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*Research Article*

## Assessment Of Vitamin D Deficiency and Dyslipidemia in Obese Patients

Yasamin Al-Qassab<sup>1\*</sup>, Tharwa H. Hasan Al-Tai<sup>1</sup>, Hayfaa S. AL-Hadithi<sup>2</sup> Ameen Abdulhasan Al-Alwany<sup>3</sup>

<sup>1\*</sup>Clinical Communicable Diseases Research Unit /College of Medicine/University of Baghdad, Baghdad, Iraq, \*e-mail: yasaminalqassab@comed.uobaghdad.edu.iq, ORCID: <https://orcid.org/my-orcid?orcid=0000-0001-6779-5379>, e-mail: tharwaaltai@comed.uobaghdad.edu.iq,

ORCID: <https://orcid.org/0000-0002-1065-4337>

<sup>2</sup>Prof.; Dept. of Microbiology, College of Medicine, University of Baghdad, Baghdad, Iraq, e-mail: haifaa.abd@comed.uobaghdad.edu.iq, ORCID: <https://orcid.org/0000-0001-9648-8933>

<sup>3</sup>Prof.; Dept. of Surgery, College of Medicine, University of Baghdad, Baghdad, Iraq, e-mail: ameen.a@comed.uobaghdad.edu.iq, ORCID: <https://orcid.org/0000-0002-3535-4023>

**\*Corresponding Author:** Dr. Yasamin Al-Qassab

\*Email: yasaminal-qassab@comed.uobaghdad.edu.iq, ORCID: <https://orcid.org/0000-0001-6779-5379>

### Abstract

**Background:** The three main methods of obtaining vitamin D are through diet, sun exposure, and supplements. The most common type is formed by the skin after exposure to UVB rays. Vitamin D is thought to work more like a prohormone or multifunctional hormone. Calcitriol enters the enterocyte by the genomic route and binds to VDR. This generates a complex that attaches to particular DNA sequences in target genes known as vitamin D responsive elements (VDREs) and forms a heterodimer with the nuclear receptor known as retinoid X receptor (RXR), which is activated by retinoic acid. One alternative pathway for vitamin D activation, at least in keratinocytes, has been identified as the 20-hydroxylation of vitamin D, which is catalyzed by the side-chain cleavage enzyme CYP11A1, which is required for steroidogenesis.

**Martial and Methods:** Between December 2022 and May 2023, the following groups were studied. control group conducted among the obese population (100) participants, 50% of them were set as case group contained obese, and 50% were healthy subjects set as a control group, they were attended to patient Baghdad Medical City Teaching Hospital / Department of Medicine Both groups gave permission for enrolling in the study, which pre-approved by the ethical committee of Clinical Communicable Diseases Research Unit /College of Medicine/University of Baghdad. Fasting blood samples withdrawn from each participant in a plane container in order to form serum, this collected later and preserved at -20 Co for later analysis process which conducted at bioMerieux-USA. Body mass index (BMI) calculated from weight and height of subjects, analytical parameters, vitamin D was assessed by VIDAS device depend on techniques of enzyme-linked fluorescence assay (ELFA) technique, while lipid profile was measured by means of Mindray.

**Results:** In this work we collected, one hundred (100) patients, included 67 % females and 33% male follow up their mean  $\pm$  SD of age (44.18 $\pm$ 18.34) years and body mass index 33.87 $\pm$ 3.93 kg/m<sup>2</sup> (figure 1). and 33% male follow up their mean  $\pm$  SD of age (44.18 $\pm$ 18.34) years and body mass index 33.87 $\pm$ 3.93 kg/m<sup>2</sup> (Figure 1).

**Conclusion:** On the basis of our results, we can conclude that low vitamin D level was found among obese people even though they were under treatment for dyslipidemia. suggested that vitamin D status should be checked regularly and supplementary doses should be administered in order to diminish harm that obese subjects could be subjected to in the current situation or in the in the coming future.

**Keywords:** Vitamin D, VIDAS, BMI, Dyslipidemia

\*Authors for correspondence: E-mail Id: yasaminal-qassab@comed.uobaghdad.edu.iq.

