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Association of serum total 25(OH)D with cardiometabolic factors among adult women

Asosiasi Serum Total 25(OH)D dengan Faktor Kardiometabolik pada Perempuan Dewasa

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ABSTRACT

Background: *Vitamin D deficiency remains common among adult women in tropical settings, but its association with cardiometabolic factors is not fully characterized.*
Objective: *To examine the association between serum total 25-hydroxyvitamin D [25(OH)D] and cardiometabolic factors among adult women.*
Methods: *This secondary cross-sectional analysis used baseline screening data collected in Bogor City, Indonesia, from 29 August to 20 September 2025 before a vitamin D3 clinical trial. Using purposive sampling, 85 nonpregnant, nonlactating, premenopausal women aged 19 to 49 years with complete data were included. Serum total 25(OH)D was measured by VIDAS 25 OH Vitamin D Total using enzyme-linked fluorescent assay. Fasting capillary lipid profiles included total cholesterol, triglycerides, HDL-C, and LDL-C. Spearman correlation was the primary bivariate analysis. Multiple linear regression with HC3 robust standard errors modeled natural log-transformed serum total 25(OH)D, with total cholesterol as the exposure and age and waist-to-hip ratio as covariates.*
Results: *Serum total 25(OH)D was <20 ng/mL in 92.9% of participants and <10 ng/mL in 60.0%; the median was 8.95 ng/mL. Total cholesterol was inversely associated with serum total 25(OH)D ($\rho = -0.293$, $p = 0.006$) and remained associated in the adjusted model ($B = -0.0027$, robust $SE = 0.0009$, $p = 0.005$).*
Conclusion: *A large proportion of this purposively sampled group had low vitamin D status. Total cholesterol showed a weak inverse association with serum total 25(OH)D, but the cross-sectional design does not establish causality.*

Keywords: *25(OH)D, adult women, cardiometabolic factors, total cholesterol, vitamin D*

ABSTRAK

Latar belakang: Defisiensi vitamin D masih banyak ditemukan pada perempuan dewasa di wilayah tropis, tetapi asosiasinya dengan faktor kardiometabolik belum sepenuhnya dipahami.
Tujuan: Menganalisis asosiasi serum total 25-hidroksivitamin D [25(OH)D] dengan faktor kardiometabolik pada perempuan dewasa.
Metode: Analisis sekunder potong lintang ini menggunakan data skrining awal yang dikumpulkan di Kota Bogor, Indonesia, pada 29 Agustus sampai 20 September 2025 sebelum uji klinis vitamin D3. Sebanyak 85 perempuan tidak hamil, tidak menyusui, dan pramenopause berusia 19 sampai 49 tahun dengan data lengkap dipilih secara purposif. Serum total 25(OH)D diukur menggunakan VIDAS 25 OH Vitamin D Total dengan

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metode enzyme-linked fluorescent assay. Profil lipid darah kapiler puasa mencakup kolesterol total, trigliserida, HDL-C, dan LDL-C. Korelasi Spearman digunakan sebagai analisis bivariat utama. Regresi linier berganda dengan galat baku robust HC3 memodelkan log natural serum total 25(OH)D, dengan kolesterol total sebagai paparan serta usia dan rasio lingkar pinggang-pinggul sebagai kovariat.
Hasil: Serum total 25(OH)D <20 ng/mL ditemukan pada 92,9% peserta dan <10 ng/mL pada 60,0%; median sebesar 8,95 ng/mL. Kolesterol total berasosiasi terbalik dengan serum total 25(OH)D (ρ = −0,293; p = 0,006) dan tetap berasosiasi pada model tersesuaikan (B = −0,0027; galat baku robust = 0,0009; p = 0,005). Kesimpulan: Sebagian besar kelompok yang dipilih secara purposif ini memiliki status vitamin D rendah. Kolesterol total menunjukkan asosiasi terbalik yang lemah dengan serum total 25(OH)D, tetapi desain potong lintang tidak membuktikan hubungan kausal.

Kata kunci: 25(OH)D, faktor kardiometabolik, kolesterol total, perempuan dewasa, vitamin D

INTRODUCTION

Vitamin D is a secosteroid hormone that plays an essential role in calcium and phosphate homeostasis. Beyond skeletal health, vitamin D is increasingly recognized for its involvement in immune regulation, inflammation, lipid metabolism, gene expression, and cardiometabolic health.[1,2] Serum total 25-hydroxyvitamin D [25(OH)D] is widely used as the main circulating biomarker of vitamin D status because it reflects vitamin D obtained from sunlight exposure, dietary intake, and supplementation.[3,4,5]

Vitamin D deficiency remains a global public health concern, including in populations living in regions with abundant sunlight. A pooled analysis of population-based studies from 2000 to 2022 showed that low vitamin D status remains widespread across geographical regions and population groups.[4] Current guidance also emphasizes that optimal serum 25(OH)D thresholds vary by clinical context and remain debated for disease prevention and routine testing.[5,6] These observations illustrate the tropical paradox: year-round sunlight availability does not necessarily translate into adequate circulating 25(OH)D.

