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Research Article

Association of Vitamin D with Insulin Resistance and Pancreatic β-Cell Function in Non-Diabetic Obese Staff: A Cross-Sectional Study

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ABSTRACT

This research explored whether vitamin D status is related to Insulin Resistance (IR) and pancreatic β-cell performance among non-diabetic obese employees at Dr. M. Djamil Padang Hospital, West Sumatra, Indonesia. The study applied a cross-sectional design conducted from January to July 2020 and involved 81 obese hospital staff without diabetes. Measurements included Fasting Blood Glucose (FBG), Fasting Insulin (FI), and Serum 25-Hydroxyvitamin D [25(OH)D], all analyzed with automated instruments. HOMA-IR and HOMA-B indices were derived from FBG and FI using standard formulas. Statistical analysis used the Spearman correlation test with a significance threshold of $p<0.05$, followed by multivariate linear regression. The participants were predominantly female (58%), aged 18–58 years, with a mean Body Mass Index (BMI) of 31.46±3.99 kg/m². Average vitamin D levels were 14.84±5.48 ng/mL, while median values for HOMA-IR and HOMA-B were 2.30 and 193.09%, respectively. The results indicated that vitamin D concentrations did not show a meaningful correlation with either HOMA-IR ($r=0.071$; $p=0.530$) or HOMA-B ($r=-0.106$; $p=0.347$). BMI demonstrated the strongest association with HOMA-IR ($\beta=0.165$), and HOMA-IR was the variable most strongly related to HOMA-B ($\beta=21.83$). Overall, this study concludes that baseline vitamin D levels are not significantly linked to insulin resistance or pancreatic β-cell function, as reflected by HOMA-IR and HOMA-B measurements, among non-diabetic obese staff at Dr. M. Djamil Padang Hospital.

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INTRODUCTION

Obesity is a persistent global health challenge. According to the Asia-Pacific guidelines, obesity is defined as having a Body Mass Index (BMI) of 25 or higher (Harbuwono *et al.* 2018; Contreras-Bolivar *et al.* 2021). The World Health Organization (WHO) has also identified obesity as a major risk factor for numerous non-communicable diseases (Harbuwono *et al.* 2018). Moreover, obesity is one of the core components of metabolic syndrome, a condition that has reached epidemic levels worldwide. Current estimates show that metabolic syndrome affects 12–37% of the Asian population and 12–26% of the European population (Sigit *et al.* 2020). In Indonesia, the prevalence of general obesity and central obesity among adults is 23.1% and 28%, respectively, both of which are strongly associated with an increased risk of diabetes and hypertension (Harbuwono *et al.* 2018).

According to the West Sumatra Health Survey conducted in 2018, 19.62% of adults in the province were classified as obese [Ministry of Health Republic of Indonesia] (MoH RI 2018). Previous evidence has demonstrated that

individuals with a higher BMI tend to have lower serum concentrations of 25(OH)D compared to those with a normal BMI (Pantovic *et al.* 2019; Dai *et al.* 2025). Additionally, insulin-resistant obesity is frequently associated with reduced circulating levels of vitamin D (Contreras-Bolivar *et al.* 2021).

Vitamin D is a fat-soluble prohormone with endocrine, autocrine, and paracrine roles that are vital for bone health. In addition to its skeletal effects, vitamin D also participates in various extraskeletal physiological processes. Deficiency in this nutrient has been associated with several disorders, including diabetes, metabolic syndrome, non-alcoholic liver disease, autoimmune conditions, hypertension, cardiovascular disease, and certain cancers (Argano *et al.* 2023). Moreover, insulin-resistant obesity is frequently linked to reduced circulating concentrations of 25(OH)D (Pramono *et al.* 2019; Contreras-Bolivar *et al.* 2021).

Several studies have identified multiple mechanisms through which vitamin D may influence glucose metabolism (Argano *et al.* 2023). Vitamin D helps regulate blood glucose by modulating both insulin secretion and insulin sensitivity (Szymczak-Pajor & Śliwińska 2019). It also plays an important role in pancreatic β-cell activity. The active form, calcitriol ($1.25(OH)_2D$), acts as a signaling molecule that binds to β-cell receptors and regulates calcium flow (Contreras-Bolivar *et al.* 2021). Furthermore, vitamin D influences insulin release through Vitamin D Receptor (VDR) expression in β-cells and affects insulin sensitivity by regulating genes involved in glucose uptake and lipid metabolism in adipose tissue and skeletal muscle (Szymczak-Pajor & Śliwińska 2019).

