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# An Imbalance of Calcium and Vitamin D Increases the Risk of Type 2 Diabetes (Review Article)

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**ABSTRACT**

***Background:*** *The relationship between vitamin D levels as well as calcium consumption together with their impact on type 2 diabetes (T2D) development has become a subject of increasing scientific investigation during the past few years. Researchers have begun studying vitamin D as it extends beyond its established function to maintain bone health into multiple body systems which include its capacity to control glucose metabolism. The connection between these two elements operates through multiple channels which include insulin secretion control by pancreatic beta cells together with changes in peripheral tissue insulin sensitivity and the body's response to ongoing low-grade systemic inflammation. Multiple observational studies together with various detailed meta-analysis studies have found that higher levels of 25-hydroxyvitamin D in circulation help to decrease the chances of developing T2D. The evidence from randomized controlled trials which serve as the ultimate method to demonstrate causation remains unclear. The clinical trials which tested different vitamin D supplementation doses failed to show a constant protective effect against developing T2D which leaves the hypothesis without confirmation. Researchers still need to identify the 25-hydroxyvitamin D concentration which most effectively controls blood sugar levels and reduces diabetes development. Researchers need to establish these thresholds so they can convert observational research results into practical clinical guidelines.*

**Keywords: Diabetes mellitus, Complications, Calcium ($Ca^{2+}$), Vitamin D.**

**Article Information**

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## 1. INTRODUCTION

### 1.1 Diabetes mellitus

Diabetes mellitus (DM) is recognized as one of the most prevalent endocrine disorders worldwide, affecting more than 100 million people which accounts for about 6% of the global population. (1). DM is broadly classified into two major types insulin-dependent diabetes mellitus IDDM and non-insulin-dependent diabetes mellitus NIDDM which is commonly known as Type II diabetes.(2). Patients with diabetes mellitus face increased chances of experiencing multiple severe health problems. The health risks include cardiovascular diseases and peripheral vascular disorders and stroke and diabetic neuropathy and chronic kidney disease which leads to renal failure and retinopathy which results in vision loss or blindness and in advanced cases of the disease patients require limb amputations due to poor circulation and infection (3). The complications of this disease lead to higher rates of both illness and death throughout the


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population.The main objective of diabetes treatment exists to extend patient lives while diminishing their symptoms and sustaining their blood sugar levels within healthy limits. (4). People with diabetes can use different medications to achieve better blood sugar control, which work alongside their dietary changes. The medical field uses biguanides and sulfonylureas as oral hypoglycemic medications, which doctors frequently prescribe to their patients. (5)Patients must take antidiabetic medications for their entire life. (6).

**1.2 Complications**

The metabolic disturbances which persist throughout diabetes mellitus progression cause damage to body tissues and blood vessels, which leads to multiple severe complications that become permanent. The process starts when chronic hyperglycemia causes blood vessels to undergo structural and functional transformations which affect both their small and large components. Patients with diabetes may experience debilitating conditions, which include diabetic retinopathy, neuropathy, nephropathy, cardiovascular disorders, and chronic lower extremity ulcers. The complications reduce quality of life while people experience greater health risks which lead to increased mortality rates (7,8).

**1.3 Calcium (Ca2+)**

Calcium ($Ca^{2+}$) serves as an essential mineral for eukaryotic cells because it maintains their typical cellular structure and operational activities [9].The substance operates as an adaptable intracellular signaling entity which controls multiple processes in biological systems. The cell requires precise control of intracellular $Ca^{2+}$ signaling because it uses this mechanism to manage critical pathways while maintaining flexible cellular responses to physiological stimuli [10]. Calcium ions are critical for all essential physiological functions which occur throughout different tissues and organ systems in the human body [11].