Adult women are an important population for vitamin D assessment because vitamin D status may be influenced by reproductive age characteristics, indoor activities, clothing practices, sunscreen use, dietary intake, adiposity, and metabolic risk factors. Indonesian city-specific studies indicate that low vitamin D status occurs in several urban settings. In Jakarta, nonpregnant women aged 18 to 40 years had a mean plasma 25(OH)D concentration of 48 nmol/L, approximately 19.2 ng/mL, and more than 60% had concentrations below 50 nmol/L.[7] In Padang, female university students showed low serum 25(OH)D concentrations in both obesity and normal nutritional status groups.[8] These data are important comparators, but they cannot be assumed to represent women in Bogor because behavioral exposure, diet, adiposity, and sampling characteristics may differ across cities.

Low serum 25(OH)D has been associated with unfavorable cardiometabolic profiles, including dyslipidemia, obesity-related markers, and central adiposity. A systematic review and dose-response meta-analysis reported that higher serum 25(OH)D levels were associated with lower odds of dyslipidemia-related outcomes, including hypertriglyceridemia and low HDL cholesterol.[9] Studies among adult women and adults in rural and urban settings have also reported associations between vitamin D status, lipid profile, adiposity, and cardiometabolic risk markers.[10,11,12] However, these associations are not consistent across populations and may be influenced by age, adiposity, physical activity, diet, sun exposure, and other contextual factors.

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Evidence that specifically integrates serum total 25(OH)D with cardiometabolic factors among adult women in Bogor remains limited. The relevance of Bogor lies not in location alone, but in the need for locally grounded evidence from an urban tropical population in which effective ultraviolet exposure, dietary intake, body composition, and daily behavior may differ from those reported in Jakarta, Padang, or other regional settings. This study therefore evaluated serum total 25(OH)D together with lipid profile, adiposity indicators, dietary intake, physical activity, and sunlight exposure within the same screening population. The local context can help refine hypotheses and guide the design of larger longitudinal or intervention studies without assuming that findings from other cities are directly transferable.[4,7,8,13]

Therefore, this study aimed to examine the association between serum total 25(OH)D and cardiometabolic factors among adult women using secondary cross-sectional baseline screening data collected before initiation of a vitamin D3 clinical trial. The analysis was intended to provide association evidence and was not designed to infer causality.

METHODS

Study design

This study was a secondary analysis of baseline screening data with a cross-sectional design. The design was appropriate for describing serum total 25(OH)D and examining its associations with cardiometabolic factors at a single time point, but it did not permit causal inference. Data were collected in Bogor City, Indonesia, from 29 August to 20 September 2025 before initiation of a vitamin D3 clinical trial.

Data source and sampling procedure

The source population comprised women aged 19 to 49 years residing in Bogor City who received recruitment information, were willing to undergo screening, and were able to attend study measurements. Recruitment was conducted through study dissemination and digital posters. Purposive sampling was used to identify women who met the eligibility criteria. Eligible participants were nonpregnant, nonlactating, premenopausal women who provided written informed consent. Exclusion criteria included current smoking, alcohol consumption, pregnancy, lactation, menopause, chronic diseases or medications known to influence vitamin D metabolism, and tanning bed use within one week before enrollment. [14,15] All 85 women who met the Stage 1 eligibility criteria and had verifiable complete baseline data for serum total 25(OH)D, anthropometrics, lipid profile, dietary intake, physical activity, and sunlight exposure during the recruitment period were included. Thus, the analytic sample of 85 participants represented the complete set of eligible women with verifiable baseline data available from Stage 1 screening during the predefined recruitment period, rather than a separately selected sample size target. No separate prevalence based sample size calculation was used for this secondary screening analysis because it was not designed to estimate population prevalence. To evaluate the analytical capacity of the available sample, a sensitivity assessment indicated that 85 participants provided approximately 80% statistical power at a two sided α of 0.05 to detect a correlation of about $|r| = 0.30$, which is conventionally considered a moderate effect size.[16]

This magnitude was close to the observed correlation between total cholesterol and serum total 25(OH)D. The multivariable model was also intentionally kept parsimonious, including only total cholesterol, age, and waist to hip ratio, to limit model complexity and reduce the risk of overfitting relative to the available sample size.[17] Accordingly, the available sample was considered suitable for evaluating associations of approximately this magnitude and for estimating the prespecified parsimonious model, although smaller

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associations may have remained undetected. Accordingly, percentages of vitamin D categories are reported as sample proportions and should not be extrapolated to all adult women in Bogor.

