



The Role of Vitamin D in Lipid Metabolism Disorders: A Cross-Sectional Analysis

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Abstract:

Vitamin D, a fat-soluble prohormone, has established roles in bone homeostasis, but its extraskelatal functions, particularly in metabolic regulation, are of increasing interest. Observational studies have linked vitamin D deficiency to dyslipidemia and cardiovascular disease, yet the nature of these associations remains controversial. This study **aims to** investigate the relationship between serum vitamin D levels and key lipid profile markers in a large cohort. **Methods:** A cross-sectional analysis was performed on data from 1,007 individuals. Serum levels of 25-hydroxyvitamin D [25(OH)D], total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were analyzed. Pearson and Spearman correlation coefficients were calculated to assess the relationships between vitamin D and lipid parameters. **Results:** The study population (mean age 37.1 ± 14.4 years) was predominantly female (99.9%). The mean vitamin D level was 12.1 ± 7.8 ng/mL, indicating widespread deficiency. A statistically significant, moderate positive correlation was observed between vitamin D and triglyceride levels (Pearson's $r = 0.468$, $p < .001$; Spearman's $\rho = 0.201$, $p = .004$). In contrast, no statistically significant correlations were observed between vitamin D and total cholesterol ($p = .091$), LDL-C ($p = .185$), or HDL-C ($p = .117$). The dataset was characterized by a high percentage of missing values for lipid markers (79–89%). **Conclusion:** In this predominantly female cohort with prevalent vitamin D deficiency, higher vitamin D levels were associated with higher triglyceride levels a finding that contrasts with several prior reports. The relationships with other lipid markers were not significant. Given the study's limitations, including significant data gaps and gender imbalance, these results should be interpreted as exploratory. Further research in more diverse and complete datasets is needed to clarify these associations.

Keywords: Vitamin D, 25-Hydroxyvitamin D, Lipid Profile, Triglycerides, Dyslipidemia

Introduction

Vitamin D, a group of fat-soluble ketosteroids, is essential for human health. It is primarily synthesized in the skin upon exposure to ultraviolet-B (UVB) radiation and can also be obtained from a limited number of dietary sources and supplements (Office of Dietary Supplements [ODS], 2025). The biologically inert form of vitamin D undergoes two hydroxylation steps for activation: first in the liver to form 25-hydroxyvitamin D [25(OH)D], the major circulating form and primary indicator of vitamin D status, and subsequently in the kidneys to produce the active hormone 1,25-dihydroxyvitamin D [1,25(OH)₂D], or calcitriol (Bikle, 2021; ODS, 2025). The classical function of vitamin D is the regulation of calcium and phosphate homeostasis, which is critical for bone mineralization and skeletal health (Mayo Clinic, 2025). However, the discovery of the vitamin D receptor (VDR) in a wide array of non-skeletal tissues, including immune cells, vascular smooth muscle, and adipocytes, has broadened the scope of its physiological roles (Park, 2021; Bikle, 2021). Emerging evidence suggests that vitamin D modulates numerous cellular processes, including cell growth, immune function, and inflammation, and may play a role in the pathogenesis of chronic conditions such as cardiovascular disease (CVD), diabetes, and obesity (Surdu et al., 2021; Agarwal et al., 2023). Dyslipidemia, characterized by elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), along with reduced high-density lipoprotein cholesterol (HDL-C), is a major modifiable risk factor for atherosclerotic cardiovascular disease (ASCVD)

