Correlations between Vitamin D Concentrations and Lipid Panels in Active Duty and Veteran Military Personnel

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Abstract

Vitamin D deficiency is common in the United States and has been associated with dyslipidemia along with additional cardiovascular conditions. Dyslipidemia raises the risk for cardiovascular disease and has been linked to symptoms of post-traumatic stress disorder (PTSD) in military personnel. The purpose of this study was to identify significant correlations between serum vitamin D and lipid panel concentrations in active duty and veteran military personnel. This analysis examined 3,053 unique cases of serum vitamin D and lipid panel assessments ordered at Womack Army Medical Center, Fort Bragg, North Carolina, from January 2012 to September 2013. Assessments were drawn within 21 days of one another. Fifty-seven percent of subjects had insufficient vitamin D status, and 36.6% had high total cholesterol according to the Army Medical Department guidelines, using 30 ng/ml for 25-hydroxyvitamin D and 200 mg cholesterol, respectively. In regression models, vitamin D was significantly positively correlated with high-density lipoprotein cholesterol in all subjects after controlling for age, gender and military status. Body mass index (BMI) was available for active personnel only, and including BMI values in analysis reduced the significance of vitamin D in the model. Vitamin D concentrations were negatively associated with total cholesterol and low-density lipoprotein in veterans only. Overall, our data suggest that vitamin D deficiency may also increase the risk for PTSD through its relationship with lipids [15,16,25].

Keywords
Cholesterol, High-density lipoprotein, Low-density lipoprotein, 25-hydroxyvitamin D, Dyslipidemia, Service members

Introduction

Vitamin D functions as a hormone in the human body. The primary source of vitamin D is endogenous synthesis in response to solar ultraviolet (UV) radiation, although individuals also rely on dietary sources, especially in winter months and those in northern latitudes [1,2]. Dietary sources include fatty fish (salmon and herring), liver, eggs, and fortified foods, such as milk and other dairy products [3,4].

Worldwide, most humans expose less than 5% of their body to UV light for adequate time to synthesize vitamin D, increasing their risk for deficiencies if dietary requirements are not met [1]. People living at latitudes greater than 40 degrees are not exposed to adequate UV exposure during the winter months to replenish vitamin D concentrations [2]. Additionally, individuals with darker skin tones (non-White), who are older, who live in areas with urban photochemical smog or always wear sunscreen are at greater risk for inadequate vitamin D status. As a result, vitamin D deficiency is widespread across civilian and military populations [5,6]. It has been estimated that between 25% and 57% of the United States (US) population are deficient in vitamin D [1,7,8].

Vitamin D has essential roles in calcium homeostasis, as well as regulation of cellular growth, function and differentiation [4]. Recent studies have identified vitamin D receptors in the liver, immune system, and skeletal and cardiac muscles, suggesting that vitamin D has widespread functions throughout the body [1]. Poor vitamin D status has been associated with diabetes, obesity, hypertension, peripheral vascular disease, coronary artery disease, stroke, insulin resistance, heart failure, and dyslipidemia [1,4,9-13].

Dyslipidemia is a known risk factor for cardiovascular disease, which is a major cause of morbidity and mortality worldwide [8,14]. Dyslipidemia has also been associated with psychological conditions, such as depression [15,16] and post-traumatic stress disorder (PTSD) in military personnel [17-24]. Since vitamin D deficiency has been linked to increased risk for dyslipidemia and depression, then vitamin D deficiency may also increase the risk for PTSD through its relationship with lipids [15,16,25].
Numerous studies have identified significant associations between 25-hydroxycholecalciferol (25(OH)D) concentrations and lipid profiles [13,26-30]. There are several potential mechanisms that connect vitamin D with lipid concentrations. Vitamin D may have a direct effect on serum lipid levels, such as affecting adipogenesis and differentiation, or vitamin D may have an indirect effect on serum lipid levels, such as through its effect on parathyroid hormone and calcium homeostasis [31]. It is also hypothesized that vitamin D may be transported by lipoproteins as vitamin D binding proteins have been discovered on lipoproteins, notably very low density lipoprotein (VLDL) [32]. Additionally, changes in levels of vitamin D binding protein have been shown to affect serum 25(OH)D levels [33]. Thus, it is plausible that circulating levels of lipoprotein have an effect on the levels of free 25(OH)D.

