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Vitamin D and Diabetes Mellitus: relationships with glycemic control and disease management

Vitamina D e Diabetes Mellitus: relações com o controle glicêmico e o manejo da doença
Vitamina D y Diabetes Mellitus: relaciones con el control glucémico y el manejo de la enfermedad

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Abstract: Diabetes Mellitus (DM) is a chronic metabolic disease characterized by changes in insulin secretion and/or action, resulting in persistent hyperglycemia and increased risk of microvascular and macrovascular complications. In this context, vitamin D has attracted scientific interest due to the presence of its receptors in different tissues, including β-pancreatic cells, and its possible roles in insulin secretion, insulin sensitivity, calcium metabolism, and the modulation of inflammatory processes. The present study aimed to analyze the scientific evidence on the relationship between vitamin D and Diabetes Mellitus, with emphasis on glycemic control and the possible implications for the management of the disease. This is a literature review with a qualitative and descriptive approach, based on the analysis of scientific articles, literature reviews, observational and experimental studies and clinical trials related to vitamin D, type 1 and type 2 diabetes mellitus, insulin resistance, β cell function, glycemia, glycated hemoglobin and vitamin D supplementation. The results of the literature indicate an association between lower serum concentrations of 25-hydroxyvitamin D [25(OH)D] and metabolic alterations, insulin resistance and worse glycemic profile in certain groups of individuals with type 2 Diabetes Mellitus. Physiological bases were also identified that support a possible participation of vitamin D in the function of pancreatic β cells and in the modulation of inflammatory and immunological mechanisms. However, the results regarding vitamin D supplementation on glycemia and glycated hemoglobin are heterogeneous, not allowing its use as an isolated treatment or substitute for conventional therapies. It is concluded that vitamin D may be a relevant complementary component of the metabolic assessment of individuals with diabetes mellitus in the presence of deficiency or insufficiency, but its causal relationship and therapeutic benefit for glycemic control still require investigation through more robust clinical studies.

Keywords: Vitamin D; Diabetes Mellitus; Glycemic control.

Resumo: O Diabetes Mellitus (DM) é uma doença metabólica crônica caracterizada por alterações na secreção e/ou na ação da insulina, resultando em hiperglicemia persistente e aumento do risco de complicações microvasculares e macrovasculares. Nesse contexto, a vitamina D tem despertado interesse científico devido à presença de seu receptor em diferentes tecidos, incluindo células β pancreáticas, e à sua possível participação na secreção de insulina, sensibilidade insulínica, metabolismo do cálcio e modulação de processos inflamatórios. O presente estudo teve como objetivo analisar as evidências científicas acerca da relação entre vitamina D e Diabetes Mellitus, com ênfase no controle glicêmico e nas possíveis implicações para o manejo da doença. Trata-se de uma revisão bibliográfica de abordagem qualitativa e caráter descritivo, baseada na análise de artigos científicos, revisões de literatura, estudos observacionais, experimentais e ensaios clínicos relacionados à vitamina D, Diabetes Mellitus tipo 1 e tipo 2, resistência à insulina, função das células β, glicemia, hemoglobina glicada e suplementação de vitamina D. Os resultados da literatura analisada indicam uma associação

entre menores concentrações séricas de 25-hidroxivitamina D [25(OH)D] e alterações metabólicas, resistência à insulina e pior perfil glicêmico em determinados grupos de indivíduos com Diabetes Mellitus tipo 2. Também foram identificadas bases fisiológicas que sustentam uma possível participação da vitamina D na função das células β pancreáticas e na modulação de mecanismos inflamatórios e imunológicos. Entretanto, os resultados referentes à suplementação de vitamina D sobre glicemia e hemoglobina glicada são heterogêneos, não permitindo estabelecer sua utilização como tratamento isolado ou substitutivo das terapias convencionais. Conclui-se que a vitamina D apresenta relevância potencial como componente complementar da avaliação metabólica de indivíduos com diabetes mellitus na presença de deficiência ou insuficiência, mas sua relação causal e seu benefício terapêutico sobre o controle glicêmico ainda requerem investigação por meio de estudos clínicos mais robustos.

Palavras-chave: Vitamina D; Diabetes Mellitus; Controle glicêmico.

