

Vitamin D3 Supplementation Status and Its Association with Serum Omentin-1, Glycemic Control, and Insulin Resistance in Women with Polycystic Ovary Syndrome: A Cross-Sectional Study

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ABSTRACT:

Background: Polycystic ovary syndrome (PCOS) is a metabolic and endocrine disorder associated with obesity, adipokine dysregulation, insulin resistance, and subfertility. Omentin-1 is an adipokine with anti-inflammatory and insulin-sensitizing properties.

Aims: To evaluate the association of vitamin D3 supplementation status with serum omentin-1 levels, glycemic control, and insulin resistance in women with PCOS.

Materials and Methods: This cross-sectional study included 105 women aged 16–40 years: 35 healthy controls, 31 women with PCOS without vitamin D3 supplementation, and 39 women with PCOS reporting daily supplementation (400 IU). Serum omentin-1, insulin, and 25-hydroxyvitamin D3 levels were measured using enzyme-linked immunosorbent assays, while fasting blood glucose was determined by colorimetry. Glucose data were available for 103 participants. Insulin resistance was assessed using HOMA-IR. Group differences were evaluated using the Kruskal–Wallis test followed by Dunn’s multiple-comparison test. Multiple linear regression analyses were adjusted for age and body mass index (BMI). **Results:** After adjustment for age and BMI, vitamin D3 supplementation status was significantly associated with higher serum omentin-1 levels ($\beta = 56.82$; 95% CI: 27.92–85.71; $P = 0.0002$), lower glucose levels ($\beta = -34.34$; 95% CI: -39.82 to -28.86; $P < 0.0001$), and lower HOMA-IR values ($\beta = -1.581$; 95% CI: -2.132 to -1.031; $P < 0.0001$). The association with insulin levels was nonsignificant ($\beta = -1.299$; 95% CI: -3.603 to 1.005; $P = 0.2644$). **Conclusion:** Vitamin D3 supplementation status was associated with higher omentin-1 levels and lower glucose and HOMA-IR values in women with PCOS. However, the observational design precludes causal inference. Prospective studies are warranted.

KEYWORDS: Glycemic control, Insulin resistance, Omentin-1, Polycystic ovary syndrome, Vitamin D3.

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1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex metabolic and endocrine disease that affects women of reproductive age, typically characterized by insulin resistance, obesity, infertility, polycystic ovaries, anovulation, excess androgens, and irregular periods. [1] Obesity, neuroendocrine disturbances, gut dysbiosis, genetics, environmental pollutants, and diet or lifestyle are among the risk factors that increase a woman's risk of developing PCOS. [2] Polycystic ovaries, elevated androgens, and irregular menstruation lead to infertility and increase the possibility of depression, cardiovascular disease, and type 2 diabetes mellitus. [3]

In addition to its effects on reproduction, PCOS is closely linked to abnormalities in adipokines, insulin resistance, and obesity. Adipokines are hormones released by adipose tissue that affect inflammation and metabolism. In women with PCOS, adipokines influence endocrine signaling and metabolism. [4] Many adipokines have been shown to modulate local ovarian steroidogenesis or to affect the regulation of the pituitary-gonadal axis. It has been documented that women with PCOS exhibit abnormalities in the regulation of adipokines secreted by adipocytes. For example, the serum levels of adiponectin are lower, and leptin is higher. [5]

Omentin-1 is one such adipokine that has garnered increasing interest due to its possible function in reducing inflammation and enhancing insulin sensitivity. [6] The importance of adipokines in PCOS has already been confirmed by recent studies using several omics techniques, which have identified single-nucleotide polymorphisms in adipokines and their receptors as sensitivity markers for PCOS. [7] In addition, epigenetic modifications passed from mother to child that alter the expression of adipokines are receiving increasing attention. However, circulating adipokine levels have been the focus of most traditional studies that have addressed the role of adipokines in PCOS. Subsequent studies investigated the gene expression of adipokines in the ovaries of women with PCOS or their

concentrations in follicular fluid. [8] To elucidate the molecular processes behind the action of adipokines in PCOS, in vitro investigations were carried out. In addition, the role of brown adipose tissue in causing and treating PCOS has received attention recently. [9] However, a common problem among women with PCOS is vitamin D insufficiency, which is a contributing cause of metabolic abnormalities in this disorder. [10] Limited evidence exists regarding the association between vitamin D3 supplementation status and circulating omentin-1 levels specifically among women with PCOS. Therefore, the current study provides population-specific observational data on this relationship and its correlation with glycemic control and insulin resistance.

2. MATERIALS AND METHODS

Data: Participants were recruited from private clinics in Nineveh Governorate between February and August 2026. The final sample consisted of 105 women, including 35 healthy women (control group) and 70 women diagnosed with PCOS. Based on the available recruitment records, it was not possible to determine the total number of women assessed for eligibility, nor the number who were excluded for specific reasons. It was not possible to prepare a complete flowchart for the selection of participants. This limited the possibility of assessing the likelihood of selection bias and prevented full compliance with the reporting requirements for participant flow according to STROBE standards.

