# THE RELATIONSHIP BETWEEN 25-OH VITAMIN D LEVEL, DYSLIPIDEMIA AND INSULIN RESISTANCE IN EASTERN PART OF TURKEY

# TÜRKİYE'NİN DOĞU BÖLGESİNDE 25-HİDROKSİ VİTAMİN D SEVİYESİ, DİSLİPİDEMİ VE İNSÜLİN REZİSTANSI ARASINDAKİ İLİŞKİ

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**ABSTRACT**

**BACKGROUND:** It has been accepted that vitamin D deficiency and insufficiency are associated with many chronic diseases. As the relationship between vitamin D, dyslipidemia and insulin resistance may provide early diagnosis and treatment of related diseases, in this study, it was aimed to investigate the relationship between vitamin D levels and dyslipidemia and insulin resistance.

**METHODS:** The study was conducted with 249 healthy volunteers in Kafkas University Departement of Internal Medicine. Laboratory examinations including Vitamin D were conducted. Insulin resistance was calculated by homeostasis model assessment-insulin resistance index. We also searched the relationship between various Vitamin D levels and age, gender, glucose and lipid parameters and insulin resistance.

**RESULTS:** Vitamin D level of the participants was higher in males than females. Vitamin D levels decreased as age increased. It was found that 3.6% of the participants had normal vitamin D levels, 14.5% had insufficiency, 45% had deficiency and 36.9 % had severe deficiency. Insulin resistance was detected in 57.6% of those with severe deficiency, 22.3% of those with deficiency, and 8.3% of those with insufficiency. None of those with normal Vitamin D levels have insulin resistance. No significant difference was found between vitamin D groups in terms of lipid parameter values. As Vitamin D decreased, fasting blood glucose and insulin values increased.
Conclusion: We concluded that although there was not a relationship between vitamin D and dyslipidemia levels, we thought that vitamin D and insulin resistance can not be considered independent of each other. Early interventions for those who are found to be deficient by vitamin D scans may also help dealing with other diseases such as metabolic syndrome and diabetes.

***Keywords*** *Vitamin D, Insulin resistance, Dyslipidemia*

**ÖZET**

**AMAÇ:** D vitamini eksikliği ve yetersizliğinin birçok kronik hastalık ile ilişkili olduğu kabul edilmektedir. D vitamini, dislipidemi ve insülin direnci arasındaki ilişki, ilgili hastalıkların erken teşhisi ve tedavisini sağlayabileceğinden, bu çalışmada D vitamini düzeyleri ile dislipidemi ve insülin direnci arasındaki ilişkiyi araştırmayı amaçladık.

**GEREÇ VE YÖNTEMLER:** Çalışma 249 sağlıklı kişi ile gerçekleştirilmiştir. Vitamin D dahil olmak üzere laboratuvar testleri yapıldı. İnsülin direnci, homeostaz model değerlendirme-insülin direnci indeksi ile hesaplandı. Ayrıca çeşitli vitamin D düzeyleri ile yaş, cinsiyet, glikoz ve lipit parametreleri ile insülin direnci arasındaki ilişkiyi araştırdık.

**BULGULAR:** Katılımcıların D vitamini düzeyi erkeklerde kadınlara göre daha yüksekti. Vitamin D seviyeleri yaş arttıkça azaldı. Katılımcıların %3,6'sında normal D vitamini düzeyleri, %14,5'inde yetersizlik, %45'ında eksiklik, %36,9'unda ciddi eksiklik saptandı. İnsülin direnci, ciddi yetersizliği olanların %57,6'sında, yetersizliği olanların %22,3'ünde ve eksikliği olanların %8,3'ünde saptandı. Normal D vitamini seviyeleri olanların hiçbiri insülin direncine sahip değildir. D vitamini grupları arasında lipid parametresi değerleri açısından anlamlı fark bulunmadı. D vitamini azaldıkça açlık kan şekeri ve insülin değerleri arttı.

**SONUÇ:** D vitamini ve dislipidemi düzeyleri arasında bir ilişki olmamasına rağmen, D vitamini ve insülin direncinin birbirinden bağımsız olarak değerlendirilemeyeceğini düşündük. D vitamini taraması ile eksik olduğu tespit edilenler için erken müdahaleler, metabolik sendrom ve diyabet gibi diğer hastalıklarla ilgilenirken yardımcı olabilir.