Variables of the study

The dependent variable was serum total 25(OH)D concentration (ng/mL). Serum total 25(OH)D was specified as the dependent variable because the primary analytical question was whether variation in vitamin D status was associated with measured cardiometabolic characteristics within the baseline screening population. Total cholesterol was designated as the principal cardiometabolic exposure in the multivariable model because it is a clinically interpretable lipid marker and previous epidemiological evidence has reported associations between circulating 25(OH)D and lipid related outcomes.[9,11,12] This statistical orientation was chosen because the analysis examined variation in vitamin D status in relation to measured cardiometabolic factors; however, designation of serum total 25(OH)D as the dependent variable and total cholesterol as the principal exposure reflects the analytical structure of the model rather than an assumed biological or causal direction. Because both variables were measured at the same time, the cross-sectional design cannot establish whether differences in total cholesterol precede changes in serum total 25(OH)D or vice versa.

For descriptive purposes, vitamin D deficiency and severe deficiency were operationally defined as <20 ng/mL and <10 ng/mL, respectively, while recognizing that outcome-specific thresholds remain debated across professional guidance.[3,5,6] The cardiometabolic variables evaluated were total cholesterol, triglycerides, high density lipoprotein cholesterol (HDL C), and low density lipoprotein cholesterol (LDL C). Triglycerides, HDL C, and LDL C were retained as secondary lipid variables in the bivariate analyses, whereas total cholesterol was carried forward as the principal lipid exposure in the parsimonious multivariable model. Other variables included age, body mass index (BMI), waist circumference, waist-to-hip ratio, nutrient intake, physical activity, sunlight exposure, sunscreen use, clothing style, and skin type.

For descriptive stratification, BMI was grouped as <23 kg/m² and ≥23 kg/m² using the 23 kg/m² Asia focused public health action point identified by the WHO Expert Consultation because cardiometabolic risk may increase at lower BMI values in Asian populations.[18] This grouping was used for risk stratification and was not intended to redefine universal BMI diagnostic categories.

Measurement and instruments

Data on participant characteristics, medical history, medications, allergies, physiological status, lifestyle habits, sunlight exposure, sunscreen use, clothing style, and Fitzpatrick skin type were collected using a structured questionnaire. Skin phototype was classified according to the conventional Fitzpatrick sun reactive skin type system, which categorizes skin responses to ultraviolet exposure into types I to VI.[19] Dietary intake was assessed using two 24 hour dietary recalls, one on a weekday and one on a weekend day. Portion size estimation was supported by household measures and the *Buku Foto Makanan* issued by the Indonesian Ministry of Health, and nutrient intake was processed using NutriSurvey software with the Indonesian Food Composition Table. The *Buku Foto Makanan*[20] and *Tabel Komposisi Pangan Indonesia*[21] were used as national reference resources for portion size estimation and nutrient composition coding.

Physical activity was assessed using a structured activity-level questionnaire. Physical activity was analyzed using the resulting activity level score and was not categorized using a clinical diagnostic threshold Body weight and height were measured to the nearest 0.1 kg and 0.1 cm using a digital scale and stadiometer, while waist and

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hip circumferences were measured using a flexible, nonelastic tape; BMI was calculated as kg/m². Waist and hip circumference measurements were referenced to standardized anthropometric procedures described by the World Health Organization, which provides guidance on measurement of waist circumference and waist-to-hip ratio.[22] BMI was interpreted using the Asia-focused public health action points of the WHO Expert Consultation, with <23 kg/m² and ≥23 kg/m² used for descriptive risk stratification in this study.[23] WHO guidance specifically addresses standardized measurement and interpretation of waist circumference and waist-to-hip ratio.

Fasting capillary blood for lipid profiling was obtained by fingertip puncture. A 40 µL sample was analyzed using the Sinocare PalmLab SLX-120 point-of-care lipid analyzer with LL-12 lipid test strips according to the manufacturer procedure.[24] Total cholesterol, triglycerides, and HDL-C were measured by the device, and LDL-C was estimated internally by the analyzer. Technical checks included instrument calibration code, strip lot and expiry verification, and adequacy of specimen volume. The four lipid variables were analyzed primarily as continuous measurements rather than classified into diagnostic dyslipidemia categories. Therefore, clinical lipid cut off values were not used to define study groups or determine eligibility. This approach avoided assigning clinical diagnoses from a point of care screening measurement and was consistent with the study objective of evaluating continuous associations with serum total 25(OH)D.

