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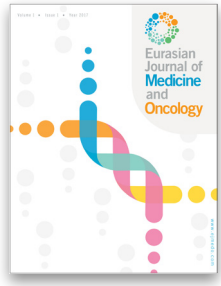


Research Article

# Assessment of the Relationship Between Serum Vitamin D Levels and Obesity in the Reproductive-Aged Women

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## Abstract

**Objectives:** The aim of this study was to examine the association between Vitamin D levels and body mass index (BMI) as an adiposity measure in the reproductive-aged women.

**Methods:** A total of 171 women were included in this comparative cross-sectional study. The subjects were classified into three groups according their BMI's: Group I; non-obese=80 (BMI<25.0 kg/m$^2$), Group II; overweight=54 (25.0<BMI<30 kg/m$^2$), and Group III; obese=37 (BMI>30 kg/m$^2$).

**Results:** Obese women possessed the lowest mean follicle stimulating hormone levels (6.26±1.46, p=0.001), and the highest luteinizing hormone (LH) levels were found in non-obese group (5.70±2.15, p=0.001). The comparison of anti-Müllerian hormone (AMH) levels yielded that there was a significant difference between non-obese and overweight women (4.96±4.02 vs. 3.11±3.03, p=0.019). The mean Vitamin D level was found to be highest in the non-obese group (10.45±7.48, p=0.043). The correlation analysis demonstrated that Vitamin D level was weak correlated with AMH level in the overweight group (r=0.285, p=0.047).

**Conclusion:** Our study showed a negative association between Vitamin D level and obesity. Vitamin D supplementation may aid to reduce the obesity incidence. Further evaluations are needed to elucidate this issue.

**Keywords:** Body mass index, obesity, vitamin D

Vitamin D is a fat-soluble vitamin and categorized as a non-essential secosteroid, as it is produced endogenously in the body by the aid of specific ultraviolet rays.[1] The main action of Vitamin D is on the quality of bone mineralization by regulation of calcium and phosphate metabolism. Vitamin D performs its biological actions through Vitamin D receptors which are identified mostly in calcium regulating tissues (skeleton and parathyroid glands intestines) and immune system (macrophages, monocytes, and T and B cells). However, these receptors are also identified in various reproductive organs, such as ovaries, uterus, placenta, testis, hypothalamus, and pituitary gland.[2, 3]

According to our current knowledge obtained by the appreciation of a global epidemic of Vitamin D insufficiency/deficiency, Vitamin D has role on pro-differentiation, anti-differentiation, proapoptosis, immunosuppression, and anti-inflammation.[4] Moreover, Vitamin D has a critical role in reproductive physiology because many physiological processes are influenced by Vitamin D. The recent data show that Vitamin D has a key role in processes involved in reproductive success.[5] Prevalence studies have been performed on Vitamin D deficiency and/or insufficiency in Turkish population. However, the majority of these studies included women, people in nursing homes, and the elderly.[6, 7]

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Obesity is a major global health problem. Its prevalence has risen steeply worldwide in recent decades. Cardiovascular diseases, hypertension, diabetes mellitus, and various kinds of cancers are associated with obesity.[8] The World Health Organization (WHO) defined body mass index (BMI) ≥25 kg/m$^2$ as overweight, BMI ≥30 kg/m$^2$ as obese. [9] In TEKHARF study, the prevalence of obesity was found 32.02%, and it is the highest mean prevalence of obesity in Turkey (men, 21.1%, and women, 43.0%). In TURDEP study, it was also found mean obesity 29.9% in women.[10]

There is little convincing evidence about the assessment of the relationship between serum Vitamin D levels and obesity in the existing literature. Tosunbayraktar et al. found that overweight and obese individuals had a very low level of Vitamin D and low Vitamin D levels were associated with obesity, especially visceral obesity.[11] They also described a Vitamin D deficiency group (<20 ng/mL) and a sufficiency group (≥20 ng/mL). In deficiency group, the mean BMI, waist circumference (WC), and fat mass were significantly higher than the sufficiency group. However, Botella-Carratero et al. demonstrated similar BMIs and WCs in patients with and without Vitamin D deficiency.[12] Therefore, the aim of the current study is to assess the association between Vitamin D concentration and BMI as an adiposity measure in the reproductive-aged women admitting a university hospital.