$Ca^{2+}$ functions as a crucial element which regulates gene expression together with cellular growth and differentiation processes.[12]. Muscle cells use calcium cycling as their main process which requires their entire system of specialized proteins to function. [13]. T2DM development has been linked to disturbances in calcium homeostasis which result from cell as well as tissue failures to manage intracellular $Ca^{2+}$ levels. [14]. The oxidative stress (OS) condition of cellular components causes this imbalance which results in greater disease progression [11–15–16].

**1.4 Vitamin D**

The scientists Goldblatt and Soames discovered and scientifically described vitamin D in 1923 which established a significant milestone for nutritional research and medical investigations. The vitamin functions as a fat-soluble substance which maintains human health through its ability to be produced by the body after sunlight exposure [17]. The skin produces vitamin D through a photochemical reaction which occurs when UV radiation via the sun interacts with the skin because most other vitamins need to be consumed through food. Vitamin D exists in two main forms: $VD_2$ and $VD_3$. The production of vitamin $D_2$ occurs through the industrial irradiation of plant-derived sterols which includes the use of ergosterol to create supplements and fortified products. The body produces vitamin $D_3$ through the process of endogenously synthesizing it from the skin precursor 7-dehydrocholesterol when exposed to ultraviolet B (UVB) sunlight radiation. The body synthesizes $VD_3$ through natural processes but people can consume it through food products which include animal-based dietary sources.

 

### 1.5 The role of vitamin D in regulating blood sugar

The examination of vitamin D effects on glucose control and metabolic processes is necessary for investigating the link between vitamin D as well as diabetes development. Research demonstrates that vitamin D functions as a vital element which helps the body maintain blood sugar control by improving insulin sensitivity of body cells according to research findings [18]. Insulin sensitivity enhancement results in muscle and adipose tissue cells achieving better insulin response which enables them to absorb glucose from blood circulation. The pancreas produces insulin through its beta cells which maintains blood glucose levels within the normal range. Cells use glucose through this process which allows them to access energy while using insulin to create energy reserves for upcoming requirements [18].

### 1.6 The relationship between vitamin D as well as diabetes

The scientific and clinical communities have shown increased interest in studying vitamin D's potential role as a diabetes mellitus preventive measure and treatment method during the last few years. The growing interest in vitamin D deficiency exists because people now understand that this deficiency leads to an increased likelihood of developing both type 1 and type 2 diabetes [19]. Researchers have discovered that vitamin D affects both bone health and the metabolic pathways which control glucose levels and insulin operation. The scientific community has developed a stronger interest in vitamin D now because researchers spent two decades conducting extensive studies about vitamin D's health impacts. Researchers conducted observational studies and clinical studies and epidemiological studies during this time to investigate how diabetic patients vitamin D levels impacted their disease progression and diabetes development. The studies have uncovered significant knowledge about how vitamin D levels affect glucose metabolism as well as insulin sensitivity and metabolic health [19].

### 1.7 The relationship between calcium as well as vitamin D with diabetes

The evidence which scientists have gathered over time indicates that vitamin D and calcium balance disturbances lead to type 2 diabetes development (20). Researchers have established this hypothesis through different methods which include experimental studies on animal models that uncovered how vitamin D impacts glucose metabolism and insulin function (21, 22). Cross-sectional studies show that people who have low vitamin D levels develop diabetes with impaired glucose tolerance, which creates a link between vitamin D deficiency and disrupted glycemic control (23). Longitudinal observational studies provide evidence about the connection between type 2 diabetes development and vitamin D deficiency because these studies show that people with low vitamin D levels have an increased risk of developing diabetes. The findings establish that inadequate vitamin D status leads to metabolic abnormalities and serves as a gateway to disease development (24). Calcium exists as an essential element which scientists believe plays a role in developing type 2 diabetes through indirect mechanisms. Cross-sectional studies demonstrate that people who consume more calcium experience weight loss because body weight operates as an insulin resistance and diabetes risk factor (25–26). Longitudinal observational studies show that people who consume more dietary calcium experience a decreased risk of developing type 2 diabetes, which creates evidence for calcium's protective effect (27, 28).

