Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 32s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

The Role of Glycemic Control and Vitamin D3 Levels in Modulating mRNA Expression of GLUT4 and IL-6 in Newly Diagnosed Type 2 Diabetes Mellitus Patients

Imam Fatkhurrohman[*1], Andi Makbul Aman[2], Husaini Umar[3], Agussalim Bukhari[4], Heri Nugroho HS[5], Himawan Sanusi[6], Mochammad Hatta[7], Andi Alfian Zainuddin[8]

[1] Postgraduate Doctoral Study Program , Hasanuddin University, Makassar

[2,3,6] Internal Medicine Study Program, Faculty of Medicine, Hasanuddin University, Makassar

[4] Clinical Nutrition Science Study Program, Faculty of Medicine, Hasanuddin University, Makassar

[5] Internal Medicine Study Program, Faculty of Medicine, Diponegoro University, Semarang

[7] Clinical Microbiology Study Program, Faculty of Medicine, Hasanuddin University, Makassar

[8] Faculty of Medicine, Hasanuddin University, Makassar

[*]**Corresponding author:**

Imam Fatkhurrohman

Email ID: Ifatkhurrohman@yahoo.com, Email ID: makbul_aman@yahoo.com

Cite this paper as: Imam Fatkhurrohman, Andi Makbul Aman, Husaini Umar, Agussalim Bukhari, Heri Nugroho HS, Himawan Sanusi, Mochammad Hatta, Andi Alfian Zainuddin, (2025) The Role of Glycemic Control and Vitamin D3 Levels in Modulating mRNA Expression of GLUT4 and IL-6 in Newly Diagnosed Type 2 Diabetes Mellitus Patients. *Journal of Neonatal Surgery*, 14 (32s), 4460-4467.

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and chronic low-grade inflammation. The molecular mechanisms of glucose transport and inflammation in T2DM are influenced by both glycemic status and vitamin D3 levels, particularly through their effects on GLUT4 and interleukin-6 (IL-6) expression. Methods: This cross-sectional study involved 34 newly diagnosed male T2DM patients aged 40–60 years. Glycated hemoglobin (HbA1c), serum 25(OH)D3, and IL-6 levels were measured using ELISA, and mRNA expression of GLUT4 was assessed by RT-qPCR. Correlation analysis and path analysis (PLS-SEM) were performed to evaluate the direct and indirect relationships among variables. Results: HbA1c levels showed a significant negative correlation with GLUT4 expression ($r = –0.711$, $p < 0.001$) and a positive correlation with IL-6 levels ($r = 0.747$, $p < 0.001$). Vitamin D3 levels mediated the effect of glycemic control on both IL-6 and GLUT4, with significant indirect effects observed ($p < 0.05$). Path analysis revealed that vitamin D3 partially mediated the relationship between HbA1c and molecular markers of inflammation and glucose transport. Conclusion: Poor glycemic control is associated with increased IL-6 and decreased GLUT4 expression in newly diagnosed T2DM patients. Vitamin D3 plays a mediating role in this mechanism and may serve as a therapeutic adjunct to modulate inflammation and glucose transport at the molecular level. These findings highlight the metabolic significance of vitamin D beyond bone health, particularly in the early management of T2DM.

Keywords: *HbA1c, Vitamin D3, GLUT4, IL-6, Type 2 Diabetes Mellitus, Gene Expression, Insulin Resistance, Inflammation*

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance and pancreatic β-cell dysfunction, which globally accounts for more than 90% of diabetes cases (WHO, 2023). The prevalence of T2DM is increasing exponentially, with an estimated 537 million sufferers in 2021 and is expected to reach 783 million in 2045, especially in developing countries such as Indonesia which ranks fifth in the world (Webber, 2021; IDF, 2021).

Insulin resistance is the root of the pathophysiology of T2DM, where impaired glucose transport into cells, especially by the GLUT4 transporter, is the main mechanism of chronic hyperglycemia (Rahmi et al., 2021). GLUT4 plays an important role in glucose homeostasis, especially in skeletal muscle and adipose tissue, but its expression is disrupted in chronic hyperglycemia. Several studies have shown that GLUT4 expression is significantly decreased in T2DM patients, which causes reduced glucose uptake by peripheral tissues (Vargas et al., 2023).