***Anahtar kelimeler:*** *Vitamin D, İnsülin rezistansı, Dislipidemi*

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## INTRODUCTION

Recently, it has been proved that vitamin D deficiency and insufficiency are associated with many chronic diseases including cancers, cardiovascular diseases, metabolic syndrome, infectious and autoimmune diseases (1). A possible mechanism of action is to induce insulin secretion and increase insulin sensitivity. Vitamin D is thought to increase insulin secretion by affecting nuclear receptors in pancreatic beta cells (2).

Serum 25-OH vitamin D levels are used as an indicator of body vitamin D stores. Accordingly, vitamin D levels above 30 ng/mL are considered adequate. Limit values are as follows: Less than 10 ng/mL= severe vitamin D deficiency, 10-19 ng/mL = vitamin D deficiency, 20-29 ng/mL = vitamin D insufficiency and ≥30 ng/mL is considered as normal vitamin D level (3). Nowadays, deficiency of vitamin D is regarded as a global epidemic (4). In a study conducted by Ucar; 20.7% of population were found to have vitamin D insufficiency and a high proportion of population (51.8%) were found to have vitamin D deficiency in Turkey (5).

Insulin resistance is defined as biological insensitivity to endogenous or exogenous insulin and is an important component of type 2 diabetes, metabolic syndrome and cardiovascular diseases (6). Onat et al. investigated the prevalence of insulin resistance by using HOMA-IR (Homeostasis Model Assesment of Insulin Resistance) formula in their study including 1534 subjects in Turkey. According to the study, it was found that the prevalence of the insulin resistance is 21% in adults without metabolic syndrome in Turkey. However, in patients with metabolic syndrome, this rate increases to 32.5% in females and 45.5% in males. Insulin resistance was found to be high in males from the age of 30 and not significantly increased with age, but it was found to be low in females during reproductive period and significantly increased with aging and menopause (7). In a study conducted by Cade et al, a positive correlation was found between vitamin D levels and insulin sensitivity (8). However, there was also a positive correlation between vitamin D levels and insulin sensitivity in subjects with normal glucose tolerance and body mass index. So, it is considered that vitamine D is associated with insulin resistance and a risk factor for diabetes and metabolic syndrome (9). Insulin secretion from pancreatic beta cells in vitamin D deficiency is decreasing. Dietary vitamin D supplementation increases insulin conversion of proinsulin as well as improves impaired insulin release. Increasing the vitamin D level from 10 ng/mL to 30 ng/mL increases the insulin sensitivity for about 60%. A decrease in insulin resistance may correct impaired glucose tolerance by reducing the load on the pancreatic beta cells (10).

Potential relationships between vitamin D, dyslipidemia, and insulin resistance may lead to early diagnosis and early treatment of related diseases, as well as to reduce disease-related complications. Therefore, the relationship between vitamin D, dyslipidemia and insulin resistance needs to be clarified. For this purpose, we aimed to investigate the relationship between vitamin D levels and dyslipidemia and insulin resistance in our study. Our study has some novelty as we examined vitamin D levels of the people in eastern part of Turkey. There are very few investigations in this area (11,12).

## MATERIAL AND METHODS

**Patients:**

A total of 249 patients were included in the study who had been applied to the outpatient clinic of Kafkas University Medical School between January 1 to March 31, 2016, without a known chronic disease, and have normal liver and kidney function tests.

Our exclusion criteria were patients having type 1 or 2 diabetes mellitus (DM), pregnant women, patients having heart failure, active infection, acute or chronic inflammatory disease, uncontrolled hypertension, history of cardiovascular or cerebrovascular event, chronic renal disease, thyroid or parathyroid disease (active or past). Subjects with malignancy, tumor lysis syndrome, chronic diseases of renal and liver, skin disorders, malabsorption, inflammatory bowel or Celiac disease (in history or nowadays), and ones taking medications that may interfere serum levels of Vit D were also excluded.

After detailed physical examination, in all subjects body weight and height were measured. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m2).

