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Chapter

Vitamin D and Its Deficiency in Saudi Arabia

Fawzi F. Bokhari and Mai Albaik

Abstract

Vitamin D is a hot topic that has attracted attention over the past 10 years, especially since a large proportion of people suffer from this nutrient deficiency. Vitamin D deficiency is estimated to be about 1 billion people all over the world and 50% in Asia and the Middle East. Saudi Arabia has also demonstrated a high prevalence of vitamin D deficiency among healthy Saudi individuals. This chapter provides, in detail, a clear and understandable identification of vitamin D, its function, source, synthesis, metabolism, status, and deficiency. The chapter also focuses on studying vitamin D deficiency in Saudi Arabia based on PubMed's initial research criteria.

Keywords: vitamin D, function, synthesis, deficiency, Saudi Arabia

1. Introduction

Vitamin D, "sunshine" vitamin, is a vital topic that has attracted great attention of many researchers and the public over the past decades, because a large proportion of the world's population is deficient in this nutritious element [1]. Vitamin D was first discovered at the beginning of the twentieth century in children with rickets [2].

Vitamin D is a prohormone steroid and belongs to the fat-soluble vitamins. It is responsible for endocrine, paracrine, and autocrine functions [1]. Vitamin D is also essential for calcium absorption, bone mineralization, calcium and phosphorus homeostasis, hormonal release, nerve conduction, and neuromuscular function [3, 4].

2. Function of vitamin D

Vitamin D is known for its crucial role in bone health about a century ago. However, it has also demonstrated its role and effectiveness in extra-skeletal roles of vitamin D during the past two decades [5].

2.1 Vitamin D and bone health

Vitamin D regulates physiological functions by controlling the metabolism of calcium and phosphates, stimulates growth, and promotes the necessary remodeling of bones and teeth [6]. Vitamin D deficiency is often associated with bone disorders (such as rickets, osteomalacia, and osteoporosis); when serum calcium decreases, the thyroid gland immediately releases parathyroid hormone (PTH),

which acts by stimulating bone reabsorption and reduction of calcium urinary excretion [7].

Vitamin D levels are positively correlated with bone mineral density (BMD) [4]. Many observational studies have reported relations between chronic lower vitamin D concentrations and poorer lower-extremity function, lower muscle strength, lower contraction speed, and lower appendicular muscle mass [8]. Vitamin D deficiency can put people at risk because of low bone mineral density, osteopenia, osteoporosis, and tooth loss [6].

2.2 Vitamin D and non-skeletal diseases

Observational studies have shown associations between the low concentration of serum vitamin D and increased risk of cancer, cardiovascular diseases, disorders of glucose metabolism, neurodegenerative diseases, and mortality [3]. Vitamin D modulates a variety of processes and regulatory systems including host defense, inflammation, and immunity and repair, especially patients with lung diseases often have low vitamin D serum level [9].

The biological effect of vitamin D on cardiac function is through reduced remodeling and fibrosis secondary to negative regulation of renin by vitamin D receptor (VDR)-linked gene regulation and through reduced cardiac metalloproteinase activities [10]. In addition, many indications support a relation between hypovitaminosis D and slower nerve conduction and poorer executive functions [8]. VDR are also expressed on immune cells (T and B cells, monocytes/macrophages, mast cells, and antigen-presenting cells) [10]. Moreover, vitamin D may exert positive effects on oral health by affecting the production of antimicrobial peptides [6].

Furthermore, many studies have demonstrated that vitamin D supplementation has a beneficial effect in decreasing the mortality rate under multiple factors, by influencing the cardiovascular system, immune system, tumor progression, and others [11].

3. Source of vitamin D

The main sources of vitamin D are our diet, supplementation, and sun exposure [2].

Two dominant forms of dietary vitamin D are vitamins D_2 (ergocalciferol) and D_3 (cholecalciferol) [12]. Vitamin D_2 is produced by plants and invertebrates after ultraviolet radiation exposure [13]. Vitamin D_3 is naturally found in many foods (such as oily fish, egg yolks, cod liver oil, cheese, mackerel, salmon, tuna fish, and beef liver), fortified foods (margarine, breakfast cereals, dairy products, orange juice), and vitamin supplements (both vitamins D_2 and D_3 are available) [1, 14, 15]. Dietary vitamin D provides only 10–20% of circulating levels of vitamin D [13].