Vitamin D, especially the active form $1,25(OH)_2D_3$, has a regulatory effect on glucose metabolism through modulation of GLUT4 expression and proinflammatory cytokines such as interleukin-6 (IL-6). Several studies have shown that low vitamin D3 levels are correlated with decreased GLUT4 expression and increased IL-6, which ultimately worsens insulin resistance (Manna & Jain, 2012; Zhang et al., 2012). IL-6 itself is a proinflammatory cytokine that plays a role in reducing insulin sensitivity and inhibiting the insulin signaling pathway through activation of the JAK/STAT pathway (Rehman et al., 2017).

Experimental studies have also shown that vitamin D has anti-inflammatory effects, increasing GLUT4 expression through genomic and nongenomic pathways, and decreasing IL-6 expression through MAP kinase inhibition and epigenetic modification (Szymczak-Pajor et al., 2020). Conversely, low vitamin D levels are known to worsen systemic inflammation and accelerate the progression of T2DM (Wimalawansa, 2018).

However, although the relationship between glycemic control, vitamin D3, GLUT4, and IL-6 has been studied separately, integrated studies assessing GLUT4 and IL-6 mRNA expression in newly diagnosed T2DM patients are still limited. Therefore, this study aims to evaluate the relationship between glycemic control (HbA1c) and vitamin D3 levels on GLUT4 and IL-6 mRNA expression, to enrich the understanding of the molecular mechanisms underlying insulin resistance and inflammation in T2DM.

2. METHOD

Research Design and Location

This study is an analytical observational study with a cross-sectional design conducted at Bhayangkara Hospital Makassar during the period January to May 2024. The main objective of this study was to evaluate the relationship between glycemic control (HbA1c) and vitamin D3 levels on GLUT4 and IL-6 mRNA expression in newly diagnosed type 2 diabetes mellitus patients.

Population and Sample

The population in this study were all male patients aged 40–60 years who were newly diagnosed with T2DM based on the American Diabetes Association (ADA) and PERKENI criteria. The sampling technique was consecutive sampling. A total of 34 subjects met the inclusion and exclusion criteria, and were willing to follow all research procedures.

Inclusion criteria:

1. Men aged 40–60 years
2. New diagnosis of T2DM (HbA1c ≥ 6.5%; GDP ≥ 126 mg/dL)
3. Have not received insulin therapy or vitamin D3 supplementation

Exclusion criteria:

1. Patients with acute/chronic infectious diseases
2. Chronic liver or kidney disease
3. Consumption of drugs that affect vitamin D metabolism
4. Autoimmune comorbidities

Research Variables

The main independent variables were HbA1c levels and serum vitamin D3 levels. The dependent variables consisted of GLUT4 mRNA expression and plasma IL-6 levels. Vitamin D3 was assessed as a mediator variable between glycemic control and molecular expression.

Examination Procedures and Techniques

1. **HbA1c** was measured using the NGSP standardized HPLC chromatography method.
2. **Vitamin D3 (25(OH)D)** was measured using the ELISA kit method according to the manufacturer's protocol.
3. **IL-6** was measured by a quantitative ELISA technique with high sensitivity.
4. **GLUT4 mRNA expression** was analyzed by RT-qPCR. RNA was extracted from whole blood samples, then converted to cDNA and amplified with GLUT4-specific primers. Expression was calculated using the ΔΔCt method with GAPDH as the housekeeping gene.

Data Processing and Analysis

Data were analyzed using SPSS and SmartPLS statistical software. Normality test was performed first with Shapiro-Wilk. Correlation between variables was evaluated using Spearman or Pearson test according to distribution. For mediation path analysis, **path analysis approach (PLS-SEM) was used** to measure the direct and indirect effects of HbA1c on GLUT4 and

IL-6 through the mediation role of vitamin D3. A p value <0.05 was considered statistically significant.

