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## RESEARCH ARTICLE

# SERUM LEVEL OF VITAMIN D3 WIDAD KADHIM MATROOD (B.SC. DIP BIOCH), AL- YARMOUK TEACHING HOSPITAL, BAGHDHD, IRAQ

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**ABSTRACT**

**Objective:-** To measure the level of the vitamin D3**. Patients and Methods:-** blood is typically collected from 100 patients via venipuncture and more recently "blood spots" from a simple finger prick from the individual being assessed. The serum (from venipuncture) or blood (from blood spot) is isolated and used in the assay being employed. There are then a number of methods to assess 25(OH)D to determine vitamin D status. **Results:-** One hundred patients were studied, 50 female (50%) and 50 male (50%), and female to male ratio was (1: 1). The age ranged from 10 years to 80 years, with a mean age of 40 years + 5 years. The majority being in the fifth decade of life constituting 27 patients (27%) . Also our study showed that the lower level of VITAMIN D3 is 4 ng /dl and the higher level is 45 ng/ dl and the mean level is 19.4 ng/ dl.



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## INTRODUCTION

Vitamin D is a group of fat-soluble secosteroids responsible for increasing intestinal absorption of calcium, magnesium, and phosphate, and multiple other biological effects (Holick, 2004). In humans, the most important compounds in this group are vitamin $D_3$ (also known as cholecalciferol) and vitamin $D_2$ (ergocalciferol) (Calvo, 2005). Cholecalciferol and ergocalciferol can be ingested from the diet and from supplements (Calvo, 2005; Vitamin, 2013; Hollis, 1996). Only a few foods contain vitamin D. The major natural source of the vitamin is synthesis of cholecalciferol in the skin from cholesterol through a chemical reaction that is dependent on sun exposure (specifically Ultra Violet B radiation). Dietary recommendations typically assume that all of a person's vitamin D is taken by mouth, as sun exposure in the population is variable and recommendations about the amount of sun exposure that is safe are uncertain in view of the skin cancer risk (Holick *et al*., 1971). Vitamin D from the diet or skin synthesis is biologically inactive; enzymatic conversion (hydroxylation) in the liver and kidney is required for activation. As vitamin D can be synthesized in adequate amounts by most mammals exposed to sufficient sunlight, it is not an essential dietary factor, and so not technically a vitamin (Hollis, 1996).

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Instead it could be considered as a hormone, with activation of the vitamin D pro-hormone resulting in the active form, calcitriol, which then produces effects via a nuclear receptor in multiple different locations (Hollis, 1996). Cholecalciferol is converted in the liver to calcifediol (25-hydroxycholecalciferol); ergocalciferol is converted to 25-hydroxyergocalciferol. These two vitamin D metabolites (called 25-hydroxyvitamin D or 25(OH)D) are measured in serum to determine a person's vitamin D status (Wolf, 2004; Pittas *et al*., 2010). Calcifediol is further hydroxylated by the kidneys to form calcitriol (also known as 1,25-dihydroxycholecalciferol), the biologically active form of vitamin D (Chung *et al*., 2009). Calcitriol circulates as a hormone in the blood, having a major role regulating the concentration of calcium and phosphate, and promoting the healthy growth and remodeling of bone. Calcitriol also has other effects, including some on cell growth, neuromuscular and immune functions, and reduction of inflammation.(5) Vitamin D has a significant role in calcium homeostasis and metabolism. Its discovery was due to effort to find the dietary substance lacking in children with rickets (the childhood form of osteomalacia) (Bjelakovic *et al*., 2014). Vitamin D supplements are given to treat or to prevent osteomalacia and rickets, but the evidence for other health effects of vitamin D supplementation in the general population is inconsistent (Bolland *et al*., 2014; Dorland's Illustrated Medical Dictionary, under Vitamin (Table of Vitamins)). The effect of vitamin D supplementation on mortality is not clear, with one meta-analysis finding a small decrease in mortality in elderly people

(About Vitamin, 2015), and another concluding no clear justification exists for recommending supplementation for preventing many diseases, and that further research of similar design is unneeded in these areas (Holick, 2007). Several forms (vitamers) of vitamin D exist. The two major forms are vitamin $D_2$ or ergocalciferol, and vitamin $D_3$ or cholecalciferol; vitamin D without a subscript refers to either $D_2$ or $D_3$ or both. These are known collectively as calciferol (Gordon *et al*., 2004). Vitamin $D_2$ was chemically characterized in 1931. In 1935, the chemical structure of vitamin $D_3$ was established and proven to result from the ultraviolet irradiation of 7-dehydrocholesterol (Lips *et al*., 2006). Chemically, the various forms of vitamin D are secosteroids, i.e., steroids in which one of the bonds in the steroid rings is broken (Lips *et al*., 2006). The structural difference between vitamin $D_2$ and vitamin $D_3$ is the side chain of $D_2$ contains a double bond between carbons 22 and 23, and a methyl group on carbon 24 (Lips *et al*., 2006). Vitamin D insufficiency affects almost 50% of the population worldwide (Rostand, 1997). An estimated 1 billion people worldwide, across all ethnicities and age groups, have a vitamin D deficiency (VDD) (Rostand, 1997; Melamed *et al*., 2008).