No prior calculation of the sample size was performed. The study sample consists of the eligible participants recruited during the study period. Women with PCOS were diagnosed according to the Rotterdam criteria and classified according to their pre-existing vitamin D3 supplementation status. The status of supplementation, dosage, and duration were determined through self-reported supplementation use dates obtained during interviews. Women receiving high-dose vitamin D3 supplements weekly or intermittently were excluded. Supplementation history, including dosage and frequency of use, was assessed through participant

interviews and self-reported information. The supplements were not independently verified through prescription records, drug dispensing records, or pill counts. Adherence and potential interruptions in supplementation have not been objectively assessed and cannot be independently confirmed. The vitamin D3 supplementation group consisted of women who were already taking 400 IU/day of cholecalciferol as part of their routine care, while the non-supplemented PCOS group were not taking vitamin D3. The decision to prescribe vitamin D3 was made independently of the research protocol. All biomarkers and clinical variables were assessed in a single study visit. To reduce measurement bias, clinical measurements and blood sample collection were performed using consistent procedures across study groups, and serum biomarkers were measured using laboratory methods described below. Potential selection bias was considered when interpreting the results because recruitment was limited to private clinics and participants were not randomly selected.

Ethical approval: Ethical approval was obtained from the Pharmaceutical Research Ethics Committee (PREC), College of Pharmacy/University of Mosul, in accordance with ethical guidelines (approval no.: PREC-26-6-20, Date: January 05, 2026). All participants provided informed consent. The study excluded women who were pregnant or nursing, smoking, having a history of alcohol use, changing medications during the study year, taking medications other than vitamin D3 supplementation, having acute or chronic conditions other than PCOS, or being unable to follow study instructions. In addition, women whose serum samples were insufficient to perform all the required biochemical analyses were excluded from the final analysis. The final study population consisted of 105 women: 35 women in the control group (healthy), 31 women with PCOS who did not take vitamin D3 supplements, and 39 women with PCOS who reported taking vitamin D3 supplements. Because fasting blood glucose measurements were not available for two women, the available glucose measurements were

limited to 103 women. No financial assistance was offered to participants to take part in the study.

Diagnostic criteria for PCOS: The diagnosis of PCOS was based on the Rotterdam criteria, which require at least two of three diagnostic features after excluding related alternative disorders. However, the availability of study records did not allow for participant-level verification of the clinical, biochemical, and ultrasound findings used to establish the diagnosis. This limitation presents a potential misdiagnosis and should be considered when interpreting the results.

Collection of samples: Following an overnight fast, five milliliters of venous blood was drawn from the PCOS and healthy women. Each participant's blood samples were promptly transferred to gel tubes, where they were incubated in a water bath for ten minutes at 37 °C. Following that, samples were separated by centrifugation at 3500 × g for 10 minutes in order to obtain the serum.

The serum samples were stored at -20 °C for approximately one month and were not thawed more than once before analysis. ELISA measurements were performed in duplicate according to the manufacturer's instructions. However, the original study records did not contain sufficient documentation to independently verify the laboratory quality-control performance, calibration procedures, or intra- and inter-assay variance coefficients. Details regarding the conditions of sample transport were also unavailable.

Laboratory investigation: Serum concentrations of omentin-1, insulin, and 25-hydroxyvitamin D3 [25(OH)D3] were measured using commercially available ELISA kits from Bioassay Technology Laboratory (BT LAB). Omentin-1 levels were measured using the Human Intelectin-1 (ITLN1) ELISA Kit (catalogue no. E0155Hu), insulin levels using the Human Insulin (INS) ELISA Kit (catalogue no. E0010Hu), and 25(OH)D3 levels using the Human 25-hydroxyvitamin D3 ELISA Kit (catalogue no. E1981Hu), according to the manufacturers' instructions. Each serum sample was

analyzed in duplicate, and the absorbance was measured at 450 nm.

According to the manufacturer's documentation, the detection range of the omentin-1 kit is between 2 and 600 ng/L with a sensitivity of 1.03 ng/L. The detection range for the insulin test kit is between 0.2 and 60 mIU/L with a sensitivity of 0.11 mIU/L. The detection range for the 25(OH)D3 kit is between 0.5 and 150 ng/mL with a sensitivity of 0.23 ng/mL. These specifications represent values reported by the manufacturer and have not been independently verified in the study laboratory. Therefore, the analytical accuracy and assay performance under the specific conditions of this study cannot be independently confirmed. This limitation should be considered when interpreting laboratory measurements.

Glucose concentrations were also measured separately using the Glucose Content Assay Kit based on o-toluidine-based colorimetry (catalogue no. AK0837-100T-96S) supplied by SunLong Biotech Co., Ltd. (Hangzhou, China), according to the manufacturer's instructions.