Ethical Considerations

This study has obtained ethical approval from the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (Ethics No.: 12/UN4.6.4.5.31/PP36/2024). All participants were given informed consent before following the research procedure.

3. RESULTS

Table 1. Description of Respondent Characteristics

Variables	n (%) | (Mean±SD)
IL6 (pg/mL)	(52.535±30.74)
10.25 - 23.56	9 (26.47)
23.57 - 51.11	8 (23.53)
51.12 - 82.24	8 (23.53)
82.25 - 96.45	9 (26.47)
GLUT4	(9,944±2,517)
5.63 - 8.05	9 (26.47)
8.06 - 10.11	8 (23.53)
10.12 - 11.94	8 (23.53)
11.95 - 14.04	9 (26.47)
Vitamin D3 (ng/mL)	(25,276±9,636)
<30 ng/mL	20 (58.82)
≥30 ng/mL	14 (41.18)
HbA1c (%)	(10,537±2,041)
<9.0 mmol/L	10 (29.41)
≥9.0 mmol/L	24 (70.59)
BMI (kg/m ^{2})	(26.19±2.814)
Normal	12 (35.29)
Overweight	18 (52.94)
Obesity	4 (11.76)

A total of 34 male patients with newly diagnosed type 2 DM aged 40–60 years were included. The majority had HbA1c levels ≥ 9% and vitamin D3 levels < 30 ng/mL, indicating poor glycemic control and a relatively high level of hypoVitaminosis D in this population.

Table 2. Relationship between Glycemic Control (HbA1c) and Research Biochemical Parameters

Parameter	HbA1c (Mean±SD)		p-value
	<9 mmol/L (n=10)	≥9 mmol/L (n=24)	
Vitamin D3	(33.36±6.01)	(21.91±8.88)	<0.001***##
IL6	(26.43±16.62)	(63.41±28.78)	<0.001***##
GLUT4	(12.05±1.44)	(9.07±2.35)	<0.001***#

'#' Independent T Test '##' Mann Whitney

<0.0001 '****' <0.001 '***' <0.01 '**' <0.05 '*' not significant 'ns'

There was a significant negative correlation between HbA1c and GLUT4 expression ($r = -0.711$; $p < 0.001$), and a significant positive correlation with IL-6 ($r = 0.747$; $p < 0.001$). This indicates that the worse the glycemic control, the lower the GLUT4 expression and the higher the IL-6 level.

Table 3. Relationship between Nutritional Status (BMI) and Biochemical Parameters

Parameter	BMI (Mean±SD)			p-value
	Normal (n=12)	Overweight (n=18)	Obesity (n=4)	
HbA1c	(11.18±1.74)	(9.81±1.95)	(11.88±2.43)	0.071ns#
Vitamin D3	(22.43±8.74)	(28.77±9.17)	(18.12±9.54)	0.052ns##
IL6	(62.89±28.16)	(40.83±28.93)	(74.17±29.49)	0.041*##
GLUT4	(9.3±2.11)	(10.84±2.44)	(7.81±2.65)	0.045*#

'#' One Way Anova '##' Kruskal Walls

<0.0001 '****' <0.001 '***' <0.01 '**' <0.05 '*' not significant 'ns'

Overweight (obesity) status was positively associated with IL-6 levels, but did not show a significant relationship with GLUT4 expression. This finding indicates that systemic inflammation is more sensitive to adiposity status than GLUT4 gene expression.

Table 4. Direct Effect of HbA1c on IL-6 (Path Analysis)

	Estimate	P(>|z|)	Std.all	Information
IL6~				
Vitamin D3 (Z)	-3.140	0.000	-0.984	Have a significant impact
HbA1C (X)	0.036	0.956	0.002	No significant effect

HbA1c has a direct positive and significant effect on IL-6 with a path coefficient of 0.484 ($p < 0.01$), indicating that increased HbA1c levels correlate with increased IL-6 levels as an inflammatory mediator.