The chemical structure of these vitamins (D_2 and D_3) is similar but differs only in their side chains (**Figure 1**). This structural difference modifies their binding to carrier protein vitamin D binding protein (DBP) and their metabolism [16]. Vitamin D_3 is significantly demonstrated more effective than D_2 in increasing serum 25-hydroxyvitamin D [25(OH)D] concentrations due to several reasons including reduced vitamin D_2 binding and metabolites to DBP in plasma, a non-physiological metabolism, and a shorter shelf life of vitamin D_2 [13]; therefore vitamin D_3 is considered the preferred choice for supplementation [14].

Sun exposure is the chief source of vitamin D via the synthesis in the skin through the action of ultraviolet B (UVB) radiation on the precursor of vitamin D_3 [4, 17]. The Commission Internationale de l'Eclairage (CIE) described the efficiency of vitamin D

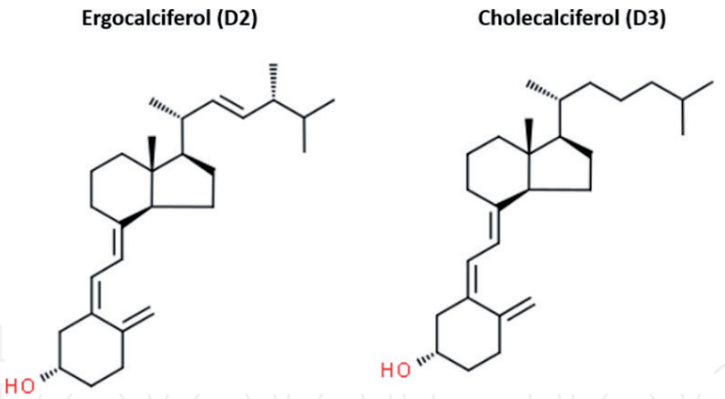

Figure 1.
The structure of vitamins (D2 and D3).

radiation as the efficiency of each wavelength to synthesize vitamin D in the skin. The CIE suggests the efficiency of UVB radiation that covers the spectral range (255–330 nm) with a maximum at about 295 nm [1]. A whole-body exposure to UVB radiation inducing the light pink color of the minimal erythema dose for 15–20 min is able to induce the production of up to 250 μg vitamin D (10,000 IU) [1, 6].

4. Synthesis and metabolism

Vitamin D, either endogenously produced (vitamin D_3) or ingested (vitamin D_2 or vitamin D_3), must be activated in order to produce its effects [5]. This biological activation is performed in a multi-step process (**Figure 2**).

Firstly, UVB radiation penetrates the epidermis and stimulates the conversion of 7-dehydrocholesterol (7-DHC) into pre-vitamin D3 [18] which undergoes thermal isomerization through a sigmatropic hydride shift into vitamin D_3 [13].

Secondly, vitamin D_2 or D_3 is specifically translocated by DBP into circulation and then to the liver for hydroxylation at carbon-25 to form 25-hydroxyvitamin D [25(OH)D] mainly by two cytochrome P-450 enzymes (CYP2R1 and CYP27A1) [5].