The following formula was then applied to determine insulin resistance based on glucose and insulin levels:

$$\text{Insulin } (\mu\text{U/mL}) \times \text{Glucose (mg/dL)} / 405 = \text{HOMA-IR.}$$

Statistical analysis: Continuous variables are presented as median and interquartile range (IQR). Differences between the three study groups were assessed using the Kruskal–Wallis test, followed by Dunn's multiple-comparison test to perform pairwise comparisons. Multiple linear regression analyses were performed in women with PCOS to assess the associations between vitamin D3 supplementation status and serum levels of omentin-1, glucose, insulin, and HOMA-IR, adjusting for age and BMI. Separate regression models were constructed for each of the dependent variables. The vitamin D3 supplementation variable was coded as follows: (0) for women who did not take vitamin D3 supplements and (1) for women who took vitamin D3

supplements at a dose of 400 IU daily. Regression coefficients (β), standard errors, 95% confidence intervals (CIs), and P values were reported. The model fit and regression assumptions were assessed using actual vs. predicted plots, residual plots, homoscedasticity plots, Q-Q plots, and formal tests of residual normality. The multiple linear regression model was assessed using variance inflation factors (VIFs). The number of observations included in each model depends on the availability of the relevant outcome data and is reported in the corresponding table. Fasting blood glucose measurements were not available for two of the participants. Therefore, the glucose and HOMA-IR regression models only included participants with available data for the relevant outcomes. A P value of less than 0.05 was considered statistically significant.

3. RESULTS

Demographic and anthropometric Characteristics: [Table 1](#) and [Fig 1](#) summarize the demographic and anthropometric characteristics of the healthy women and PCOS women (age, BMI, duration of infertility, and number of children) for both groups (with or without vitamin D3 supplementation). Body mass index (BMI) varied between the study groups, with BMI being higher in the PCOS group and the vitamin D3 group compared to the healthy control group. Furthermore, women with PCOS who reported taking vitamin D3 supplements have a significantly higher BMI than the healthy control group ($p = 0.0395$). Delayed conception was observed in PCOS (with or without vitamin D3 supplementation) compared to the healthy control group (PCOS vs. control: $p = 0.0055$; PCOS + vitamin D3 vs. control: $p = 0.0467$). Additionally, the average number of children was significantly lower among women with PCOS who reported taking vitamin D3 supplements compared to women with PCOS who did not report taking supplements ($p = 0.0422$; [Table 1](#)).

Table 1: Anthropometric parameters of the studied groups.

Parameters	Control	PCOS	Control vs PCOS (p)	PCOS + vitamin D3	Control vs PCOS+vitamin D3 (p)	PCOS vs PCOS+vitamin D3 (p)
Age (years)	29 (19.5–34)	29 (27–34)	NS	27 (24–29)	NS	NS
BMI (kg/m ²)	18.82 (17.78–30.67)	27.06 (25.39–29.30)	NS	29.30 (25.30–31.59)*	*P= 0.0395	NS
Duration of infertility (months)	5 (0–15)	27 (0–72)**	**P= 0.0055	18 (0–60)*	*P= 0.0467	NS
Number of children	0 (0–2)	1 (0–3)	NS	0 (0–1)#	NS	#P= 0.0422

PCOS = polycystic ovary syndrome, BMI = body mass index. Values are presented as median (25th-75th percentile). Pairwise comparisons were performed using Dunn's multiple-comparison test after the Kruskal–Wallis test. *Represents a significant variation compared to the control group (*P<0.05, **P<0.01), and # represents a significant variation compared with untreated women (#P<0.05). NS = not statistically significant.

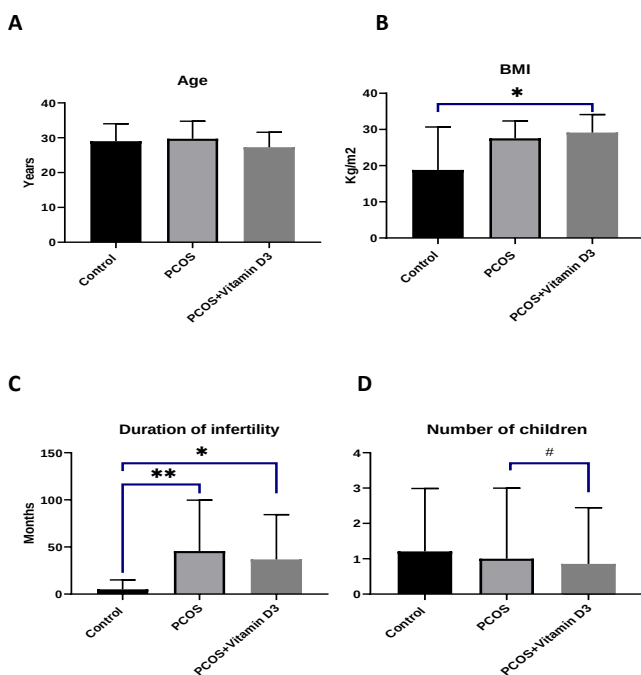


Fig 1: Anthropometric parameters of the studied groups. (A) Age, (B) Body mass index (BMI), (C) Duration of infertility, and (D) Number of children.

Table 2: Serum levels of omentin-1, glucose, insulin, HOMA-IR, and 25(OH)D3 in the studied groups.

