



# The Effect of Vitamin D Deficiency and Thyroid Hormone on Regulating Blood Glucose Levels in Type II Diabetic Patients in Salaheddin Hospital, Tripoli

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## ABSTRACT

Blood glucose levels rise as insulin secretion declines, which is regarded as a major problem for numerous medical disorders. Diabetes patients' inability to control their blood glucose levels is also attributed to thyroid malfunction and vitamin D deficiency. This study aimed to confirm the possible interaction of these three crucial factors: patients' blood glucose regulation, thyroid dysfunction, and vitamin D deficiency. A descriptive analysis test was performed. The study tested a cohort of hospital visitors from February 2023 to April 2024. One hundred samples of men and women, all older than thirty, were tested. The IBM SPSS Statistics 26 (SPSS IBM) program was utilized to assess the study's results. The variables' Pearson correlation coefficient was ascertained, and the sigma test ( $\sigma$ ) was computed to assess the quality of the different datasets. Results revealed that out of the 100 patients, 42% had vitamin D insufficiency, 10% had HbA1c, and 30% had an imbalance in thyroid hormones. From the correlation coefficient test, the study concluded that there is no systematic linear association between vitamin D3 and the thyroid hormones. Additionally, there is no systematic linear relationship between vitamin D3 and T3 or T4. However, according to the results, there was a statistically significant correlation between Ca and vitamin D3 and between HbA1c and TSH. However, HbA1c does not systematically correlate with T3, T4, or Ca levels. In conclusion, results demonstrate that low vitamin D has no discernible impact on thyroid hormones, which may exacerbate the symptoms of diabetes.

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## INTRODUCTION

Recent medical research has focused on how various physiological factors, particularly vitamin D deficiency and thyroid dysfunction, influence metabolic homeostasis and blood glucose regulation in patients, thereby highlighting their potential role in diabetes mellitus. Vitamin D, known for its importance in calcium homeostasis and bone health, has also been linked to glucose metabolism, insulin sensitivity, and pancreatic beta-cell function [1]. The relationship between vitamin D deficiency and insulin resistance may involve inherited genetic polymorphisms, including those in the vitamin D-binding protein, vitamin D receptor, and vitamin D 1 alpha-hydroxylase genes. Furthermore, vitamin D is thought to play a role in immune system regulation, inflammation, and glucose homeostasis, possibly influenced by low calcium levels, obesity, or elevated parathyroid hormone levels [1,2]. Epidemiological studies have noted a higher prevalence of vitamin D deficiency among individuals with diabetes. In addition, thyroid hormones are critical in

managing metabolism, energy expenditure, and substrate utilization, with dysfunction leading to metabolic disturbances that may impact glucose homeostasis. Despite the significance of vitamin D deficiency and thyroid dysfunction, their combined effects on blood glucose regulation remain insufficiently explored. Research into the interaction between these factors may uncover important insights [3]. Additionally, understanding these elements may lead to more effective management strategies for metabolic disorders and reduce associated healthcare costs [4]. A thorough review of the mechanisms involved in blood glucose regulation by insulin and the implications of dysregulation has been discussed, addressing vitamin D's roles and the consequences of deficiency [5]. Numerous studies have highlighted associations between vitamin D deficiency and blood glucose levels. Some clinical trials suggest that vitamin D supplementation may positively influence glucose control [6]. Research indicates that vitamin D enhances insulin sensitivity and

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beta-cell function, affecting two key abnormalities in type 2 diabetes: impaired insulin secretion and insulin resistance [7]. The interplay between vitamin D deficiency and type 2 diabetes pathogenesis has been emphasized, as low levels of vitamin D have been linked to increased risk for insulin resistance [8].

Type 2 diabetes manifests as elevated blood glucose levels due to insulin-secretion impairment and insulin resistance, leading to chronic complications if not managed effectively [9]. Timely intervention is essential to maintain glucose levels and prevent complications [10]. Cross-sectional studies since the 1980s have associated lower vitamin D levels with increased type 2 diabetes prevalence, and initial findings indicate that vitamin D may benefit insulin sensitivity in older adults. Additionally, a significant inverse correlation exists between serum 25(OH)D levels and type 2 diabetes prevalence across diverse ethnic populations [11].