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activity. Apart from the kidneys, the mitochondrial CYP27B1 protein is expressed by epithelial cells, endocrine glands, immune system cells, osteoblasts, and chondrocytes [8]. In the duodenum and cecum, the transcellular pathway predominates. 1,25 dihydroxy vitamin D (1,25(OH)<sub>2</sub>D) predominantly regulates this pathway; however, it also regulates certain components of the paracellular pathway, including the claudins 2 and 12 [9]. One alternative pathway for vitamin D activation, at least in keratinocytes, has been identified as the 20-hydroxylation of vitamin D, which is catalyzed by the side-chain cleavage enzyme CYP11A1, which is required for steroidogenesis. The product 20 OHD or its metabolite 20,23(OH)<sub>2</sub>D appears to have activity similar to 1,25(OH)<sub>2</sub>D, at least for some tasks [10]. The chicken gut was the initial site of discovery of a binding protein that was subsequently named the VDR. Subsequently, the protein was found in additional tissues such as the kidney, bone, and parathyroid glands. The molecular characteristics of this protein, such as its capacity to bind to DNA and its retention in chromatin, were discovered some years later. These characteristics showed that this protein functioned in transcriptional regulation and that it resembled other receptors for known steroids hormones [11]. The activation of VDR by 1,25(OH)<sub>2</sub>D<sub>3</sub> is necessary for the genomic activities of vitamin D. Epigenome modifications result in changes to the transcriptome and proteome. Therefore, in a typical in vitro vitamin D stimulation experiment, when supraphysiological doses of 10–100 nM 1,25(OH)<sub>2</sub>D<sub>3</sub> are administered to a cell culture model, it takes several hours to observe the physiological effects of the nuclear hormone. This is due to the length of time needed for the production of proteins and RNA. [12,13]. This modifies the transcription of mRNA genes, resulting in de novo protein synthesis and non-genomic consequences. It is believed that 1,25D binding to a receptor on the plasma membrane is the mechanism of action behind these typically quick reactions (minutes to hours) [14]. It initiates a series of events leading to the phosphorylation of intracellular proteins or the production of a second messenger (arachidonic acid, cAMP, diacylglycerol, and inositol triphosphate). Certain receptors can be found in numerous additional tissues and cell types, such as the immune system, as well as in the traditional target organs of vitamin D that are crucial for its calcemic effects, such as the kidney, bone, gut, and parathyroid [15]. The side chain structure of vitamins D<sub>2</sub> and D<sub>3</sub> is the only difference between

## Introduction

The three main methods of obtaining vitamin D are through diet, sun exposure, and supplements. The most common type is formed by the skin after exposure to UVB rays. Vitamin D is thought to work more like a prohormone or multifunctional hormone. This is because vitamin D is involved in many different body processes. Numerous metabolic pathways, including the cardiovascular, immune, and endocrine systems, have been shown to function better when calcitriol is present. There is evidence that vitamin D also contributes to depression, pain, and cancer [1]. The biological effects of calcitriol are classified as non-genomic, mediated by fast signal transduction pathways triggered by VDR in the cytoplasm or cell membrane, and genomic, mediated by VDR transcriptional effects in the target cells' nucleus [2,3,4]. Calcitriol enters the enterocyte by the genomic route and binds to VDR. This generates a complex that attaches to particular DNA sequences in target genes known as vitamin D responsive elements (VDREs) and forms a heterodimer with the nuclear receptor known as retinoid X receptor (RXR), which is activated by retinoic acid. VDREs are found in a large number of human genes that are involved in endocrine and autocrine/paracrine processes, such as immune regulation, apoptosis, cell differentiation (including keratinocyte differentiation), and inhibition of cell proliferation [5,6]. According to the Institute of Medicine (IOM), 97.5% of the population, or those between the ages of one and seventy-seven, may get enough sun exposure from 600 International Units (IU)/day(d). The lowest amount of sun exposure was assumed while calculating the DRIs. This recommended dietary allowance (RDA) corresponds to a serum 25(OH)D concentration of  $\geq 50$  nmol/L, which is necessary to maintain bone health. However, the data gathered did not provide any strong evidence linking larger intakes to health advantages or outcomes other than skeletal health. Additionally, all European population groups aged one year and older are advised to consume an appropriate 600 IU/d by the European Food Safety Agency (EFSA)[6,7]. Although it happens in other tissues as well, the kidneys are the primary site of the second hydroxylation. The enzyme 1-OHase converts the physiologically inactive form of vitamin D, 25(OH)D, into the physiologically active form, 1,25-dihydroxyvitamin D [1,25(OH)<sub>2</sub>D] (calcitriol). This 1 $\alpha$ -hydroxylation process is carried out by CYP27B1, the only CYP known to have 1-OHase