This pandemic of hypovitaminosis D can mainly be attributed to lifestyle and environmental factors that reduce exposure to sunlight, which is required for ultraviolet-B (UVB)-induced vitamin D production in the skin. Black people absorb more UVB in the melanin of their skin than do white people and, therefore, require more sun exposure to produce the same amount of vitamin D (Autier, 2007). The high prevalence of vitamin D insufficiency is a particularly important public health issue because hypovitaminosis D is an independent risk factor for total mortality in the general population (Heaney, 2011). Emerging research supports the possible role of vitamin D against cancer, heart disease, fractures and falls, autoimmune diseases, influenza, type-2 diabetes, and depression. Many health care providers have increased their recommendations for vitamin D supplementation to at least 1000 IU. [21] A meta-analysis published in 2007 showed that vitamin D supplementation was associated with significantly reduced mortality.[22] In this review, we will focus on the biology of vitamin D and summarize the mechanisms that are presumed to underlie the relationship between vitamin D and its clinical implications.[22]

**Patients and Methods:-** blood is typically collected from 100 patients via venipuncture and more recently "blood spots" from a simple finger prick from the individual being assessed. The serum (from venipuncture) or blood (from blood spot) is isolated and used in the assay being employed. There are then a number of methods to assess 25(OH)D to determine vitamin D status. Commercially available assays include: 1-High pressure liquid chromatography mass spectrometry (LC-MS/MS) 2-Radioimmunoassay (RIA) 3-Enzyme immunoassay (EIA) 4-Competitive protein binding assay (CPBA) 5-Automated chemiluminescent protein binding assay (CLPBA) 6-Chemiluminescent immunoassay (CLIA) The normal level of the vitamin is 30-100 ng/dl, and the insufficient is 10- 30 ng/dl, and the deficient is less than 10 ng/dl.

## RESULTS

One hundred patients were studied, 50 female (50%) and 50 male (50%) as shown in Table 1, and female to male ratio was (1: 1).

The age ranged from 10 years to 80 years, with a mean age of 40 years + 5 years. The majority being in the fifth decade of life constituting 27 patients (27%) as shown in Table 2. Also our study showed that the lower level of VITAMIN D3 is 4 ng /dl and the higher level is 45 ng/ dl and the mean level is 19.4 ng/ dl.

**Table 1. SEX distribution of patients**

| SEX | TOTAL | % |
|---|---|---|
| FEMALE | 50 | 50% |
| MALE | 50 | 50% |

**Table 2. AGE distribution of patients**

| Age group (Years) | No of patient | % |
|---|---|---|
| 1 – 10 | 0 | 0 |
| 11 -20 | 15 | 15% |
| 21 – 30 | 15 | 15% |
| 31 – 40 | 20 | 20% |
| 41- 50 | 27 | 27% |
| 51-60 | 14 | 14% |
| 61-70 | 6 | 6% |
| 71-80 | 3 | 3% |
| Total | 100 | 100% |

## DISCUSSION

OUR study showed that 14 patients have less than 10 ng/ dl, 59 patients have vitamin level between 10-30 ng/dl, and 27 have vitamin level between 30-100 ng/ dl. However, it is note worthy that numerous scientific researchers disagree with the guidelines and propose that they are too conservative and outdated. From available literature and our own findings, it is evident that there is great inter-individual variation in basal vitamin D concentrations in participants tested at the same point of the year. Before discussion of mechanisms to improve vitamin D status, there are a number of considerations that must be taken into account. As with the measurement units used for circulating metabolites of vitamin D, there is more than one unit of measurement for supplemental vitamin D. It is extremely important to distinguish between international units (IU) and µg. One hundred IU = 2.5 µg vitamin D2/D3. Clearly, confusing these two could result in extremely large or very low and ineffective doses of vitamin D. A further consideration before supplementation is whether vitamin D2 or D3 is more effective.

Quite simply, vitamin D3 is approximately 87% more potent in raising and maintaining serum 25(OH)D concentrations and produces 2- to 3-fold greater storage of vitamin D than does an equimolar amount of D2.[23] Data suggests vitamin D deficiency is endemic and its frequency is on the rise, a consistent observation in athletic sub-groups. This may primarily be owing to a sun-shy lifestyle and poor dietary sources of vitamin D. The cost of vitamin D deficiency is sub-optimal biological function in many tissues and therefore measurement is necessary in athletes, particularly during the winter months when sun exposure is low. Appropriate biochemical methods for vitamin D assessment should be adopted and correct interpretation of results implemented. The current RDI for vitamin D (600 IU/d) is unlikely to be efficacious to prevent deficiency in the absence of sun exposure. If necessary (upon presentation of serum 25(OH)D <75 nmol/L) it is advisable to supplement with oral vitamin D3 with doses reflecting the necessity of change required in serum 25(OH)D.

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