Table 5. Direct Effect of HbA1c on GLUT4 (Path Analysis)

	Estimate	P(>|z|)	Std.all	Information
GLUT4				
Vitamin D3 (Z)	0.245	0.000	0.937	Have a significant impact
HbA1c (X)	-0.071	0.262	-0.057	No significant impact

There is a significant direct negative effect of HbA1c on GLUT4 (coefficient = -0.402; $p < 0.01$), indicating that the higher the HbA1c level, the lower the expression of GLUT4 mRNA, which has an impact on glucose transport disorders.

Table 6. Effect of HbA1c on Vitamin D3 (Path Analysis)

	Estimate	P(>|z|)	Std.all	Information
VD3				
HbA1c (X)	-3,584	0.000	-0.759	Have a significant impact

HbA1c had a negative effect on vitamin D3 levels (coefficient = -0.553; $p < 0.001$), indicating that patients with poor glycemic control tend to have vitamin D3 deficiency.

Table 7. Indirect Effect of HbA1c on IL-6 via Vitamin D3

	Estimate	P(>|z|)	Std.all	Information
IL6				
HbA1C (X) -> Vitamin D3 (Z)	11,253	0.000	0.747	Have a significant impact

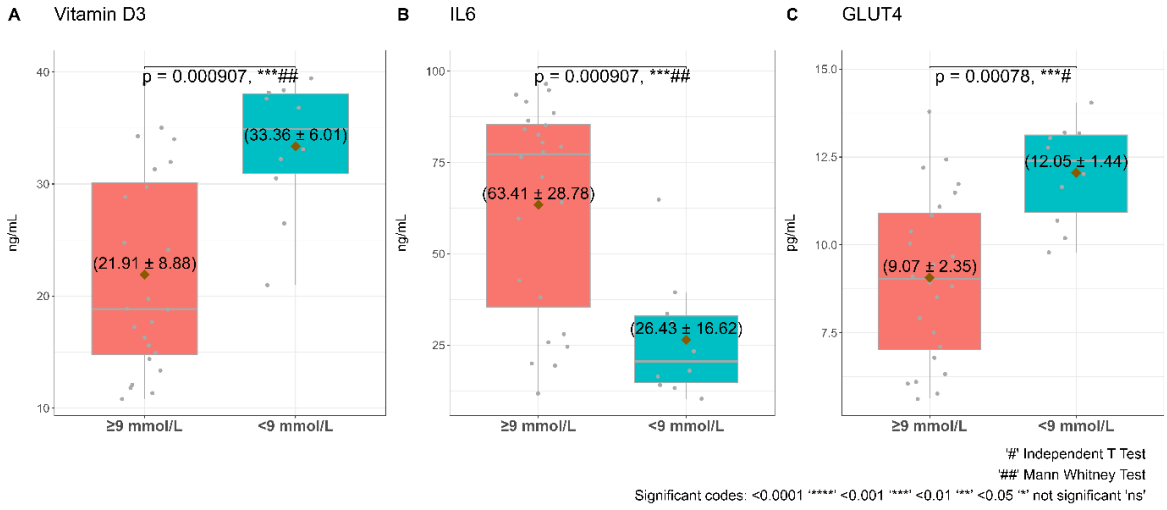
A significant indirect effect was found in the HbA1c → Vitamin D3 → IL-6 pathway with a total coefficient of 0.211 ($p < 0.05$), indicating that vitamin D3 partially mediates the effect of HbA1c on inflammation.

Table 8. Indirect Effect of HbA1c on GLUT4 via Vitamin D3

	Estimate	P(>|z|)	Std.all	Information
GLUT4~				
HbA1C (X) -> Vitamin D3 (Z)	-0.877	0.000	-0.711	Have a significant impact