## Methods

### Study Population

In the present study, the purposive sampling was preferred as a non-probability sampling, and this comparative cross-sectional study was conducted with the participation of 171 women admitted to the Hitit University Hospital, Corum, Turkey between November 1, 2015, and March 31, 2016. The ethics committee of Ankara Numune Hospital approved the study that was in accordance with the Declaration of Helsinki 2013 Brazil version (20796219–724.087). The written informed consents were collected from all participant women before inclusion in the study. Attention was paid to the fact that participant women showed similar clothing styles. The inclusion criteria were accepted as <45 years of age and no history of menopause, premature ovarian failure, pelvic surgery, endometriosis, ovarian masses, smoking, current use of medications known to affect reproductive functions, chronic systemic diseases, and hyperprolactinemia. Exclusion criteria were accepted as the presence of coexisting systemic diseases, drug or hormone usage, diseases that could affect Vitamin D levels, such as parathyroid disease, hyperprolactinemia, hypothyroidism, Cushing's disease or congenital adrenal hyperplasia, pregnancy, and lactation.

At the initial visit, height, weight, waist, and hip circumferences (HCs) of the participant women were measured after a 12 h fasting with the same scale and by the same observer. The BMI was calculated using the standard equation (kilogram per meters squared). The WC was measured in the standing position, midway between the lower margin of the last rib and iliac crest, at mid exhalation. The HC was measured at the widest point of the hip/buttocks area with the measuring tape parallel to the floor. The waist-to-hip ratio (WRH) was determined by dividing WC by HC.

The participant women were categorized into three groups according their BMI's: Group I; non-obese=80 (BMI <25.0 kg/m$^2$), Group II; overweight=54 (25.0<BMI<30 kg/m$^2$), and Group III; obese=37 (BMI>30 kg/m$^2$)

### Blood Samples and Assays

The blood samples of the participants were obtained from the antecubital vein after overnight fasting between 08:00 am and 10:00 am in the early follicular phase on days 2 to 5. The blood samples were collected into 5 mL serum separator tubes (BD Vacutainer, Becton Dickinson, New Jersey, USA). The samples allowed to clot completely at room temperature and then centrifuged within 30 min at 3000 rpm for 20 minutes. The serums were analyzed on a daily basis for estradiol (E2), follicle-stimulating hormone (FSH), LH, total testosterone (TT), 17-hydroxyprogesterone (17OHP), and dehydroepiandrosterone sulfate (DHEAS) with an electrochemiluminescence immunoassay (ECLIA) method using an autoanalyzer (Cobas 6000, E 601 Roche Diagnostics, GmbH, Mannheim, Germany). To obtain minimal fluctuations in samples, serum for anti-Mullerian hormone (AMH) measurements was frozen at –20°C within 2 h for a maximum of 7 days and then analyzed. All analyses of AMH samples were also performed on a weekly basis by the ECLIA method using an autoanalyzer (Cobas 6000, E 601 Roche Diagnostics, GmbH, Mannheim, Germany). Serum Vitamin D levels were also measured by liquid chromatography-tandem mass spectrometry (Waters Quattro Premier mass spectrometer-Milford, Massachusetts, USA).