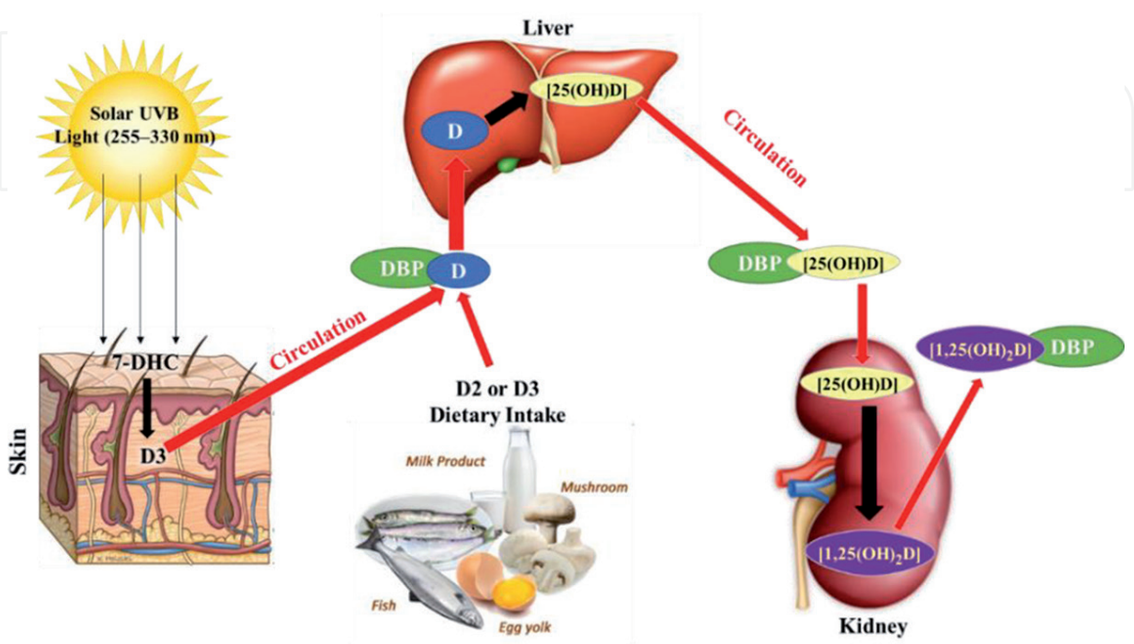

Figure 2.
A diagram illustrating the different sources and synthesis of vitamin D.

[25(OH)D] is an inactive and the most abundant circulating form of vitamin D, and it is generally measured when assessing vitamin D status which has a circulating half-life of about 15 days [2].

Thirdly, 1,25-dihydroxyvitamin D [1,25$(OH)_2$D], the biologically active form of vitamin D, is generated through second hydroxylation that takes place in the kidney [6] by the enzyme cytochrome P-450 (CYP27B1) monooxygenase 25(OH)D-1-α-hydroxylase [13] [1,25$(OH)_2$D], which serves as a hormone to regulate a variety of cellular functions in other organs or acts inside the kidneys in an autocrine and/or paracrine fashion [5]. Several factors regulate the levels of [1,25$(OH)_2$D], 25(OH) D-1-α-hydroxylase (whose hydroxylation is activated by PTH), calcitonin (which is inhibited by serum levels of calcium), phosphorus, and [1,25$(OH)_2$D] itself [19].

Finally, [1,25$(OH)_2$D] enters the cell by diffusion and binds and activates the VDR [20].

5. Vitamin D receptor (VDR)

VDR is a member of the nuclear hormone receptor superfamily and acts as a ligand-inducible transcription factor as well as its non-genomic actions outside of the nucleus [18, 20]. VDRs are present mostly in body organs such as the colon, small intestine, bone, breast, brain, pancreas, pituitary, and muscles [12].

The widespread distribution of VDRs and production of calcitriol may interpret the increasing number of diseases related to vitamin D deficiency [12]. Binding of calcitriol to VDR prompts the transcription of vitamin D-responsive genes (at least 913 genes) involved in cell proliferation, differentiation, function, and the renin-angiotensin system [2, 21].

VDR forms a heterodimer complex with the retinoid X receptor (RXR) capable of binding to a vitamin D response element (VDRE) in the promoter region of a target gene and thereby regulates transcription of more than thousand genes [20].

6. Factors affecting vitamin D synthesis

Many factors affect vitamin D synthesis and its concentration [1, 3, 6, 11, 17, 19, 20]: aging (age decreases the capacity of the skin to produce vitamin D due to lower availability of 7-DHC), season of the year (autumn and winter), weather conditions (cloudiness), geographical locations (higher latitude), sun exposure, sunscreen (with a protection factor of 30 reduces above 95% of vitamin D synthesis in the skin), skin pigmentation (darker skin needs 3–5 times longer sun exposure to synthesize the same amount of vitamin D than light skin since melanin absorbs UVB radiation), genetic factors (SNPs and mutations), skin damage (burns decrease its production), adiposity (obesity has reduced vitamin D levels), workplace (indoor vs. outdoor), lifestyle, physical activity, clothing habits, air pollution, smoking, diet and calcium intake, vitamin D supplements, and individual height.

7. Vitamin D status

Vitamin D status is best determined by measuring serum 25-hydroxyvitamin D [25(OH)D]; a level higher than 50 nmol/L (30 ng/mL) contributes to the optimal calcium absorption, fall prevention, and prevention of the fracture [4, 22]. Below

this level, PTH levels increase blood circulation, causing secondary hyperparathyroidism, and increase the risk of osteoporosis and fractures leading to bone loss. In addition, moderate increase of PTH may also enhance insulin resistance, weight gain, hypertension, left ventricular hypertrophy, and acute phase response, increasing the risk of ischemic arrhythmias and cardiovascular mortality [4].