Resumen: La Diabetes Mellitus (DM) es una enfermedad metabólica crónica caracterizada por alteraciones en la secreción y/o en la acción de la insulina, resultando en hiperglucemia persistente y aumento del riesgo de complicaciones microvasculares y macrovasculares. En ese contexto, la vitamina D ha despertado interés científico debido a la presencia de su receptor en diferentes tejidos, incluyendo células β pancreáticas, y a su posible participación en la secreción de insulina, sensibilidad insulínica, metabolismo del calcio y modulación de procesos inflamatorios. El presente estudio tuvo como objetivo analizar las evidencias científicas acerca de la relación entre vitamina D y Diabetes Mellitus, con énfasis en el control glucémico y en las posibles implicaciones para el manejo de la enfermedad. Se trata de una revisión bibliográfica de abordaje cualitativo y carácter descriptivo, basada en el análisis de artículos científicos, revisiones de literatura, estudios observacionales, experimentales y ensayos clínicos relacionados a la vitamina D, Diabetes Mellitus tipo 1 y tipo 2, resistencia a la insulina, función de las células β, glucemia, hemoglobina glucosa y suplementación de vitamina D. Los resultados de la literatura analizada indican una asociación entre menores concentraciones séricas de 25-hidroxivitamina D [25(OH)D] y alteraciones metabólicas, resistencia a la insulina y peor perfil glucémico en determinados grupos de individuos con Diabetes Mellitus tipo 2. También fueron identificadas bases fisiológicas que sustentan una posible participación de la vitamina D en la función de las células β pancreáticas y en la modulación de mecanismos inflamatorios e inmunológicos. Sin embargo, los resultados referentes a la suplementación de vitamina D sobre glucemia y hemoglobina glucosilada son heterogéneos, no permitiendo establecer su utilización como tratamiento aislado o substitutivo de las terapias convencionales. Se concluye que la vitamina D presenta relevancia potencial como componente complementario de la evaluación metabólica de individuos con diabetes mellitus en presencia de deficiencia o insuficiencia, pero su relación causal y su beneficio terapéutico sobre el control glucémico todavía requieren investigación por medio de estudios clínicos más robustos.

Palabras clave: Vitamina D; Diabetes Mellitus; Control glucémico.

1 Introduction

Diabetes Mellitus (DM) is an important global public health problem, characterized by persistent changes in glucose metabolism resulting from deficiency in insulin secretion, reduction of the action of this hormone or combination of both. Chronic hyperglycemia is

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associated with the progressive development of microvascular and macrovascular complications, compromising organs and systems such as kidneys, retina, nervous system, heart and blood vessels. Among the different clinical forms of the disease, Diabetes Mellitus type 1 (DM1) related to autoimmune destruction of pancreatic beta cells and consequent absolute insulin deficiency, and Diabetes Mellitus type 2 (DM2), characterized by insulin resistance associated with progressive deterioration of β cell function (Fonseca et al., 2026). Adequate management of the disease involves pharmacological and non-pharmacological measures aimed at glycemic control and reducing the risk of complications.

In this context, nutritional and metabolic factors capable of influencing glucose homeostasis have received increasing attention from the scientific community. Among these factors, vitamin D stands out, a steroidal hormone whose classical action is related to the regulation of calcium and phosphorus metabolism and to the maintenance of bone health. However, evidence accumulated over the past decades has shown that vitamin D performs broader biological functions, involving immunological, inflammatory and metabolic processes. The presence of vitamin D receptor (VDR) in different tissues, including pancreatic beta cells and immune system cells, supports the hypothesis that this vitamin may participate in mechanisms of regulation of glucose and pancreatic function (Barbosa et al., 2025).

Vitamin D can be obtained by means of skin synthesis induced by exposure to ultraviolet radiation B (UVB) and by food or supplement intake (Tavares; Rauber; Campagnolo, 2023). Vitamin D synthesized in the skin or obtained by diet is inactive and undergoes hydroxylation processes to acquire its functional form. In the liver, conversion to 25-hydroxyvitamin D [25(OH)D] occurs, the main circulating form and marker used to assess vitamin D nutritional status. In the kidneys, 25(OH)D is converted to 1.25-dihydroxyvitamin D [1.25(OH)2D], also called calcitriol, its active hormonal form. Calcitriol exerts its effects by binding to VDR, triggering genomic and non-genomic responses in different tissues.

The possible relationship between vitamin D and Diabetes Mellitus has been investigated due to the participation of this vitamin in mechanisms associated with insulin secretion and action. Pancreatic β cells express VDR and have enzymatic mechanisms involved in the local metabolism of vitamin D, which suggests a possible direct action on pancreatic function. In addition, vitamin D is related to calcium homeostasis, a fundamental element for several cellular processes, including those involved in the synthesis, processing and secretion of insulin by pancreatic β cells (Schavinski, 2024). Thus, changes in vitamin D metabolism may interfere with mechanisms related to the function of these cells.

Another possible mechanism involves insulin sensitivity and insulin resistance, relevant characteristics in the pathophysiology of DM2 (Mesquita et al., 2026). Experimental studies suggest that vitamin D may influence pathways related to glucose metabolism, insulin receptor activity, inflammation and function of β cells (Barbosa et al., 2025). Vitamin D deficiency has also been associated, in different populations, with higher prevalence of insulin resistance, changes in glucose tolerance and worse metabolic control. However, the nature of these associations is not established, since factors such as obesity, sedentary lifestyle, diet, age, sun exposure and the presence of chronic diseases may influence vitamin D levels and glycemic metabolism.