Experimental evidence further supports vitamin D's role in insulin sensitivity and secretion, with observations of impaired glucose tolerance in high-vitamin-D diets and deficient vitamin D receptors in knockout mice [12]. Although observational studies suggest a link, randomized trials for vitamin D supplementation in type 2 diabetes are lacking, indicating a need for further intermediate investigations [13,14]. Type 2 diabetes is a rising global health challenge, estimated to affect 171 million people by 2000, with projections for this number to double by 2030 [15]. Understanding diabetes prevention through the regulation of glucose levels and the effects of vitamin D is crucial, especially given the rise in cases observed in adolescents and its associated costs to healthcare [16]. Additionally, high glucose intake may negatively affect vitamin D metabolism, while evidence suggests a role for vitamin D supplementation in mitigating cardiovascular disease and improving blood glucose levels [17].

Thyroid hormones significantly impact insulin and glucose regulation, with both hypo- and hyperthyroidism contributing to disrupted glucose homeostasis, particularly in populations prone to marginal thyroid dysfunction and type 2 diabetes [18]. Vitamin D insufficiency impacts over half the population, with implications for various health issues, including autoimmune diseases and cardiovascular conditions [19-21]. Insulin resistance, often related to vitamin D deficiency, is also linked to conditions such as obesity and metabolic syndrome, emphasizing the need for further understanding of the connections among these disorders [11,13,28]. Ultimately, while clinical studies reveal reductions in metabolic parameters with vitamin D supplementation, the specific levels required for

glycemic improvement remain to be fully defined [18].

Recent medical research has increasingly focused on the interplay of physiological factors, particularly vitamin D deficiency and thyroid dysfunction, in influencing metabolic homeostasis and blood glucose regulation. Vitamin D, traditionally known for its role in calcium homeostasis and bone health, has also been implicated in glucose metabolism, insulin sensitivity, and pancreatic beta-cell function [1]. Its deficiency may relate to genetic variations in the vitamin D-binding protein, vitamin D receptor, and vitamin D 1 alpha-hydroxylase genes. Additionally, vitamin D is believed to modulate immune functions and inflammation, which can further impact glucose homeostasis through pathways influenced by factors such as calcium levels and obesity [1]. Epidemiological studies have highlighted a higher prevalence of vitamin D deficiency among diabetic individuals. Thyroid hormones also play a crucial role in metabolism, energy regulation, and substrate utilization. Dysfunction in the thyroid, whether through hypothyroidism or hyperthyroidism, can disrupt glucose control, making its relationship with diabetes significant. However, research has yet to thoroughly explore how vitamin D deficiency and thyroid dysfunction overlap in their effects on blood glucose regulation [10]. A greater understanding of these interactions may inform better management of metabolic disorders and reduce healthcare costs [26]. Multiple studies have found links between vitamin D levels and blood glucose regulation, with some clinical trials indicating that vitamin D supplementation can positively influence glucose control [5]. Vitamin D's role in enhancing insulin sensitivity and improving beta-cell function highlights its importance in addressing two key abnormalities in type 2 diabetes: impaired insulin secretion and insulin resistance [8].

The connection between vitamin D deficiency and the risk of developing insulin resistance has gained recognition, adding to the understanding of type 2 diabetes pathology [5]. Chronic high blood glucose levels can lead to complications, underscoring the need for timely interventions to maintain blood glucose within safe limits [23, 24]. Since the 1980s, studies have indicated a correlation between lower vitamin D levels and increased type 2 diabetes prevalence, with evidence suggesting the benefits of vitamin D for older adults' insulin sensitivity. Additionally, there is a significant inverse relationship between serum 25(OH)D levels and type 2 diabetes prevalence across various ethnic groups [29]. While observational studies suggest a link between vitamin D and glycemic control, randomized trials assessing vitamin D supplementation specifically for type 2 diabetes

management remain limited, indicating a need for further investigation [25]. The growing global challenge of type 2 diabetes, affecting millions, necessitates a deeper understanding of how vitamin D regulation can aid in prevention and treatment [12]. Moreover, thyroid hormones significantly influence insulin and glucose management, with both hypo- and hyperthyroidism leading to disrupted glucose balance, especially in aging populations susceptible to thyroid dysfunction and diabetes [28]. Vitamin D insufficiency affects a significant portion of the population, with adverse implications for health, including autoimmune diseases and cardiovascular conditions [15, 16, 19, 22]. Furthermore, insulin resistance—often linked to vitamin D deficiency—correlates with obesity and metabolic syndrome, signaling a pressing need for understanding these relationships [11, 13, 29]. While clinical studies demonstrate reductions in metabolic parameters with vitamin D supplementation, the specific levels for optimal glycemic improvement remain to be clarified [18].