the formation of osteoblasts, the inhibition of parathyroid hormone release, and calcium absorption from the intestine, which results in the mineralization of the bone matrix. In calcium-deficient environments, 1,25(OH)<sub>2</sub>D mobilizes calcium reserves from the bone Research Unit /College of Medicine/University of Baghdad. Study variables were age, gender, height, and weight. Fasting blood samples withdrawn from each participant in a plane container in order to form serum, this collected later and preserved at -20 C° for later analysis process which conducted at bioMerieux-USA. Body mass index (BMI) calculated from weight and height of subjects, analytical parameters, vitamin D was assessed by VIDAS device depend on techniques of enzyme-linked fluorescence assay (ELFA) technique, while lipid profile was measured by means of Mindray BS 480 device and reagents and analysis conducted through the protocol of the manufacturing company.

**Statistical analysis:** The statistical analyses were carried out using SPSS (version 20.0 for Windows, SPSS, Chicago, IL, USA), a statistical tool designed for the social sciences. The mean and standard deviation can be used to illustrate numerical data. When representing qualitative data, the phrases count and percentage are useful since they let one assess how two groups differ from one another. Using a student's t-test, the link between the qualitative data was studied. P values less than 0.05 were ultimately used to determine statistical significance.

**Results:**

In this work we collected, one hundred (100) patients, included 67 % females and 33% male follow up their mean ± SD of age (44.18±18.34) years and body mass index 33.87±3.93 kg/m<sup>2</sup> (figure 1). and 33% male follow up their mean ± SD of age (44.18±18.34) years and body mass index 33.87±3.93 kg/m<sup>2</sup> (Figure 1).

them. The prohormone function is present in both forms, and the variations have no effect on metabolism (i.e., activation). Dietary lipids and vitamin D (either D<sub>2</sub> or D<sub>3</sub>) are typically absorbed in the small intestine [16]. The main biological effects of 1,25-(OH)<sub>2</sub> vitamin D are by promoting monocytic cells to grow into osteoclasts, assisting in maintaining serum calcium levels within normal ranges [17]. Similarly, compensatory secondary hyperparathyroidism improves renal conversion of 25OHD to maintain normal or slightly higher plasma levels of 1,25(OH)<sub>2</sub>D in the event of low vitamin D [18]. Obesity develops when energy intake mostly stored as triglycerides exceeds energy expenditure and is a key risk factor for major illnesses. Obesity is a complicated characteristic that is impacted by genes, age, physical activity, developmental stage, and food [19,20,21]. In white adipose tissue, the balance between lipid removal and storage governs triglyceride turnover. Different turnover rates between visceral white adipose tissue and abdominal subcutaneous white adipose tissue may create metabolic issues in the overweight/obese condition [22]. Triglycerides, which are made of glycerol and energy-dense fatty acids esterified, are the primary type of lipid storage in white adipocytes. Triglyceride hydrolysis, or lipolysis, releases fatty acids into the bloodstream from fat cells [23].

**Material and method:**

Between December 2022 and May 2023, the following groups were studied. control group conducted among the obese population (100) participants, 50% of them were set as case group contained obese, and 50% were healthy subjects set as a control group, they were attended to patient Baghdad Medical City Teaching Hospital / Department of Medicine Both groups gave permission for enrolling in the study, which pre-approved by the ethical committee of Clinical Communicable Diseases