### Statistical Analysis

All analyses were performed using Statistical Packages for the Social Sciences (SPSS) software version 21 (SPSS Inc. Chicago, USA). The continuous variables were first evaluated for normality of statistical distribution by Shapiro-Wilk test. While the one-way analysis of variance was used for the normally distributed continuous variables, the Kruskal–Wallis test was used for the abnormally distributed continuous variables. When the Kruskal–Wallis test indicated statistically significant differences, the causes of those differences were determined using a Bonferroni-

adjusted Mann–Whitney U-test. Categorical data were analyzed by Pearson's Chi-square test, and Fisher's exact test was applied if the expected frequency was <5 in >20% of all cells. The continuous variables were presented as the mean±standard deviation or median and 25–75$^{th}$ percentiles, and the categorical variables were presented as a percentage. The Pearson or Spearman correlation analysis, where appropriate, was used to study the correlations between Vitamin D levels and study variables. P<0.05 was considered as statistically significant.

## Results

The components of anthropometric and biochemical parameters are demonstrated in Table 1. There was no difference in ages, E2, thyroid stimulating hormone, prolactin, TT, 17OHP, and DHEAS between the groups (p>0.05). As expected, obese women had the highest mean WC, HC values (101.46±12.68, p<0.001 and 116.57±7.68, p<0.001, respectively), and the lowest waist to HC ratio (WHR) mean was found in non-obese group (0.83±0.06, p<0.05). Interestingly, obese women possessed the lowest mean FSH levels (6.26±1.45, p=0.05), and the highest LH levels were found in non-obese group (7.59±2.98, p<0.05). The comparison of AMH levels yielded that there was a statistically significant difference between non-obese and overweight women (4.96±4.02 vs. 3.11±3.03, p=0.019). The mean Vitamin D concentration was found to be lowest in the obese group (6.61±2.58, p<0.05).

The correlation analyses showed that Vitamin D weakly and significantly correlated with AMH only in overweight group (r=0.285, p=0.047), as shown in Table 2. Other study parameters did not demonstrate any correlation with Vitamin D.

## Discussion

In our study, we found a weakly association between serum Vitamin D level and overweight women. Only in the overweight group, there was a weak but significant correlation between serum Vitamin D levels and AMH. There was no correlation between Vitamin D and other study parameters. Our study also demonstrated an inverse relationship between Vitamin D levels and BMI as the previous studies.[12–14]

**Table 1.** Comparisons of anthropometric and biochemical parameters between BMI categories

| | Group I=non-obese (BMI<25.0 kg/m²) (n=80) | Group II=overweight (25<BMI<30 kg/m²) (n=54) | Group III=obese (BMI≥30.0 kg/m²) (n=37) | p | | |
|---|---|---|---|---|---|---|
| | | | | 1 versus 2 | 1 versus 3 | 2 versus 3 |
| Age (years) | 30.24±5.40 | 32.3±5.24 | 30.16±6.23 | 0.113 | | |
| Education (%) | | | | | | |
| Primary school | 45.0 | 63.0 | 73.0 | 0.042* | 0.002* | 0.416 |
| High school | 25.0 | 24.1 | 21.6 | | | |
| University | 30.0 | 12.9 | 5.4 | | | |
| Place of residence (%) | | | | | | |
| Urban | 91.3 | 87.0 | 78.4 | 0.207 | | |
| Rural | 8.7 | 13.0 | 21.6 | | | |
| WC (cm) | 81.75±9.81 | 93.89±8.23 | 101.46±12.68 | <0.001* | <0.001* | 0.002* |
| HC (cm) | 98.39±7.71 | 107.13±6.01 | 116.57±7.68 | <0.001* | <0.001* | <0.001* |
| WHR | 0.83±0.06 | 0.87±0.06 | 0.88±0.09 | 0.001* | 0.015* | 0.928 |
| FSH (IU/L) | 7.14±2.04 | 7.99±2.67 | 6.26±1.45 | 0.085 | 0.041* | 0.001* |
| LH (IU/L) | 7.59±2.98 | 5.70±2.15 | 6.03±3.21 | 0.001* | 0.036* | 0.834 |
| E2 (pg/mL) | 43.50 (30.00–56.75) | 37.50 (27.75–51.50) | 35.00 (25.00–49.50) | 0.099 | | |
| TSH | 1.80 (1.40–2.87) | 1.95 (0.97–2.31) | 2.10 (1.40–3.15) | 0.143 | | |
| Prolactin | 14.55 (11.00–21.67) | 12.80 (8.77–17.86) | 14.70 (10.00–24.60) | 0.165 | | |
| TT (ng/dL) | 22.70 (16.00–32.75) | 22.30 (13.55–31.65) | 28.40 (15.50–39.10) | 0.343 | | |
| 17OHP (ng/dL) | 0.70 (0.50–1.10) | 0.60 (0.40–0.92) | 0.70 (0.50–0.84) | 0.569 | | |
| DHEAS (mcg/dL) | 217.00 (148.50–281) | 205.00 (165.75–279.00) | 182.00 (137.50–287.50) | 0.779 | | |
| AMH (ng/dL) | 4.96±4.02 | 3.11±3.03 | 4.51±4.16 | 0.019* | 0.827 | 0.205 |
| Vitamin D (ng/mL) | 10.45±7.48 | 7.90±4.97 | 6.61±2.58 | 0.043* | <0.001* | 0.294 |