Parameters	Control	PCOS	PCOS+vitamin D3	Control vs PCOS	Control vs PCOS+vitamin D3	PCOS vs PCOS+vitamin D3
Omentin-1 (ng/L)	201.1 (173.3–245.4)	198.9 (173.3–211.0)	263.3 (197.8–300.0)##	NS	NS	##p = 0.002
Glucose (mg/dL)	97 (85.75–102.8)	117 (113.5–123)****	85 (80.25–89.75)*****	****P < 0.0001	**p = 0.0067	####P < 0.0001
Insulin (μIU/mL)	6.885 (5.300–8.918)	15.31 (13.40–18.07)****	13.60 (10.20–14.50)****	****P < 0.0001	****P < 0.0001	NS
HOMA-IR	1.573 (1.306–1.991)	4.780 (4.030–5.287)****	2.796 (2.167–3.248)*****	****P < 0.0001	***P = 0.0008	####P < 0.0001

PCOS = polycystic ovary syndrome. Values are shown as median (25th-75th percentile). *Represents a significant variation compared to the control group (*P<0.05, **P<0.01), and # represents a significant variation compared with untreated women (#P<0.05) as set by Kruskal-Wallis test followed by Dunn's multiple-comparison test.

Validation of serum levels of glucose, insulin, and 25(OH)D3, omentin-1, and insulin resistance: As shown in [Fig 2](#), women with PCOS who received vitamin D3 supplements had significantly higher serum levels of omentin-1 compared to women with PCOS who did not receive vitamin D3 supplements (P= 0.002). They also had significantly lower serum glucose (P= 0.0067) and HOMA-IR (P= 0.0008), while serum insulin levels did not differ significantly between the two groups. ([Table 2](#))

25(OH)D3	28 (24–32.25)	27 (23–33)	29 (24.50–38.50)	NS	NS	NS
(ng/mL)						

The values are presented as median (25th–75th percentile). Overall differences between groups were assessed using the Kruskal–Wallis test, followed by Dunn’s multiple-comparison test to perform pairwise comparisons. The P-value columns report the pairwise comparisons specified in their headings. The P values are two-sided, and $P < 0.05$ was considered statistically significant. *Represents a significant variation compared to the control group (** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$), and # represents a significant variation compared with untreated women (### $P < 0.01$, #### $P < 0.0001$). PCOS = polycystic ovary syndrome; 25(OH)D3 = 25-hydroxyvitamin D3; HOMA-IR = Homeostatic Model Assessment of Insulin Resistance.

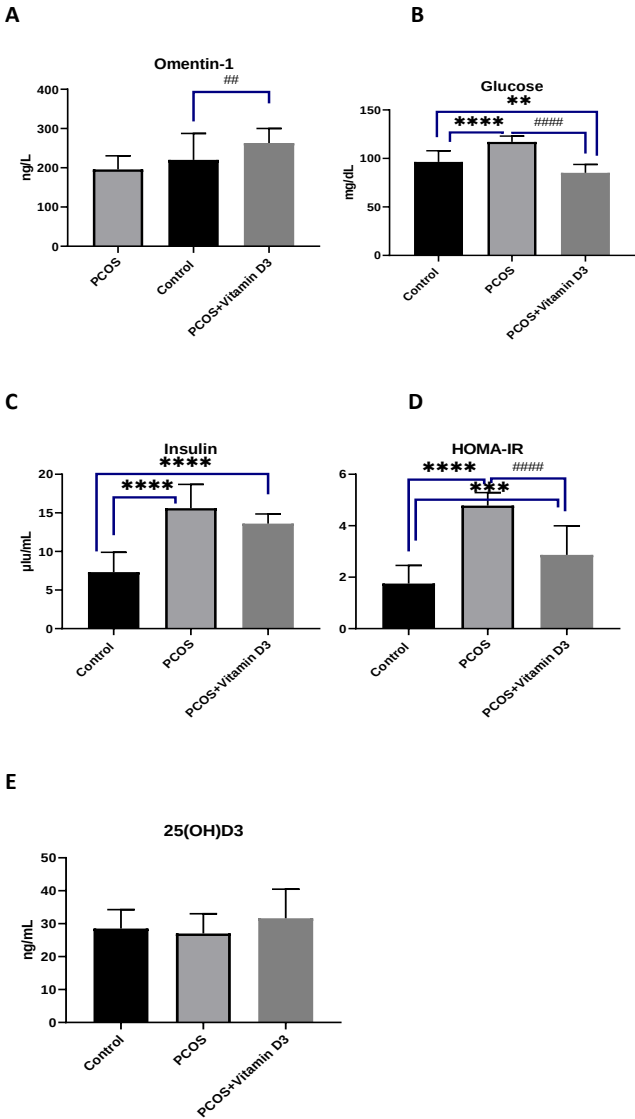


Fig 2: Serum levels of (A) Omentin-1, (B) Glucose, (C) Insulin, (D) HOMA-IR, and (E) 25(OH)D3. PCOS = polycystic ovary syndrome; HOMA-IR = Homeostasis Model Assessment of Insulin Resistance. Values are shown as median (25th-75th percentile). *Represents a significant variation compared to the control group ($P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$), and # represents a significant variation compared with untreated women (### $P < 0.01$, #### $P < 0.0001$) as set by Kruskal-Wallis test followed by Dunn’s multiple-comparison test.**

Multiple linear regression analysis: Multiple linear regression analysis was performed to assess the associations between age, BMI, vitamin D3 supplementation status, and serum levels of omentin-1, glucose, insulin, and HOMA-IR. After adjusting the results for age and BMI, a significant association was found between vitamin D3 supplementation status and higher serum omentin-1 levels ($\beta = 56.82$, 95% CI: 27.92–85.71, $P = 0.0002$) and lower glucose levels ($\beta = -34.34$, 95% CI: -39.82 to -28.86, $P < 0.0001$). Vitamin D3 supplementation was also significantly associated with lower HOMA-IR values ($\beta = -1.581$, 95% CI: -2.132 to -1.031, $P < 0.0001$). However, the association with insulin levels was not statistically significant ($\beta = -1.299$, 95% CI: -3.603 to 1.005, $P = 0.2644$). Age or BMI were not significantly associated with omentin-1, glucose, insulin, or HOMA-IR levels in the adjusted models. In general, vitamin D3 supplementation status was associated with differences in omentin-1, glucose, and HOMA-IR levels, but not insulin levels, after adjusting for age and BMI (Table 3).