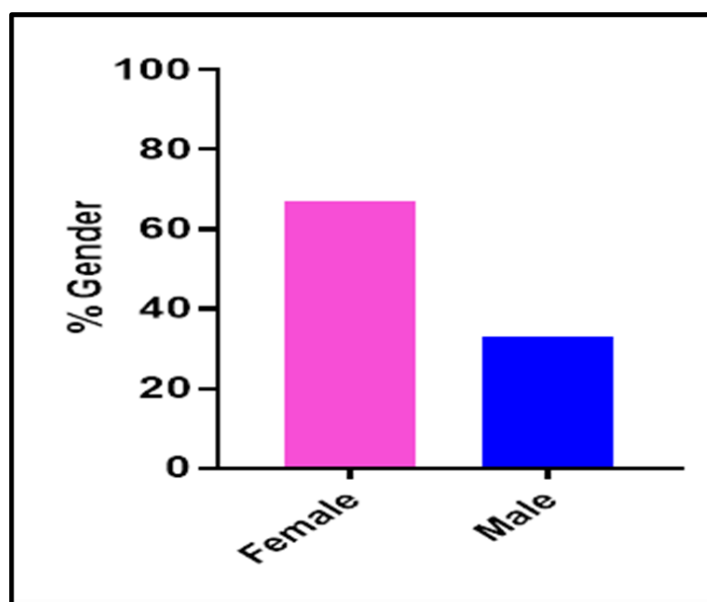


Figure 1: Percentage of different gender distribution among study participant

Body mass index BMI, vitamin D, and lipid profile data for obese group and non-obese (control) were compared through independent T-test, there was a significant decreased in vitamin D and HDL-C levels in obese subjects while there was no statistical significant observed with other parameters (Figure 2A, B) and (table 1).

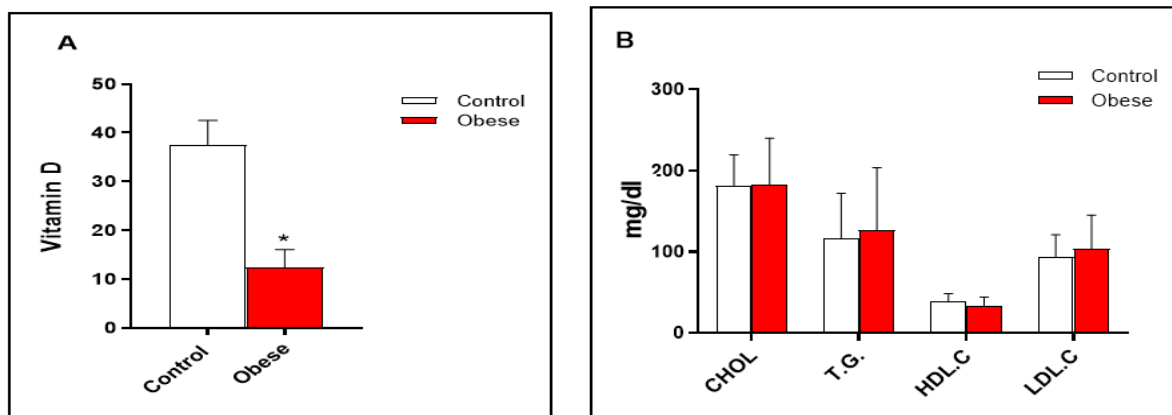


Figure 2: (A, B) The data, which represent the mean ± standard deviation of three separate trials conducted in duplicate, are presented. Results that are statistically significant are denoted by an asterisk.

Table 1: Independent T-test for measured parameters

| Parameters  | Case         | Control      | P-value |
|-------------|--------------|--------------|---------|
|             | (Mean±SD)    | (Mean±SD)    |         |
| BMI         | 35.86±3.95   | 37.00±3.84   | 0.158   |
| VIT D       | 13.45±3.64   | 39.55±17.60  | 0.000*  |
| Cholesterol | 183.20±56.88 | 181.88±37.82 | 0.897   |
| T.G         | 127.29±76.45 | 116.28±56.19 | 0.438   |
| HDL.C       | 34.05±10.33  | 38.55±10.24  | 0.035** |
| LDL.C       | 103.98±41.46 | 93.18±27.95  | 0.123   |

\*=highly Significant, \*\*=Significant difference at p value < 0.05.

Furthermore, no significant difference was observed between obese males and females in BMI, Vitamin D, Triglyceride and total cholesterol while there was a significant statistical difference only in HDL (Figure3 A, B).