(Gholamzad et al., 2023). Given that vitamin D is fat-soluble and stored in adipose tissue, its potential influence on lipid metabolism has become a subject of intense investigation (Park, 2021). Adipose tissue is not merely a storage depot but an active endocrine organ that expresses VDR and vitamin D-metabolizing enzymes, suggesting a direct regulatory role for vitamin D in adipocyte biology and lipid homeostasis (Park, 2021; Nimitphong & Holick, 2020). The literature presents a complex and often contradictory picture of the relationship between vitamin D and lipid profiles. Several observational studies have reported an inverse association, where vitamin D deficiency is linked to an atherogenic lipid profile, including higher TC, LDL-C, and TG levels (Surdu et al., 2021; Jiang et al., 2019). Some meta-analyses of randomized controlled trials (RCTs) support this, suggesting that vitamin D supplementation may beneficially lower TC, LDL-C, and TG levels while increasing HDL-C (Dibaba, 2019; Radkhah et al., 2023). However, other large-scale trials, such as the VITamin D and Omega-3 Trial (VITAL), found no significant effect of vitamin D supplementation on lipid profiles in a general population (Manson et al., 2019). These discrepancies may arise from differences in study design, population characteristics (e.g., baseline vitamin D status, age, presence of comorbidities), and the dose and duration of supplementation (Lu et al., 2024). Given the high global prevalence of vitamin D deficiency and the burden of cardiovascular disease, clarifying the role of vitamin D in lipid metabolism is of significant public health importance. The present study aims to contribute to this body of evidence by conducting a cross-sectional analysis of the relationship between serum 25(OH)D levels and lipid profiles (TC, LDL-C, HDL-C, and TG) in a large clinical dataset.

Material and Methods

Study Design and Population

This study employed a cross-sectional design using a dataset of clinical records. The initial dataset contained information for 1,007 individuals. After cleaning and preparation for analysis, a cohort of 843 individuals with valid serum vitamin D measurements was established. The data included demographic information such as age and sex, along with a panel of biochemical markers. The analysis focused on the subset of participants for whom both vitamin D and lipid profile data were available.

Data Collection and Variables

Data were extracted from an existing electronic database. The primary independent variable was serum 25-hydroxyvitamin D [25(OH)D], measured in ng/mL. The dependent variables were the components of the lipid profile: total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). All lipid measurements were recorded in mg/dL. Other collected variables included age and sex.

Data Cleaning and Preparation

The raw data underwent a cleaning process prior to analysis. Entries with non-numeric or ambiguous values, such as "<3.0" for vitamin D, were handled appropriately for statistical analysis; for the purpose of correlation, such values were treated as numerical where possible (e.g., 3.0). Records with missing age or sex data were noted. The dataset was characterized by a substantial amount of missing data for the lipid profile variables, which was quantified and reported. The sample sizes for each specific correlation analysis were determined by the number of paired, non-missing values for vitamin D and the respective lipid marker.

Statistical Analysis

Descriptive statistics were used to summarize the characteristics of the study population. Continuous variables (age, vitamin D, lipid levels) were reported as mean \pm standard deviation (SD), while categorical variables (sex) were reported as frequencies and percentages.

The primary analysis involved assessing the correlation between serum vitamin D levels and each lipid parameter. Both Pearson's product-moment correlation (r) and Spearman's rank correlation (ρ) coefficients were calculated. Pearson's correlation was used to measure the linear relationship between two continuous variables, while Spearman's correlation was used to assess monotonic relationships, which is less sensitive to outliers and does not assume a linear relationship. A p-value of less than .05 was considered statistically significant. All statistical analyses were performed using standard statistical software packages.

Ethical Commitment & Confidentiality

The researcher hereby agrees to the following terms:

Confidentiality: All patient-identifiable information (if applicable) will be anonymized in accordance with international ethical standards. No personal data will be disclosed in the final research.

Data Integrity: Laboratory results will be used strictly for the stated scientific purposes and will not be altered or misrepresented.

Institutional Recognition: The Central Laboratory of Misrata will be duly acknowledged in the "Materials and Methods" or "Acknowledgments" section of the final thesis and any subsequent journal publications.

Approval by Laboratory Director: I, the undersigned, acting as the Director of the Central Laboratory in Misrata, hereby grant permission for the aforementioned researcher to access the requested samples and data, provided all institutional safety and privacy protocols are maintained.