The purpose of this study was to identify significant correlations between serum vitamin D and lipid panel concentrations in active duty and veteran military personnel. Establishing a link between vitamin D status and dyslipidemia in service members is important to the treatment of cardiovascular disease and may play a role in reducing the risk for PTSD.

Materials and Methods

This retrospective study examined 3,053 unique cases of serum vitamin D assessments ordered at Womack Army Medical Center, Fort Bragg, North Carolina, between January 2012 and September 2013 (Figure 1). Inclusion criteria were active duty or veteran military personnel with a lipid panel measured within 21 days of vitamin D assessment. Age at the time of test as well as military status (active duty or veteran) was identified for all subjects, while body mass index (BMI) was available for only 1,350 active-duty subjects. Race and ethnicity identifiers were too limited to include in analysis.

Measurement of vitamin D and lipid panel assessments were conducted through Womack Army Medical Center. Serum 25(OH)D concentrations were determined by liquid chromatography-tandem mass spectrometry (Quest Diagnostics, Chantilly, VA) with a detection limit of 4 ng/ml and 8.3% coefficient of variation. Lipid concentrations were determined by enzymatic, colorimetric method (Roche Diagnostics, Indianapolis, IN) with a detection limit of 3.86 mg/dL and 1.6% coefficient of variation. Since serum 25(OH)D has a half-life of three weeks, subjects were included only if lipid panel assessment was conducted within 21 days of vitamin D assessment. Therefore, no adjustment for season was warranted.

Vitamin D and lipid concentrations were categorized according to the laboratory ranges used by the US Army Medical Department (AMEDD) standards of care [5]. The AMEDD laboratory ranges follow guidelines from the Endocrine Society Clinical Practice Guideline that defines deficient as 25(OH)D less than 20 ng/ml, insufficient as 20-29 ng/ml, and sufficient as 30-100 ng/ml [32]. AMEDD guidelines define high total cholesterol (TC) as > 200 mg/dL, above optimal low-density lipoprotein (LDL-C) as > 130 mg/dL, high triglycerides (TG) as > 150 mg/dL, and low high-density lipoprotein (HDL-C) as < 35 mg/dL [5]. The BMI categories, underweight (< 18.5 kg/m²), normal (18.5-24.99 kg/m²), overweight (25.00-29.99 kg/m²), and obese (≥ 30.00 kg/m²) were defined based on the international classification of adult overweight, overweight and obesity according to BMI guidelines, published by the World Health Organization [34,35]. We recognize the controversy in defining vitamin D deficiency and have used AMEDD guidelines for all laboratory assessments since those were the ranges defined by the Army for this population.

Data were analyzed for descriptive statistics and correlations using SPSS version 20.0 (SPSS, Inc., Chicago, IL). Summary statistics for categorical variables included frequencies, means, and standard deviations. Marginal relationships between age, vitamin D, and HDL-C, LDL-C, TC and TG were assessed using Pearson’s correlations. Student’s t-test and ANOVA were used to compare means. General linear models were used to test the effect of vitamin D on lipid concentrations. In these models, age of soldiers was a covariate, and active duty vs. veteran was a fixed factor. BMI was included as a covariate in step-wise regression. Interactions were tested and removed from the models if they were not statistically significant at a level of p = 0.05. This study was approved by Womack


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Results

We examined medical records for 3,053 subjects who met study criteria: 2,468 (80.8%) male and 585 (19.2%) females. Mean age was 45.8 ± 15.1 years; 1,125 (36.9%) of the subjects were veterans and 1,928 (63.1%) were active duty. The overall mean for 25(OH)D concentrations was 28.78 ± 11.72 ng/ml. Six-hundred twenty-three (20.4%) of subjects were vitamin D deficient; 1127 (36.9%) were vitamin D insufficient; and 1302 (42.7%) were vitamin D sufficient. High TC was found in 1118 (36.6%) of subjects; above optimal LDL-C was 45.8 ± 15.1 years; 1,125 (36.9%) of the subjects were veterans.

Table 1: Service Member age, vitamin D concentrations, and BMI according to 25-hydroxyvitamin D status

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Deficient &lt; 20 mg/ml (n = 623)</th>
<th>Insufficient 20-29 mg/ml (n = 1127)</th>
<th>Sufficient 30-100 mg/ml (n = 1302)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average age</td>
<td>42.57 ± 13.22**</td>
<td>44.81 ± 14.56**</td>
<td>46.14 ± 15.96**</td>
</tr>
<tr>
<td>p-value</td>
<td>&lt; 0.001</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

BMI only available for 1,350 of the active duty service members.