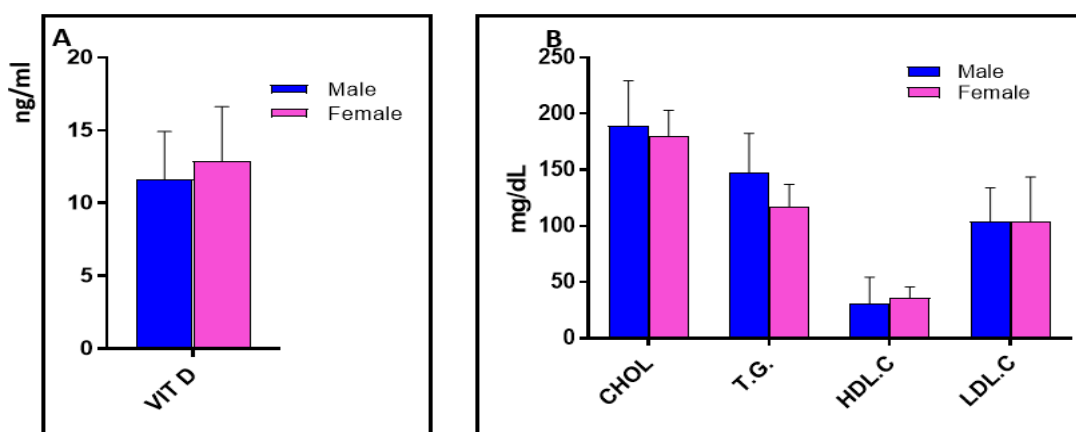


Figure 3: (A, B) The data are shown as mean ± standard deviation and are indicative of three independent experiments carried out in duplicate.

Table 2: Independent T-test for parameters between males and females

| Parameters  | Male         | Female       | P-value |
|-------------|--------------|--------------|---------|
|             | (Mean±SD)    | (Mean±SD)    |         |
| BMI         | 33.20±3.49   | 34.20±4.14   | 0.331   |
| VIT D       | 11.60±3.32   | 12.87±3.75   | 0.188   |
| Cholesterol | 189.40±68.28 | 180.10±50.93 | 0.555   |
| T.G         | 147.60±88.68 | 116.89±68.28 | 0.145   |
| HDL.C       | 30.55±9.035  | 35.80±10.59  | 0.052   |
| LDL.C       | 104.10±45.82 | 103.93±39.72 | 0.988   |

Presented values are the mean ±SD. Significant difference at p value < 0.05

All Lipid parameters were positively correlated with age (table 3,4). Serum vitamin D levels showed a negative correlation with body mass and low-density lipoprotein while the positive correlation with triglyceride, total cholesterol, and High-density lipoprotein.

**Table 3: Correlation between the study parameters and age**

| Parameters  | R-value | P-value |
|-------------|---------|---------|
| VIT D       | 0.016   | 0.905   |
| Cholesterol | 0.103   | 0.433   |
| T.G         | 0.077   | 0.563   |
| HDL.C       | 0.141   | 0.282   |
| LDL.C       | 0.240   | 0.065   |

Presented values are the mean ±SD. Significant difference at p value < 0.05

**Table 4: Relationship between research parameters and vitamin D**

| Parameter               | R-value | P-value |
|-------------------------|---------|---------|
| Vitamin D/ BMI          | -0.337  | 0.034   |
| Vitamin D/ Cholesterol  | 0.014   | 0.916   |
| Vitamin D/ Triglyceride | 0.150   | 0.256   |
| Vitamin D/ LDL          | -0.073  | 0.581   |
| Vitamin D/ HDL          | 0.374   | 0.033   |