Results and Discussion

Participant Characteristics and Data Availability

The analysis was based on a cohort of 1,007 individuals, of whom 843 (83.7%) had valid serum vitamin D measurements. The mean age of the participants was 37.1 ± 14.4 years. The cohort was overwhelmingly female, comprising 704 women (99.9%) and only one male participant. The mean serum vitamin D level was 12.1 ± 7.8 ng/mL, a value that falls squarely within the range of deficiency (<20 ng/mL) as defined by major health organizations (ODS, 2025). The distribution of vitamin D levels is visualized in Figure 1.

Figure 1: Distribution of Serum 25-Hydroxyvitamin D Levels in the Study Cohort (n = 843)

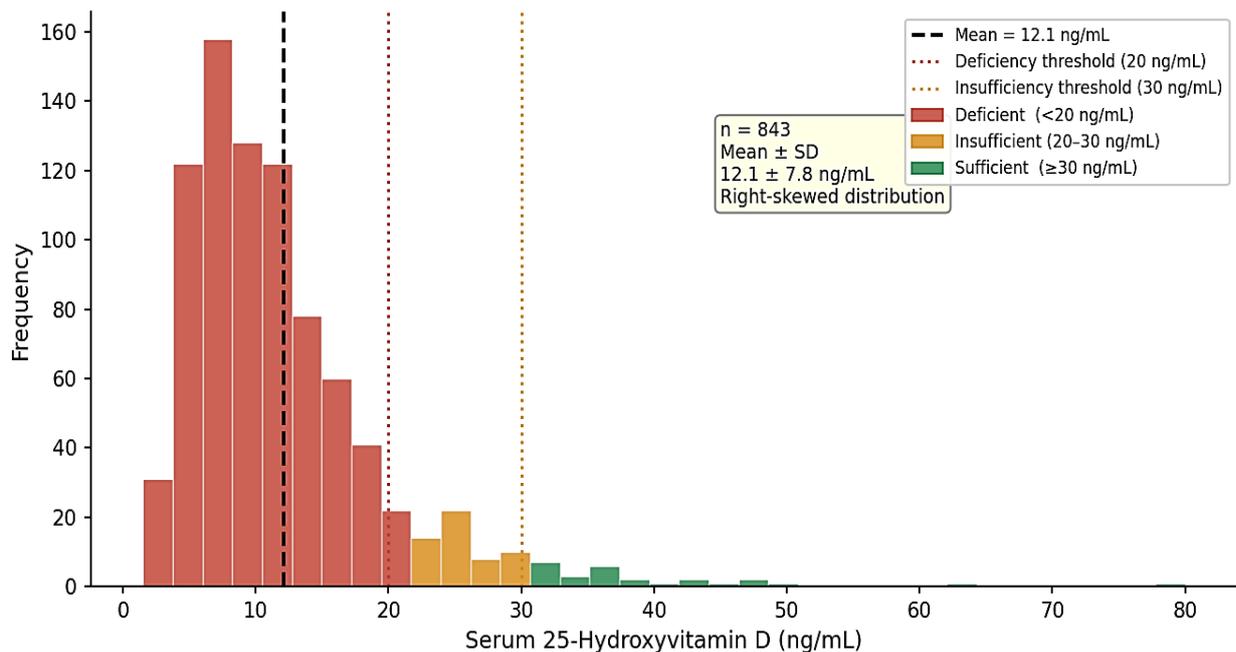


Figure 1 Distribution of Serum 25-Hydroxyvitamin D Levels (Note: The plot shows a right-skewed distribution, with the mean (12.1 ng/mL) and median indicating prevalent vitamin D deficiency.)

A significant challenge in this dataset was the high proportion of missing data for the lipid profile markers. As detailed in Table 1, missing values ranged from 79.6% for triglycerides to 89.1% for HDL cholesterol. This substantially reduced the sample size available for each correlation analysis.