Venous blood was collected for serum total 25(OH)D, which was quantified using the VIDAS 25 OH Vitamin D Total enzyme-linked fluorescent assay on a Mini VIDAS analyzer.[25] Serum total 25(OH)D was used as the circulating biomarker of vitamin D status. For descriptive reporting, concentrations <20 ng/mL and <10 ng/mL were operationally classified as vitamin D deficiency and severe deficiency, respectively, based on established vitamin D literature, while acknowledging that current professional guidance does not support a single universal 25(OH)D threshold for all health outcomes.[3,5,6] Current Endocrine Society guidance explicitly notes that outcome-specific 25(OH)D thresholds have not been established for generally healthy adults, therefore, this cautious wording is more appropriate than presenting these cutoffs as universal clinical standards.

Data collection

Data collection began with study dissemination and recruitment. Prospective participants received an explanation of the study objectives, procedures, benefits, risks, confidentiality, the right to refuse, and the right to withdraw. Written informed consent was obtained before interviews, anthropometric measurements, or blood collection. Trained enumerators collected questionnaire, dietary, physical activity, and sunlight exposure data and performed anthropometric measurements using standardized procedures. Dietary intake was collected using two 24 hour dietary recalls conducted on one weekday and one weekend day. Portion size estimation was assisted by household measures and the *Buku Foto Makanan,*[20] while nutrient intake was subsequently processed using NutriSurvey and the *Tabel Komposisi Pangan Indonesia,*[21] consistent with the procedures described in the Measurement and Instruments section.

Anthropometric measurements were performed using the same standardized procedures specified in the Measurement and Instruments section. Body weight and height were measured using a digital scale and stadiometer and recorded to the nearest 0.1 kg and 0.1 cm, respectively. Waist and hip circumferences were measured using a flexible, nonelastic tape following standardized anthropometric measurement procedures, and the measurements were subsequently used to calculate BMI and waist -to-hip ratio.[22]

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Capillary lipid testing was conducted by trained personnel according to the device protocol. Fasting capillary blood was obtained by fingertip puncture, and a 40 µL specimen was applied to the LL 12 lipid test strip and analyzed using the Sinocare PalmLab SLX 120 point of care lipid analyzer according to the manufacturer protocol.[24] Before analysis, technical checks included verification of the instrument calibration code, test strip lot and expiry date, and adequacy of specimen volume. Total cholesterol, triglycerides, and HDL C were measured by the device, whereas LDL C was estimated internally by the analyzer. Venous blood sampling for serum total 25(OH)D was performed by certified healthcare professionals at Ratnalisa Clinical Laboratory, Bogor City, in accordance with laboratory standard operating procedures. Serum total 25(OH)D was quantified using the VIDAS 25 OH Vitamin D Total enzyme linked fluorescent assay on a Mini VIDAS analyzer, consistent with the analytical method specified in the Measurement and Instruments section.[25] Samples requiring transport were handled using a cold chain to preserve analytical quality. These procedures were applied consistently across participants to minimize procedural variation during data collection and laboratory assessment.

Ethical considerations

The study protocol was approved by the Health Research Ethics Committee of the Faculty of Medicine and Health, Universitas Muhammadiyah Jakarta (No. 105/PE/KE/FKK UMJ/VIII/2025). Before data collection, all participants received a detailed briefing and provided written informed consent. To ensure confidentiality, participant identities were anonymized using unique research codes within the dataset.

Data analysis

Data analysis was conducted in three stages. First, univariate analysis was used to describe study variables. Categorical variables were presented as frequencies and percentages. Numerical variables were assessed using histograms, Q-Q plots, and the Shapiro-Wilk test. Variables with an approximately normal distribution were presented as mean ± standard deviation, whereas skewed variables were presented as median and interquartile range.

Second, bivariate analysis examined associations between continuous variables and serum total 25(OH)D. Spearman's rank correlation was the primary analysis because serum total 25(OH)D was skewed and Spearman correlation does not require normally distributed variables. All conventional lipid variables, namely total cholesterol, triglycerides, HDL-C, and LDL-C, were examined. Pearson correlation was used as a sensitivity analysis to assess whether the direction of the main linear associations was comparable under a parametric assumption and was not treated as a second primary hypothesis test. Differences in serum total 25(OH)D between the BMI <23 kg/m² and BMI ≥23 kg/m² groups were assessed using the Kruskal-Wallis test.

Third, ordinary least squares multiple linear regression was performed with natural log-transformed serum total 25(OH)D as the dependent variable. Serum total 25(OH)D was positioned as the dependent variable because it represents the principal circulating biomarker of vitamin D status and the analytical objective was to examine variation in vitamin D status in relation to measured cardiometabolic characteristics. Total cholesterol was treated as the principal lipid exposure in the multivariable model because it is a clinically interpretable cardiometabolic marker, represents an integrated measure of circulating cholesterol, and previous epidemiological evidence has reported associations between circulating 25(OH)D and lipid-related outcomes.[9,11,12] Total cholesterol was the lipid exposure carried forward into the final multivariable model

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because it showed the clearest and most consistent bivariate association with serum total 25(OH)D among the four lipid variables examined.