Blood was drawn after 12 hour of overnight fasting, at 08.30 a.m. for fasting plasma glucose (FPG), serum total cholesterol (TC) and high density lipoprotein cholesterol (HDL-C), triglyceride (TG), and hemoglobin A1c (HbA1c), urea, creatinine, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), calcium, phosphorus, parathormone (PTH).

Patients were divided into 3 groups in terms of the age, 18 to 35 years, 36 to 50 years and over 50 years old. Vitamin D values were divided into groups according to the limit values and 4 groups were formed as normal, vitamin D insufficiency, vitamin D deficiency and severe vitamin D deficiency.

In this study, insulin resistance was calculated using the HOMA-IR formula as Insulin (μU/ml) x Glucose (mg/dl) /405 and it was accepted as HOMA-IR> 2.5 (13).

Written consent of the study subjects, and ethics committee approval from our center were obtained. This study was performed according to the Helsinki decleration 2008.

**Laboratory methods:**
Serum 25-Hydroxvitamin D (25 (OH) D), insulin, HbA1c, parathormone (PTH) levels were determined by Cobas e 411 (Roche Diagnostics GmbH, Mannheim, Germany). Serum AST, ALT, alkaline phosphatase (ALP), urea, creatinine, calcium, phosphorus, LDL, triglyceride, total cholesterol, HDL levels were determined by a biochemical analyzer Cobas 6000 C501 (Roche Diagnostics GmbH, Mannheim, Germany).

**Statistical Analysis:**
Statistical analysis was performed using SPSS 19.0 for Windows (SPSS, Inc, Chicago, USA) packet program. Pearson chi-square, Yates corrected Chi-square and Fisher tests were used to compare categorical variables. Continuous variables were compared with nonparametric tests (Mann-Whitney U and Kruskal Wallis Test). The relationship between variables was assessed using the Spearman Correlation Test. Statistical significance level was accepted as $p < 0.05$.

**RESULTS**
Of the 249 people included in the study, 84 (33.7%) were male and 165 (66.3%) were female. The mean age was 46.69 ± 13.26 years. Twenty two point five percent of subjects were 18-35 years, 36.1% were 36-50 years old and 41.4% were over 50 years old. The distribution of values of the subjects according to sex and age groups are given in **Table 1.**

AST, ALT, urea, creatinine, TG, Ca, vitamin D levels of men were statistically higher than women and HDL-C were lower. Among the age groups, urea, creatinin, FBG, HbA1c and ALP was found to be different and it was observed that ALP levels increased significantly with increasing age.

When vitamin D levels of subjects were examined, it was seen that 3.6% was normal, 14.5% had vitamin D insufficiency, 45% had vitamin D deficiency and 36.9% had severe vitamin D deficiency. In men, normal vitamin D levels, vitamin D insufficiency, vitamin D insufficiency and severe vitamin D insufficiency rates were found to be 3.6%, 14.5%, 45% and 36.9%, respectively. While 46.1% of the women had severe deficiency, 37 % had deficiency and 13.9% had deficiency and 3% had normal vitamin D levels. The difference between gender was statistically significant ($p < 0.001$). When vitamin D levels were compared according to age groups, there was no significant difference between the groups.

**Table 2** compares vitamin D levels of subjects with TC, LDL-C, HDL-C and TG levels. According to this, no significant difference was found between vitamin D groups in terms of lipid parameter values ($p > 0,05$). When the correlation between vitamin D and TC, LDL-C, HDL-C and TG values of subjects was evaluated, between vitamin D level and LDL-C (r : -0,145, p: 0,022), TG (r:-0,158, p=0,012) and TC (r: 0,139, p: 0,029) showed a weak negative correlation **(Table 3, Figure1).**

There was a significant difference between the groups in terms of all of these variables when the vitamin D groups are compared with HbA1c, fasting blood glucose, insulin and HOMA-IR levels ($p < 0,05$). The correlation between the groups was evaluated. While negative weak correlation was detected between vitamin D and HbA1c (r=0,230, p<0,001), negative moderate correlation was found between vitamine D and glucose (r:-0,355, p<0,001), insulin (r:-0,455, p<0,001) and HOMA-IR levels (r:-0,482, p<0,001). As vitamin D decreased, fasting blood glucose, fasting insulin and HOMA-IR values increased **(Table 4, Figure 2).**

Subjects' BMI values were divided into 3 groups; less than 25kg/m2, 25 to 30 kg/m2 and over 30 kg/m2. When the relationship between BMI and insulin resistance was examined, insulin resistance was observed in 17.4% of the subjects with BMI less than 25 kg/m2, 34.6% in those with BMI between 25-30 kg/m2 and 59.3% in those with BMI over 30 kg/m2 (p<0,05).