According to the Committee of the Institute of Medicine (IOM, USA) and the Endocrine Society [19, 23], vitamin D status defined the values lower than 50 nmol/L (20 ng/mL) as vitamin D deficiency (VDD), while values between 50 and 75 nmol/L are indicated to vitamin D insufficiency (VDI), and values equal or above 75 nmol/L (30 ng/mL) is described as adequate or sufficient vitamin D. The IOM adds extra criterion which is severe VDD with 25 nmol/L (10 ng/mL).

Serum [25(OH)D] test is detected by using high-performance liquid chromatography/mass-spectrometry (LC/MS) methodology [4, 19] which is recommended by the National Diet and Nutrition Survey [19]. The high cost for vitamin D detection hampers the diagnosis of vitamin D deficiency. There is a great need to develop a specific and cheap testing method [4].

8. Vitamin D deficiency

Most studies have identified the vitamin D insufficiency (VDI) at concentrations of [25(OH)D] less than 75 nmol/L (30 ng/mL) and vitamin D deficiency (VDD) at concentrations below 50 nmol/L (20 ng/mL) [1, 4, 10, 17].

Vitamin D deficiency is still a highly prevalent disorder. It is estimated that ~1 billion people are deficient or have insufficient levels of vitamin, in spite of foods fortified with vitamin D and wide supplement intake [2].

VDD is widespread in the whole world as well as predominant in Asia and in the Middle East (more than 50% of the population is VDD and about 75% is VDI) [4]. VDD is found in 30–50% of otherwise healthy middle-aged to elderly adults [11].

Deficiency of vitamin D can result from many reasons such as dietary inadequacy of vitamin D, poor absorption and use, increased requirement, increased excretion and catabolism, limited sunlight exposure, and inefficient production in the skin. Dietary deficiency of vitamin D is associated with milk allergy, lactose intolerance, ovo-vegetarianism, and veganism [1, 4]. In addition, various diseases affect the bioavailability of vitamin D, such as gastrointestinal disorders which limit its absorption; kidney and liver diseases can prevent the activation of the parenteral vitamin D or impair the conversion of vitamin D into its active metabolites [4].

Severe VDD in adults leads to osteomalacia while in children leads to rickets, defective bone mineralization, increased bone turnover, increased risk of fractures [4], impaired reproductive function, and production of gonadal hormone that may affect other organs, e.g., gastrointestinal and renal calcium handling, renal CYP27B1 activity, and bone function [20].

In the critical care condition, VDD has been associated with adverse outcomes such as infections, longer length of stay, acute kidney injury, and higher mortality [10].

9. Vitamin D deficiency in Saudi Arabia

A total of 132 articles studying the deficiency of vitamin D in Saudi Arabia were identified based on the initial PubMed search criteria. About 20 studies have investigated the vitamin D deficiency in healthy individuals living in Saudi Arabia during

Status of women	Prevalence of VDD [25(OH)D] <50 nmol/L	Age group (years)	City of Saudi Arabia	Year of study	Ref.
Premenopausal	80.5%	20–40	Tabuk	2018	[24]
	62%	>21	Riyadh	2017	[25]
	67.8%	19–25	Tabuk	2016	[26]
	74.8%	18–50	Riyadh	2015	[27]
	100%	19–40	Riyadh	2012	[28]
	78.2%	20–50	Jeddah	2011	[29]
	99.03%	18–22	Dammam	2009	[30]
	41.2%	≥18	Riyadh	2008	[23]
Postmenopausal	85%	50–79	Jeddah	2011	[29]
	86.2%	>60	Riyadh	2006–2011	[31]
Pregnant women	88%	>16	Riyadh	2011–2012	[32]
	64.2%	20–40	Al Khafji	2011	[33]
	50%	20–49	Riyadh	2010	[34]

To convert (nmol/L) into (ng/ml), divide the value by 2.496.

Table 1.
Prevalence reports on vitamin D deficiency in Saudi healthy women.

