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REVIEW ARTICLE

Impact of vitamin D on insulin sensitivity and glycemic homeostasis in patients with type 2 diabetes

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ABSTRACT

Introduction: type 2 diabetes represents a growing public health problem, in which vitamin D has emerged as a potential modulator of glucose metabolism and insulin sensitivity.
Objective: to describe the role of vitamin D in glycemic homeostasis and insulin resistance in patients with type 2 diabetes.
Methods: a literature review was conducted through the search of sources from different databases. Studies meeting the selection criteria were identified and selected, followed by an analysis of those that allowed for a comprehensive and adequate approach to the topic.
Development: the analyzed studies show that low levels of 25-hydroxyvitamin D are associated with greater insulin resistance, poorer glycemic control, and an increased risk of progression from prediabetes to type 2 diabetes. Pancreatic beta cells express vitamin D receptors, suggesting a direct effect on insulin secretion. In addition, vitamin D exhibits anti-inflammatory properties that could mitigate the chronic inflammation linked to obesity and metabolic syndrome. However, results regarding supplementation are heterogeneous and influenced by methodological differences, doses used, and analytical techniques employed to measure vitamin D levels.
Conclusions: vitamin D may play a protective role in type 2 diabetes by improving insulin sensitivity and pancreatic function. Nevertheless, the current evidence remains inconclusive, justifying the need for standardized studies that allow the establishment of clear clinical recommendations.

Keywords: Diabetes Mellitus, Type 2; Homeostasis; Insulin Resistance; Vitamin D.

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INTRODUCTION

Vitamin D has gained relevance in the context of type 2 diabetes mellitus (T2DM) due to its role in glucose metabolism regulation and insulin sensitivity. Recent studies suggest that vitamin D deficiency may be a significant risk factor for T2DM development, as low levels of this vitamin have been associated with greater insulin resistance and impaired glycemic control.[1,2]

Vitamin D receptors are present in various tissues, including pancreatic beta cells, suggesting that vitamin D may influence insulin secretion and overall metabolic function. Additionally, vitamin D has demonstrated anti-inflammatory properties, which could help mitigate the chronic inflammation associated with obesity and diabetes.[1]

Despite growing evidence supporting the relationship between vitamin D and T2DM, further research is needed to establish clear recommendations regarding supplementation and vitamin D level management in the general population and in individuals at risk of developing diabetes.[3] Several mechanisms have been proposed through which vitamin D might influence the development of this disease, including improving insulin sensitivity, enhancing insulin secretion by pancreatic beta cells, and exerting anti-inflammatory and antioxidant effects.[4]

Currently, the international scientific community is focused on elucidating the potential mechanisms by which vitamin D influences glucose metabolism, insulin sensitivity, and pancreatic beta-cell function. This context motivated the present review, which aimed to describe the role of vitamin D in glycemic homeostasis and insulin resistance in patients with type 2 diabetes.

METHODS

This study was designed as a systematic review of the scientific literature, conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. The search period was limited to January 2010 through December 2024 to encompass the most recent and relevant evidence on the impact of vitamin D on insulin sensitivity and glycemic homeostasis in T2DM patients.

Information sources included the databases PubMed/MEDLINE, SciELO, ScienceDirect, Google Scholar, LILACS, and BVSALUD. Additionally, secondary references from key article bibliographies and grey literature (theses, technical reports, and institutional documents) were reviewed to broaden thematic coverage and reduce publication bias.

The search strategy was constructed using keywords and Boolean operators, employing terms such as: "Type 2 diabetes mellitus" OR "T2DM" AND "Vitamin D" OR "25-hydroxyvitamin D" AND "Insulin" OR "Insulin sensitivity" OR "Glucose metabolism" OR "Supplementation." Articles published in Spanish, English, and Portuguese were considered to include relevant literature in the main scientific languages of Ibero-American and Anglo-Saxon regions.

Inclusion criteria encompassed original studies (clinical trials, observational studies, meta-analyses, and systematic reviews) published within the defined timeframe that directly addressed the relationship between vitamin D and type 2 diabetes. Excluded were duplicates, articles without full-text access, irrelevant studies, and those outside the search period.

The selection process was carried out in several stages: first, titles and abstracts were screened to exclude non-relevant studies; subsequently, full texts of potentially eligible articles were evaluated. Initially, 1,245 records were identified; 312 were removed as duplicates and 587 excluded for not meeting inclusion criteria. Finally, 346 articles underwent full-text review, and 42 studies met all criteria for inclusion in the qualitative synthesis. The procedure was documented using a PRISMA flow diagram (Fig. 1), detailing the phases of identification, screening, eligibility, and inclusion.