In our study; insulin resistance was detected in 57.6% of those with severe vitamin D deficiency, 22.3% of those with vitamin D deficiency and 8.3% of those with vitamin D insufficiency. Insulin resistance was not detected in any of the patients with normal vitamin D levels. As the degree of vitamin D deficiency increased, the incidence of insulin resistance also increased **(Table 5).**

**DISCUSSION**
The mean vitamin D level of subjects in our study was 13.5 ng/mL. Vitamin D levels were found to be 15 ng/mL in males and 12,8 ng/mL in females. The difference of vitamin D levels between gender was statistically significant. Severe vitamin D deficiency is much more common in women than in men. This suggests that vitamin D deficiency is a more serious problem for women. As age increases, vitamin D deficiency seems to increase, but there is no significant difference in terms of vitamin D groups among the ages. There are few studies specifically investigating VitD levels of the people who live in Eastern part of our country (11,12) and also comparing those levels in eastern and western parts of Turkey. Very low levels of vitamin D levels were found in children of the eastern part of Turkey as 5.8 ng/mL (11). Gür et al found significantly lower VitD levels in pregnant women living in Erzurum than those levels of pregnant women from İzmir (12).

In an English study, more than half of the adults were found to have vitamin D deficiency in the winter-spring period and severe vitamin D deficiency was detected in 16% of the adults (14). In a study conducted in South Korea, the mean level of vitamin D was found to be 25.8 ng/mL (15). In our study, the severity of severe vitamin D deficiency was twice as much as in the England, while the average vitamin D level was half the level of South Korea. In almost all studies, with normal Turkish individuals, vitamin D levels were found to be below normal limits (16,17).

The season when the study was performed, genetical variations, our clothing style, limited intake of food high in vitamin D, lack of outdoor physical activity due to the season must be considered as the reason of hypovitaminosis D in our country. In 2 studies of ours we found 14.3 ng/mL and 19 ng/mLvitamin D levels in normal people (18,19). Although this suggests that vitamin D deficiency is a serious problem for our country, the fact that the climate conditions of Kars province are strenuous and our study was carried out