Presented values are the mean ±SD. Significant difference at p value < 0.05

### Discussion

Obesity is a chronic health issue that is becoming more prevalent globally and is currently acknowledged as an epidemic. In Sudan, undernutrition and obesity are paradoxically coexisting as major health concerns, especially in metropolitan areas. Between 30 and 65 percent of adult Indians living in cities are overweight, obese, or have abdominal obesity. [24]. The typical dyslipidemia associated with obesity is characterized by raised triglycerides (TG), decreased HDL-C with HDL dysfunction, and normal or slightly elevated LDL-C with increased small dense LDL [25]. Considering BMI and other variables a meta-analysis conducted data of apparent health obese to find out relationships between vitamin D and selected considerations, it revealed that there was an inversely correlation between vitamin D and BMI, which has been demonstrated only among women [26,27]. An analysis of the relationship between BMI and vitamin D levels in the population under study revealed an inverse correlation between the two. There are several possible explanations for this, one of which is that increased body fat increases the sequestration of lipid-soluble vitamin D and serves as a reservoir for it, which lowers its bioavailability [28]. Additionally, it has been noted that there is a larger inverse relationship between fat content and serum 25(OH)D concentration than there is between vitamin D and BMI [29]. As an adaptive reaction to higher body weight, obese individuals have increased lean body mass in addition to fat mass. Through in vivo research, it was discovered that 33% of 25(OH)D was stored in fat and 20% in muscle, indicating that muscle may serve as an additional human vitamin D reservoir [30]. Another theory links obesity to reduced outdoor exercise, less exposure to sunlight, or clothing choices that prevent the skin from producing enough vitamin D [31]. Another hypothesis is that hepatic steatosis in obese individuals may cause the liver to synthesize 25-hydroxyvitamin D at a slower pace [32]. Alternatively, increased amounts

of circulating leptin and interleukin 6, primarily released by adipose tissue, may inhibit 25(OH)D production through their receptors [33].

In a retrospective study, a sample of obese and control participants' body mass index (BMI), lipid levels, and vitamin D deficiency were evaluated. When comparing individuals with inadequate vitamin D levels to those with acceptable levels, the mean lipid levels of the former group were found to be considerably higher: Total cholesterol was measured at 165 ± 28.6 mg/dL, while low-density lipoprotein was measured at 92.7 ± 25.7 mg/dL, compared to 80.8 ± 21.4 mg/dL (P = 0.007). Triglycerides were measured at 148.9 ± 97.1 mg/dL, while 90.6 ± 40.7 mg/dL (P = 0.0000). The participants who were overweight or obese had a mean triglyceride level of 148.9 mg/dL, which falls into the dyslipidemic range. There were no statistically significant differences in lipid levels between the vitamin D adequate and vitamin D deficient groups in the underweight and healthy weight participants with low BMI [34]. These findings partially corroborate our research, which found that obese individuals had lower vitamin D levels than non-obesity individuals (p value= 0.000) and that low levels of HDL-C significantly differed from the control group (p value= 0.035). However, no significant differences were found in cholesterol, triglycerides, or LDL-C. Given that our contributions were admitted to the hospital for observation, it's possible that the dyslipidemia medicine is having a positive effect. This study discovered a favorable correlation with HDL, triglycerides, and cholesterol and a negative correlation with LDL. This result somewhat agrees with another study that discovered a relationship between low vitamin D and elevated triglycerides and greater LDL in men and women, respectively [35].

**Conclusion:** On the basis of our results, we can conclude that low vitamin D level was found among obese people even though they were under treatment for

dyslipidemia. suggested that vitamin D status should be checked regularly and supplementary doses should be administered in order to diminish harm that obese subjects could be subjected to in the current situation or in the in the coming future.