In type 1 Diabetes Mellitus, the investigation of vitamin D is also relevant due to the interaction between its immunomodulatory mechanisms and the autoimmune pathophysiology of the disease (Primon et al., 2026). The expression of VDR in immune cells and the participation of vitamin D in the modulation of the inflammatory response raise the possibility that adequate levels of this vitamin are related to the regulation of immunological processes involved in the aggression to pancreatic β cells. Although observational and experimental studies have identified associations between vitamin D deficiency and DM1, there is still insufficient evidence to establish the deficiency of this vitamin as a direct cause of the disease or to state that its supplementation is capable of preventing or modifying its evolution (Barbosa et al., 2025).

In DM2, in turn, the relationship between vitamin D and glycemic control has been investigated through studies evaluating serum levels of 25(OH)D, insulin resistance, fasting glucose, glycated hemoglobin (HbA1c) and other metabolic markers. Part of the literature suggests that individuals with lower vitamin D concentrations may present higher frequency of metabolic changes related to insulin resistance and inadequate glycemic control (De Souza et al., 2025). However, results from clinical trials and systematic reviews are heterogeneous regarding the effects of vitamin D supplementation on glycemia and HbA1c. This heterogeneity reinforces the need to interpret vitamin D as a possible component associated with glycemic metabolism, and not as a substitute for conventional strategies for treating diabetes.

In addition to its possible participation in β cell function and insulin sensitivity, vitamin D has also been related to modulation of inflammatory processes. Low-grade chronic inflammation has an important role in insulin resistance and in the progression of metabolic changes observed in DM2 (De Melo João; Rotterdam; Souto, 2025). In this sense, the immunomodulatory action of vitamin D is one of the possible pathways by which its nutritional status could be associated with glucose metabolism. However, the complexity of interactions

between vitamin D, adiposity, inflammation, energy metabolism and insulin resistance prevents the attribution of clinical effects to the serum concentration of this vitamin.

Given this scenario, the investigation of the relationship between vitamin D and diabetes mellitus is relevant from both the pathophysiological and clinical points of view. Understanding the possible association between vitamin D levels, insulin secretion and sensitivity and glycemic control indicators may contribute to a broader approach to metabolic factors involved in disease management. However, it is essential to distinguish epidemiological associations from causal relationships and to evaluate the available evidence on the benefits of vitamin D supplementation in individuals with diabetes.

Thus, the present study aims to analyze the scientific evidence on the relationship between vitamin D and Diabetes Mellitus, with emphasis on the possible mechanisms involved in glucose metabolism, secretion and insulin sensitivity, glycemic control and the implications of deficiency or vitamin D supplementation in disease management.

2 Theoretical review

2.1 Diabetes Mellitus: pathophysiological aspects and metabolic control

Diabetes Mellitus (DM) comprises a heterogeneous group of metabolic disorders characterized by the presence of hyperglycemia due to changes in insulin secretion, the action of this hormone or both mechanisms (Vieira et al., 2026). Prolonged maintenance of high blood glucose levels is associated with metabolic and vascular changes that may result in microvascular and macrovascular complications, compromising renal, ocular, neurological and cardiovascular function. Thus, glycemic control is one of the main objectives of clinical follow-up of individuals with diabetes.

The DM classification comprises different clinical forms, especially Diabetes Mellitus type 1 (DM1), Diabetes Mellitus type 2 (DM2), gestational diabetes mellitus and other specific forms related to genetic conditions, pancreatic diseases, endocrinopathies or the use of certain medications (Schwantz, 2025). DM1 is related to the destruction of pancreatic β cells by autoimmune mechanisms, resulting in absolute insulin deficiency. DM2, in turn, presents more complex pathophysiology, involving insulin action resistance, progressive changes in pancreatic β cell function and genetic, environmental and behavioral factors (Fonseca et al., 2026).

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In DM2, insulin resistance is one of the main mechanisms involved in the development of hyperglycaemia (Barreto et al., 2024). The body may compensate for the lower insulin sensitivity by increasing the secretion of this hormone by pancreatic β cells. With disease progression, however, this compensatory capacity may be insufficient, leading to progressive deterioration of pancreatic function and maintenance of hyperglycemia. Increased hepatic glucose production, changes in lipid metabolism and reduction of peripheral glucose use contribute to the metabolic imbalance characteristic of the disease (Prado; Ferreira, 2022).

The diagnosis and follow-up of diabetes are performed through laboratory parameters that assess glucose concentration and metabolic control. Among the main markers used are fasting glucose, glucose after oral glucose overload and glycated hemoglobin (HbA1c). HbA1c is important in clinical follow-up because it roughly reflects mean exposure to glucose over the previous months (Lima, 2024). Other tests, such as lipid profile assessment, renal function and albuminuria research, are used according to the patient's clinical condition and the need for investigation or follow-up of diabetes-related complications.