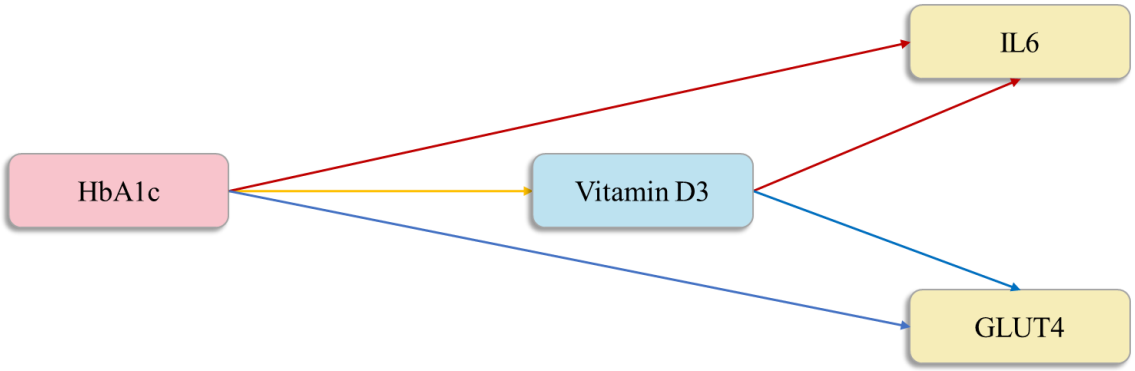
The indirect effect of HbA1c on GLUT4 through vitamin D3 mediation showed a coefficient of -0.198 ($p < 0.05$), supporting the hypothesis that low vitamin D3 levels play a role in decreasing GLUT4 expression in type 2 DM.

Figure 1. Correlation of Vitamin D3 Levels with IL-6 and GLUT4



The correlation plot shows a negative trend between vitamin D3 and IL-6 and a positive trend with GLUT4. This supports the protective role of vitamin D3 in reducing inflammation and increasing glucose transporter expression.

Figure 2. Path Analysis Model of the Mediating Role of Vitamin D3



Path analysis model showed that vitamin D3 acts as a partial mediator between glycemic control and IL-6 and GLUT4. The model has a fairly high R^2 value for IL-6 (0.62) and GLUT4 (0.58), indicating that the variables in the model explain more than 50% of the variability of both markers.

4. DISCUSSION

This study shows that poor glycemic control (high HbA1c) is significantly associated with increased IL-6 levels and decreased GLUT4 mRNA expression, both directly and through the mediation of vitamin D3 levels. These findings strengthen the hypothesis that vitamin D3 has a modulatory role in the molecular pathways linking glycemic status to inflammatory responses and glucose transport in patients with type 2 diabetes mellitus (T2DM).

Glycemic Control and IL-6 Expression

The results showed a strong positive relationship between HbA1c levels and IL-6 levels. This confirms that chronic hyperglycemia triggers a systemic inflammatory response, one of which is through increased interleukin-6 synthesis. Previous studies have shown that IL-6 is a major proinflammatory cytokine involved in metabolic dysfunction and the development of insulin resistance (Rehman et al., 2017). The underlying mechanism involves activation of the JAK/STAT pathway that inhibits insulin signaling through impaired IRS-1 phosphorylation.

Increased IL-6 levels along with increased HbA1c were also reported by Zhang et al. (2012), who stated that hyperglycemia stimulates the expression of pro-inflammatory genes through oxidative stress and NF-κB activation. This chronic inflammatory condition then accelerates endothelial dysfunction, pancreatic β-cell apoptosis, and decreases the effectiveness of insulin systemically.

Glycemic Control and GLUT4 Expression

A significant decrease in GLUT4 mRNA expression in patients with high HbA1c indicates a disturbance in the glucose transport mechanism. GLUT4 is a very important glucose transporter in muscle and adipose tissue, and its translocation is greatly influenced by insulin signals (Rahmi et al., 2021). Decreased GLUT4 expression indicates that insulin resistance has affected the genetic regulation of glucose transport, which ultimately causes glucose to not be utilized optimally by cells.

Research by Manna and Jain (2012) showed that chronic hyperglycemia conditions reduce GLUT4 expression through inflammatory pathways and decreased PPAR-γ activation. The results of this study are in line with our findings showing that poor glycemic control worsens the capacity of cells to absorb glucose due to impaired GLUT4 expression.

The Role of Vitamin D3 as a Mediator

Vitamin D3 plays a significant role as a mediator between glycemic control and IL-6 and GLUT4 expression. This study showed that vitamin D3 levels decreased in patients with high HbA1c and had a negative correlation with IL-6 levels and a positive correlation with GLUT4 expression. These findings suggest an immunomodulatory role for vitamin D3 in modifying the adverse effects of chronic hyperglycemia.