Data extraction and analysis were performed systematically, collecting key variables such as author, publication year, methodological design, sample size and characteristics, type of intervention or exposure, and main outcomes related to insulin sensitivity, glycemic control, and pancreatic beta-cell function. Evidence synthesis was conducted qualitatively, integrating findings from selected studies. In cases where data were homogeneous and comparable, an exploratory meta-analysis was considered; however, methodological heterogeneity limited quantitative integration in several sections.

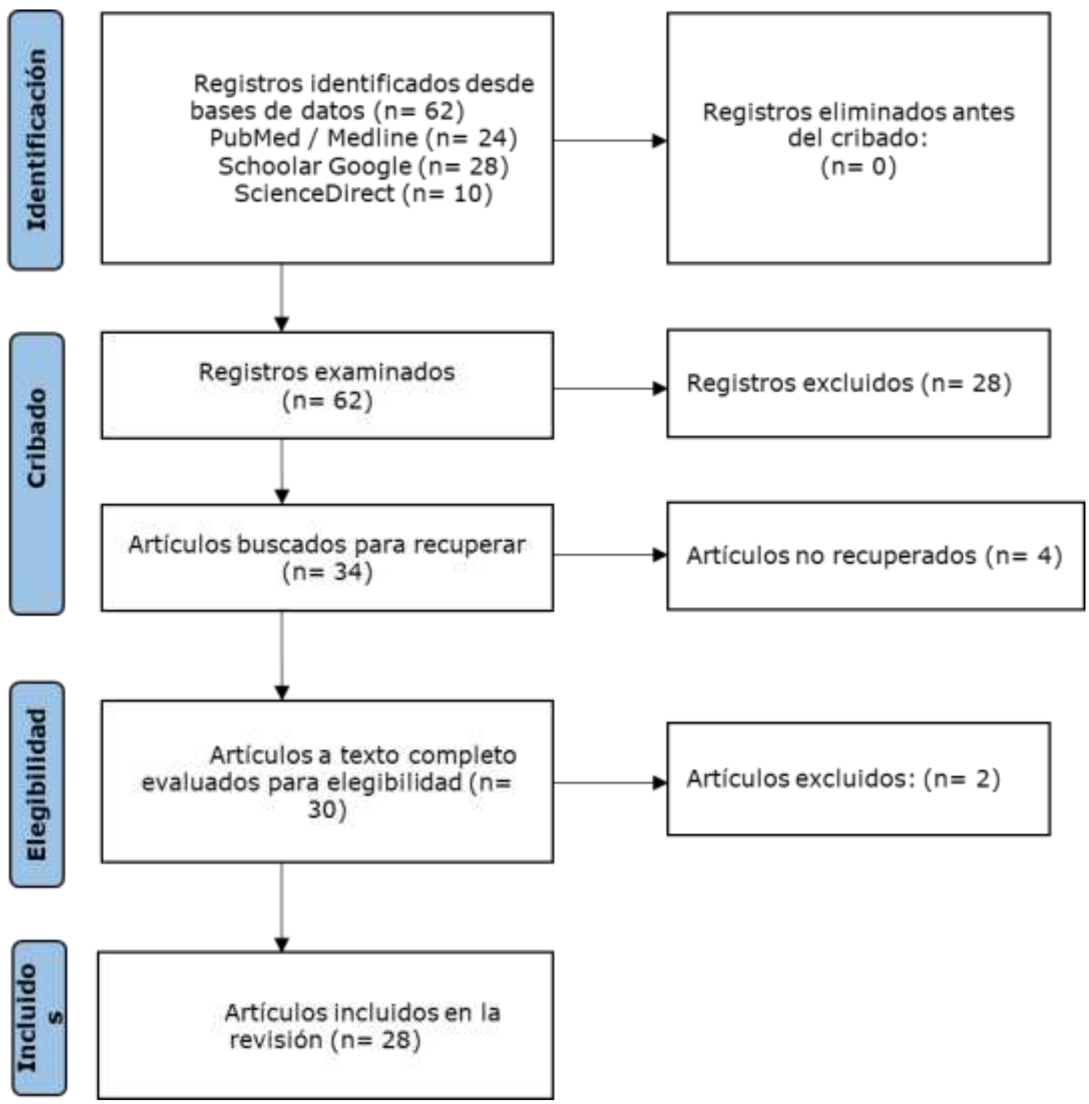

Fig. 1. PRISMA flow diagram for study selection.

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DEVELOPMENT

Vitamin D is a fat-soluble compound with multiple functions in the body that extend beyond calcium metabolism.[4] It is obtained through sun exposure, diet, or supplementation. Its metabolism involves several key steps that convert vitamin D into its active form, necessary for diverse biological functions.