The designation of serum total 25(OH)D as the dependent variable and total cholesterol as the exposure reflects the analytical orientation of the regression model and should not be interpreted as evidence that total cholesterol causally determines vitamin D status. Because exposure and outcome were measured at the same baseline time point, the cross-sectional design cannot establish temporality, causal direction, or reverse causation. Triglycerides, HDL-C, and LDL-C remained part of the bivariate analysis and were not interpreted as absent from the study. Age and waist-to-hip ratio were retained as covariates on biological grounds because both may be related to vitamin D status and lipid metabolism. Age was retained because age-related differences in lifestyle, sunlight exposure, body composition, and metabolic characteristics may influence both serum 25(OH)D and lipid concentrations, making age a plausible confounding variable. Waist-to-hip ratio was retained because it captures central fat distribution, which is biologically relevant to both vitamin D status and cardiometabolic metabolism and may provide information on adiposity that is not fully represented by body weight alone.[11,12,26,27]

BMI was not entered simultaneously with waist-to-hip ratio because they represent overlapping dimensions of adiposity. Including both measures in the same parsimonious model could introduce redundant information and unnecessary collinearity; waist-to-hip ratio was therefore retained to represent central adiposity. LDL-C was not added alongside total cholesterol because of conceptual and statistical overlap. Because LDL-C contributes substantially to total cholesterol and was estimated internally by the point-of-care analyzer rather than measured independently, simultaneous inclusion of both variables could further increase redundancy without adding sufficient independent information**.** LDL-C was not added alongside total cholesterol because of conceptual and statistical overlap. Dietary variables were not entered simultaneously because two 24-hour recalls provide limited representation of usual intake, and adding many predictors to a sample of 85 could increase overfitting and unstable estimates. Physical activity, sunlight exposure, sunscreen use, clothing style, and other behavioral variables were also not entered simultaneously because the study was not powered for a high-dimensional adjustment model, several of these measures were questionnaire based, and excessive adjustment relative to the available sample could reduce precision and produce unstable coefficients.

Their exclusion from the final model does not imply that they are biologically unimportant. Variable selection therefore considered biological relevance, confounding potential, redundancy, measurement limitations, and model parsimony rather than bivariate p-values alone. The final model was intentionally restricted to total cholesterol as the principal exposure and age and waist-to-hip ratio as covariates to balance control of key biologically plausible confounding with model stability and interpretability. This parsimonious approach was used to reduce the risk of overfitting in the available sample.[17] HC3 heteroskedasticity-consistent robust standard errors were applied.[28,29] Statistical significance was set at $p < 0.05$. Analyses were performed using IBM SPSS Statistics version 27.

RESULTS

The analysis included 85 adult women with a mean age of 26.58 ± 8.62 years and a mean BMI of 24.31 ± 5.79 kg/m². Using the Asia-focused 23 kg/m² action point for

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descriptive risk stratification, 39 participants had a BMI <23 kg/m² and 46 had a BMI ≥23 kg/m². Baseline characteristics are presented in Table 1.

Table 1. Baseline Characteristics of Participants (n = 85)

Characteristic	Total	BMI < 23 kg/m² (n = 39)	BMI ≥ 23 kg/m² (n = 46)
Age, years	26.58 ± 8.62	21.46 ± 1.74	30.91 ± 9.71
Body mass index, kg/m²	24.31 ± 5.79	19.25 ± 1.89	28.60 ± 4.31
Waist circumference, cm	79.45 ± 11.94	69.38 ± 4.77	87.99 ± 9.19
Waist-to-hip ratio	0.84 ± 0.06	0.83 ± 0.05	0.85 ± 0.06
Total cholesterol, mg/dL	144.69 ± 38.69	136.44 ± 34.98	151.70 ± 40.65
Triglycerides, mg/dL	124.00 (103.00–136.00)	105.25 (94.00–137.00)	172.00 (136.00–187.50)
HDL-C, mg/dL	46.67 ± 17.30	48.08 ± 9.83	45.48 ± 21.77
LDL-C, mg/dL	73.72 ± 32.98	63.23 ± 30.14	82.61 ± 32.96
Serum 25(OH)D, ng/mL	8.95 (8.09–11.60)	8.62 (8.09–13.07)	8.09 (8.09–12.38)
Vitamin D intake, µg/day	0.90 (0.30–1.46)	0.30 (0.09–6.18)	1.40 (0.47–3.45)
Calcium intake, mg/day	192.90 (131.42–279.39)	193.25 (117.25–313.80)	191.10 (123.70–387.73)
Physical activity score	1.69 ± 0.52	1.65 ± 0.50	1.73 ± 0.54

* Data are presented as mean ± SD or median (IQR), as appropriate. BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; 25(OH)D, 25-hydroxyvitamin D; SD, standard deviation; IQR, interquartile range.