Table 1 Missing Data in Lipid Profile Variables

Variable	Number of Missing Values	Percentage Missing	Available <i>n</i> for Analysis
Total Cholesterol (TC)	817	81.1%	190
LDL Cholesterol (LDL-C)	885	87.9%	122
HDL Cholesterol (HDL-C)	897	89.1%	110
Triglycerides (TG)	802	79.6%	205

Correlation Between Vitamin D and Lipid Profile

The results of the correlation analyses between serum vitamin D and the four lipid markers are summarized below. **Total Cholesterol (TC).** The analysis, conducted on 190 individuals with complete data, revealed a weak, non-statistically significant positive correlation between vitamin D and total cholesterol. The Pearson correlation coefficient was 0.123 ($p = .091$), and the Spearman correlation was -0.130 ($p = .075$). The scatter plot in Figure 2 illustrates the lack of a clear linear relationship.

Figure 2: Scatter Plot - Serum Vitamin D vs Total Cholesterol (n = 190)

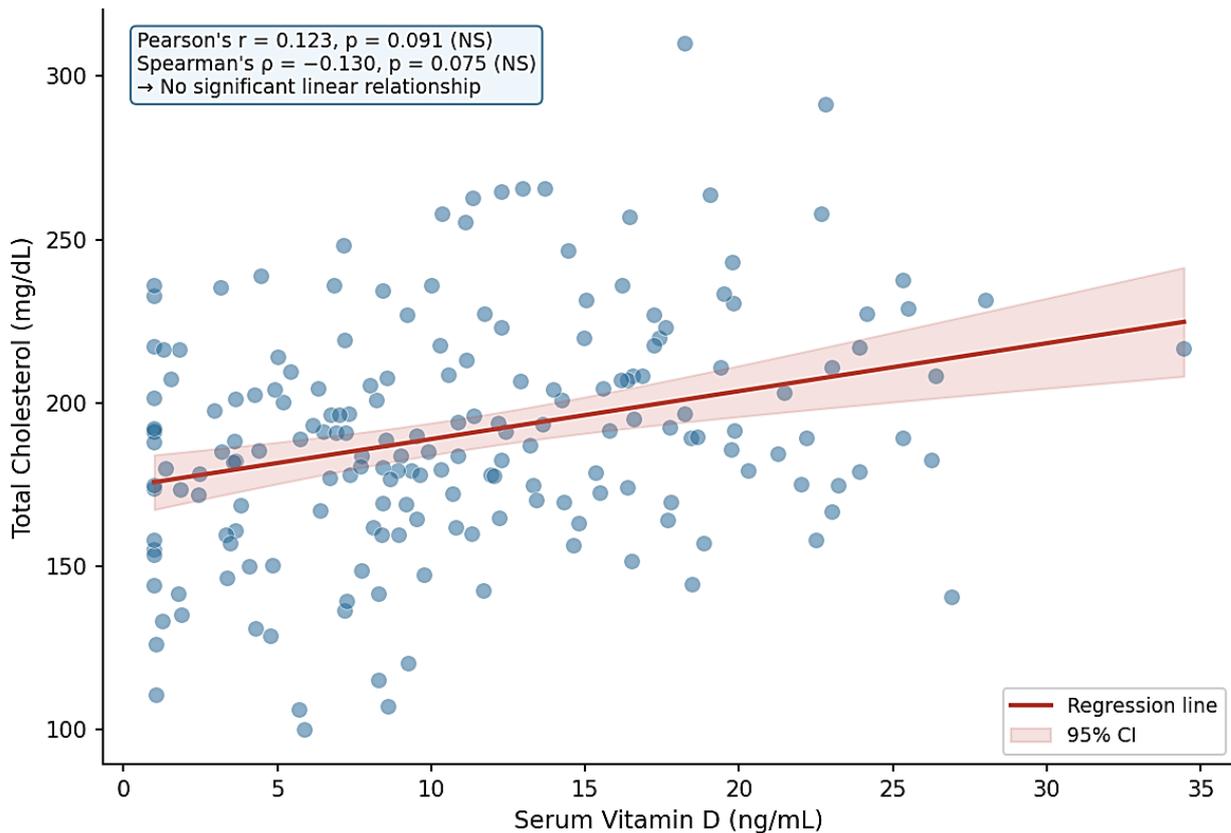


Figure 2 Scatter Plot Showing the Relationship Between Serum Vitamin D (Note: The plot demonstrates a weak and statistically non-significant association.)