Recent evidence indicates that obesity-related immune alterations may contribute to vitamin D deficiency (Infante *et al.* 2019; Ghaseminejad-Raeini *et al.* 2023). Vitamin D has been reported to reduce inflammation and oxidative stress (Sharma *et al.* 2025). It also protects pancreatic β-cells from oxidative damage and apoptosis, both of which are crucial for maintaining normal insulin secretion (Infante *et al.* 2019; Fathi *et al.* 2022). Calcidiol, or 25(OH) D, represents the primary circulating form of vitamin D, and its concentration is commonly used to assess vitamin D status (Contreras-Bolivar *et al.* 2021). Various organizations have proposed guidelines regarding optimal serum 25(OH)D levels (Zeng *et al.* 2021). Maintaining plasma vitamin D levels at ≥30 ng/mL is recommended for overall health (Contreras-Bolivar *et al.* 2021; Zeng *et al.* 2021). The reference values for 25(OH)D are as follows: deficiency <20 ng/mL, insufficiency 21–29 ng/mL, sufficiency/adequate 30–100 ng/mL, high or hypervitaminosis D >100 ng/mL, and toxicity >150 ng/mL (Muneer *et al.* 2022).

The Homeostatic Model Assessment (HOMA) provides an approach for estimating insulin resistance (HOMA-IR) and pancreatic β-cell performance (HOMA-B) (Kartika *et al.* 2024). This method is non-invasive and relies on mathematical calculations derived from Fasting Blood Glucose (FBG) and Fasting Insulin (FI) measurements (Gołacki *et al.* 2022). Earlier investigations examining the link between vitamin D status and either HOMA-IR or HOMA-B have yielded mixed results across various populations. To better understand this relationship in our setting, the present study sought to determine whether vitamin D levels are associated with insulin resistance and β-cell function, assessed through HOMA indices, among non-diabetic obese employees at Dr. M. Djamil Padang Hospital, West Sumatra, Indonesia.

METHODS

Design, location, and time

This cross-sectional research was carried out from January to July 2020. The required minimum sample size was determined using a single-sample correlation formula (Bujang & Baharum 2016). All participants were employees of Dr. M. Djamil Central Hospital in Padang, West Sumatra. The inclusion criteria consisted of: (1) adults aged 18–60 years and (2) a BMI of ≥25.00 kg/m², classified as obese according to the Asia-Pacific BMI standards (Harbuwono *et al.* 2018; Okawa *et al.* 2024). Participants were excluded if they had: (a) a history of diabetes; (b) Fasting Blood Glucose (FBG) ≥126.0 mg/dL [American Diabetes Association-ADA] (ADA 2020; Perkeni 2021); (c) current use of oral vitamin D supplements; (d) pregnancy; or (e) liver or kidney disorders. Ethical clearance for this study was provided by the Ethics Committee of Dr. M. Djamil Central Hospital, Padang (No. 361/KEPK/2019) in November 2019. Written informed consent was obtained from all participants prior to data collection.

Sampling

Venous blood samples were obtained by a trained phlebotomist following an overnight fast to measure FBG and FI. After collection, the samples were centrifuged at room temperature at 3,500 rpm for 15 minutes. The resulting serum was divided into two aliquot tubes. The first aliquot, designated for FBG analysis, was processed immediately after centrifugation, while the second aliquot, intended for assessing vitamin D (25(OH)D) and FI levels, was stored at −20°C until analysis. Participants with FBG values ≥126 mg/dL were excluded after laboratory testing, in accordance with diagnostic criteria for diabetes mellitus (ADA 2020; Perkeni 2021).

Data collection

Hexokinase method. FBG was analyzed on the same day of collection using a chemical analyzer based on the hexokinase technique (Cobas Integra 400 Plus Analyzer).

Chemiluminescent Microparticle Immunoassay (CMIA). The second aliquot was stored at −20°C for the assessment of fasting insulin (FI) and total 25-hydroxyvitamin D [25(OH)D]. Both FI and 25(OH)D concentrations were measured using the CMIA method (Abbott ARCHITECT (Abbott Laboratories 2018).