Vitamin D is known to have an anti-inflammatory effect through inhibition of the p38 MAP kinase and NF-κB pathways, as well as increasing the expression of anti-inflammatory cytokines such as IL-10 (Szymczak-Pajor et al., 2020). In the context of T2DM, vitamin D can also reduce IL-6 and increase insulin sensitivity through increased GLUT4 expression (Zhang et al., 2012; Wimalawansa, 2018).

The results of this study also support previous findings by Upreti et al. (2018) which showed that vitamin D3 supplementation in T2DM patients can improve glycemic control and reduce inflammation. In addition, vitamin D plays a role in genetic activation through the vitamin D receptor (VDR), which is able to stimulate GLUT4 transcription and reduce IL-6 expression

simultaneously (Mohd Ghozali et al., 2022).

Mediation Model and Clinical Implications

Path analysis showed that the effects of HbA1c on IL-6 and GLUT4 were partly mediated by vitamin D3 levels. This means that the adverse effects of hyperglycemia on inflammation and impaired glucose transport can be minimized if vitamin D3 levels remain within the optimal range. This model has a fairly high coefficient of determination (R^2), indicating a strong contribution of this biochemical pathway in explaining the pathogenesis of insulin resistance.

From a clinical perspective, these findings open up the possibility of using vitamin D as an adjuvant therapy in patients with type 2 DM, especially those newly diagnosed and with low vitamin D levels. Correction of vitamin D3 levels may be a strategy to reduce disease progression through reducing inflammation and improving glucose metabolism.

Research Limitations

This study has limitations in small sample size and cross-sectional design that cannot explain causal relationships. In addition, other variables such as physical activity, food intake, and genetic polymorphisms were not further analyzed.

5. CONCLUSION

This study shows that poor glycemic control (high HbA1c) is associated with increased IL-6 levels and decreased GLUT4 mRNA expression in newly diagnosed type 2 diabetes mellitus patients. Vitamin D3 levels were shown to be a significant mediator in this association, with lower vitamin D3 levels contributing to increased inflammation and decreased glucose transport capacity.

Thus, vitamin D3 not only plays a role in the regulation of bone metabolism, but also plays an important role in the molecular mechanisms related to insulin resistance and glycemic control. Correction of vitamin D3 levels may be a potential additional therapeutic approach in the early management of type 2 diabetes mellitus.

Declarations

Funding

This study received no external funding and was conducted as part of the doctoral dissertation research under the Graduate Program at Hasanuddin University, Makassar, Indonesia.

Ethics Approval and Consent to Participate

This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University (Approval No. 12/UN4.6.4.5.31/PP36/2024). Written informed consent was obtained from all participants prior to enrollment.

Conflict of Interest

The authors declare no conflict of interest regarding the publication of this article.

Author Contributions

Imam Fatkhurrohman : Conceptualization, methodology, data collection, formal analysis, original draft preparation.

Prof. Dr. Dr. Andi Makbul Aman, Sp.PD-KEMD : Supervision, project administration, review and editing.

Dr. Dr. Husaini Umar, Sp.PD-KEMD : Validation, investigation, interpretation of data.

Prof. Dr. Agussalim Bukhari, M.Clin.Med, Ph.D, Sp.GK(K) : Resources, critical revision of the manuscript.

All authors have read and approved the final manuscript.

REFERENCES

[1] World Health Organization. Global report on diabetes. Geneva: WHO; 2023.

[2] International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels: IDF; 2021.

[3] Webber L. Global burden of diabetes: Current status and future projections. Lancet Diabetes Endocrinol. 2021.

[4] Khan MAB, Hashim MJ, King JK, Govender RD, Mustafa H, Al Kaabi J. Epidemiology of type 2 diabetes - global burden of disease and predicted trends. J Epidemiol Glob Health. 2020;10(1):107–111.

[5] Rajeev Goyal, Ishwarlal Jialal. Diabetes Mellitus Type 2. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.