Regression diagnostics and sensitivity analyses: For the omentin-1 regression model ($n = 70$), the residuals did not significantly deviate from the normal distribution ($W = 0.984$, $P = 0.521$). However, the Breusch-Pagan test showed heteroscedasticity ($P = 0.011$). Accordingly, standard errors consistent with heteroscedasticity (HC3) were used in the sensitivity analysis. The association between vitamin D3 status and omentin-1 remained statistically significant ($\beta = 56.82$, robust standard error = 14.66, 95% CI: 27.54–86.09, $P < 0.001$), which is consistent with the results of the conventional regression model.

Table 3. Multiple linear regression analysis of the relationship between vitamin D3 supplementation and metabolic and biochemical parameters in women with PCOS.

Outcome	Predictor	β	SE	95% CI	P value
Omentin-1	Age	0.2373	1.500	-2.758 to 3.233	0.8748
	BMI	-0.3552	1.414	-3.179 to 2.468	0.8024
	Vitamin D3	56.82	14.47	27.92 to 85.71	0.0002
Glucose	Age	0.2259	0.2839	-0.3413 to 0.7931	0.4291
	BMI	0.3608	0.2664	-0.1715 to 0.8931	0.1805
	Vitamin D3	-34.34	2.744	-39.82 to -28.86	<0.0001
Insulin	Age	0.1657	0.1196	-0.07317 to 0.4046	0.1707
	BMI	-0.1199	0.1128	-0.3450 to 0.1053	0.2917
	Vitamin D3	-1.299	1.154	-3.603 to 1.005	0.2644
HOMA-IR	Age	0.04655	0.02853	-0.01045 to 0.1035	0.1077
	BMI	-0.01022	0.02677	-0.06370 to 0.04326	0.7039
	Vitamin D3	-1.581	0.2757	-2.132 to -1.031	<0.0001

β = unstandardized regression coefficient; SE = Standard error; CI = confidence interval; HOMA-IR = homeostatic model assessment of insulin resistance; D3 = vitamin D3 supplementation. Multiple linear regression models were adjusted for age and BMI. D3 supplements were coded as 0 = no vitamin D3 supplement and 1 = vitamin D3 supplement (400 IU/day). The number of participants included in each regression model varied according to data availability: omentin-1 (n = 70), glucose (n = 68), insulin (n = 70), and HOMA-IR (n = 68).

The residual diagnostics for the glucose regression model indicated a deviation from the normal distribution ($W = 0.964$, $P = 0.045$). The Breusch-Pagan test did not show statistically significant heteroscedasticity ($LM = 4.220$, $P = 0.239$). Visual inspection of the residual vs. fitted and normal Q-Q plots also showed the possibility of heteroscedasticity and deviations from the normal distribution at the ends of the distribution. The Cook's distance identified five observations that exceeded the screening threshold set at $4/n$, with a maximum value of 0.167. These values have been flagged for further evaluation rather than being automatically excluded. In a sensitivity analysis using standard errors consistent with heteroscedasticity (HC3), the relationship between vitamin D3 status and glucose remained statistically significant ($\beta = -34.34$, robust standard error = 3.16, 95% CI: -40.66 to -28.02, $P < 0.0001$).

As for the insulin regression model (n = 70), the residuals deviated from the normal distribution (Shapiro-Wilk $W = 0.935$, $P = 0.0013$), with noticeable departures from the reference line, particularly at the upper end of the normal

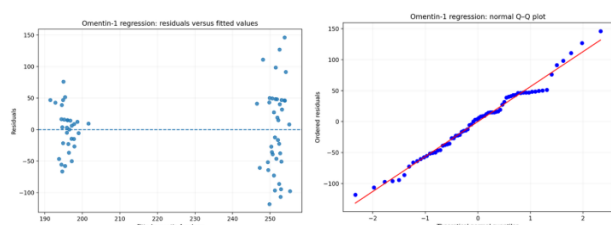
distribution plot (Q-Q plot). The Breusch-Pagan test did not show statistically significant heteroscedasticity ($P = 0.296$). The Cook's distance identified six observations that exceeded the screening threshold set at $4/n$, with a maximum value of 0.100. These observations have been flagged for further evaluation rather than being automatically excluded. In the sensitivity analysis using (HC3), the correlation between vitamin D3 status and insulin remained statistically nonsignificant ($\beta = -1.299$, robust standard error = 1.155, 95% CI: -3.604 to 1.006, $P = 0.265$), which is consistent with the results of the conventional regression model.