**Table 1 Characteristics of the subjects according to gender and age groups**

| | **Gender** | | **p*** | **Age** | | | **p**** | |
|---|---|---|---|---|---|---|---|---|
| | Men | Women | | 18-35 | 36-50 | Above50 | | Total |
| AST (U/I) | 20.5±6.5 | 18.2±5.7 | **0.004** | 20.0±7.2 | 18.7±5.8 | 18.7±5.6 | NS | 19.0±6.0 |
| ALT (U/I) | 23.7±11.2 | 17.9±8.3 | **<0.001** | 20.6±13.4 | 20.1±9.2 | 19.1±7.7 | NS | 19.8±9.7 |
| Urea(mg/dL) | 32.8±7.8 | 28.8±10.4 | **<0.001** | 24.3±6.4 | 28.5±8.0 | 34.8±10.5 | **<0.001** | 30.2±9.8 |
| Creatinine (mg/dL) | 0.9±0.1 | 0.7±0.1 | **<0.001** | 0.7±0.1 | 0.7±0.1 | 0.8±0.1 | **0.027** | 0.7±0.1 |
| FBG (mg/dL) | 92.8±7.4 | 92.3±7.8 | NS | 89.6±5.8 | 92.4±8.5 | 94.0±7.5 | **0.004** | 92.4±7.7 |
| FI (µu/mL) | 9.1±5.2 | 10.2±5.5 | NS | 10.2±6.4 | 9.5±5.2 | 10.0±4.9 | NS | 9.9±5.4 |
| HbA1c (%) | 5.4±0.3 | 5.4±0.4 | NS | 5.2±0.3 | 5.5±0.2 | 5.5±0.4 | **<0.001** | 5.4±0.3 |
| BMI(kg/m2) | 26.5±2.9 | 26.9±3.3 | NS | 24.6±3.6 | 26.9±2.5 | 27.8±2.9 | NS | 26.7±3.2 |
| HOMA-IR | 2.1±1.3 | 2.3±1.2 | NS | 2.2±1.4 | 2.2±1.2 | 2.3±1.2 | NS | 2.2±1.3 |
| T-C (mg/dL) | 187.9±32.4 | 194.0±35.7 | NS | 170.5±33.1 | 196.3±35.5 | 199.9±29.9 | **<0.001** | 192.0±34.7 |
| LDL -C (mg/dL) | 117.7±28.6 | 119.8±30.6 | NS | 99.3±28.9 | 123.4±30.0 | 126.0±25.7 | **<0.001** | 119.1±29.9 |
| HDL-C (mg/dL) | 42.2±11.0 | 50.2±12.2 | **<0.001** | 48.2±12.2 | 46.8±10.9 | 47.7±13.8 | NS | 47.5±12.4 |
| TG(mg/dL) | 140.1±65.1 | 120.2±52.8 | **0.023** | 114.5±59.0 | 130.6±56.0 | 130.6±58.5 | NS | 126.9±57.8 |
| Ca (mg/dL) | 9.3±0.3 | 9.2±0.4 | **0.005** | 9.2±0.3 | 9.2±0.3 | 9.2±0.4 | NS | 9.2±0.3 |
| P (mg/dL) | 3.3±0.4 | 3.4±0.4 | NS | 3.4±0.4 | 3.3±0.4 | 3.4±0.4 | NS | 3.4±0.4 |
| PTH (pg/mL) | 47.2±13.8 | 54.8±18.9 | **0.003** | 50.3±16.6 | 51.5±17.3 | 54.0±18.5 | NS | 52.2±17.7 |
| Vit D (µg/L) | 15.0±7.1 | 12.8±7.4 | **0.002** | 14.6±8.7 | 13.2±6.6 | 13.3±7.2 | NS | 13.5±7.4 |
| ALP (U/L) | 85.0±20.5 | 80.0±25.4 | NS | 73.9±21.2 | 79.6±22.3 | 87.8±25.3 | **0.001** | 81.7±23.9 |

*Mann Whitney U Test, **Kruskal Wallis Test

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, FBG: Fasting blood glucose, FI: Fastinginsulin, HbA1c: Hemoglobin A1c, HOMA-IR: Homeostasis model assessment- insulin resistance index, TC: Total cholesterol, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, TG: Triglyceride, Ca: Calcium, P: Phosphorus, PTH: Parathyroid hormone, VitD: Vitamin D, ALP: Alkaline phosphatase. Data are presented as mean ± SD

**Table 2 Comparison of TC, LDL-C, HDL-C, TG, FBG, FI, HbA1c and HOMA-IR values of vitamin D groups in the subjects**

| VitD | TC | LDL-C | HDL-C | TG | FBG | FI | HbA1c | HOMA-IR |
|---|---|---|---|---|---|---|---|---|
| Severe deficiency (n=92) | 197.9±30.4 | 123.1±26.4 | 47.0±11.9 | 138.4±64.0 | 95.2±7.5 | 12.7±6.1 | 5.5±0.3 | 2.9±1.4 |
| Deficiency (n=112) | 190.8±37.6 | 119.5±32.5 | 46.5±12.1 | 123.5±55.1 | 92.0±7.3 | 8.5±4.5 | 5.4±0.3 | 1.9±1.1 |
| Insufficiency (n=36) | 182.2±35.7 | 108.1±30.5 | 51.3±14.2 | 113.9±49.8 | 88.4±6.9 | 7.4±2.9 | 5.4±0.6 | 1.6±0.6 |
| Normal (n=9) | 185.6±25.4 | 116.5±18.6 | 48.2±12.2 | 104.4±35.5 | 86.0±5.7 | 7.3±2.9 | 5.3±0.2 | 1.5±0.6 |
| P | NS | NS | NS | NS | <0.001 | <0.001 | 0.007 | <0.001 |

VitD: Vitamin D, TC: Total cholesterol, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, TG: Triglyceride FBG: Fasting blood glucose, FI: Fasting insulin, HbA1c: Hemoglobin A1c, HOMA-IR: Homeostasis model assessment- insulin resistance index. Data are presented as mean ± SD. p: Significance level, *Kruskal Wallis Test