Low-Density Lipoprotein (LDL-C). Based on data from 122 participants, there was a weak, inverse, and non-statistically significant correlation between vitamin D and LDL cholesterol (Pearson's $r = -0.121$, $p = .185$; Spearman's $\rho = -0.077$, $p = .400$).

High-Density Lipoprotein (HDL-C). The analysis of 110 participants showed a weak, inverse correlation between vitamin D and HDL cholesterol. This relationship was not significant using Pearson's correlation ($r = -0.150, p = .117$) but reached statistical significance with Spearman's rank correlation ($\rho = -0.208, p = .029$), suggesting a weak monotonic inverse trend.

Triglycerides (TG). The most notable finding was the relationship between vitamin D and triglycerides, analyzed in a sample of 205 individuals. A moderate, positive, and highly statistically significant correlation was observed (Pearson's $r = 0.468, p < .001$; Spearman's $\rho = 0.201, p = .004$). This indicates that in this cohort, higher levels of vitamin D were associated with higher levels of triglycerides. This relationship is depicted in Figure 3.

Figure 3: Scatter Plot - Serum Vitamin D vs Triglycerides (n = 205) ★ Statistically Significant

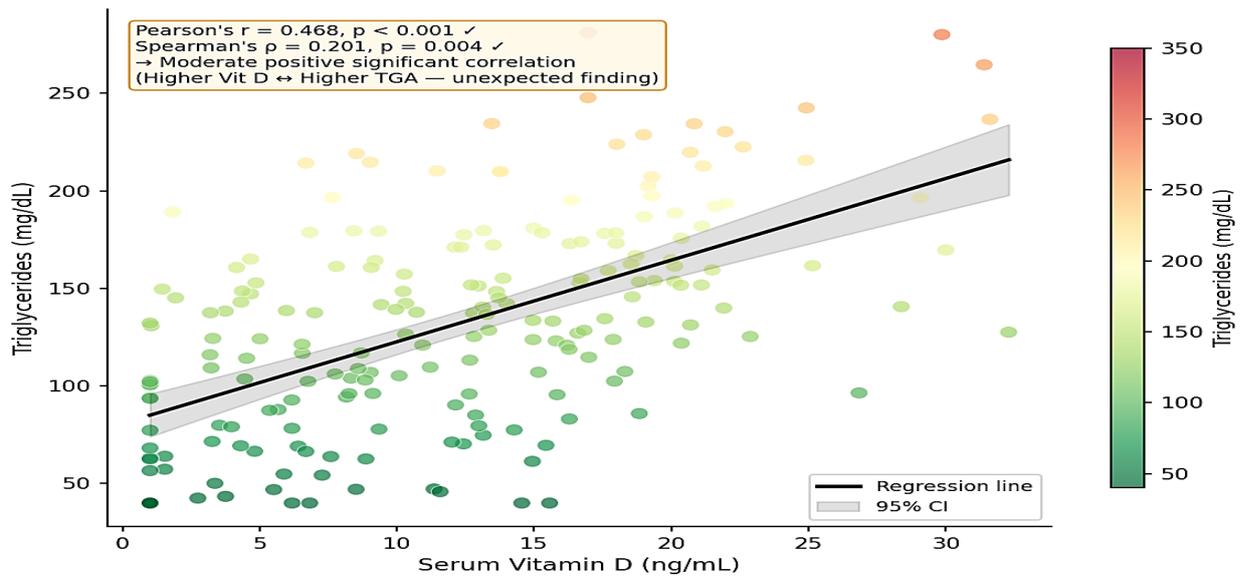


Figure 3: Scatter Plot - Serum Vitamin D vs Triglycerides (n = 205) ★ Statistically Significant