The values of Variance Inflation Factor (VIF) were low in all three regression models, ranging from 1.044 to 1.111 for omentin-1, 1.039 to 1.114 for glucose, and 1.044 to 1.111 for insulin, suggesting no evidence of concerning multicollinearity between the predictors.

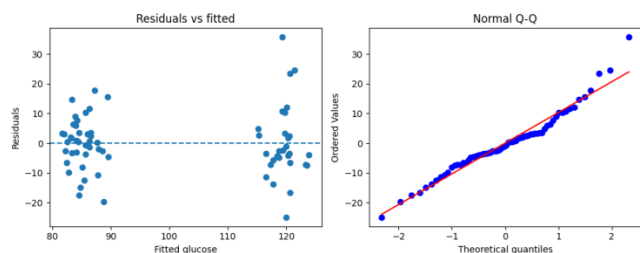
In general, the sensitivity analyses, which used robust standard errors of the HC3 type, did not lead to substantial changes in the conclusions regarding the relationship between vitamin D3 status and omentin-1, glucose, and

insulin. The relationships with glucose and omentin-1 remained statistically significant, while the relationship with insulin remained statistically insignificant. However, deviations from the normal distribution of residuals in the glucose and insulin models, as well as the presence of heteroscedasticity in the omentin-1 model, should be considered when interpreting the regression results. Influence diagnostics also identified some observations that require further evaluation to determine whether any of them have an undue influence on the model's estimates (Fig 3).

A



B



C

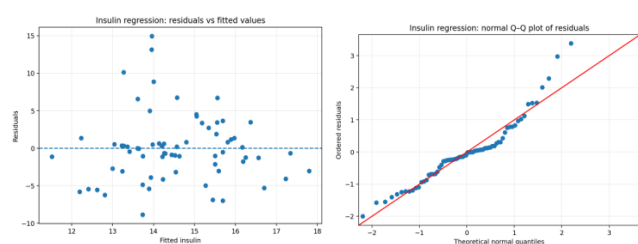


Fig 3: Diagnostic plots of regression models for omentin-1, glucose, and insulin. (A) Omentin-1 model; (B) Glucose model; and (C) Insulin model. Each of these sections displays both a residuals vs. fitted plot and the normal Q-Q plot. These plots were used to assess residual patterns, the likelihood of heteroscedasticity, and any deviations from the normal distribution of residuals.

We assessed the impact of observations that exceed the Cook's distance screening threshold by comparing regression estimates from the entire dataset with those

obtained in sensitivity analyses excluding the flagged observations. Sensitivity analyses showed that excluding these observations did not materially change the direction, magnitude, or statistical significance of the regression estimates. Therefore, the results were considered robust to the presence of potentially influential observations, and all observations were retained in the primary analyses unless there was an independent reason related to data quality for exclusion.

4. DISCUSSION

The complex features of polycystic ovary syndrome make it difficult for specialists to determine the underlying cause and identify the key indicators needed to diagnose the condition and its related metabolic consequences. The major variations in PCOS are glucose intolerance and insulin resistance, which affect other potential biomarkers, such as adipokines (omentin-1). [11] The current study aimed to evaluate the association between vitamin D3 supplementation and serum omentin-1 levels, as well as its association with glycemic control and insulin resistance, in women with polycystic ovary syndrome. Omentin-1, a 296-amino-acid adipokine produced by a gene located on chromosome 1q22-q23, is the primary circulating form of omentin-1 in human serum and has been linked to type 2 diabetes. Omentin-1 is found in blood and is primarily expressed in visceral tissue and, to a lesser extent, in subcutaneous tissue. [12] Previous studies, including Rothermel et al., have reported that omentin-1 levels were lower in overweight individuals and that there was an inverse relationship between omentin-1 and body mass index. [13] Reduced expression of the omentin-coding gene has also been reported in visceral adipose tissue in overweight individuals, which may reflect the inflammatory environment associated with obesity, including increased levels of tumor necrosis factor- α and interleukin-1. [14] In this study, serum omentin-1 levels were numerically lower in women with PCOS who did not take vitamin D3 supplements than in healthy women. However, this difference was not statistically significant. Omentin-1 levels

in women with PCOS who reported taking vitamin D3 were significantly higher than those in the non-supplemented PCOS group ($P = 0.002$), while the difference between the supplemented PCOS group and the control group was not statistically significant ([Table 2](#)). These results differ from studies that reported decreased serum omentin-1 levels in women with PCOS [15, 16]. Differences between studies may reflect variations in participant characteristics, body fat percentage, metabolic status, and study methods. However, these results should not be interpreted as evidence that taking vitamin D3 supplements directly increases omentin-1 levels, because supplementation was not randomized and the study was cross-sectional.

Metabolic results showed that women with PCOS who did not take vitamin D3 supplementation had higher serum glucose, insulin, and HOMA-IR values compared to healthy controls ([Table 2](#)). Women with PCOS who reported supplementation had lower glucose and HOMA-IR values than the non-supplemented PCOS group, while insulin levels did not differ significantly between the two PCOS groups. These results are consistent with the established association between PCOS and metabolic disorders, including insulin resistance. [17, 18] However, the lack of a significant difference in omentin-1 between the non-supplemented PCOS group and controls suggests that the relationship between omentin-1 and metabolic status may not be uniform among all women with PCOS. Although experimental studies suggest that omentin-1 may affect insulin-stimulated glucose uptake through pathways involving Akt signaling and GLUT4 translocation, these mechanisms were not investigated in the current study and therefore cannot directly explain the observed group differences. [19] Previous studies have reported inconsistent findings regarding the effects of glucose and insulin on omentin-1 production. Wurm et al. found no significant change in plasma omentin-1 levels before and two hours after glucose intake [20], whereas Tan et al. reported that glucose and insulin reduce omentin synthesis and secretion in an *ex vivo* study. [21] These differences

suggest that the relationship between glucose, insulin, and omentin-1 may depend on the experimental setup and physiological context.