HOMA-IR and HOMA-B. Indices of insulin resistance and β-cell function were calculated using standard equations derived from FBG and FI values. HOMA-IR was determined using the formula: [FBG (mg/dL) × FI (μU/mL)] / 405, while HOMA-B was calculated as [360 × FI (μU/mL)] / [FBG (mg/dL) − 63].

This study applied the Internal Medicine Department's cut-off from Dr. M. Djamil Padang General Hospital, where a HOMA-IR value above 2.0 indicates insulin resistance (Decroli *et al.* 2018). The HOMA-B index reflects pancreatic β-cell performance, with normal values ranging from 70–150% (Rahman *et al.* 2019; Kartika *et al.* 2024). A HOMA-B value below 70% suggests impaired β-cell function, whereas values exceeding 150% indicate β-cell overactivity or hyperinsulinemia (Rahman *et al.* 2019).

Data analysis

Data were analyzed using IBM SPSS Statistics 21.0 (IBM Corp., Armonk, NY, USA). Normality was assessed with the Kolmogorov–Smirnov test. Numerical variables are reported as mean±Standard Deviation (SD) or as median with minimum–maximum ranges, while categorical variables are summarized as frequencies and percentages. Associations were examined using the Spearman correlation test ($p<0.05$), and potential confounders were evaluated through multivariate linear regression. This study did not stratify participants by sex or age group.

RESULTS AND DISCUSSION

Characteristics of respondents

This study included 81 respondents, whose characteristics are presented in Table 1. Participants were between 18 and 58 years old, with most falling within the 18–35 age group (81%), followed by 36–50 years (2.3%) and over 50 years (6.2%). Females made up 58% of the sample. The larger proportion of women reflects the general gender distribution of staff at Dr. M. Djamil Hospital. National data also indicate that, among adults over 18 years, the prevalence of obesity is consistently higher in women than in men (MoH RI 2018; Harbuwono *et al.* 2018). Similarly, previous research has shown that Indonesian women experience metabolic syndrome more frequently than men (Sigit *et al.* 2020).

This study found a higher prevalence of obesity among young adults aged 18–35 years. Rising obesity rates in this age group can have long-term health impacts, increasing the likelihood of chronic diseases later in life (Abbasifard *et al.* 2023). Young adulthood is marked by significant lifestyle and behavioral transitions, which may contribute to unhealthy habits such as low physical activity, poor dietary choices, and smoking—behaviors that can carry lasting health consequences (Abbasifard *et al.* 2023).

Body mass index

The mean BMI of participants was 31.46±3.99 kg/m² (Table 1). Based on the Asia–Pacific BMI classification, 34% of respondents were categorized as obese class I (BMI 25.0–29.9 kg/m²), while 47% were classified as obese class II (BMI ≥30.0 kg/m²). Two respondents had a BMI greater than 40.0 kg/m²; both were young adults aged 18 and 20 years. The BMI profile observed in this study was comparable to findings reported by Sahasrabuddhe *et al.* (2017). Previous research has similarly grouped participants using various BMI classification schemes. For example, Kavadar *et al.* categorized individuals as overweight

Table 1. Characteristics of respondents

Variable	f (%) (n=81)	Median* (Min.–Max.)
Gender		
Man	34 (42.0)	
Woman	47 (58.0)	
Age (years)		28 (18–58)
BMI (kg/m^2) (mean±SD)	31.46±3.99	
Obese I (25.0–29.9)	34 (42.0)	
Obese II (≥30.0)	47 (58.0)	
Fasting Insulin (µU/mL)		10.90 (3.30–48.40)
Fasting Blood Glucose (mg/dL) (mean±SD)	84.98±10.05	
Normal (<100 mg/dL)	75 (92.6)	
IFG (100–125)	6 (6.7)	
25 (OH) D Levels (ng/mL) (mean±SD)	14.84±5.48	
Deficiency (<20 ng/mL)	69 (85.2)	
Insufficient (20–29 ng/mL)	11(13.6)	
Sufficient (30–100 ng/mL)	1 (1.2)	
HOMA-IR		2.30 (0.60–12.00)
Insulin Sensitive (≤2.0)	31 (38.3)	
Insulin Resistance (>2.0)	50 (61.7)	
HOMA-B (%)		193.09 (60.83–981.00)
Dysfunction (<70%)	2 (2.5)	
Normal (70%–150%)	24 (29.6)	
Hiperinsulinemia (>150%)	55(67.9)	

BMI: Body Mass Index; Homa-IR: Homeostasis Model Assessment of Insulin Resistance; HOMA-B: Homeostasis Model Assessment of Beta-Cell Function; IFG: Impaired Fasting Glucose; Min.: Minimumn; Max.: Maximum; SD: Standard Deviation; *: Data was abnormal based on Korlomogorv-Smirnov normality test ($p<0.05$), data was presented by Median (minimum value–maximum value)

(BMI 25.0–29.9 kg/m²) and non-diabetic obese (BMI ≥30.0 kg/m²) (Kavadar *et al.* 2015).