Table 2: Service Member age, vitamin D concentrations, lipid concentrations, and BMI according to military status

<table>
<thead>
<tr>
<th>Active Duty</th>
<th>Veteran</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>37.60 ± 8.93</td>
<td>59.45 ± 13.61</td>
</tr>
<tr>
<td>25(OH)D (ng/ml)</td>
<td>27.79 ± 11.28</td>
<td>30.47 ± 12.55</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.62 ± 4.11</td>
<td>-</td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td>194.33 ± 40.76</td>
<td>180.16 ± 42.09</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>115.94 ± 36.74</td>
<td>102.43 ± 36.79</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>54.18 ± 16.37</td>
<td>51.59 ± 15.75</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>123.17 ± 80.16</td>
<td>128.36 ± 88.10</td>
</tr>
</tbody>
</table>

*BMI only available for 1,350 of the active duty service members.

Table 3: Service Member age, vitamin D concentrations, lipid concentrations, and BMI according to gender

<table>
<thead>
<tr>
<th>Male</th>
<th>Female</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>46.81 ± 15.31</td>
<td>41.41 ± 13.20</td>
</tr>
<tr>
<td>25(OH)D (ng/ml)</td>
<td>28.85 ± 4.10</td>
<td>38.94 ± 8.33*</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.32 ± 4.01</td>
<td>26.14 ± 3.59</td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td>186.87 ± 41.92</td>
<td>107.78 ± 37.13</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>53.63 ± 16.38</td>
<td>51.96 ± 15.82</td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td>123.17 ± 80.16</td>
<td>128.36 ± 88.10</td>
</tr>
</tbody>
</table>

*BMI only available for 1,350 of the active duty service members.

Table 4: Service Member age, vitamin D concentrations, lipid concentrations, and BMI according to BMI category

<table>
<thead>
<tr>
<th>Underweight &lt; 18.5 kg/m²</th>
<th>Obese ≥ 30.0 kg/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D (ng/ml)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td></td>
</tr>
<tr>
<td>TC (mg/dl)</td>
<td></td>
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<tr>
<td>LDL-C (mg/dl)</td>
<td></td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td></td>
</tr>
<tr>
<td>TG (mg/dl)</td>
<td></td>
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</tbody>
</table>

Table 5: General linear models to predict HDL-C levels in veteran and active duty military personnel

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>HDL-C in all subjects</th>
<th>HDL-C in activity duty subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D</td>
<td>β</td>
<td>ρ</td>
</tr>
<tr>
<td>Age</td>
<td>0.075</td>
<td>0.007</td>
</tr>
<tr>
<td>Gender</td>
<td>15.467</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Active/Veteran</td>
<td>3.310</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BMI</td>
<td>-3.31</td>
<td>-0.683</td>
</tr>
</tbody>
</table>

by gender (Figure 2), serum 25(OH)D had a stronger correlation with HDL-C ($r = 0.087; p = 0.038$) in the female population than in the male population ($r = 0.050; p = 0.013$). However, adding BMI to the regression model in active duty subjects reduced the significance of 25(OH)D on HDL-C ($p = 0.06$).

LDL-C and TC concentrations were significantly negatively correlated with 25(OH)D concentrations ($p < 0.001; r = -0.08195$ and $p < 0.0001; r = -0.06004$, respectively). After adjustment for military status, 25(OH)D had a significant negative linear effect on LDL-C and TC concentrations in veterans only ($p < 0.001$ and $p = 0.0002$ respectively). TG levels were not significantly correlated with 25(OH) D concentrations.

**Discussion**

In this study, we found that the majority of service members tested for vitamin D had deficient/insufficient status. Furthermore, service members with low vitamin D had higher TC, higher LDL-C, and lower HDL-C compared to those with sufficient vitamin D. When adjusted for age, gender, and status, 25(OH)D had a significant negative linear effect on LDL-C and TC concentrations in veterans only ($p < 0.001$ and $p = 0.0002$ respectively). TG levels were not significantly correlated with 25(OH) D concentrations.