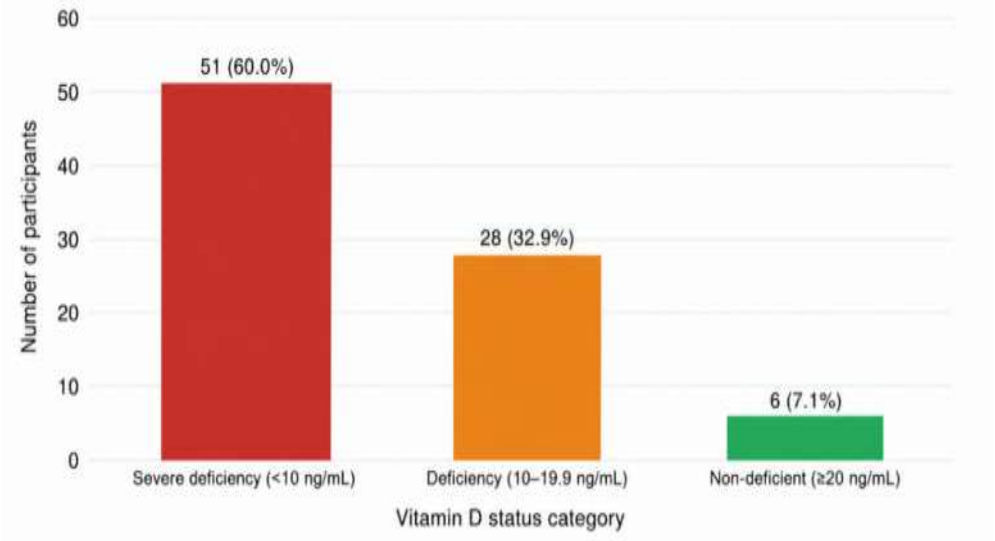

Figure 1. Distribution of serum total 25(OH)D categories in the study sample

Serum total 25(OH)D was low in most participants, with a median concentration of 8.95 ng/mL. A concentration <20 ng/mL was observed in 79 participants (92.9%), including 51 participants (60.0%) with concentrations <10 ng/mL. These percentages describe the purposively recruited study sample and are not estimates of population prevalence in Bogor. Serum total 25(OH)D did not differ significantly between the two BMI groups (Kruskal-Wallis H = 0.110, p = 0.740). Total cholesterol showed a weak inverse association with serum total 25(OH)D (Spearman's ρ = −0.293, p = 0.006), and Pearson sensitivity analysis showed the same direction (r = −0.219, p = 0.044). Triglycerides (ρ = −0.121, p = 0.270), HDL-C (ρ = 0.085, p = 0.438), and LDL-C (ρ = −0.192, p = 0.079) were not significantly associated with serum total 25(OH)D. Several

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nutrient-intake variables showed associations in rank-based analysis, but these findings were treated as exploratory because two 24-hour recalls are sensitive to day-to-day variation, portion-size estimation, and recall bias.

Given the large proportion of participants with low vitamin D status, subsequent analyses evaluated associations between serum total 25(OH)D and the measured cardiometabolic variables and nutrient intakes. Primary Spearman correlation results are presented in Table 2.

Table 2. Spearman Correlations of Selected Variables with Serum Total 25(OH)D

Variable	Spearman's ρ	p-value
Total cholesterol	−0.293	0.006
Triglycerides	−0.121	0.270
HDL-C	0.085	0.438
LDL-C	−0.192	0.079
Vitamin A intake	−0.240	0.027
Calcium intake	−0.237	0.029
Vitamin C intake	−0.224	0.040
Magnesium intake	−0.210	0.054
Waist-to-hip ratio	0.153	0.163
BMI	0.014	0.899

* Spearman's rank correlation was the primary bivariate analysis. All conventional lipid variables measured in the study are shown. Pearson correlation was used as a sensitivity analysis; for total cholesterol, the direction was unchanged (r = −0.219, p = 0.044). BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; 25(OH)D, total 25-hydroxyvitamin D.

Multiple linear regression using the natural logarithm of serum total 25(OH)D yielded a statistically significant model. The model included age, total cholesterol, and waist-to-hip ratio and demonstrated an R^2 of 0.139, adjusted R^2 of 0.107, and model p = 0.007. Higher total cholesterol was associated with lower log-transformed serum total 25(OH)D (B = −0.0027, robust SE = 0.0009, p = 0.005). Waist-to-hip ratio showed an unexpected positive coefficient (B = 1.494, robust SE = 0.656, p = 0.025). Because the data were cross-sectional, these regression coefficients describe adjusted associations and do not establish causal direction. Results are presented in Table 3.