**Conflict of Interest:** There are no conflicts of interests

**Funding:** Self

**Ethical clearance:** is not required

**Reference:**

1. Ellison DL, Moran HR. Vitamin D: Vitamin or Hormone? Nurs Clin North Am. 2021 ;56(1):47-57. doi: 10.1016/j.cnur.2020.10.004.
2. Del Valle, H. B., Yaktine, A. L., Taylor, C. L., and Ross, A. C. (Eds.). Dietary reference intakes for calcium and vitamin D. 2011. doi: 10.17226/13050.
3. Haussler MR, Jurutka PW, Mizwicki M, Norman AW. Vitamin D receptor (VDR)mediated actions of 1 $\alpha$ ,25(OH)<sub>2</sub> vitamin D<sub>3</sub>: genomic and non-genomic mechanisms. Best Pract Res Clin Endocrinol Metab. 2011;25(4):543-59. doi: 10.1016/j.beem.2011.05.010.
4. Trochoutsou AI, Kloukina V, Samitas K, Xanthou G. Vitamin-D in the immune system: genomic and non-genomic actions. Mini Rev Med Chem. 2015;15(11):953-63. doi: 10.2174/1389557515666150519110830.
5. Christakos S, Dhawan P, Verstuyf A, Verlinden L, Carmeliet G. Vitamin D: metabolism, molecular mechanism of action, and pleiotropic effects. Physiol Rev. 2016;96(1):365-408. doi: 10.1152/physrev.00014.2015.
6. Vanda C, Joana A, Alejandro S, Elisabete R. Vitamin D – from the pro-hormone to the biological actions. ACTA PORTUGUESA DE NUTRIÇÃO. 2019;(19): 50-54. doi:10.21011/apn.2019.1909.
7. EFSA Panel on Nutrition, Novel Foods and Food Allergens (NDA); Turck D, Bohn T, Castenmiller J, de Henauw S, Hirsch-Ernst KI, Knutsen HK, Maciuk A, Mangelsdorf I, McArdle HJ, Pentieva K, Siani A, Thies F, Tsabouri S, Vinceti M, Lanham-New S, Passeri G, Craciun I, Fabiani L, De Sousa RF, Martino L, Martínez SV, Naska A. Scientific opinion on the tolerable upper intake level for vitamin D, including the derivation of a conversion factor for calcidiol monohydrate. EFSA J. 2023;21(8):e08145. doi: 10.2903/j.efsa.2023.8145.
8. Bikle DD. Vitamin D metabolism, mechanism of action, and clinical applications. Chem Biol. 2014;21(3):319-29. doi: 10.1016/j.chembiol.2013.12.016.
9. Wongdee K, Chanpaisaeng K, Teerapornpantakit J, Charoenphandhu N. Intestinal Calcium Absorption. Compr Physiol. 2021;11(3):2047–2073.
10. Bikle DD. Vitamin D metabolism, mechanism of action, and clinical applications. Chem Biol. 2014;21(3):319-29. doi: 10.1016/j.chembiol.2013.12.016.
11. Pike JW, Christakos S. Biology and Mechanisms of Action of the Vitamin D Hormone. Endocrinol Metab Clin North Am. 2017 ;46(4):815-843. doi: 10.1016/j.ecl.2017.07.001.
12. Carlberg, C. Vitamin D genomics: From in vitro to in vivo. Front. Endocrinol. 2018, 9, 250.
13. Carlberg C. Vitamin D and Its Target Genes. Nutrients. 2022;14(7):1354. doi: 10.3390/nu14071354.
14. Dursun E, Gezen-Ak D. Vitamin D receptor is present on the neuronal plasma membrane and is co-localized with amyloid precursor protein, ADAM10 or Nicastrin. PLoS One. 2017;12(11):e0188605. doi: 10.1371/journal.pone.0188605.
15. Sha S, Degen M, Vlaski T, Fan Z, Brenner H, Schöttker B. The Safety Profile of Vitamin D Supplements Using Real-World Data from 445,493 Participants of the UK Biobank: Slightly Higher Hypercalcemia Prevalence but Neither Increased Risks of Kidney Stones nor Atherosclerosis. Nutrients. 2024;16(14):2251. doi: 10.3390/nu16142251.
16. Silva MC, Furlanetto TW. Intestinal absorption of vitamin D: a systematic review. Nutr Rev. 2018;76(1):60-76. doi: 10.1093/nutrit/nux034.
17. Sun Y, Alessandrini L, Angeloni S, Del Bianco E, Sagratini G. From 7-dehydrocholesterol to vitamin D<sub>3</sub>: Optimization of UV conversion procedures toward the valorization of fish waste matrices. Food Chem X. 2024 ;22:101373. doi: 10.1016/j.fochx.2024.101373.
18. Goswami R. Primer on the metabolic bone diseases and disorders of mineral metabolism. Indian J Med Res. 2016,144(3):489–90. doi: 10.4103/0971-5916.198664.
19. Loos RJF, Yeo GSH. The genetics of obesity: from discovery to biology. Nat Rev Genet. 2022 ;23(2):120-133. doi: 10.1038/s41576-021-00414-z.
20. Obici S. Minireview: Molecular targets for obesity therapy in the brain. Endocrinology. 2009;150(6):2512-7. doi: 10.1210/en.2009-0409.
21. Yazdi FT, Clee SM, Meyre D. Obesity genetics in mouse and human: back and forth, and back again. PeerJ. 2015;3:e856. doi: 10.7717/peerj.856.
22. Tchernof A, Després JP. Pathophysiology of human visceral obesity: an update. Physiol Rev. 2013 ;93(1):359-404. doi: 10.1152/physrev.00033.2011.
23. Mittal B. Subcutaneous adipose tissue & visceral adipose tissue. Indian J Med Res. 2019,149(5):571-573. doi: 10.4103/ijmr.IJMR\_1910\_18.
24. Misra A, Bhardwaj S. Obesity and the metabolic syndrome in developing countries: focus on South Asians. Nestle Nutr Inst Workshop Ser. 2014;78:133-40. doi: 10.1159/000354952..
25. Howard BV, Ruotolo G, Robbins DC. Obesity and dyslipidemia. Endocrinol Metab Clin North Am. 2003;32(4):855-67. doi: 10.1016/s0889-8529(03)00073-2.
26. Galuşca D, Popoviciu MS, Babeş EE, Vidican M, Zaha AA, Babeş VV, Jurca AD, Zaha DC, Bodog F. Vitamin D Implications and Effect of Supplementation in Endocrine Disorders: Autoimmune Thyroid Disorders (Hashimoto’s Disease and Grave’s Disease), Diabetes Mellitus and