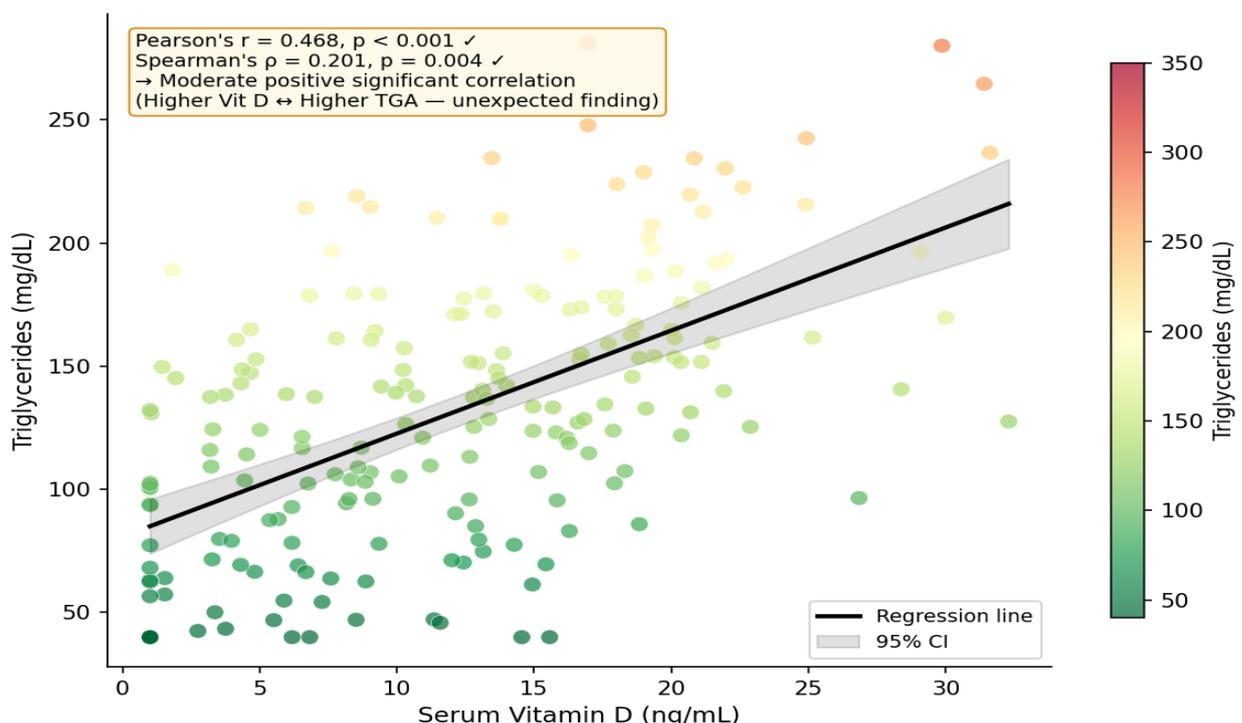


Figure 3: Scatter Plot - Serum Vitamin D vs Triglycerides (n = 205) ★ Statistically Significant