Subjects	Prevalence of VDD [25(OH)D] <50 nmol/L	Age group (years)	City of Saudi Arabia	Year of study	Ref.
Men	66.7%	20–40	Tabuk	2018	[24]
	59%	>21	Riyadh	2017	[25]
	74.4%	18–50	Riyadh	2015	[27]
	92%	20–23	Riyadh	2013	[35]
	87.8%	20–74	Jeddah	2012	[36]
	92.6%	18–22	Dammam	2009	[30]
	32.5%	≥25	Eastern Province	2009	[37]
Newborns	90%	Neonates	Riyadh	2013	[38]
	88%	Neonates	Riyadh	2011–2012	[32]
Children and adolescents	63%	1–6	Makkah	2015	[39]
	92.5% girls 79.3% boys	13–17	Riyadh	2015	[27]
	97.8% girls 92.8% boys	6–15	Western, central, and eastern regions (8 provinces)	2013–2014	[40]
	62.65% girls 40.6% boys	≤15	Different regions	2013	[41]
	86.27%	4–15	Jeddah	2010	[42]

To convert (nmol/L) into (ng/ml), divide the value by 2.496.

Table 2.
Prevalence reports on vitamin D deficiency in healthy men, newborns, children, and adolescents living in Saudi Arabia.

the past 10 years. These studies have demonstrated a noticeably high prevalence of VDD in Saudi women (41.2–100%, **Table 1**) compared to Saudi men (32.5–92.6%, **Table 2**). Deficiency of vitamin D was not only limited to adults but also included newborns (88–90%), children, and adolescents (40.6–97.8%) (**Table 2**).

10. Vitamin D toxicity and safety limits

Excessive oral supplementation and food fortification of vitamin D may lead to toxicity because it raises plasma [25(OH)D] concentrations that exceed DBP binding capacity, and free [25(OH)D] concentrations have direct effects on gene expression once it enters the target cells [13].

Upper safe limit is 5000 IU a day (and some considered it to be 10,000 IU), and the toxicity does not manifest serum levels below 120 ng/mL (300 nmol/L). To reach the latter levels, one must ingest vitamin D in excess of 50,000 IU daily for several months. Thus, the safety of doses to 5000 IU a day is assured [4].

11. Conclusion

In conclusion, this chapter summarizes that vitamin D is a fat-soluble prohormone and has skeletal and extra-skeletal functions. The main sources of vitamin D are sun exposure and diet. Two common types of vitamin D are vitamins D2 (ergocalciferol) and D3 (cholecalciferol). Vitamins D2 or D3 must be activated to produce its effects in a multi-step process. Vitamin D status is determined by serum 25-hydroxyvitamin D [25(OH)D]; the value lower than 50 nmol/L (30 ng/mL) contributes to vitamin D deficiency. Severe vitamin D deficiency leads to osteomalacia in adults, rickets in children, and an increased risk of fractures.

Like other countries in the world, Saudi Arabia suffers from vitamin D deficiency. This chapter illustrates the terrible deficiency of vitamin D for the Saudi population for both genders and for different age groups.

To improve the status of vitamin D deficiency, distinct strategies should be applied to raise the vitamin D stored as a routine measurement through sunlight exposure by increasing daily outdoor activity. Moreover, nutritionists should emphasize increased dairy intake, vitamin D supplementation, calcium supplementation, and vitamin D-fortified foods. Finally, effective educational programs are needed at the Saudi national level to raise public awareness of the serious vitamin D deficiency problem.

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References

[1] Mostafa WZ, Hegazy RA. Vitamin D and the skin: Focus on a complex relationship: A review. Journal of Advanced Research. 2015;**6**(6):793-804

[2] Müller DN, Kleinewietfeld M, Kvakan H. Vitamin D review. Journal of the Renin-Angiotensin-Aldosterone System. 2011;**12**(2):125-128

[3] Autier P, Boniol M, Pizot C, Mullie P. Vitamin D status and ill health: A systematic review. The Lancet Diabetes & Endocrinology. 2014;**2**(1):76-89

[4] Wimalawansa SJ. Associations of vitamin D with insulin resistance, obesity, type 2 diabetes, and metabolic syndrome. The Journal of Steroid Biochemistry and Molecular Biology. 2018;**175**:177-189

[5] Sutaria N, Liu C-T, Chen TC. Vitamin D status, receptor gene polymorphisms, and supplementation on tuberculosis: A systematic review of case-control studies and randomized controlled trials. Journal of Clinical & Translational Endocrinology. 2014;**1**(4):151-160