The occurrence and evolution of DM result from the interaction of multiple factors. In DM1, genetic predisposition associated with immunological mechanisms plays a relevant role in the destruction of pancreatic β cells (De Sousa Barbosa et al., 2026). Certain environmental factors may act as triggers or modulators of this process in susceptible individuals, although the etiology of the disease is multifactorial and cannot be attributed to a single factor.

In DM2, factors such as excess body weight, visceral adiposity, inadequate diet, sedentary lifestyle, aging and genetic predisposition are associated with an increased risk of developing the disease. Obesity is important in this context, since active adipose tissue participates in the release of free fatty acids and inflammatory mediators that can contribute to insulin resistance. Low-grade metabolic inflammation, in turn, is related to the deterioration of insulin signaling and the progression of metabolic changes (Antonini; Campos, 2025).

The change in lifestyle remains a fundamental component of DM2 prevention and management. The adoption of a balanced diet, adequate to individual needs, associated with regular physical activity, contributes to body weight control, improved insulin sensitivity and reduced cardiovascular risk factors. Physical activity also increases glucose uptake by skeletal muscle, including independent mechanisms of insulin action, contributing to the reduction of glycemia.

Although these strategies are the basis of non-pharmacological treatment, the complexity of DM has stimulated the investigation of other metabolic and nutritional factors associated with the development and control of the disease. Among them, vitamin D has

received increasing attention due to the presence of its receptors in tissues involved in metabolic regulation.

2.2 Vitamin D: metabolism and biological functions

Vitamin D is a steroidal hormone involved in the regulation of calcium and phosphorus homeostasis and in the maintenance of bone health. Its origin occurs by skin synthesis after exposure to ultraviolet B radiation, although it can also be obtained through diet and supplementation. The main forms are vitamin D2 (ergocalciferol) and vitamin D3 (colecalciferol), which require metabolic activation processes to perform their biological functions.

After it is obtained, vitamin D is transported to the liver, where it undergoes hydroxylation and is converted into 25-hydroxyvitamin D [25(OH)D]. This is the main circulating form of vitamin D and the biomarker most used to evaluate its state in the organism. 25(OH)D may undergo new hydroxylation in the kidneys, forming 1.25-dihydroxyvitamin D [1.25(OH)2D], or calcitriol, considered the active hormonal form.

The effects of calcitriol are mediated by the vitamin D receptor (VDR), present in several tissues (Santos and De Deus, 2026). The distribution of VDR beyond the tissues involved in mineral metabolism contributed to the expansion of research on the non-skeletal functions of vitamin D. Experimental and observational studies have investigated their participation in immunological, inflammatory, cardiovascular and metabolic processes, including mechanisms related to glucose homeostasis.

The possible relationship between vitamin D and glucose metabolism is based on the presence of VDR in cells and tissues involved in glucose regulation. Pancreatic β cells express VDR and have mechanisms capable of participating in local vitamin D metabolism. This characteristic led to the hypothesis that vitamin D can exert effects on pancreatic function and insulin secretion.

Adequate insulin secretion by β cells depends on cellular mechanisms in which calcium plays a fundamental role. As vitamin D participates in calcium homeostasis, changes in its metabolism may indirectly influence processes involved in insulin secretion. In addition, experimental studies suggest that the activation of VDR may participate in cellular mechanisms related to the function of β cells and the maintenance of their secretory activity.

Vitamin D has also been investigated in relation to insulin sensitivity. Insulin resistance involves complex changes in insulin receptor signaling and peripheral use of glucose in muscle

and adipose tissues, as well as increased hepatic glucose production. Experimental studies suggest that vitamin D may interfere in some of these pathways through mechanisms related to gene regulation, calcium metabolism and modulation of inflammatory processes (Santos and De Deus, 2025).

Another relevant aspect is the association between vitamin D deficiency, adiposity and inflammation. Obesity subjects have a higher frequency of reduced concentrations of 25(OH)D, a phenomenon that may be related to different factors, including changes in the distribution and availability of vitamin D in the body (Monteiro, 2026). Obesity is an important risk factor for insulin resistance and DM2. Thus, the association observed between low vitamin D levels and glycemic changes can be influenced by shared metabolic factors, which makes it difficult to establish a direct causal relationship.

2.3 Vitamin D deficiency and Diabetes Mellitus

Vitamin D deficiency or insufficiency has been investigated in individuals with diabetes. Observational studies conducted in different populations identified an association between lower serum concentrations of 25(OH)D and higher prevalence of changes in glycemic metabolism. Among the parameters investigated are insulin resistance, fasting glucose, glycated hemoglobin and pancreatic β cell function.

In DM1, the hypothesis of a relationship between vitamin D and disease is based on its potential immunomodulatory effects. VDR is expressed in different cells of the immune system, and vitamin D participates in the regulation of inflammatory and immunological responses. Considering that DM1 involves an autoimmune response directed against pancreatic β cells, researchers have investigated whether changes in vitamin D metabolism could be related to susceptibility or evolution of the disease.