Diabetes mellitus is a chronic metabolic disorder described by diminished insulin secretion or action, resulting in elevated blood glucose levels. Despite progression in diabetes management, achieving optimal glycemic control remains a challenge for many patients. Emerging evidence suggests that vitamin D deficiency and thyroid dysfunction may contribute to the deregulation of glucose metabolism in diabetic individuals. However, the precise mechanisms underlying these associations and the potential therapeutic implications are not fully understood. As a result, the purpose of this study is to look into the relationship between vitamin D deficiency, thyroid dysfunction, and glucose regulation in diabetic patients, with the ultimate goal of gaining new insights into the pathophysiology of diabetes and identifying potential adjunctive therapeutic strategies to improve glycemic control and metabolic outcomes in this population.

This study was conducted to understand the potential relationships and interactions between three key factors: vitamin D deficiency, thyroid dysfunction, and blood glucose regulation. Also, to Assess the prevalence of vitamin D deficiency and thyroid dysfunction among diabetic patients within a specific population.

## Methods

### Study design

This study is a descriptive-analytics study. The study was performed in Salahuddin Hospital, Tripoli, Libya. Random samples from the hospital's ongoing records were analyzed to determine the extent to which vitamin D deficiency affects the functioning of the thyroid

glands or the development of diabetes from their analysis records.

### Time of the study

The study was conducted between February 2023 and April 2024.

### Study Setting

The study was based on examining a group of 100 diabetics over 30 years old males and females. Blood tests were conducted for them to determine the level of HbA1c, 25 OH vitamin (D), stimulating hormone (TSH) levels, T4 and T3, and Calcium (Ca) levels. All participants were tested from February 2023 to April 2024.

### Selection for Criteria

The inclusion criteria were as follows: Type 2 DM patients were tested for 25 OH vitamin D, HbA1c, free T4, free T3, thyroid-stimulating hormone (TSH) levels, and Ca. Among variables, HbA1c parameters, which are the most related to blood glucose regulation, were selected. Vitamin D was tested, and this test was linked to other tests.

### Statistical Analysis

When evaluating the findings obtained in the study, the IBM SPSS Statistics 26 (SPSS IBM) program was used. The Pearson correlation coefficient was determined between the variables. Six Sigma was also calculated to determine the quality of the various datasets. All variables had their prevalence and percentages assessed, as well as their arithmetic mean and standard deviation.

Six Sigma was calculated. The term "Six Sigma" refers to a statistical concept that measures how far a given process deviates from perfection. In a Six Sigma process, the goal is to have no more than 3.4 defects per million opportunities. This level of quality corresponds to achieving a process capability level of  $6\sigma$  (sigma), where  $\sigma$  is the standard deviation of the process.

## Results and Discussion

The study addresses an important gap in understanding the multifactorial nature of type 2 diabetes management. The project aimed to investigate the roles of vitamin D deficiency and thyroid dysfunction in type 2 diabetes. After analyzing the various results of vitamin D and linking them to cases of diabetes, it was discovered that there is no substantial relationship between vitamin D deficiency and diabetes, which is consistent with much prior research.

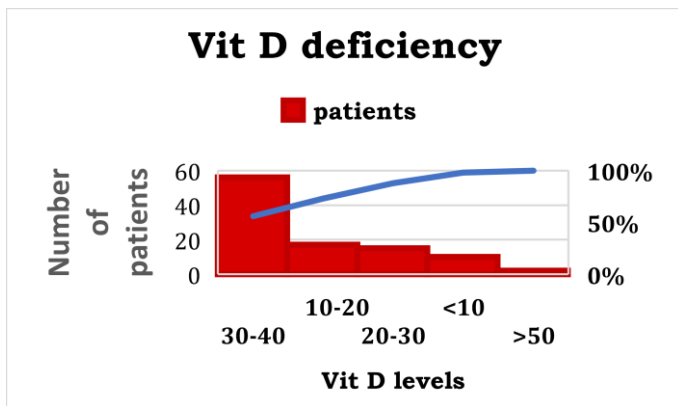
The total number of samples in the questionnaire was 100, including 29 males and 71 females with type 2 diabetes. All were over 30 years old as in Table (1).

