrenia. *Psychiatry Res* 233: 175-179.
  29. Zhu DM, Liu Y, Zhang AG, Chu ZX, Wu Q, Li H, et al. (2015) High levels of vitamin D in relation to reduced risk of schizophrenia with elevated C-reactive protein. *Psychiatry Res* 228: 565-570.
  30. Kassab M, Alkhatib AJ, AbuBaker A, Shotar A, Bjørklund G (2015) Vitamin D deficiency and its associated predictors among patients visiting primary health care center. *Res J Biol Sci* 10: 78-80.
  31. Mithal A, Wahl DA, Bonjour JP, Burckhardt P, Dawson-Hughes B, Eisman JA, et al. (2009) Global vitamin D status and determinants of hypovitaminosis D. *Osteoporos Int* 20: 1807-1820.
  32. Aucoin M, Weaver R, Thomas R, Jones L (2013) Vitamin D status of refugees arriving in Canada. *Can Fam Physician* 59: e188-e194.
  33. Abdel-Wareth L, Haq A, Turner A, Khan S, Salem A, et al. (2013) Total vitamin D assay comparison of the Roche Diagnostics "Vitamin D total" electrochemiluminescence protein binding assay with the Chromsystems HPLC method in a population with both D2 and D3 forms of vitamin D. *Nutrients* 5: 971-980.
  34. Holvik K, Meyer HE, Haug E, Brunvand L (2005) Prevalence and predictors of vitamin D deficiency in five immigrant groups living in Oslo, Norway: the Oslo Immigrant Health Study. *Eur J Clin Nutr* 59: 57-63.
  35. Erkal MZ, Wilde J, Bilgin Y, Akinci A, Demir E, et al. (2006) High prevalence of vitamin D deficiency, secondary hyperparathyroidism and generalized bone pain in Turkish immigrants in Germany: identification of risk factors. *Osteoporos Int* 17: 1133-1140.

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