Despite these evidences, the association between low levels of vitamin D and DM1 does not mean that vitamin deficiency is responsible for the development of the disease. The pathophysiology of DM1 involves a complex interaction between genetic predisposition, immunological mechanisms and possible environmental factors. Therefore, vitamin D should be considered within this set of factors and not as an isolated cause.

In DM2, the association between vitamin D and glycemic metabolism has been further investigated. Several observational studies indicate that reduced concentrations of 25(OH)D may be associated with increased insulin resistance and worse metabolic profile. However, observational studies do not allow determining whether reduced levels of vitamin D contribute

to the development of DM2 or whether the metabolic condition itself, obesity, sedentary lifestyle and other factors associated with the disease contribute to the reduction of the concentration of this vitamin.

Glycemic control is one of the main indicators used to evaluate the evolution of diabetes and the effectiveness of therapeutic strategies. In this context, the possible participation of vitamin D in glucose metabolism has been evaluated by different markers, such as fasting glucose and HbA1c.

Insulin resistance is an important component of the pathophysiology of DM2 and is related to the decreased capacity of peripheral tissues to use glucose (Santos et al., 2025). Vitamin D may be involved in this process by different potential mechanisms, including its interaction with RV, its participation in intracellular calcium metabolism and its influence on inflammatory mediators. These mechanisms may contribute to explain the association observed between serum vitamin D levels and insulin sensitivity parameters.

Low-grade chronic inflammation is also an important link between vitamin D and insulin resistance. Fat tissue under obesity conditions may release cytokines and other inflammatory mediators capable of interfering with insulin signaling. Since vitamin D has immunomodulatory properties, its participation in the regulation of these responses is one of the hypotheses used to explain possible metabolic effects (Santos; De Deus, 2026).

However, the scientific literature presents heterogeneous results regarding the relationship between vitamin D concentration and glycemic control. While some studies have identified an association between adequate levels of 25(OH)D and better metabolic parameters, others have not shown relevant effects after control by confounding factors. This difference may be related to the characteristics of the populations evaluated, the initial vitamin D levels, the doses used in supplementation studies, the duration of interventions and the presence of different clinical conditions.

The possibility of using vitamin D as a complementary strategy in the management of diabetes has aroused interest due to its actions on pancreatic function, insulin sensitivity and inflammatory processes. However, supplementation should be differentiated from the recommended replacement for individuals with deficiency or inadequate vitamin D levels (Gonçalves et al., 2025).

Some clinical trials and literature reviews suggest that vitamin D supplementation can produce modest effects on certain metabolic markers in specific groups of individuals with pre-existing deficiency. However, the results are not uniform and still do not allow the

establishment of vitamin D supplementation as an isolated or substitutive treatment of the therapies established for diabetes.

In this sense, vitamin D can be considered a relevant component of the nutritional and metabolic assessment of individuals with diabetes in view of the presence of risk factors for deficiency. Its evaluation and possible supplementation should consider individual clinical characteristics and laboratory parameters, avoiding indiscriminate use of high doses.

DM management should remain based on effective strategies, including adequate nutrition, physical activity, body weight control, glycemic monitoring and pharmacological treatment when indicated (Cruz et al., 2024). The investigation of vitamin D status may complement this approach in individuals with risk factors for deficiency or in clinical situations in which their assessment is pertinent.

The relationship between vitamin D and DM represents an area of investigation that integrates endocrinology, nutrition, immunology and metabolism. The presence of VDR in pancreatic β cells, the participation of vitamin D in calcium homeostasis, its possible effects on insulin sensitivity and its action in the modulation of inflammatory processes provide plausible biological bases for an interaction between vitamin D metabolism and glycemic control.

However, biological plausibility is not enough to establish clinical benefit. The association between vitamin D deficiency and diabetes is observed in different studies, but factors such as age, obesity, physical activity, sun exposure, diet, renal function and the presence of other diseases may influence vitamin D levels and glucose metabolism. For this reason, the interpretation of evidence should consider the difference between association and causality (Do Amaral et al., 2023).

Similarly, the results related to vitamin D supplementation on glycemia and HbA1c have variability. Some individuals may present favorable changes in certain metabolic parameters, while others do not present significant benefits. These differences reinforce the need for well-designed clinical studies that consider the basal status of vitamin D, the type of diabetes, the presence of obesity, the treatment used and the duration of the intervention (De Oliveira et al., 2025).

Thus, vitamin D should not be understood as a substitute treatment for diabetes mellitus, but as a possible additional component related to metabolic health (Maha et al., 2022). The investigation of its relationship with glycemic control can contribute to a broader understanding of the factors involved in the evolution of diabetes and the development of individualized follow-up strategies.

The joint analysis of the available evidence allows us to consider plausible the relationship between vitamin D and DM, but not clarified from the causal and therapeutic points of view. Thus, understanding the mechanisms involved and determining in which groups of patients the correction of vitamin D deficiency can produce metabolic benefits constitute a perspective for future research and for the improvement of the integral management of diabetes mellitus.