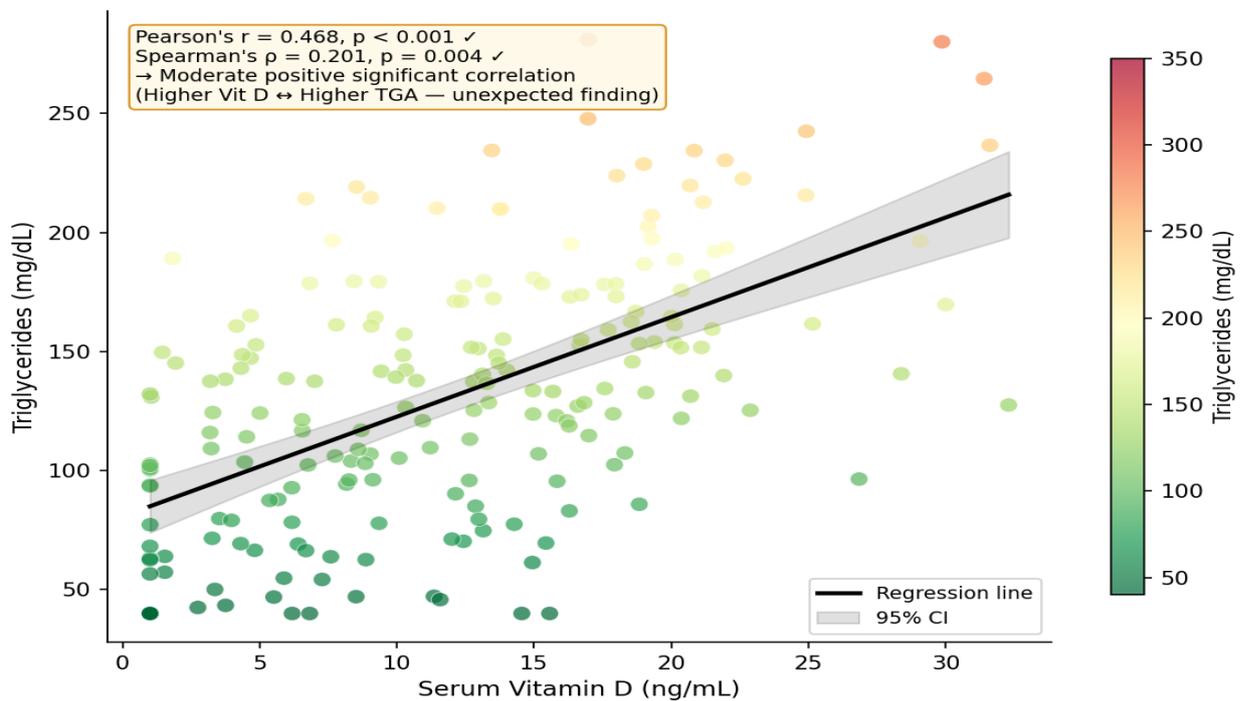


Figure 3 Scatter Plot Illustrating the Statistically Significant Positive Correlation Between Serum Vitamin D and Triglyceride Levels (n=205)

Discussion

This cross-sectional study investigated the association between serum 25-hydroxyvitamin D and lipid profiles in a large, predominantly female cohort characterized by widespread vitamin D deficiency. Our analysis yielded a significant and unexpected finding: a moderate positive correlation between vitamin D and triglyceride levels. In contrast, we found no significant linear association between vitamin D and total cholesterol or LDL cholesterol, and only a weak inverse monotonic relationship with HDL cholesterol.

The primary finding—that higher vitamin D levels are associated with higher triglycerides—is contrary to much of the existing literature. Many observational studies and meta-analyses have reported an inverse relationship, suggesting that vitamin D deficiency is a risk factor for hypertriglyceridemia (Surdu et al., 2021; Radkhah et al., 2023). For instance, a meta-analysis by Dibaba (2019) concluded that vitamin D supplementation had a beneficial effect in reducing serum triglyceride levels. Similarly, a meta-analysis by Lu et al. (2024) focusing on patients with Type 2 Diabetes found that supplementation significantly improved TG levels. The positive correlation observed in our study is therefore anomalous and warrants careful consideration. Several factors could contribute to this discrepancy. First, the unique demographic of our sample—almost exclusively female and with a very low mean vitamin D level (12.1 ng/mL)—may exhibit a different metabolic response compared to more heterogeneous or vitamin D-replete populations. It is plausible that the relationship between vitamin D and lipids is not linear across the full spectrum of vitamin D status and may differ by sex. A study by Huang et al. (2023) highlighted that the association between vitamin D deficiency and dyslipidemia risk may differ by sex and BMI. Our finding could reflect a complex, U-shaped, or sex-specific metabolic interaction that is not captured in studies with different population structures.