BMI: Body mass index; WC: Waist circumference; WHR: Waist-to-hip circumference ratio; HC: Hip circumference ratio; FSH: Follicle-stimulating hormone; LH: Luteinizing hormone; E2: Estradiol; TSH: Thyroid-stimulating hormone; TT: Total testosterone; 17OHP: 17-hydroxyprogesterone; DHEAS: Dehydroepiandrosterone sulfate; AMH: Anti-Müllerian hormone. *p-values indicate statistically significant (p<0.05).

**Table 2.** Pearson's correlation analyses of Vitamin D with the study parameters

| | | Group I =non-obese (BMI <25.0 kg/m²) (n=80) | Group II=overweight (25<BMI<30 kg/m²) (n=54) | Group III=obese (BMI ≥30.0 kg/m²) (n=37) |
|---|---|---|---|---|
| Age (years) | r | 0.164 | –0.108 | 0.131 |
| | p | 0.134 | 0.462 | 0.439 |
| WC (cm) | r | –0.068 | –0.196 | 0.093 |
| | p | 0.536 | 0.176 | 0.583 |
| HC (cm) | r | –0.136 | –0.171 | –0.055 |
| | p | 0.214 | 0.239 | 0.747 |
| WHR | r | 0.048 | –0.100 | 0.129 |
| | p | 0.664 | 0.493 | 0.447 |
| E2 (pg/mL) | r | 0.132 | 0.214 | 0.008 |
| | p | 0.228 | 0.140 | 0.964 |
| FSH (IU/L) | r | 0.141 | 0.063 | –0.206 |
| | p | 0.197 | 0.668 | 0.222 |
| LH (IU/L) | r | 0.099 | 0.023 | 0.030 |
| | p | 0.365 | 0.874 | 0.858 |
| TT (ng/dL) | r | –0.129 | 0.015 | –0.048 |
| | p | 0.241 | 0.920 | 0.779 |
| 17 OHP (ng/dL) | r | 0.036 | 0.004 | –0.006 |
| | p | 0.745 | 0.981 | 0.973 |
| DHEAS (mcg/dL) | r | –0.054 | –0.162 | 0.176 |
| | p | 0.623 | 0.266 | 0.297 |
| AMH (ng/dL) | r | –0.204 | 0.285 | 0.104 |
| | p | 0.061 | 0.047* | 0.539 |

BMI: Body mass index; WC: Waist circumference; WHR: Waist-to-hip circumference ratio; E2: Estradiol; FSH: Follicle-stimulating hormone; LH: Luteinizing hormone; TT: Total testosterone; 17OHP: 17-hydroxyprogesterone; DHEAS: Dehydroepiandrosterone sulfate; AMH: Anti-Müllerian hormone. *p-values indicate statistically significant (p<0.05).