**Table 3 Correlation between vitamin D and lipid values of the subjects**

| | | VitD | TC | LDL-C | HDL-C | TG |
|---|---|---|---|---|---|---|
| **VitD** | r<br>p | - | -0.139<br>0.029 | -0.145<br>0.022 | 0.106<br>NS | -0.158<br>0.012 |
| **TC** | r<br>p | -0.139<br>0.029 | - | 0.925<br><0.001 | 0.158<br>0.012 | 0.396<br><0.001 |
| **LDL-C** | r<br>p | -0.145<br>0.022 | 0.925<br><0.001 | - | -0.036<br>NS | 0.269<br><0.001 |
| **HDL-C** | r<br>p | 0.106<br>0.094 | 0.158<br>0.012 | -0.036<br>NS | - | -0.417<br><0.001 |
| **TG** | r<br>p | -0.158<br>0.012 | 0.396<br><0.001 | 0.269<br><0.001 | -0.417<br><0.001 | - |

VitD: Vitamin D, TC: Total cholesterol, LDL-C: Low density lipoprotein cholesterol, HDL-C: High density lipoprotein cholesterol, TG: Triglyceride

**Table 4 Correlation between Vitamin D values and FBG, FI, HbA1c and HOMA-IR**

| | | Vit D | FBG | FI | HbA1c | HOMA-IR |
|---|---|---|---|---|---|---|
| **Vit D** | r<br>p | - | -0.355<br><0.001 | -0.455<br><0.001 | -0.230<br><0.001 | -0.482<br><0.001 |
| **FBG** | r<br>p | -0.355<br><0.001 | - | 0.261<br><0.001 | 0.319<br><0.001 | 0.393<br><0.001 |
| **FI** | r<br>p | -0.455<br><0.001 | 0,261<br><0,001 | - | 0.192<br>0.002 | 0.987<br><0.001 |
| **HbA1c** | r<br>p | -0.230<br><0.001 | 0.319<br><0.001 | 0.192<br>0.002 | - | 0.233<br><0.001 |
| **HOMA-IR** | r<br>p | -0.482<br><0.001 | 0.393<br><0.001 | 0.987<br><0.001 | 0.233<br><0.001 | - |

VitD: Vitamin D, FBG: Fasting blood glucose, FI: Fasting insulin, HbA1c: Hemoglobin A1c, HOMA-IR: Homeostasis model assessment- insulin resistance index.

**Table 5 Comparison of the Vitamin D groups with insulin resistance**

| | | Insulin Resistance | | | |
|---|---|---|---|---|---|
| | | Negative | | Positive | |
| | | Frequency | % | Frequency | % |
| Vitamin D | Severe deficiency (<10 ng/ml) | 39 | 42.4 | 53 | 57.6 |
| | Deficiency (10-19 ng/ml) | 87 | 77.7 | 25 | 22.3 |
| | Insufficiency (20-29 ng/ml) | 33 | 91.7 | 3 | 8.3 |
| | Normal (≥30 ng/ml) | 9 | 100.0 | 0 | 0.0 |