Fasting insulin concentration and fasting plasma glucose levels

The respondents' FI levels ranged from 3.30 µU/mL to 48.40 µU/mL (Table 1). Thirty-three individuals (40%) recorded FI levels ≥10.0 µU/mL, and six exceeded 20.0 µU/mL. Variations in FI values are widely reported in the literature. Fasting insulin levels above 20 µU/mL indicate hyperinsulinemia (Thomas *et al.* 2019). Insulin secretion operates through two primary mechanisms: tonic secretion and biphasic secretion. Basal or tonic secretion is influenced by fluctuations in glucose levels and occurs independently of external glucose stimulation, whereas biphasic secretion represents a direct response to exogenous glucose intake (Guyton & Hall 2016).

The mean FBG observed in this study was 84.98±10.05 mg/dL. All participants were nondiabetic and reported no history of hypertension. Among individuals with obesity who do not have diabetes or hypertension, elevated insulin secretion—manifested as hyperinsulinemia—tends to predominate over insulin resistance. This pattern supports the view that hyperinsulinemia functions as a self-reinforcing process and is more likely the initiating or primary abnormality that eventually contributes to the development of insulin

resistance, rather than simply a compensatory reaction to it (Thomas *et al.* 2019).

Vitamin D level

This study evaluated respondents' baseline 25(OH)D concentrations using established reference ranges and vitamin D status categories (Muneer *et al.* 2022). The mean 25(OH) D level was 14.84±5.48 ng/mL (Table 1). Most participants (85.2%) were vitamin-D-deficient (<20 ng/mL), including 17 individuals (20.98%) with severe deficiency (<10 ng/mL) (Sahasrabuddhe *et al.* 2017). Additionally, 11 respondents (13.6%) showed insufficient levels, and only one met the criteria for sufficiency. Despite these findings, all participants were generally healthy and did not exhibit clinical manifestations typically associated with severe vitamin D deficiency.

Reliable data on vitamin D levels and status in Indonesian adults remain limited. However, existing evidence in younger populations shows that hypovitaminosis D affects about 33% of Indonesian children and adolescents, with a notably higher prevalence in females (60%) compared to males (40%) (Octavius *et al.* 2023).

HOMA-IR and HOMA-B values and vitamin D status

Median value of HOMA-IR was 2.30 with minimum and maximum values being 0.60 and 12.00, respectively. HOMA-B had a median value of 193.09% where the minimum and maximum values were 60.83% and 981.00%, respectively (Table 1).

The HOMA-IR values of the respondents are in the range of 0.60–12.00. The study established the HOMA-IR cut-off at ≤2.0 as insulin sensitive and >2.0 as insulin resistant (Decroli *et al.* 2018). A total of 31 respondents (38.3%) were insulin-sensitive, and 50 respondents (61.73%) were insulin-resistant. This study proves that obese individuals are at risk of insulin resistance (Gołacki *et al.* 2022).

Most respondents (61.7%) were classified as having insulin resistance (HOMA-IR > 2.0). Among those with vitamin D deficiency, 59.4% also showed insulin resistance. Interestingly, even the one respondent with sufficient vitamin D still exhibited insulin resistance, as shown in Table 2. Insulin resistance appeared across all vitamin D status categories. Statistical analysis based on vitamin D stratification could not be performed due to distribution limitations. The relationship between 25(OH)D and insulin-responsive tissues is complex and can be affected by numerous confounding factors (Szymczak-Pajor & Śliwińska 2019).