Obesity, insulin resistance, and inflammation are closely related and may contribute to the metabolic disorders associated with PCOS. [22-25] Women with PCOS are also at increased risk of metabolic complications, including heart attacks, type 2 diabetes, and obesity. [3] Therefore, further studies into the pathophysiology of PCOS are needed to improve understanding of these mechanisms and to guide preventive and therapeutic strategies. [26]

Previous studies have reported decreased levels of omentin-1 in overweight women with PCOS and decreased omentin-1 expression in plasma and adipose tissue of women with PCOS. [11, 21] Tan et al. also reported that glucose and insulin reduce the synthesis and secretion of omentin-1 in a dose-dependent manner in a pilot study. [21] Furthermore, an inverse relationship between omentin-1 levels and BMI has been reported in previous research. However, in this study, BMI was not significantly associated with age-adjusted serum levels of omentin-1. This suggests that the relationship between obesity and omentin-1 levels may vary among study populations and that BMI alone may not fully explain differences in omentin-1 levels among women with PCOS. Notably, vitamin D3 supplementation status was significantly associated with higher levels of omentin-1 after adjusting for age and BMI. Nevertheless, given the cross-sectional design and the non-randomized supplementation status, this association should not be interpreted as a causal effect of vitamin D3 supplementation.

PCOS is a multifactorial disorder with diverse clinical and metabolic manifestations, and its management requires an individualized approach. Available treatment options depend on the patient's clinical presentation and treatment goals. [27] However, relatively few studies have specifically investigated the association between vitamin D3 supplementation and serum omentin-1 levels in women with PCOS.

In this study, vitamin D3 supplementation status was significantly associated with lower glucose and HOMA-IR values, but not with insulin levels, after adjusting for age and BMI. Previous studies investigating vitamin D3 supplementation and metabolic outcomes have reported mixed results. Niroomand et al. revealed a significant decrease in insulin resistance and a non-significant decrease in blood glucose levels after six months of taking a high-dose vitamin D3 supplementation. [28] Interestingly, Peterson et al. reviewed the evidence regarding vitamin D deficiency and insulin resistance in obese adolescents and noted the inconsistency of results in studies that deal with vitamin D3 supplementation, which underscores the need for more well-designed clinical trials. [29] Al-Shami et al. reported an association between low vitamin D levels and increased insulin resistance in patients with type 2 diabetes mellitus. However, their cross-sectional findings do not establish the effects of vitamin D3 supplementation on HOMA-IR or glucose levels. [30] These differences may reflect differences in study groups, vitamin D3 dosage, duration of supplementation, baseline metabolic status, and study design. Therefore, the current findings add to the existing evidence but do not prove that vitamin D3 supplementation directly improves metabolic outcomes in women with PCOS.

Previous studies have reported inconsistent effects of vitamin D3 supplementation on glucose regulation and insulin resistance. Bhatt et al. showed that in women with prediabetes and obesity, after 78 weeks of taking vitamin D3 supplements, serum glucose levels decreased, while insulin and insulin resistance remained unchanged. [31] However, Nazarian et al. showed that taking vitamin D supplements improves insulin sensitivity in patients with impaired glucose regulation. [32] Other studies have indicated no significant metabolic improvement following supplementation. [33] These differences may be attributed to variations in the characteristics of the study groups, baseline vitamin D levels, supplementation regimens, and study designs. In the current study, vitamin D3

supplementation status was significantly associated with lower levels of glucose and HOMA-IR index, but without a statistically significant difference in insulin levels after adjusting the results for age and BMI. Because the study relied on a cross-sectional design, these results should be interpreted as reflecting correlational relationships and not as evidence of a therapeutic effect.

An important finding of this study is that associations between vitamin D3 supplementation status and metabolic outcomes remained statistically significant after adjusting for age and BMI. In the adjusted regression models ([Table 3](#)), vitamin D3 supplementation status was significantly associated with higher serum omentin-1 levels ($\beta = 56.82$, 95% CI: 27.92–85.71, $P = 0.0002$) and lower glucose levels ($\beta = -34.34$, 95% CI: -39.82 to -28.86, $P < 0.0001$). Vitamin D3 supplementation was also significantly associated with lower HOMA-IR values ($\beta = -1.581$, 95% CI: -2.132 to -1.031, $P < 0.0001$). However, the association with insulin levels was not statistically significant ($\beta = -1.299$, 95% CI: -3.603 to 1.005, $P = 0.2644$). These results indicate that the observed associations with omentin-1, glucose, and HOMA-IR persisted after accounting for age and BMI. However, residual confounding by other factors cannot be ruled out.