## 2. METHODOLOGY

A thorough examination of the MEDLINE database was performed to discover English-language studies which investigated the connection between vitamin D and calcium levels and type 2 diabetes mellitus (T2DM). The review included observational studies which measured vitamin D and calcium levels through serum 25-OHD concentrations and dietary vitamin D and calcium and dairy product consumption. The studies investigated T2DM occurrence and development across various population groups. The research included randomized controlled trials which studied non-pregnant adults to determine how vitamin D and calcium supplementation affected glucose homeostasis and metabolic regulation.The analysis needed to maintain evidence quality required certain publication types to be excluded from analysis. The analysis excluded letters abstracts as well as conference proceedings which had not been completely published in scientific journals that underwent peer review (29).

## 3. RESULTS

### 3.1 Pancreatic Beta Cell Function

The scientific evidence shows that vitamin D functions as a vital component for healthy pancreatic beta cells that produce and release insulin. Vitamin D enhances insulin response during glucose stimulation because it enables beta cells to increase their insulin production when blood glucose levels rise according to research findings. The study indicates that vitamin D does not significantly impact resting insulin levels that occur during baseline conditions (30,31). The active form of vitamin D known as 1,25-dihydroxyvitamin D (1,25-OHD) directly influences beta cell activity by binding to vitamin D receptors that exist on pancreatic beta cells. The mechanism works through receptor binding of vitamin D, which enables the vitamin to activate its target proteins that control insulin secretion through gene expression regulation. The enzyme 1-alpha-hydroxylase, which is present in beta cells, enables the local activation of vitamin D within those cells, as vitamin D exists in its active form. The research shows that beta cells can convert vitamin D from its inactive state into its active form, which allows them to control vitamin D function within their immediate environment (32). Vitamin D affects insulin secretion through direct mechanisms by interrupting insulin secretion processes, which require the vitamin D to control calcium levels. The substance regulates calcium levels that exist outside the cell while it affects how calcium moves through cell membranes, which includes pancreatic beta cells. Beta-cell function relies on calcium signaling because insulin secretion depends on calcium presence (33). The research studies conducted on various populations have produced results that remain inconsistent. The research conducted on certain cohorts found that vitamin D deficiency leads to decreased insulin secretion after glucose consumption (34–35), while other studies showed no correlation (36,37). There has been a lack of agreement among intervention studies, which have produced different findings. The results of multiple randomized controlled trials with short durations, which tested vitamin D supplementation, demonstrated that the treatment improved insulin secretion (34,35,37,38). The results showed no significant change (35,37,39).

### 3.2 Insulin Resistance

The beneficial effects of vitamin D on insulin function occur through various biological pathways active in the body. The direct effects of vitamin D enable cells to operate better through insulin receptors which create better cell functions to handle insulin together with glucose intake (40). Calcium functions as an essential element for insulin-responsive tissues which include skeletal muscle as well as adipose tissue because it

 

enables the insulin binding process to trigger intracellular activities (41–42). The processes which depend on these specific calcium levels need extremely precise control because even tiny changes to the optimal [$Ca^{2+}$] range will block insulin functions (43). Insulin resistance develops when primary insulin target tissues experience disruptions in their intracellular calcium levels ([$Ca^{2+}$]i) (54–56). Insulin signal transduction pathway failures (42,45) produce this effect because they block glucose transporter type 4 (GLUT-4) activity which delivers glucose into cells (44,46). Epidemiological evidence shows that there exists a connection between vitamin D status and insulin sensitivity. The body of research via multiple cross-sectional studies shows that low vitamin D levels lead to insulin sensitivity reduction (59–37,47,48). The observational studies show that participants who have high vitamin D or calcium levels display lower insulin resistance (50,48). The connection between vitamin D and calcium intake to insulin resistance does not appear in every study (48) because different research populations and methodologies produce different results. The research studies which executed randomized controlled trials to explore how vitamin D or calcium supplements affect insulin resistance produced results that lack consistency. The studies demonstrated no positive effects on insulin sensitivity after vitamin D supplementation (37,51,52) but the research showed some benefits which included better insulin performance together with lower insulin resistance (53,54).