The normal range of Vit D is 30 to 50 ng/mL (75 to 125 nmol/L). Vitamin D deficiency may be categorized: as Mild deficiency: Less than 20

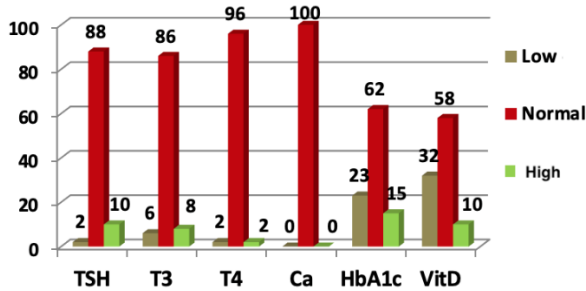
nanograms per milliliter. Moderate deficiency: Less than 10 nanograms per milliliter. Severe deficiency: Less than 5 nanograms per milliliter. Among the study's 100 samples, the number of patients with vitamin D moderate deficiency < 10 nmol/L was (10%), the number of patients with vitamin D mild deficiency was (32%) < 20 nmol/L, and the number of people who did not suffer from vitamin D deficiency 20-50 nmol/L was (58%) as in Figure (1, 2).

**Table 1. The number of cases in different age periods.**

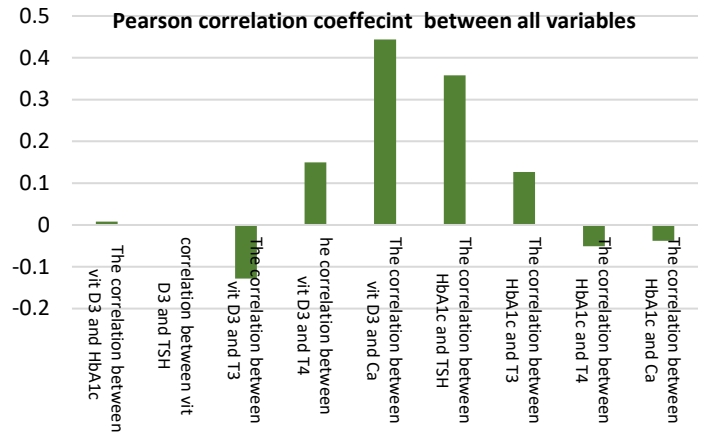
Percentage	Cases	Gender		Ages
		Male	Female	
%23	23	0	23	40-30
%26	26	14	12	50-40
%24	24	10	14	60-50
%27	27	5	22	60>



**Figure 1. The result of the analysis of 25 OH vitamin D.**



**Figure 2. A blood test results for HbAc1, Vitamin D, Thyroid hormones TSH, T3, T4, and Calcium.**



**Figure 3. Pearson correlation coefficient between all variables**

The analysis presented in Figure 3 illustrates the correlation between vitamin D3 and HbA1c, as determined by the Pearson correlation coefficient, yielding a value of 0.0078. This result is well below the tabulated Pearson value of 0.205, indicating a lack of a systematic linear relationship between these two variables.

This study addresses a significant gap in our understanding of the multifactorial aspects of managing type 2 diabetes. The objective was to explore the roles of vitamin D deficiency and thyroid dysfunction in the context of type 2 diabetes. A comprehensive analysis of vitamin D levels in conjunction with diabetes cases revealed no substantial relationship between vitamin D deficiency and diabetes, aligning with findings from previous research [2].

In exploring the correlation between vitamin D3 and thyroid-stimulating hormone (TSH), the calculated Pearson value of -0.0728 again falls below the tabulated threshold of 0.205, confirming the absence of a significant linear relationship. These results corroborate similar findings from studies conducted by Billic-Komarica and Beciragic in 2012 and Elgazar in 2019 [2, 8]. Notably, the findings indicate no association between vitamin D levels and thyroid hormones, reaffirming that a deficiency in vitamin D does not significantly impact thyroid function, consistent with earlier research by Chahardoli et al. in 2019 [3].

The correlation between vitamin D3 and triiodothyronine (T3) produced a Pearson value of -0.1279, which is also smaller than the tabulated Pearson value of 0.205, further validating the null hypothesis of no systematic linear relationship between the two variables. Additionally, the Pearson value for the correlation between vitamin D3 and thyroxine (T4) was found to be 0.15, remaining below the tabulated value of 0.205, leading to acceptance of the null hypothesis once more, in agreement with Chahardoli's 2019 research [3].