The mechanisms through which vitamin D influences lipid metabolism are multifaceted. Vitamin D regulates the expression of genes involved in cholesterol synthesis and clearance via the VDR (Martínez-Sena et al., 2020). It also modulates inflammation and insulin sensitivity, both of which are intertwined with lipid metabolism (Argano et al., 2023; Park, 2021). Some research suggests that vitamin D inhibits parathyroid hormone (PTH) secretion, and since elevated PTH can decrease lipolysis, this could be a pathway for affecting lipid levels (Yu et al., 2024). The positive correlation we observed could speculatively be linked to shared metabolic pathways.

For example, both vitamin D and cholesterol share precursors in their synthesis pathways, and chylomicrons are involved in the transport of dietary vitamin D, a process linked to triglyceride metabolism (Park, 2021). However, without further mechanistic data, any explanation for our finding remains speculative.

Our null findings regarding total cholesterol and LDL are consistent with some, but not all, previous research. The VITAL trial, a large-scale RCT, found no benefit of vitamin D supplementation on major cardiovascular events or lipid profiles (Manson et al., 2019). This suggests that in a generally healthy, albeit vitamin D-insufficient population, the influence of vitamin D on cholesterol may be minimal or non-existent. Conversely, other meta-analyses have found that supplementation does reduce TC and LDL-C (Dibaba, 2019; Radkhah et al., 2023). The weak, inverse association between vitamin D and HDL observed in our study (significant only with Spearman's test) also contrasts with studies that report a positive effect of supplementation on HDL levels (Lu et al., 2024). These inconsistencies across studies underscore the complexity of the relationship and suggest that it may be modified by factors such as baseline health status, obesity, and genetic predisposition.

Limitations

This study has several important limitations that must be acknowledged. The most significant is the vast amount of missing data for the lipid profile variables, with 80–90% of participants lacking these measurements. This drastically reduces the statistical power and may introduce selection bias, as the individuals with complete data may not be representative of the entire cohort. Second, the cross-sectional design precludes any inference of causality; we can only report associations. Third, the extreme gender imbalance (99.9% female) severely limits the generalizability of our findings to men. Fourth, the study did not control for numerous potential confounders, such as body mass index (BMI), diet, physical activity, medication use (e.g., statins), and menopausal status, all of which can influence both vitamin D levels and lipid profiles. Obesity, in particular, is a known confounder, as it is associated with both lower vitamin D levels (due to volumetric dilution) and dyslipidemia (Park, 2021; Gholamzad et al., 2023).

Conclusion

In this cross-sectional analysis of a predominantly female population with a high prevalence of vitamin D deficiency, we identified a statistically significant positive correlation between serum 25-hydroxyvitamin D and triglyceride levels. This finding is contrary to many published reports and suggests a complex, and perhaps population-specific, relationship. No significant associations were found with total cholesterol or LDL cholesterol. Due to major limitations, including extensive missing data and a lack of demographic diversity, these results should be interpreted with caution. They highlight the need for further well-designed prospective studies and RCTs with complete data collection across diverse populations to unravel the true nature of vitamin D's role in lipid metabolism and its potential implications for cardiovascular health. Future research should focus on identifying effect modifiers such as sex, BMI, and baseline vitamin D status to reconcile the conflicting evidence in the field.

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