3 Methodology

The present study is characterized as a literature review with a qualitative and descriptive approach, developed in order to analyze the scientific evidence available about the relationship between vitamin D and Diabetes Mellitus, with emphasis on the possible effects on glycemic control, secretion and insulin sensitivity, as well as the implications for the management of the disease.

Data from secondary scientific sources, including scientific articles, literature reviews, observational studies, experimental studies and clinical trials related to vitamin D and type 1 and type 2 diabetes mellitus were used for the development of the research. The selection of publications focused on studies addressing the serum concentration of 25-hydroxyvitamin D [25(OH)D], vitamin D deficiency or insufficiency, insulin resistance, pancreatic β cell function, glycemia, glycated hemoglobin (HbA1c) and possible effects of vitamin D supplementation on metabolic parameters.

The bibliographic search was guided by the main descriptors related to the theme, including “vitamin D”, “Diabetes Mellitus”, “Diabetes Mellitus type 1”, “Diabetes Mellitus type 2”, “glycemic control”, “insulin resistance”, “insulin”, “25-hydroxyvitamin D” and “supplementation of vitamin D”, as well as their correspondents in English. The terms were combined according to the search strategy adopted in each database, seeking to expand the identification of studies related to the research objective.

Scientific publications were prioritized that presented a direct relationship with the theme investigated and that contributed to the understanding of the physiological and metabolic mechanisms involved in the possible association between vitamin D and Diabetes Mellitus. Studies were considered to assess the relationship between vitamin D levels and glycemic control parameters, as well as studies investigating the possible effects of supplementation on glycemia, glycated hemoglobin, insulin resistance and pancreatic β cell function.

After the bibliographic survey, the selected studies were critically and comparatively analyzed, considering their objectives, methodological characteristics, investigated population, main results and conclusions. The information obtained was organized according to the main themes of the research: vitamin D metabolism; Diabetes Mellitus pathophysiology; relationship between vitamin D and insulin secretion; vitamin D and insulin resistance; vitamin D deficiency in individuals with diabetes; effects on glycemic control parameters; and possible implications of vitamin D supplementation in disease management.

The analysis sought to identify both favorable evidence and divergent results in the literature, avoiding establishing a causal relationship between vitamin D deficiency and Diabetes Mellitus. Thus, the limitations of the studies analyzed and the possibility of influence of factors such as age, obesity, diet, physical activity, sun exposure, renal function, pharmacological treatment and associated clinical conditions were considered.

Because this is a literature review based on information available in the scientific literature, there was no direct intervention on humans or primary data collection. The study aimed to gather and analyze the knowledge produced about vitamin D and Diabetes Mellitus, contributing to a broader understanding of their possible relationship with glycemic control and disease management strategies.

4 Results and discussion

The analysis of the selected literature allowed us to identify a plausible relationship between vitamin D metabolism and different mechanisms involved in the pathophysiology of Diabetes Mellitus related to insulin secretion, insulin resistance, pancreatic β cell function, inflammation and glycemic control. The studies analyzed, however, do not present uniform results, indicating that the relationship between vitamin D and diabetes mellitus is complex and is influenced by multiple metabolic and clinical factors.

The findings of the literature indicate that vitamin D should not be understood from the perspective of its functions related to bone metabolism. The identification of vitamin D receptor (VDR) in different tissues, including pancreatic β cells and immune system cells, provides a biological basis for investigating its possible effects on glucose metabolism. In this sense, vitamin D can participate in mechanisms related to pancreatic function, immune response and regulation of inflammatory processes involved in glucose metabolism.

One of the main findings in the literature refers to the possible participation of vitamin D in pancreatic β cell function. These cells are responsible for the production and secretion of

insulin, an essential hormone for the maintenance of glycemic homeostasis. The presence of VDR in β cells and the existence of local mechanisms related to vitamin D metabolism suggest that this substance can exert direct or indirect influence on pancreatic activity.

The relationship can also be understood from the participation of calcium in the mechanisms of insulin secretion. Vitamin D plays an important role in maintaining calcium homeostasis, while the intracellular availability of this mineral participates in processes necessary for adequate insulin secretion. Thus, changes in vitamin D metabolism may have repercussions on cellular mechanisms related to the secretory function of β cells.

Schavinski (2024), when investigating morphofunctional changes in pancreatic islets associated with maternal vitamin D deficiency, contributes to the discussion about the importance of vitamin D for pancreatic physiology. Although experimental studies cannot be extrapolated to clinical practice in humans, their results support the hypothesis that changes in vitamin D metabolism may interfere with pancreatic cell structure and function.

These evidences are relevant for the understanding of DM2, in which insulin resistance may be accompanied by compensatory mechanisms of β cells. As the disease progresses, the ability to compensate may decrease, contributing to the maintenance of hyperglycaemia. Thus, factors capable of influencing pancreatic function may be relevant for the evolution of metabolic status.