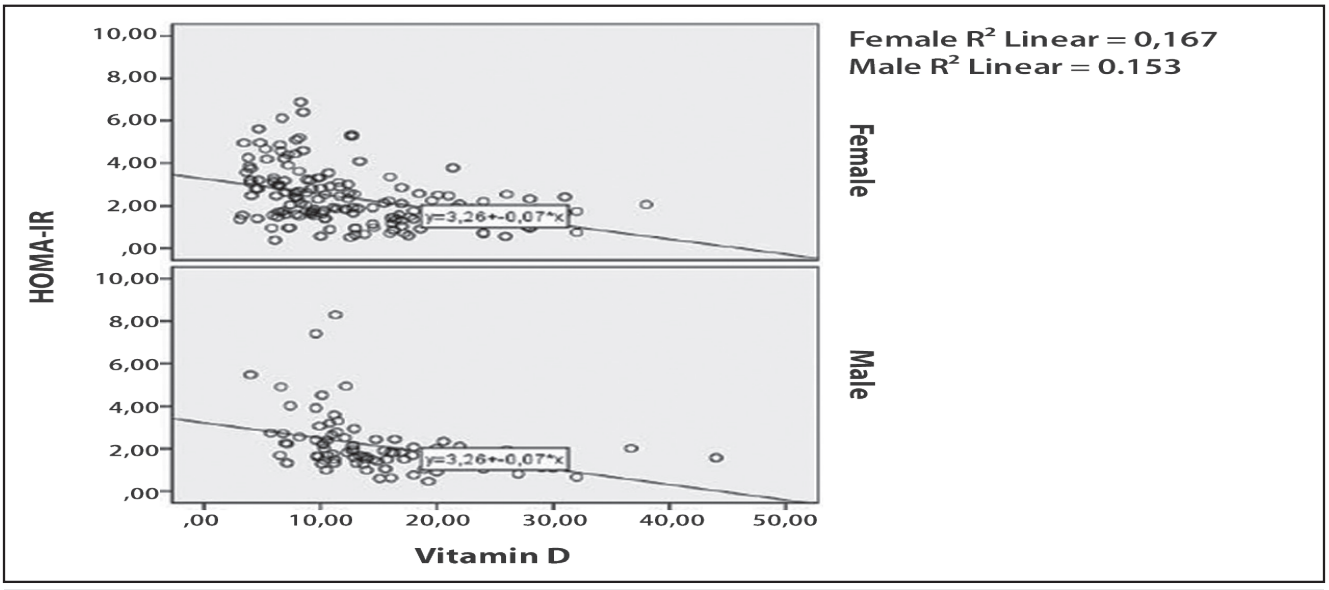


**Figure 1 Correlation between Vitamin D and LDL-C values of the subjects**

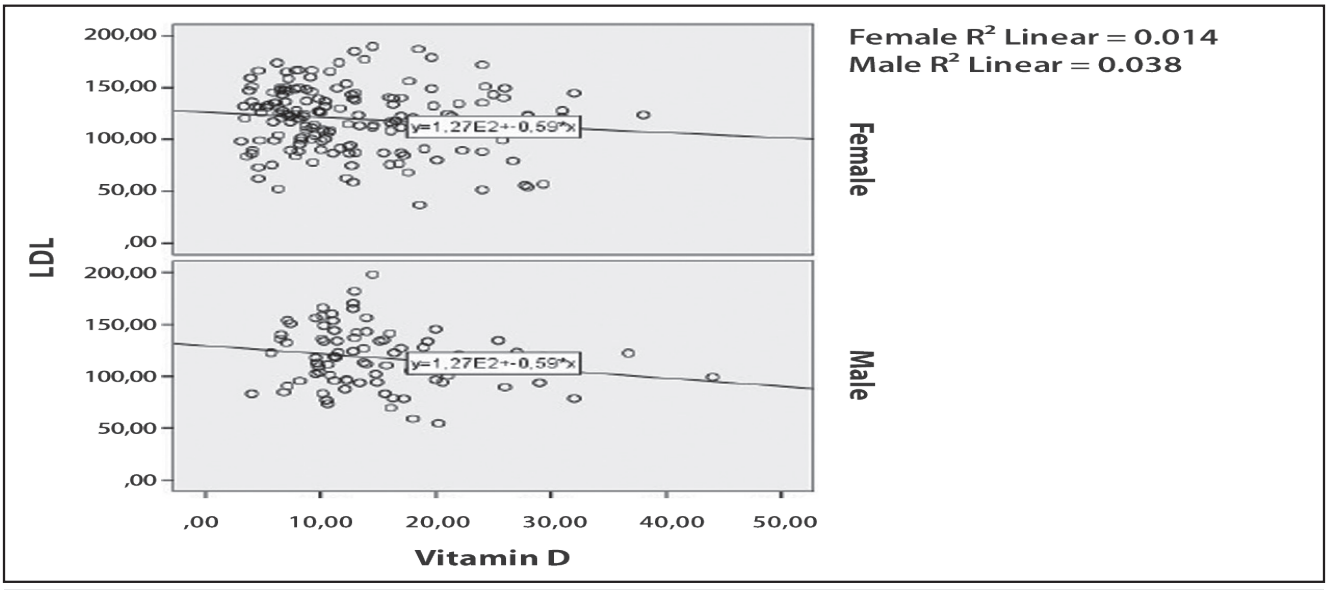


**Figure 2 Correlation between Vitamin D and HOMA- IR values of the subjects**

between January and March may be a reason.