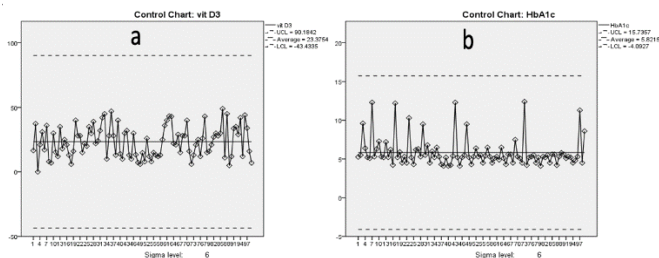
Conversely, the computed Pearson value for the correlation between vitamin D3 and calcium (Ca) was 0.4439, surpassing the tabulated value



of 0.205 and indicating a statistically significant relationship between these two variables. This finding aligns with studies by Billic-Komarica, Beciragic, and Junuzovic in 2012, as well as Elgazar in 2019 [2, 8].

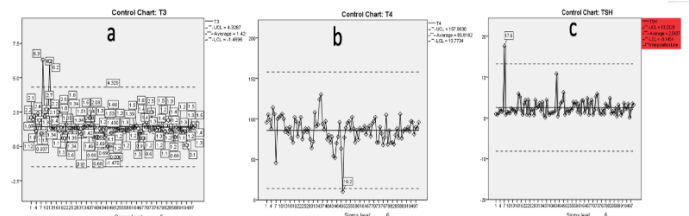
Furthermore, we observed that the calculated Pearson value for the correlation between HbA1c and TSH was 0.3581, which exceeds the tabulated value of 0.205. Consequently, we reject the null hypothesis and support the alternative hypothesis, which posits a statistically significant connection between these two variables. This study reveals a robust positive correlation between thyroid hormones and diabetes, as evidenced by significant Pearson coefficient values relating HbA1c to TSH and T3. These results underscore the consequential relationship between T3 and TSH levels in the context of diabetes, consistent with prior studies by Billic-Komarica, Beciragic, and Junuzovic in 2012 and Elgazar et al. in 2019 [2, 8].

The computed Pearson value for the correlation between HbA1c and T is 0.1267, which is less than the tabulated Pearson value of 0.205, indicating no systematic linear relationship between these two variables. Our results suggest a notable relationship between goiter and diabetes, particularly through the significant association observed between HbA1c and T3, supported by research from Billic-Komarica, Beciragic, and Junuzovic 2012, and Elgazar et al. 2019 [2, 8]. Similarly, the computed Pearson value for the correlation between HbA1c and T4 was recorded at -0.0509, again falling below the tabulated threshold of 0.205, further substantiating the lack of a systematic linear relationship. Lastly, the computed Pearson value for the correlation between HbA1c and calcium was -0.0375, remaining below the tabulated value of 0.205, which indicates no significant linear relationship between these two variables, thus reinforcing the findings reported in studies by Billic-Komarica, Beciragic, and Junuzovic 2012, as well as Elgazar et al. 2019.



**Figure 4. Six Sigma for: a: vitamin D3, and b: HbA1c data.**

Figure 4, which displays the 6 sigma values obtained using the SPSS program, all presented values were found naturally, with no value deviating from normal, indicating the quality of the vitamin D3 data and HbA1c data,



**Figure 5. Six Sigma a (T3) data, Six Sigma b (T4), and Six Sigma c (TSH) data.**

The results in Figure 5 represent the six sigma values generated with the SPSS program, indicating a divergence from the data's normal range. We also see that the values 6.3 and 6.2 have surpassed the maximum value, 5.4, indicating a flaw in the T3 data, as in Figure (5-a). The graph above represents the six sigma values generated with the SPSS program, indicating a divergence from the data's normal range. We also see that the value of 10.2 has surpassed the minimum value 10, indicating a flaw in the T4 data, as in Figure (5-b). The graph above shows the six sigma values generated by the SPSS programmer, which indicate a deviation from the data's normal range. We may also notice that the figure of 17.6 exceeds the maximum value 12, indicating a problem in the TSH data, as in Figure (5-c).

**Conclusion**

The issue delves into the intricate interaction between vitamin D insufficiency, thyroid dysfunction, and their combined effect on blood glucose management in people with type 2 diabetes. The findings show that vitamin D shortage has no clear impact on thyroid hormones, which play a crucial role in regulating blood glucose levels and may worsen diabetic symptoms. Understanding these interactions can reveal new factors that influence diabetes care in addition to traditional risk factors. Studying the relationship between vitamin D, thyroid function, and blood glucose levels has allowed us to develop innovative techniques for controlling type 2 diabetes and improving patient outcomes.

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**Conflicts of Interest**

All authors declare that they have no conflicts of interest.

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