- Obesity. *Medicina*. 2022; 58(2):194. doi:10.3390/medicina58020194.
27. Delle Monache S, Di Fulvio P, Iannetti E, Valerii L, Capone L, Nespoli MG, Bologna M, Angelucci A. Body mass index represents a good predictor of vitamin D status in women independently from age. *Clin Nutr*. 2019;38(2):829-834. doi: 10.1016/j.clnu.2018.02.024.
28. Vanlint S. Vitamin D and obesity. *Nutrients*. 2013;5(3):949-56. doi: 10.3390/nu5030949.
29. Shadid ILC, Guchelaar HJ, Weiss ST, Mirzakhani H. Vitamin D beyond the blood: Tissue distribution of vitamin D metabolites after supplementation. *Life Sci*. 2024;355:122942. doi: 10.1016/j.lfs.2024.122942.
30. Vimalaswaran KS, Berry DJ, Lu C, Tikkanen E, Pilz S, Hiraki LT and Cooper JD, Causal relationship between obesity and vitamin D status: bi-directional Mendelian randomization analysis of multiple cohorts. *PLoS Med*. 2013;10(2):e1001383. doi: 10.1371/journal.pmed.1001383
31. Zhang J, Cao ZB. Exercise: A Possibly Effective Way to Improve Vitamin D Nutritional Status. *Nutrients*. 2022;14(13):2652. doi: 10.3390/nu14132652.
32. El Khoudary SR, Samargandy S, Zeb I, Foster T, de Boer IH, Li D, Budoff MJ. Serum 25-hydroxyvitamin-D and nonalcoholic fatty liver disease: Does race/ethnicity matter? Findings from the MESA cohort. *Nutr Metab Cardiovasc Dis*. 2020;30(1):114-122. doi: 10.1016/j.numecd.2019.09.004.
33. Cătoi AF, Iancu M, Pârvu AE, Cegan AD, Bidian C, Chera EI, Pop ID, Macri AM. Relationship between 25 Hydroxyvitamin D, Overweight/Obesity Status, Pro-Inflammatory and Oxidative Stress Markers in Patients with Type 2 Diabetes: A Simplified Empirical Path Model. *Nutrients*. 2021; 13(8):2889. doi: 10.3390/nu13082889.
34. Kumaratne M, Early G, Cisneros J. Vitamin D Deficiency and Association With Body Mass Index and Lipid Levels in Hispanic American Adolescents. *Glob Pediatr Health*. 2017, 7;4:2333794X17744141. doi: 10.1177/2333794X17744141
35. Wang Y, Si S, Liu J, Wang Z, Jia H, Feng K, Sun L, Song SJ. The Associations of Serum Lipids with Vitamin D Status. *PLoS One*. 2016;11(10):e0165157. doi: 10.1371/journal.pone.0165157.