### 3.3 Inflammation

The scientific community now recognizes that T2DM develops from a combination of genetic risk factors and ongoing low-level inflammation throughout the body(55,56,57). The development of insulin resistance stems from the disease process which involves this type of inflammatory response. The body continues to produce high levels of inflammatory mediators such as cytokines which disrupt insulin signaling pathways, leading to diminished cellular responsiveness toward insulin. The increased production of pro-inflammatory cytokines causes insulin resistance which also leads to pancreatic beta cell damage through cellular stress induction and apoptosis induction, thereby hindering insulin production. Vitamin D functions through these processes to decrease the strength of inflammatory reactions which then protect both insulin function and beta cell viability from harm. (54,58-59).

## 4. DISCUSSION

Researchers have studied vitamin D supplementation together with calcium and found that this treatment improves metabolic function in adults who have a high risk of developing type 2 diabetes. Vitamin D intake linked to better disposition index which measures insulin secretion together with insulin sensitivity while it also improves insulin secretion. Research findings indicate that some studies have discovered a connection between vitamin D supplementation and decreased progression of HbA1c levels in diabetic patients who have higher diabetes risk. The metabolic characteristics of the studied group showed no significant improvement from the treatment which used calcium alone. The evidence suggests that vitamin D has a significant ability to delay the transition from prediabetes to full development of type 2 diabetes. The main pathological changes that occur in T2DM development show two key features which involve developing insulin resistance and losing pancreatic beta cell functionality. Experimental research which tested vitamin D effects through in vitro studies and in vivo studies demonstrates that this vitamin affects both essential operational pathways. Vitamin D deficiencies prevent

pancreatic beta cells from releasing insulin in response to glucose stimulation (60,61–63).

The research proves that vitamin D supplementation reverses all insulin secretion effects present in these models (60,62–64) because it controls beta cell activity. The active form of vitamin D 1,25-dihydroxyvitamin D binds to vitamin D receptors VDRs which beta cells express to produce its direct effects on pancreatic beta cells. The human insulin gene contains vitamin D response elements which serve as transcriptional control points for its expression as well as 1,25-dihydroxyvitamin D activates human insulin gene transcription. The findings present molecular evidence that vitamin D directly regulates both insulin production and release. Vitamin D exists as both active systemic vitamin D as well as locally active one which pancreatic beta cells produce through their 25-hydroxyvitamin D-1α-hydroxylase CYP27B1 enzyme activity (69).

The local activation mechanism shows evidence that vitamin D functions through autocrine and paracrine pathways inside the pancreatic gland. Beta-cell function receives indirect regulation from vitamin D because it manages calcium homeostasis which directly affects insulin secretion through calcium-dependent mechanisms. The calcium balance of a cell directly impacts its ability to release insulin and respond to beta-cell stimuli because insulin secretion depends on calcium availability (70). Vitamin D regulates extracellular calcium as well as intracellular calcium levels to create the necessary conditions for optimal insulin secretion. The active vitamin D metabolites present in peripheral insulin-sensitive tissues like skeletal muscle as well as adipose tissue can enhance insulin sensitivity through several distinct pathways. The pathways include insulin receptor expression upregulation (68), transcription factor activation for glucose metabolic and homeostatic processes (71).

## 5. CONCLUSION

The evidence from scientific studies shows that vitamin D maybe involved in developing type 2 diabetes mellitus (T2DM) as its scientific understanding grows. The different vitamin D status levels which result from deficiency and metabolic changes and biological action disruptions will affect major disease pathways that control insulin responsiveness and pancreatic beta-cell operational capacity.

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### 8. Conflicts of interest

There are no conflicts of interest.

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