Several studies have reported inverse associations between omentin-1 levels and glucose intolerance or insulin resistance [34–36], while some studies have reported increases in omentin-1 levels after vitamin D3 supplementation. However, the evidence is inconsistent. One study found that vitamin D intervention did not increase omentin-1 levels in prediabetic patients, although subgroup analysis indicated significantly higher omentin-1 levels among patients with low baseline serum 25(OH)D3 concentrations. [37] Similarly, Cheshmazar et al. reported no significant change in serum omentin-1 levels after the intervention. [38] In contrast, Jafari et al. showed a significant increase in omentin-1 levels in postmenopausal women with type 2 diabetes after consuming vitamin D-fortified yogurt daily for 12 weeks. [39] Additionally, Amirkhizi et al. reported a positive relationship between

omentin-1 levels and vitamin D. [40] Taken together, these results suggest that the relationship between vitamin D3 status or supplementation and omentin-1 may vary according to the study population and intervention characteristics.

Interestingly, the lack of a statistically significant difference in serum 25(OH)D3 levels between the two PCOS groups is worth considering. Although participants in the supplemented group reported taking vitamin D3 at a dose of 400 IU/day, the relatively low supplement dose may have contributed to the lack of a detectable difference in serum 25(OH)D3 concentrations. However, the extent of its contribution cannot be determined based on these data. Furthermore, serum 25(OH)D3 levels may be affected by baseline vitamin D status, sunlight exposure, food intake, and individual differences in absorption and metabolism. Because adherence to supplementation and potential interruptions were not objectively verified, and serum 25(OH)D3 concentrations prior to supplementation were unavailable, misclassification of supplementation status may also have contributed to the observed results. These potential explanations should be considered with caution because adherence and relevant determinants of vitamin D status were not directly assessed in this study.

Limitations: This study has a cross-sectional observational design, which prevents causal conclusions about vitamin D3 supplementation and metabolic outcomes. The relatively small sample size and the fact that recruitment was limited to private clinics in Nineveh Governorate may have led to bias in selection and reduced the possibility of generalizing the results. The incompleteness of the recruitment records also prevented the determination of the total number of women who were screened and excluded for each reason, which limited the assessment of participant selection and full reporting of participant flow in accordance with STROBE recommendations. In addition, fasting blood glucose measurements were not available for two of the participants, leaving 103 observations for this result, and the exclusion of women with insufficient serum samples

may have led to further selection bias. The status, dosage, and duration of supplement use were based on self-reporting and were not independently verified, which may lead to misclassification of exposure. Nutritional factors, physical activity, body composition changes, and molecular mechanisms were not directly assessed. Finally, no prior calculation of sample size was performed, which may have reduced the accuracy of effect estimates and the ability to detect smaller correlations. Therefore, results that are not statistically significant should be interpreted with caution. Larger prospective studies with appropriate sample size calculations are needed to confirm these correlations.

Although we verified the final study participants' data against the original participant dataset, the available recruitment records were insufficient to fully reconstruct the recruitment process, including the total number of women screened and the number excluded for each reason. The supporting documentation for the diagnosis of PCOS was reviewed as thoroughly as possible. However, it was not possible to independently verify, through the available study documents, the detailed diagnostic records, specific clinical assessments, laboratory tests, ultrasound findings, and exclusion procedures used by the diagnosing physician. Consequently, the completeness of the diagnostic documentation and the likelihood of misclassification cannot be fully assessed. These limitations should be considered when interpreting the results. Future studies should use standardized diagnostic criteria and maintain complete records of screening, eligibility, diagnosis, and enrollment to improve reproducibility and reporting in accordance with STROBE recommendations.

5. CONCLUSIONS

In this cross-sectional observational study, reported vitamin D3 supplementation status among women with PCOS was associated with higher serum omentin-1 levels and lower glucose and HOMA-IR values after adjustment for age and BMI. No statistically significant adjusted correlation was observed with serum insulin levels. However, serum 25(OH)D3 concentrations did not differ significantly

between the supplemented and non-supplemented PCOS groups, and supplementation status was based on self-report. Therefore, these results should be interpreted as adjusted observational correlations and not as evidence of a causal effect of vitamin D3 supplementation. More prospective studies and randomized controlled trials are needed to determine whether vitamin D3 supplementation is associated with changes in omentin-1 levels, blood glucose control, and insulin resistance in women with PCOS.

Abbreviations:

25(OH)D3: 25-Hydroxyvitamin D3; B: Regression coefficient;
BMI: Body mass index; CI / Cis: Confidence interval(s)
ELISA: Enzyme-linked immunosorbent assay
FBG: Fasting blood glucose; Fig.: Figure
HC3: Heteroscedasticity-consistent covariance estimator 3
HOMA-IR: Homeostatic model assessment of insulin resistance
IQR: Interquartile range; IU: International unit
LM: Lagrange multiplier; mg/dL: Milligrams per deciliter
mIU/L: Milli-international units per liter
ng/L: Nanograms per liter; ng/mL: Nanograms per milliliter
NS: Not significant; PCOS: Polycystic ovary syndrome
P: Probability value; Q-Q plot: Quantile–quantile plot
SE: Standard error; VIF: Variance inflation factor
W: Shapiro–Wilk test statistic;
μIU/mL: Micro-international units per milliliter

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