A high prevalence of hipovitaminosis D was noted in diabetics (20-22). In a study of ours we demonstrated significantly lower vitamin D levels in type 2 DM patients than the controls (18). It was stated that a deranged vitamin D status may reflect an increased risk of type 2 diabetes mellitus (T2DM) in the general population (21). Moreover data from adults showed that hypovitaminosis D is correlated with the development of impaired glucose tolerance (24) and impaired fasting glucose (25). In concordance with the literature when we compared all vitamin D groups were with HbA1c, FBG, insulin and HOMA-IR levels we found that as the degree of Vitamin D deficiency increased HbA1c, FBG, insulin and HOMA-IR levels also increased.

Studies support a role for vitamin D in both secretion and sensitivity of insulin (26,27). Liu and co-workers found that, compared with the participants in the lowest tertile category of plasma 25(OH)D, those in the highest tertile category had 12.7% lower HOMA-IR score (28). Ford et al. also demonstrated that vitamin D status was inversely associated with insulin resistance (29). In our study while insulin resistance was detected in 57.6% of those with severe vitamin D deficiency, 22.3% of those with vitamin D deficiency and 8.3% of those with vitamin D insufficiency it was not detected in any of the patients with normal vitamin D levels.

As the level of vitamin D deficiency increases, the incidence of insulin resistance also increases. We examined the correlation between vitamin D, glucose, insulin and HOMA-IR values. Accordingly, there was a negative weak correlation between vitamin D and HbA1c levels and a negative moderate correlation between vitamin D levels and FBG, insulin and HOMA-IR levels. As vitamin D decreased, FBG, FI and HOMA-IR values increased. Unlike our study Erdönmez and colleagues evaluated serum vitamin D and insulin resistance where they did not find any correlation between serum vitamin D levels and insulin resistance in all groups (30). But there were studies supporting our findings. In a study conducted in Chinese with volunteers without glucose intolerance an inverse relationship between vitamin D and insulin resistance was found, and they stated that the level of low vitamin D was a risk factor for many metabolic diseases (31). In Badawi and colleagues' study there was an inverse relationship between plasma vitamin D level and insulin resistance for both gender (32).

There are conflicting results about the relationship of hypovitaminosis D and levels of TC, LDL-C, HDL-C, TG and apoliporotein A-1 (29,33). In another study of ours in metabolic syndrome patients, TC, LDL-C, and TG levels were higher and HDL-C levels were statistically lower than the control group, where vitamin D levels were lower than the control group (19). In the present study there was no significant difference between vitamin D groups and lipid parameters. In addition, dyslipidemia was detected in 68.9% of those with normal vitamin D levels and 80.9% of those with vitamin D deficiency. The difference is not statistically significant.

In a study conducted by Chadhuri and colleagues, it was shown that the incidence of dyslipidemia in those with vitamin D deficiency was higher than those without vitamin D deficiency (34). In a study conducted by Zhang et al. there was a significant reverse relationship between vitamin D and LDL but not between HDL and TG (32). Although there are many researches in the literature showing the relationship between vitamin D and LDL (31,33,34) , there are also researches that do not support this relationship (38). Our findings support that vitamin D levels are not associated with lipid parameters.

There are a few limitations of this study. One is the moderate sample size. Second, laboratory values evaluated in this study represents only one point in time. Third, we performed the study in winter season, it is obvious that seasonal variations could have influenced the results. Fourth, the gold standard for the measurement of insulin sensitivity is the use of the euglycemic clamp; we demonstrated insulin resistance by an indirect method; HOMA-IR. Fifth, although the effect of vitamin D supplementation on dyslipidemia and insulin resistance were controversial we did not have the chance of studying those parameters after vitamin D was given. Finally, the findings are limited to our groups, which included only adults from an eastern part of Turkey, so our results may not be applicable to all our country or other nationalities.

In conclusion, findings in this study showed that vitamin D, and insulin resistance can not be considered independent of each other, but dyslipidemia is related to vitamin D levels. Further prospective studies are needed to evaluate the effect of vitamin D on metabolic parameters in eastern part of Turkey.

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