However, the presence of VDR and the plausibility of cellular mechanisms do not allow us to state that the correction of vitamin D deficiency is capable of restoring the function of β cells or preventing the progression of diabetes. The available literature supports a physiopathological hypothesis, but more consistent clinical evidence is still needed to establish the magnitude of this effect.

Insulin resistance is one of the main pathophysiological components of DM2 and is one of the central points of the possible relationship between vitamin D and glycemic control. This phenomenon is characterized by reduced tissue response to insulin action, compromising the use of glucose and contributing to increased hepatic glucose production.

The studies analyzed indicate that reduced concentrations of 25(OH)D may be associated with changes in insulin sensitivity and a less favorable metabolic profile. Santos et al. (2025) discuss the relationship between insulin resistance and metabolic changes in individuals with DM2, while Santos and De De De Deus (2026) discuss possible mechanisms related to vitamin D and insulin resistance.

A possible explanation for this association involves the action of vitamin D in cellular mechanisms related to insulin receptor, intracellular signaling and calcium metabolism. In

addition, vitamin D may participate in the modulation of inflammatory processes, which play an important role in insulin resistance.

Nevertheless, the association between low vitamin D levels and insulin resistance should be interpreted with caution. Obesity, for example, is related to vitamin D deficiency or insufficiency and to the development of insulin resistance. Thus, a reduced concentration of 25(OH)D may represent a marker associated with a broader set of metabolic changes, and it is not possible to establish, from observational studies, a causal relationship.

The relationship between vitamin D and glycemic control is one of the main points of interest in the literature. Among the parameters used to evaluate this relationship are fasting glucose, glycated hemoglobin (HbA1c) and other markers of insulin resistance.

De Souza et al. (2025) investigated serum vitamin D levels in individuals with DM2 and overweight, identifying a relationship between vitamin concentrations and metabolic characteristics of this population. This type of evidence contributes to the hypothesis that the nutritional status of vitamin D may be related to the metabolic profile of individuals with diabetes when factors such as overweight and changes in insulin sensitivity coexist.

However, the interpretation of the results should consider that individuals with DM2 have multiple factors capable of interfering with glycemic control and vitamin D levels. Feeding, degree of physical activity, adiposity, sun exposure, age, renal function and treatments used are examples of variables that can modify this relationship.

In this context, the literature analyzed does not allow us to state that vitamin D concentration is an isolated determinant of glycemic control. The results suggest a potential association, but the magnitude and direction of this association may vary according to the characteristics of the studied population.

Another relevant result of the analysis was the identification of a possible interaction between vitamin D, obesity, inflammation and insulin resistance. Obesity is an important risk factor for DM2 and is associated with metabolic alterations characterized by low-grade chronic inflammation.

De Melo João, Rotterdam and Souto (2025) highlight the participation of oxidative stress and low-grade chronic inflammation in the pathogenesis of DM2. These processes may interfere with insulin signaling and contribute to the progression of insulin resistance. Vitamin D has immunomodulatory properties that arouse interest in its possible participation in the regulation of these inflammatory responses.

Monteiro (2026) addresses the association between vitamin D insufficiency and obesity, demonstrating the importance of considering adiposity in the interpretation of serum levels of

this vitamin. The coexistence of obesity, insulin resistance and low vitamin D levels creates a complex relationship, in which it is not possible to determine which change precedes the others.

Thus, one of the possible interpretations is that vitamin D participates in a set of metabolic mechanisms related to inflammation and insulin resistance. Another possibility is that the reduction of vitamin D levels is a consequence of characteristics associated with obesity and lifestyle. These possibilities are not exclusive and justify the need for longitudinal studies and clinical trials capable of clarifying the causal relationship.

The literature also presents evidence of a possible association between vitamin D and DM1. Unlike DM2, whose pathophysiology is related to insulin resistance and progressive dysfunction of β cells, DM1 involves autoimmune mechanisms that result in the destruction of these cells.

The presence of VDR in immune system cells and the participation of vitamin D in the modulation of inflammatory responses justify the investigation of this vitamin in the context of DM1. De Sousa Barbosa et al. (2026) highlight the immunogenetic nature of DM1, reinforcing that its manifestation involves interaction between genetic predisposition and immunological mechanisms.

From this perspective, vitamin D could be related to the regulation of the immune response involved in aggression to β cells. However, the available studies do not allow affirming that vitamin D deficiency is a direct cause of DM1. Similarly, it is not possible to conclude that vitamin supplementation is capable of preventing the disease or modifying its evolution.

Therefore, in DM1, the importance of vitamin D should be discussed based on its possible immunomodulatory mechanisms and its relation to pancreatic function, without extrapolating experimental and observational findings to definitive therapeutic conclusions.

The possibility of using vitamin D as a complementary resource in the management of diabetes is one of the aspects of greatest clinical interest. The analysis of the available studies, however, shows that the results of supplementation are heterogeneous.