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In our study, 36.6% subjects had high TC; 26.5% subjects had above optimal LDL-C; 22% subjects had low HDL-C; and 25.8% subjects had high TG. Current prevalence of dyslipidemia in military personnel in the US is lacking. In the general US adult population, it is estimated that 38.4% have high TC, 32.2% have high LDL-C, 25.1% have high TG, and 17% have low HDL-C based on statistics gathered from a variety of national surveys [39]. Prevalence of abnormal lipids in our sample is consistent with this analysis, although we found a larger prevalence of low HDL-C in our study sample. One military study analyzed autopsy results from US service members ($n = 3,832$) who died in support of Operations Enduring Freedom or Operation Iraqi Freedom/New Dawn (excluding cause of death labeled “suicide,” “natural,” or “undetermined”), to include analysis of atherosclerosis and dyslipidemia [40]. Coronary or aortic atherosclerosis was present in 12.1% of the service members, while less than 1% ($n = 28$) service members had dyslipidemia. Of those with dyslipidemia, 50% had atherosclerosis. Lastly, 166 (4.3%) of the service members were obese, according to their BMI. These findings were not consistent with our results, as we found higher prevalence of obesity in active duty members and higher rates of dyslipidemia in both active duty and veteran service members.

When adjusted for military status, vitamin D had a significant effect on both TC and LDL-C in veterans only. In our study population, the veteran group was significantly older, had significantly lower TC and LDL-C, and had significantly higher 25(OH)D concentrations than the active duty group. TC levels typically decrease with age by mechanisms not entirely understood [41]. It is hypothesized that this decrease results from the prevalence of chronic illness in the older population rather than the physiological changes associated with age. Like TC levels, vitamin D also has been shown to decrease with age [34]. Somewhat unexpectedly, our results showed that age was significantly greater in subjects with sufficient vitamin D status. Since veterans are free from active duty responsibilities and uniform requirements, they may have had more sunlight exposures, but since we did not have data on outdoor activity, this hypothesis cannot be confirmed.

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**Figure 2:** Scatter plot illustrating the correlation between 25(OH)D and HDL-C separated by gender

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Overall, 20.4% of military personnel in our sample were vitamin D deficient, and 57.3% were deficient/insufficient, despite the southern latitude of 35.1°N. Data on the prevalence of vitamin D deficiency in the US military are limited, although vitamin D deficiency is common among the US population [36]. One study reported that 31.1-33.5% of serum samples from active duty service members had 25(OH)D less than 20ng/ml, when adjusted for season of sample analysis [37]. Another study found that 57% of female military recruits entering basic training had serum 25(OH)D concentrations less than 30ng/ml, and their 25(OH)D concentrations decreased after eight weeks of outdoor training in the Southeastern US [38]. Our results were consistent with both of these American military studies.
In our sample, the strongest relationship was a positive correlation between vitamin D and HDL-C concentrations. Our findings of a significant positive association between serum 25(OH)D and HDL-C concentrations is consistent with other military and civilian publications [27,29,42]. In US civilian men and women, as 25(OH)D was a significant predictor for HDL-C after adjusting for age, sex, waist circumference, physical activity score, alcohol consumption, smoking, and vitamin D supplementation [27]. Furthermore, no significant associations were found between 25(OH)D and LDL-C or total cholesterol. In civilian Norwegian subjects 25 years and older, a significant positive association between 25(OH)D and HDL-C was identified and maintained over 13 years in this longitudinal analysis [42]. In Belgian military personnel, there was a significant positive correlation between serum 25(OH)D and HDL-C in women, but not in men [29]. Our data showed a positive correlation between 25(OH)D and HDL-C in both men and women. One cause of this discrepancy could be the smaller sample size in the Belgian study (n = 358) compared to our much larger sample size (n = 3,053).

Furthermore, one intervention study found that supplementing vitamin D in deficient Indian children significantly improved HDL-C concentrations [43]. These authors hypothesize that vitamin D regulates cholesterol-carrying macrophages involved in reverse cholesterol transport, thus supporting a role in HDL-C synthesis. Alternatively, some researchers suggest that vitamin D may be a marker of chronic nonspecific illness rather than a direct contributor to any specific disease, noting that unhealthy people have vitamin D deficiency due to decreased sunlight exposure [13]. However, unhealthy subjects have not supported the relationship between 25(OH)D and HDL-C, as type 2 diabetic civilian subjects from Iran did not have a significant positive association between 25(OH)D and HDL-C [44].