[6] Uwitonze AM, Murererehe J, Ineza MC, Harelimana EI, Nsabimana U, Uwambaye P, et al. Effects of vitamin D status on oral health. The Journal of Steroid Biochemistry and Molecular Biology. 2017;**175**:190-194

[7] Tardelli VS, do Lago MPP, da Silveira DX, Fidalgo TM. Vitamin D and alcohol: A review of the current literature. Psychiatry Research. 2017;**248**:83-86

[8] Annweiler C, Henni S, Walrand S, Montero-Odasso M, Duque G, Duval GT. Vitamin D and walking speed in older adults: Systematic review and meta-analysis. Maturitas. 2017;**106**:8-25

[9] El-Shafey BI, El-Srougy HA. Does serum 25 hydroxy vitamin D level play a role in COPD? The Egyptian Journal of Chest Diseases and Tuberculosis. 2014;**63**(1):43-47

[10] Langlois PL, Szwec C, D'Aragon F, Heyland DK, Manzanares W. Vitamin D supplementation in the critically ill: A systematic review and meta-analysis. Clinical Nutrition. 2017;**37**(4):1238-1246

[11] Haimi M, Kremer R. Vitamin D deficiency/insufficiency from childhood to adulthood: Insights from a sunny country. World Journal of Clinical Pediatrics. 2017;**6**(1):1

[12] Narula N, Marshall JK. Management of inflammatory bowel disease with vitamin D: Beyond bone health. Journal of Crohn's and Colitis. 2012;**6**(4):397-404

[13] Dalan R, Liew H, Tan WKA, Chew DE, Leow MK-S. Vitamin D and the endothelium: Basic, translational and clinical research updates. IJC Metabolic & Endocrine. 2014;**4**:4-17

[14] Tripkovic L, Lambert H, Hart K, Smith CP, Bucca G, Penson S, et al. Comparison of vitamin D2 and vitamin D3 supplementation in raising serum 25-hydroxyvitamin D status: A systematic review and meta-analysis. The American Journal of Clinical Nutrition. 2012;**95**(6):1357-1364

[15] Nicholson I, Dalzell AM, El-Matary W. Vitamin D as a therapy for colitis: A systematic review. Journal of Crohn's and Colitis. 2012;**6**(4):405-411

[16] Bikle D. Vitamin D metabolism, mechanism of action, and clinical applications. Chemistry and Biology. 2014;**21**(3):319-329

[17] Rey C, Sánchez-Arango D, López-Herce J, Martínez-Camblor P, García-Hernández I, Prieto B, et al.

Vitamin D deficiency at pediatric intensive care admission. Jornal de Pediatria (Versão em Português). 2014;**90**(2):135-142

[18] Cui X, Gooch H, Petty A, McGrath JJ, Eyles D. Vitamin D and the brain: Genomic and non-genomic actions. Molecular and Cellular Endocrinology. 2017;**453**:131-143

[19] Urrutia-Pereira M, Solé D. Vitamin D deficiency in pregnancy and its impact on the fetus, the newborn and in childhood. Revista Paulista de Pediatria. 2015;**33**(1):104-113

[20] Lorenzen M, Boisen IM, Mortensen LJ, Lanske B, Juul A, Jensen MB. Reproductive endocrinology of vitamin D. Molecular and Cellular Endocrinology. 2017;**453**:103-112

[21] Kurasawa K. Bone quality and biochemical markers. Clinical Calcium. 2005;**15**(6):959-964

[22] Albaik M, Khan JA, Iyer AP. Vitamin D status in Saudi population: A minireview. Indian Journal of Applied Research. 2016;**6**(1):629-632

[23] Alsuwadia AO, Farag YM, Al Sayyari AA, Mousa DH, Alhejaili FF, Al-Harbi AS, et al. Prevalence of vitamin D deficiency in Saudi adults. Saudi Medical Journal. 2013;**34**(8):814-818

[24] Altowijri A, Alloubani A, Abdulhafiz I, Saleh A. Impact of nutritional and environmental factors on vitamin D deficiency. Asian Pacific Journal of Cancer Prevention;**19**(9):2569-2574

[25] Alkhenizan A, Mahmoud A, Hussain A, Gabr A, Alsoghayer S, Eldali A. The relationship between 25 (OH) D levels (Vitamin D) and bone mineral density (BMD) in a Saudi population in a community-based setting. PLoS One. 2017;**12**(1):e0169122