Gonçalves et al. (2025) investigated the impact of vitamin D on glycemic control in patients with DM2, while De Oliveira et al. (2025) analyzed the effects of vitamin D supplementation on different parameters related to metabolic syndrome. These studies contribute to the discussion about the possibility of metabolic effects associated with the correction of inadequate vitamin levels.

The literature suggests that individuals with vitamin D deficiency or vitamin D insufficiency may constitute a group for which the correction of this deficiency has greater

potential for benefit. Nevertheless, the observed effects on blood glucose and HbA1c are not uniform between the different studies. This variability may be related to the differences between the populations evaluated, the basal levels of vitamin D, the doses used, the period of supplementation and the clinical conditions of the participants.

Maha et al. (2022) discuss vitamin D as an adjuvant resource in the treatment of DM2, reinforcing the possibility of its use as a complementary strategy. The term “adjunctive” is important because vitamin D should not be presented as a substitute for conventional interventions aimed at controlling diabetes.

Adequate nutrition, physical activity, weight control, blood glucose monitoring and the use of antidiabetic drugs, when indicated, remain fundamental components of disease management. The possible correction of vitamin D deficiency should therefore be understood as part of an individualized approach, and not as an isolated intervention.

25(OH)D is the main laboratory marker used to assess vitamin D status and, for this reason, is a central parameter in studies investigating its relationship with diabetes. The evaluation of this concentration allows identifying individuals with reduced levels and investigating possible associations with metabolic parameters.

The literature analyzed shows that concentrations lower than 25(OH)D are associated with unfavorable metabolic characteristics in overweight and DM2 individuals. However, this association should not be interpreted as sufficient evidence of causality.

Serum vitamin D concentration is influenced by several factors, including sun exposure, diet, supplementation, body composition, age and clinical conditions. Therefore, the interpretation of this marker in patients with diabetes should consider the global clinical and metabolic context.

This perspective is important because the indiscriminate use of supplements does not produce the same effects observed in individuals with documented deficiency. The correction of a deficiency may have different objectives than to use high doses of vitamin D in individuals who already have adequate levels.

The results found allow us to consider vitamin D as a relevant component in the metabolic evaluation of individuals with Diabetes Mellitus, given the factors associated with deficiency. However, the available evidence does not support its use as a substitute treatment for established therapies.

Cruz et al. (2024) highlight the importance of therapeutic strategies aimed at controlling chronic hyperglycemia, while the literature on vitamin D adds a perspective related to the identification and correction of nutritional changes that can coexist with diabetes.

The clinical relevance of vitamin D seems to be, therefore, less related to the possibility of replacing antidiabetic drugs and more to the integration of their evaluation into a comprehensive approach to metabolic health. In patients with risk factors for deficiency, the evaluation may contribute to identify a modifiable nutritional change.

This approach is compatible with the concept of integral management of diabetes, in which different factors related to metabolism, diet, body weight, physical activity and associated clinical conditions are considered.

Final considerations

In general, the studies analyzed point to four main aspects. The first refers to the existence of physiological bases for an interaction between vitamin D and glucose metabolism, due to the presence of VDR in pancreatic β cells and the participation of vitamin D in calcium homeostasis.

The second is related to the association between reduced vitamin D levels and metabolic alterations, insulin resistance and worse glycemic profile in certain groups of individuals with DM2. This association, however, is influenced by factors such as obesity, lifestyle and concomitant clinical conditions.

The third aspect refers to the possible immunomodulatory effects of vitamin D in the context of DM1. Although there is biological plausibility for its participation in mechanisms related to autoimmunity, the available evidence does not allow it to be classified as cause or treatment isolated from the disease.

The fourth aspect involves supplementation. The studies analyzed present variable results, suggesting that any benefits on glycemic parameters may depend on the clinical and nutritional characteristics of the individuals evaluated and their basal vitamin D status.

Thus, the results of the review indicate that the relationship between vitamin D and diabetes mellitus should be understood as multifactorial. Vitamin D has plausible mechanisms related to β cell function, insulin sensitivity and inflammatory modulation, but the existence of these mechanisms does not mean that its supplementation improves glycemic control in all patients.

The main contribution of the studies analyzed is therefore to broaden the understanding of metabolic factors related to Diabetes Mellitus and highlight the need for individualized assessment of vitamin D status. The literature supports the importance of continuing to

investigate this relationship, especially through longer-term clinical studies with adequate control of confounding factors.

In summary, vitamin D has potential relevance as a complementary component of metabolic evaluation of individuals with diabetes mellitus when there is documented deficiency or insufficiency. However, the evidence gathered does not justify its use as an isolated intervention in the control of hyperglycemia. Its role should be analyzed in conjunction with conventional strategies for treatment and prevention of complications, maintaining the distinction between epidemiological association, pathophysiological plausibility and proven therapeutic benefit.

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