In our study, adding BMI to the active duty model reduced the significance of vitamin D on HDL-C in activity duty service members. Therefore, BMI likely explained much of the connection between 25(OH)D and HDL-C, since BMI was significantly negatively correlated with both 25(OH)D (r = 0.117; p < 0.001) and HDL-C (r = 0.683; p < 0.001) concentrations. Since vitamin D is fat soluble and is easily taken up into adipose tissue, individuals with more body fat have a greater storage capacity for vitamin D that could result in lower concentrations of circulating 25(OH)D [27,45]. Low vitamin D in obese individuals may also stem from these individuals having less motility or ability to participate in outdoor activities and thus have inadequate sun exposure [30]. Additionally, higher body fat has been associated with lower HDL-C concentrations [46]. However, other research has found that 25(OH)D continued to be a significant predictor for HDL-C when physical characteristics were included in the analysis [27].

The relationship between poor vitamin D status and dyslipidemia has implications beyond cardiovascular disease, as dyslipidemia has been associated with psychological conditions, including depression [26,27] and PTSD in military personnel [28,31,34-37]. Studies have shown that soldiers with combat-related PTSD have significantly higher TC, LDL-C, and TG and lower HDL-C levels compared to soldiers without PTSD [17,19,23,24]. Dyslipidemia was found in younger active duty military personnel (mean age 31) as well as older veterans [17,18,23]. It is hypothesized that dyslipidemia may increase activity of the noradrenergic system and thereby aggravate symptoms of PTSD [17], which is supported by a strong positive correlation between noradrenaline and lipid levels [23]. Additionally, civilian research has shown that vitamin D deficiency increases risk for cognitive deficits and depression, although no link has been established between vitamin D status and symptoms of PTSD [47]. Since evidence suggests that vitamin D deficiency increases the risk for dyslipidemia and depression, then vitamin D deficiency may also increase the risk for PTSD although this hypothesis requires further research [15,16,25].

Other factors such as diet and exercise may impact this observed relationship. Exercise has been shown to improve both serum vitamin D and lipid profile concentrations [48]. Plant-based diets, especially, vegan diets, have also been shown to improve serum lipid profile concentrations [49,50]. However, vitamin D is found in only small concentrations in plant-based foods, and vegans have been shown to have low serum vitamin D levels despite improved serum lipid profile concentrations [49,50]. While we did not have data on our subjects diet and exercise habit, a recent study on the US military found that 31.2% of soldiers fail to meet the recommended amount of moderate aerobic exercise per week (at least 150 minutes per week); 43% fail to meet the recommended amount of vigorous aerobic training (at least 75 minutes per week); and 53% fail to meet the recommended amount of strength training per week (at least three days per week) [51]. Furthermore, only 10.8% of soldiers eat at least three servings of fruit per week, and 12.9% eat at least three servings of vegetables per day [51]. These habits likely play a role in the serum vitamin D levels and lipid panel concentrations of the US military population.

This study has several limitations. First it is a retrospective analysis, and we had no access to data on sun exposure, vitamin D consumption, medical history or medication data (including hyperlipidemic treatments and vitamin D supplements) for subjects. Therefore, we were able to identify correlations but not causal relationships. BMI data were available for active duty subjects only, and BMI may not accurately represent true body composition in active military personnel. Finally, since the study only included military personnel with medically ordered vitamin D and lipid assessments, the sample may not be representative of the entire military population. The study was strengthened by including only assays completed within 3 weeks of one another to control for season of vitamin D analysis, as well as only including one location to control for latitude variances.

Future research should prospectively examine the prevalence of vitamin D deficiency and dyslipidemia in active duty and veteran military personnel. Furthermore, longitudinal analyses should establish clinically significant links between vitamin D status and lipid profiles and if these factor influence PTSD symptoms. Further research should also clarify mechanisms that connect altered lipid profiles and body fat with vitamin D deficiency. Lastly, intervention studies to treat subjects with vitamin D deficiency and dyslipidemia would better explain these hormonal and lipid metabolism relationships.

In conclusion, serum 25(OH)D was significantly positively associated with HDL-C in all subjects after controlling for age, gender and military status. However, including BMI values in active duty personnel reduced the significance of 25(OH)D. Serum 25(OH)D concentrations were negatively associated with LDL-C and TC in veterans only. Overall, our data suggest that lipoprotein concentrations are complex and may be influenced by vitamin D status in military service members. Therefore, future research should aim to explain the correlation between vitamin D and cholesterol concentrations in service members to determine if improving vitamin D status would also improve cholesterol status.

Acknowledgments

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References