The HOMA-B value range of the respondents is 60.83%–981.00%. We revealed that most of the respondents (67.9%) had hyperinsulinemia (HOMA-B >150%), two respondents (2.5%) had pancreatic cell dysfunction (HOMA-B <70%), and 24 respondents (29.6%) had normal HOMA-B (70%–150%) (Table 1). This HOMA-B data is quite surprising. We found a respondent with an extreme value of HOMA-B (981.0%). Subjects who tend to have hypoglycemia have high insulin levels (Thomas *et al.* 2019). Hyperinsulinemia is a compensatory response of pancreatic β-cells in maintaining euglycemic conditions via β-cell proliferation (Mezza *et al.* 2019). Two respondents have HOMA-B <70% and are included in the IFG respondents, indicating dysfunction of pancreatic-cell failure. Another respondent had normal FBG and HOMA-B levels lower than the reference value.

Table 2. Distribution of vitamin D and HOMA-IR stratification of respondent

Vitamin D status	HOMA-IR category		Total (n=81)
	Insulin sensitive (HOMA-IR <2) n (%)	Insulin resistance (HOMA-IR≥2) n (%)	
Deficiency	28 (40.6)	41 (59.4)	69
In sufficiency	3 (27.3)	8 (72.7)	11
Sufficiency	0 (0)	1 (100)	1
Total	31 (38.27)	50 (61.73)	81

Homa-IR: Homeostasis Model Assessment of Insulin Resistance

Among respondents with vitamin D deficiency, 68.1% exhibited hyperinsulinemia, while 29.0% had normal HOMA-B and 2.9% showed pancreatic β-cell dysfunction. Additionally, seven of 11 participants (63.6%) with insufficient vitamin D and the single participant (100%) with sufficient vitamin D also presented with hyperinsulinemia, as summarized in Table 3.

There are limited studies evaluating HOMA-B in non-diabetic individuals. Corica *et al.* revealed that the mean HOMA-B value of overweight and obese children was 313.8 (186.1) % (Corica *et al.* 2019). Sharan *et al.* stated that patients with new-onset T2DM in a non-diabetic population and found HOMA-B levels but did not include the mean HOMA-B values (Sharan *et al.* 2018). The onset time and length of obese body are risk factors of impaired β-cell function in obese individuals in early adulthood, regardless of the onset age (Rahman *et al.* 2019; Inaishi & Saisho 2020; Correa-Burrows *et al.* 2021).

Relationship between vitamin D levels and HOMA-IR

Spearman correlation analysis showed no significant association between vitamin D levels and HOMA-IR ($p>0.05$). Most data were concentrated at vitamin D levels <20 ng/mL, and participants with the highest and lowest HOMA-IR scores had similar vitamin D levels (17.3 ng/mL vs. 17.6 ng/mL). Likewise, participants with the highest and lowest vitamin D levels shared similar HOMA-IR values (2.5 vs. 2.3). Evidence regarding the relationship between vitamin D and HOMA-IR in non-diabetic obese individuals is inconsistent. While some studies report a negative and significant correlation, this study found no correlation ($r=0.053$; $p=0.071$). Similarly, Kavadar *et al.* observed a non-significant association ($r=-0.145$; $p=0.080$) (Kavadar *et al.* 2015), Stoica *et al.* (2020) reported no correlation in non-diabetic overweight adult females ($r=0.041$; $p=0.442$) and Carvalho-Rassbach *et al.* (2019) also found no association.

Relationship between vitamin D levels and HOMA-B

Spearman analysis showed no significant correlation between vitamin D levels and HOMA-B in non-diabetic obese adults ($r=-0.106$; $p=0.347$). Among respondents with vitamin D deficiency, HOMA-B values were mostly above the reference range (>150%). Participants with the highest and lowest HOMA-B values had similar vitamin D levels (11.6 ng/mL vs. 10.4 ng/mL), while the individual with sufficient vitamin D had a HOMA-B of 464.4%. This pilot study is among the first in Indonesia to examine the relationship between vitamin D and HOMA-B in non-diabetic obese adults, revealing no association ($r=-0.106$; $p=0.347$). These findings differ from Corica *et al.* (2019), who reported a significant inverse relationship in overweight and obese children ($r=-0.31$; $p=0.001$), and from Sharan *et al.* (2018), who found a negative association in newly diagnosed T2DM subjects ($r=-0.514$).

This study demonstrated that vitamin D is not an independent predictor of insulin resistance (HOMA-IR) or hyperinsulinemia (HOMA-B) in non-diabetic obese adults. None of the participants reported using vitamin D supplements. Although most had serum vitamin D levels below the recommended optimal range, they showed no clinical signs of deficiency. Baseline vitamin D status did not explain differences in insulin resistance or pancreatic β-cell function. Regression analysis indicated that other confounding factors had a stronger impact, as detailed in Table 4.