Several studies revealed that circulating Vitamin D levels were lower in obese individuals when compared to non-obese individuals.[12, 14–16] Serum Vitamin D level was lower in when BMI is ≥30 kg/m².[12, 15] After adjusting for age, sex, laboratory batch, and month of measurement in a bi-directional genetic study, it was shown that each unit increase in BMI being associated with 1.15% lower concentration of Vitamin D and this study suggested that higher BMI led to lower Vitamin D levels.[16] A statistically significant inverse association between BMI and serum Vitamin D concentrations was confirmed by a meta-analysis.[17] On the other hand, a negative correlation was found between Vitamin D level and percentage of body fat in the study by Arunabh et al.[18] The cause of these inconsistent results may be the limitation of adiposity measures to indirect anthropometric measures such as BMI,[19] WC, and WRH.[20]

There is little information on the relationship between total body surface area and Vitamin D concentrations. To the best of our knowledge so far, this relationship has been examined only in one study.[21] A significant positive relationship was found between height, body surface area, and Vitamin D concentration. The probable cause of this relationship was increased synthesis of Vitamin D in the skin due to the increased body surface.

Several mechanisms can cause low Vitamin D concentration in obese individuals. The action of the high content of body fat as a reservoir for lipid soluble Vitamin D is one of the hypothesis. High body fat concentration increases the sequestration of Vitamin D and causes low bioavailability.[20] It is also demonstrated that there is a negative correlation between fat content and Vitamin D concentration and this association is stronger than that between Vitamin D and BMI.[17] Volumetric dilution effect is another theory.[22] Some authors suggest that obesity is associated with decreased sunlight exposure, limited outdoor activity, or clothing habits that limit cutaneous Vitamin D synthesis.[23] According to another hypothesis, due to hepatic steatosis the synthesis of 25-hydroxy-Vitamin D by liver may occur at a lower rate in obese subjects.[24] Another explanation is that adipose tissue secretes most of circulating leptin and interleukin-6 and these may have inhibitory effect on Vitamin D synthesis through their receptors.[25] Leptin is a very

important cause of obesity; Vitamin D has a very essential role in leptin generation. Vitamin D can cause obstacles to leptin synthesis. By this mechanism depletion of Vitamin D can increase appetite and cause obesity.[26]

There are some limitations to our study. First; as the study was designed cross-sectionally, it does not prove a causal relationship. Second; two important factors affecting serum Vitamin D level, sunlight exposure and Vitamin D intake were not measured. Furthermore, our results may have been affected by the differences between obese and non-obese subjects. The last limitation is that all analyses are based on single-occasion Vitamin D measurements. Furthermore, BMI and WC are the most common indicators to estimate obesity. However, both can lead to bias in measuring adiposity.[27] Hence, when BMI is used as an obesity indicator association between Vitamin D, overall obesity can be underestimated.

In conclusion, we found a negative association between Vitamin D concentration and obesity. Vitamin D supplementation may aid to reduce the obesity incidence. Further evaluations are needed to prove the concept of maintaining an increased Vitamin D status for decreasing BMI.

**Disclosures**

**Ethics Committee Approval:** The ethics committee of Ankara Numune Hospital approved the study that was in accordance with the Declaration of Helsinki 2013 Brazil version (20796219-724.087).

**Peer-review:** Externally peer-reviewed.

**Conflict of Interest:** The authors declare that they have no conflict of interest.

**Authorship Contributions:** Concept – U.G., Z.O.I.; Design – U.G., H.A.I.; Supervision – U.G., Z.O.I.; Materials – U.G., H.A.I.; Data collection &/or processing – U.G., Z.O.I, H.A.I.; Analysis and/or interpretation – U.G., H.A.I.; Literature search – U.G., Z.O.I.; Writing – U.G., Z.O.I., H.A.I.; Critical review – U.G., Z.O.I., H.A.I.

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