[26] Alzaheb RA, Al-Amer O. Prevalence and predictors of hypovitaminosis D among Female University students in Tabuk, Saudi Arabia. Clinical Medicine Insights: Women's Health. 2017;**10**:1-7

[27] Al-Daghri NM, Al-Saleh Y, Aljohani N, Alokail M, Al-Attas O, Alnaami AM, et al. Vitamin D deficiency and cardiometabolic risks: A juxtaposition of Arab adolescents and adults. PLoS One. 2015;**10**(7):e0131315

[28] Al-Mogbel ES. Vitamin D status among adult Saudi females visiting Primary Health Care Clinics. International Journal of Health Sciences (Qassim). 2012;**6**(2):116-126

[29] Ardawi MS, Qari MH, Rouzi AA, Maimani AA, Raddadi RM. Vitamin D status in relation to obesity, bone mineral density, bone turnover markers and vitamin D receptor genotypes in healthy Saudi pre- and postmenopausal women. Osteoporosis International. 2011;**22**(2):463-475

[30] Al-Elq AH. The status of Vitamin D in medical students in the preclerkship years of a Saudi medical school. Journal of Family and Community Medicine. 2012;**19**(2):100-104

[31] Hussain AN, Alkhenizan AH, El Shaker M, Raef H, Gabr A. Increasing trends and significance of hypovitaminosis D: A population-based study in the Kingdom of Saudi Arabia. Archives of Osteoporosis. 2014;**9**:190

[32] Fouda MA, Turkestani IZ, Almusharraf S, Al-Ajlan A, Angkaya-Bagayawa FF, Sabico S, et al. Extremely high prevalence of maternal and neonatal vitamin D deficiency in the Arab population. Neonatology. 2017;**112**(3):225-230

[33] Aly YF, El Koumi MA, Abd El Rahman RN. Impact of maternal vitamin D status during pregnancy on the prevalence of neonatal vitamin

D deficiency. Pediatric Reports. 2013;**5**(1):e6

[34] Al-Faris NA. High prevalence of Vitamin D deficiency among pregnant Saudi women. Nutrients. 2016;**8**(2):77

[35] BinSaeed AA, Torchyan AA, AlOmair BN, AlQadhib NS, AlSuwayeh FM, Monshi FM, et al. Determinants of vitamin D deficiency among undergraduate medical students in Saudi Arabia. European Journal of Clinical Nutrition. 2015;**69**(10):1151-1155

[36] Ardawi MS, Sibiany AM, Bakhsh TM, Qari MH, Maimani AA. High prevalence of vitamin D deficiency among healthy Saudi Arabian men: Relationship to bone mineral density, parathyroid hormone, bone turnover markers, and lifestyle factors. Osteoporosis International. 2012;**23**(2):675-686

[37] Sadat-Ali M, AlElq A, Al-Turki H, Al-Mulhim F, Al-Ali A. Vitamin D levels in healthy men in eastern Saudi Arabia. Annals of Saudi Medicine. 2009;**29**(5):378-382

[38] AlFaleh KM, Al-Manie AM, Al-Mahmoud HF, Al-Razqan HM, Al-Mutlaq AB, Al-Rumaih SA, et al. Prevalence of vitamin D deficiency in Saudi newborns at a tertiary care center. Saudi Medical Journal. 2014;**35**(2):178-182

[39] Kensarah OA, Jazar AS, Azzeh FS. Hypovitaminosis D in healthy toddlers and preschool children from Western Saudi Arabia. International Journal for Vitamin and Nutrition Research. 2015;**85**(1-2):50-60

[40] Al Shaikh AM, Abaalkhail B, Soliman A, Kaddam I, Aseri K, Al Saleh Y, et al. Prevalence of vitamin D deficiency and calcium homeostasis in Saudi children. Journal of Clinical Research in Pediatric Endocrinology. 2016;**8**(4):461-467

[41] Tuffaha M, El Bcheraoui C, Daoud F, Al Hussaini HA, Alamri F, Al Saeedi M, et al. Deficiencies under plenty of Sun: Vitamin D status among adults in the Kingdom of Saudi Arabia, 2013. North American Journal of Medical Sciences. 2015;**7**(10):467-475

[42] Mansour MM, Alhadidi KM. Vitamin D deficiency in children living in Jeddah, Saudi Arabia. Indian Journal of Endocrinology and Metabolism. 2012;**16**(2):263-269