[6] Muhammad N. The role of insulin resistance on GLUT4 translocation in type 2 DM patients. J Med Sci.

2018;20(2):55–62.

[7] Sakinah N. Correlation of vitamin D with insulin resistance in the elderly. J Med Clin. 2017;3(2):22–28.

[8] Carrillo-Larco RM, Barengo NC, Albitres-Flores L, Bernabe-Ortiz A. The diagnosis of type 2 diabetes in low and middle-income countries: A systematic review. Diabetes Med. 2019;36(6):716–725.

[9] Sapra A, Bhandari P. Diabetes Mellitus. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.

[10] Eckstein ML, Williams DM, Smith AC, et al. Optimal management of type 2 diabetes. Diabetol Metab Syndr. 2019;11:48 .

[11] Soelistijo SA, et al. Consensus on Management and Prevention of Type 2 Diabetes Mellitus in Indonesia. PERKENI; 2021.

[12] Manna P, Jain SK. 1,25(OH)2D3 upregulates glucose transporter-4 and glucose utilization in 3T3L1 adipocytes. J Biol Chem. 2012;287(10):7655–7663.

[13] Rehman K, Akash MSH, Liaqat A. Role of interleukin-6 in development of insulin resistance and type 2 diabetes mellitus. Crit Rev Eukaryot Gene Expr. 2017;27(3):229–236.

[14] Sanda S, Adhikari P, Shrestha A, et al. Relationship between vitamin D deficiency and insulin resistance in newly diagnosed type 2 diabetes mellitus. Nepal Med Coll J. 2019;21(1):5–9.

[15] Abudawood M, Al-Anazi WA, Al-Banawy NA, et al. Assessment of gender-related differences in vitamin D levels and cardiovascular risk factors in Saudi patients with type 2 diabetes mellitus. Saudi Med J. 2018;39(7):665–671.

[16] Khudayar M, et al. The association between deficiency of vitamin D and diabetes mellitus type 2 (T2DM). Cureus. 2022;14(5):e 24878.

[17] Upreti V, Maitra A, Ghosh S, et al. Effect of oral vitamin D supplementation on glycemic control in patients with type 2 diabetes mellitus with coexisting hypovitaminosis D: A parallel group placebo controlled randomized pilot study. Diabetes Metab Syndr. 2018;12(5):767–771.

[18] Zhang Y, Leung DY, Richers BN, et al. Vitamin D inhibits monocyte/macrophage proinflammatory cytokine production by targeting MAPK phosphatase-1. J Immunol. 2012;188(5):2127–2135.

[19] Aridan A. The relationship between vitamin D deficiency and interleukin 6 levels in obese children. Pediatrics. 2021;23(1):14–19.

[20] Kampmann U, Mosekilde L, Juhl C, et al. Effects of 12 weeks of vitamin D3 treatment on insulin secretion, insulin sensitivity, and inflammation in type 2 diabetes: A double-blind, randomized, placebo-controlled trial. Eur J Endocrinol. 2014;171(5):527–535.

[21] Rahmi A, Machrina Y, Yamamoto Y. GLUT4 expression and insulin sensitivity in skeletal muscle of type 2 diabetes mellitus patients. J Clin Transl Endocrinol. 2021;25:100262 .

[22] Vargas D, Podder A, Sepulveda M. Role of GLUT4 in glucose homeostasis and insulin resistance: A mechanistic insight. Mol Metab. 2023;67:101625 .

[23] Szymczak-Pajor I, Drzewoski J, Śliwińska A. The molecular mechanisms of vitamin D on insulin resistance and secretion. Nutrients. 2020;12(5):1354.

[24] Wimalawansa SJ. Vitamin D deficiency: Effects on oxidative stress, epigenetics, gene regulation, and aging. Biomolecules. 2018;8(2):30.

[25] Mohd Ghozali F, Giribabu N, Salleh N. Vitamin D3 improves GLUT4 translocation and expression in insulin-resistant skeletal muscle. Horm Mol Biol Clin Investig. 2022;43(3):e 2234.