Analysis using multivariate linear regression

Multivariate linear regression for HOMA-IR, including vitamin D levels, age, sex, and BMI, is presented in Table 4. The analysis showed that BMI was the strongest predictor of increased HOMA-IR ($\beta=0.165$), independent of vitamin D levels. Similarly, HOMA-IR had an independent effect on HOMA-B ($\beta=21.83$).

As shown in Table 4, baseline vitamin D levels were not a direct contributor to insulin resistance (HOMA-IR) in non-diabetic obese adults. Instead, BMI was the strongest factor associated with increased HOMA-IR, independent of vitamin D status. Chronic obesity can induce oxidative stress and inflammation, impairing insulin secretion and pancreatic β-cell function, but initial vitamin D levels showed no association (Corica *et al.* 2019). Additionally, HOMA-IR independently affected HOMA-B (Table 4). Insulin resistance elevates plasma glucose, increasing β-cell demand and triggering compensatory hyperinsulinemia to maintain euglycemia in the prediabetic state (Hudish *et al.* 2019; Gołacki *et al.* 2022). In this study,

Table 3. Vitamin D status and HOMA-B category of respondents

Vitamin D status	HOMA-IR category			Total (n=81)
	Hyperinsulinemia (HOMA-B >150%) n (%)	Normal function (HOMA-B 70%–150%) n (%)	Dysfuction cell β (HOMA-B <70%) n (%)	
Deficiency	47 (68.1)	20 (29.0)	2 (2.9)	69
In sufficiency	7 (63.6)	4 (36.4)	0 (0)	11
Sufficiency	1 (100)	0 (0)	0 (0)	1
Total	55 (67.9)	24 (29.6)	2 (2.5)	81

Homa-B: Homeostasis Model Assessment of Beta-Cell Function; HOMA-IR: Homeostasis Model Assessment of Insulin Resistance

Table 4. Multivariate linear regression analysis for HOMA-IR and HOMA-B on some variables (Vitamin D quantities, age, sex, BMI, and HOMA-IR)

Dependent variable	Independent variable	β Coefficient (initial stage)	*p*	β Coefficient (elimination stage)			
				1st stage	2nd stage	3rd stage	4th stage
HOMA-IR	25(OH)D quantities	0.044	0.264	0.057	–	–	–
	BMI	0.178	0.285	0.177	0.165*	–	–
	Age	-0.033	<0.001	-0.037	-0.033	–	–
	Sex	-0.25	0.091	–	–	–	–
HOMA-B	25(OH)D quantities	-3.68	0.340	-3.67	-4.34	–	–
	BMI	4.48	0.354	4.47	–	–	–
	Age	0.030	0.987	–	–	–	–
	Sex	-61.33	0.153	-61.09	-60.50	-36.57	–
	HOMA-IR	16.01	0.139	15.98	20.05	20.22	21.83*

*p<0.05 as significant; BMI: Body Mass Index; HOMA-RI: Homeostatic Model Assessment of Resistance of Insulin; HOMA-B: Homeostatic Model Assessment of β-cell function; 25(OH)D: 25-hydroxy vitamin D; Analysis HOMA-IR: 1st stage: Removed the sex variable; 2nd stage: Removed 25(OH)D, and found that BMI was the most significant variable (p<0.05) related to HOMA-IR (β coefficient 0.165); Analysis HOMA-B: 1st stage: Removed the age variable; 2nd stage: Removed the BMI variable; 3rd stage: Remove the 25(OH)D variable; 4th stage: Removed the gender variable, and found that HOMA-IR was the most significant variable (p<0.05) related to HOMA-B (β coefficient 21.83)

hyperinsulinemia was observed in nearly all participants, highlighting obesity and insulin resistance as primary contributors to β-cell dysfunction (Sharan *et al.* 2018; Gołacki *et al.* 2022).

CONCLUSION

This study indicates that BMI is primarily associated with HOMA-IR, which in turn is mainly linked to HOMA-B. Baseline vitamin D levels showed no significant relationship with insulin resistance or β-cell function, as measured by HOMA-IR and HOMA-B, in non-diabetic obese staff at Dr. M. Djamil Padang Hospital.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest with any institutions.

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