Table 3. Multivariable Linear Regression of Log-Transformed Serum 25(OH)D

Variable	B	Robust SE	p-value	95% CI
Constant	1.348	0.563	0.019	0.228 to 2.468
Age	0.0045	0.0043	0.292	-0.0039 to 0.0130
Total cholesterol	-0.0027	0.0009	0.005	-0.0046 to -0.0008
Waist-to-hip ratio	1.494	0.656	0.025	0.189 to 2.799

* Dependent variable = natural log of serum 25(OH)D. Robust standard errors were used to reduce heteroskedasticity bias. Model fit: R^2 = 0.139, adjusted R^2 = 0.107, p = 0.007. SE, standard error; CI, confidence interval; 25(OH)D, 25-hydroxyvitamin D.

DISCUSSION

Vitamin D Status in the Study Sample

A large proportion of the screened women had low vitamin D status: 92.9% had serum total 25(OH)D <20 ng/mL and 60.0% had concentrations <10 ng/mL. Because participants were selected purposively, these proportions describe the study sample and should not be interpreted as population prevalence for adult women in Bogor. The median concentration of 8.95 ng/mL was lower than the mean plasma 25(OH)D of 48 nmol/L, approximately 19.2 ng/mL, reported among nonpregnant women in Jakarta,

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where more than 60% had concentrations below 50 nmol/L.[7] Research among female university students in Padang also reported low serum 25(OH)D concentrations in both obesity and normal nutritional status groups.[8] These comparisons indicate that low vitamin D status occurs across Indonesian urban settings, while also showing that magnitude differs by population, assay, sampling strategy, age distribution, and metabolic characteristics.

The low vitamin D status observed in this sample is biologically and epidemiologically plausible. Serum total 25(OH)D reflects not only sunlight availability but also effective skin exposure, indoor activity, clothing habits, sunscreen use, dietary intake, adiposity, skin type, and other contextual factors. [13,30]

In urban women, these influences can converge so that circulating 25(OH)D remains low even when ultraviolet radiation is geographically available. The Bogor findings therefore complement evidence from Jakarta, Padang, and Southern Thailand rather than assuming that all tropical populations have the same exposure profile. [7,8,13]

Association with Total Cholesterol

The inverse association between total cholesterol and serum total 25(OH)D was the most consistent lipid finding. Its magnitude was weak rather than strong: Spearman's ρ of −0.293 indicates that higher total cholesterol tended to accompany lower serum total 25(OH)D, but substantial individual variation remained. The adjusted coefficient was also statistically significant after controlling for age and waist-to-hip ratio. In contrast, triglycerides, HDL-C, and LDL-C did not show statistically significant Spearman associations in this sample. Similar inverse patterns between vitamin D status and lipid-related outcomes have been reported in women and broader adult populations.[9,10,11,12,31] The consistency of the total-cholesterol direction supports biological plausibility, but it does not justify describing the association as clinically deterministic.

Several pathways may contribute to an association between serum total 25(OH)D and total cholesterol. Vitamin D and cholesterol share a metabolic connection through 7-dehydrocholesterol, a precursor involved in cutaneous vitamin D synthesis and cholesterol-related pathways. [6,32] Vitamin D signaling has also been discussed in relation to inflammatory activity, insulin sensitivity, adipocyte function, hepatic lipid handling, and gene expression involved in lipid metabolism.[1,2,33] Nevertheless, an observational association does not demonstrate that low vitamin D status causes higher cholesterol. Recent meta-analyses of randomized trials continue to show mixed findings, with some effects on triglycerides or selected subgroups but no consistent improvement in total cholesterol, LDL-C, or HDL-C.[34,35,36] Therefore, the present finding should be interpreted as an association rather than evidence that vitamin D supplementation is a lipid-lowering treatment.

Waist-to-Hip Ratio (WHR) in Relation to Serum 25(OH)D

The positive association between waist-to-hip ratio and log-transformed serum total 25(OH)D was opposite to the direction generally reported in the literature and should be interpreted cautiously. Most previous studies and meta-analyses have shown that central adiposity is associated with lower vitamin D status when assessed using waist circumference, waist-to-hip ratio, abdominal obesity, or body-composition indicators.[37,26,27] The current literature therefore does not provide a clear biological expectation for a positive association.

Several factors may explain this unexpected coefficient. The sample size was modest, and most participants had serum total 25(OH)D values clustered in a very low range, which limited biological contrast. Waist-to-hip ratio is also an indirect anthropometric measure and cannot distinguish visceral fat, subcutaneous fat, lean mass, or overall

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body composition. Small measurement differences in waist or hip circumference can influence the ratio, and residual confounding or model sensitivity may remain. The positive coefficient may therefore reflect sampling variability, measurement limitations, or characteristics specific to this screening sample rather than a reproducible physiological pattern.

For these reasons, the waist-to-hip ratio result is considered exploratory and hypothesis-generating. It should not be interpreted as evidence against the more commonly reported inverse relation between adiposity and vitamin D status. Larger studies using direct measures of body composition are needed to clarify this association in adult women living in tropical urban settings**.**

Serum 25(OH)D as a Continuous Variable

Modeling serum total 25(OH)D as a continuous variable preserved information across the observed range. Because most participants had low concentrations, dichotomizing vitamin D status would have compressed the data, reduced statistical power, and obscured variation within the low range. Methodological literature has emphasized that categorizing continuous variables can lead to information loss and reduced sensitivity for detecting associations.[38,39]

This approach was also conceptually appropriate because vitamin D status is biologically continuous. Serum total 25(OH)D reflects cumulative contributions from sunlight exposure, dietary intake, metabolism, adiposity, and tissue distribution.[4,6] Clinical categories remained useful for describing the study sample, but continuous modeling was more informative for evaluating association patterns and did not require treating one threshold as universally applicable.

Scientific Implications

Scientifically, this study indicates that tropical residence should not be used as a proxy for adequate vitamin D status among adult women. The large sample proportion with low serum total 25(OH)D and the weak inverse association with total cholesterol provide locally specific evidence that can support hypothesis generation regarding vitamin D and cardiometabolic health. The modest association strength, purposive sampling, and cross-sectional design require a restrained interpretation. Future research should use larger representative samples and longitudinal or intervention designs to evaluate temporality, reproducibility, clinical relevance, and possible mechanisms.[4,5,6,7, 8,9,10,11,12]

The findings also distinguish observational association from intervention effects. Randomized-trial meta-analyses have not shown a consistent reduction in total cholesterol or LDL-C after vitamin D supplementation, although selected effects on triglycerides or HDL-C have been reported in some populations.[35],[36] The current inverse association should therefore be used to generate and refine hypotheses, not to infer that increasing serum 25(OH)D will lower total cholesterol.

Practical Implications for Nutrition and Public Health

Practically, the findings support attention to nutritional risk among adult women who have limited effective sunlight exposure, low dietary vitamin D intake, predominantly indoor activity, or clinical indications for vitamin D evaluation. They do not justify universal vitamin D screening or vitamin D supplementation as a lipid-lowering treatment. Current guidance does not support routine serum 25(OH)D testing in generally healthy adults without established indications.[5,6]

Counseling may emphasize safe sunlight exposure, adequate dietary intake, regular physical activity, and appropriate dyslipidemia management according to established clinical recommendations. Vitamin D supplementation, when clinically indicated, should be directed toward correction of documented deficiency and individual clinical needs

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rather than presumed treatment of elevated total cholesterol. These recommendations are practical responses to nutritional and cardiometabolic risk, not causal effects demonstrated by this cross-sectional analysis.

Strength and limitations

Strengths of the study include objective measurement of serum total 25(OH)D, assessment of all four conventional lipid variables, standardized anthropometry, consideration of diet, physical activity, and sunlight exposure, and analysis of a local adult female screening population. The use of a continuous 25(OH)D outcome and HC3 robust standard errors also improved the transparency of the analytical approach.

Several limitations define the scope of inference. The cross-sectional design precludes causal interpretation, purposive sampling limits generalizability, and the 85 participants represented all eligible complete screening records rather than a prevalence-based probability sample. Two 24-hour recalls may not represent usual dietary intake, and some behavioral variables were self-reported. Lipid profiling used a point-of-care capillary method, with LDL-C estimated internally by the device, so results may not be directly interchangeable with reference laboratory methods. The unexpected positive waist-to-hip ratio coefficient may reflect sampling variability, residual confounding, or measurement limitations. These constraints do not invalidate the observed associations, but they require confirmation in larger representative cohorts with longitudinal follow-up and more precise body-composition assessment.

CONCLUSION

A large proportion of this purposively sampled group had low vitamin D status, with 92.9% of participants having serum total 25(OH)D <20 ng/mL and 60.0% having concentrations <10 ng/mL. Total cholesterol showed a weak inverse association with serum total 25(OH)D in the primary Spearman analysis and remained inversely associated in the adjusted regression model. Triglycerides, HDL-C, and LDL-C did not show statistically significant bivariate associations with serum total 25(OH)D. Because the study was cross-sectional and used purposive sampling, the findings describe associations within this sample and do not establish causality or population prevalence. Larger representative longitudinal studies are needed to assess temporality and clinical relevance.

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