Vitamin D is synthesized in the skin from 7-dehydrocholesterol upon exposure to ultraviolet B (UVB) radiation from sunlight. This initial form is known as vitamin D_3 (cholecalciferol). It can also be obtained from dietary sources such as fatty fish, liver, egg yolks, and fortified foods.[5] Dietary vitamin D may be either D_2 (ergocalciferol) or D_3. Once ingested, vitamin D is transported to the liver, where it is converted into 25-hydroxyvitamin D (25(OH)D)—the primary circulating form of vitamin D in the blood. Subsequently, 25(OH)D is transported to the kidneys, where it is converted into 1,25-dihydroxyvitamin D (1,25$(OH)_2$D), the biologically active form of vitamin D. This process is regulated by several factors, including serum calcium and phosphate levels.[4,6]

Vitamin D and its metabolites circulate in the blood bound to proteins, primarily vitamin D-binding protein (DBP). Most circulating metabolites are protein-bound, which limits their access to target cells and prolongs their half-life in the body. Vitamin D performs several biological functions that may be linked to the development and progression of T2DM.[7,8]

Possible causes of the widespread vitamin D deficiency/insufficiency currently observed include lifestyle patterns involving more time spent indoors, increasing urbanization (as opposed to open spaces), the beneficial use of sunscreen, and rising obesity rates. A statistically significant inverse correlation exists between vitamin D deficiency and overweight/obesity, increasing the likelihood of developing T2DM.[9]

T2DM is a chronic disease that affects how the body uses glucose—a sugar that serves as the primary energy source. In T2DM, the body becomes resistant to insulin or fails to produce enough insulin to maintain normal blood glucose levels. This can lead to hyperglycemia, which, if inadequately controlled, may result in serious complications such as cardiovascular disease, kidney damage, neuropathy, and vision problems. The global prevalence of T2DM has been steadily increasing. According to the World Health Organization (WHO), the number of people with diabetes rose from 108 million in 1980 to 422 million in 2014, with the fastest growth occurring in low- and middle-income countries.[10]

Specific studies report varying T2DM prevalence depending on population and setting. For instance, a study conducted in residential care centers found a T2DM prevalence of 21,7 % among participants.[11] In Ecuador, prevalence was reported at 5,7 %, progressively increasing with age and associated with factors such as body mass index and family history.[12]

The literature reports that vitamin D may play a role in preventing T2DM in high-risk individuals.[13] An association has been described between vitamin D concentrations, glucose homeostasis, and diabetes progression. Specifically, an inverse relationship was observed between glycated hemoglobin (HbA1c) levels and 25(OH)D concentrations in individuals aged 35–74 years without a known history of diabetes. For this reason, some authors propose screening for vitamin D insufficiency in subjects with elevated HbA1c—and vice versa.[14]

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Other studies have indicated that vitamin D may influence insulin sensitivity and pancreatic beta-cell function—critical factors in T2DM pathogenesis. It has been proposed that vitamin D could help restore normal insulin production and improve the body's response to insulin.[4,15,16]

Several studies have shown that higher levels of 25-hydroxyvitamin D (25(OH)D) are inversely associated with insulin resistance and T2DM risk. This association arises from the convergence of multiple mechanisms:[3,4,13,17]

- Receptor expression: Pancreatic beta cells express vitamin D receptors (VDR) and 1α-hydroxylase, indicating that vitamin D can directly influence insulin production and secretion. Vitamin D may help maintain the function of these cells, which is essential for glucose regulation.
- Restoration of insulin production: Animal model studies have demonstrated that vitamin D can restore normal insulin production under deficiency conditions, suggesting a protective role in beta-cell function.
- Reduction of inflammation: Vitamin D possesses anti-inflammatory properties that may be beneficial in the context of T2DM. Chronic inflammation has been linked to insulin resistance and beta-cell dysfunction. By reducing inflammation, vitamin D may help improve glycemic homeostasis.

25-Hydroxyvitamin D Test [25(OH)D Assay]

The normal range for 25-hydroxyvitamin D is measured in ng/mL. Many experts recommend a level between 20 and 40 ng/mL, while others suggest a range of 30 to 50 ng/mL.[18] For accurate and reliable measurement of 25-hydroxyvitamin D, the following must be considered:

- Sample type: Serum
- Collection tube: Red-top tube
- Sample volume: 0,5 mL
- Collection instructions: Centrifuge and aliquot serum into a plastic vial within 2 hours of sample collection.
- Minimum sample volume: 0,25 mL

The most common measurement methods include immunoassays such as ELISA and CLIA, as well as advanced techniques like high-performance liquid chromatography (HPLC) and liquid chromatography–tandem mass spectrometry (LC-MS/MS).

Immunoassays

- ELISA (Enzyme-Linked Immunosorbent Assay): Uses specific antibodies to detect and quantify vitamin D. It is relatively easy to automate and low-cost, but may suffer from specificity and sensitivity issues, particularly in distinguishing between vitamin D metabolites.[19,20] ELISA sensitivity for vitamin D measurement is generally very high, reaching up to 99,4 % in some studies, indicating its ability to detect nearly all individuals with true vitamin D deficiency. Specificity is also high, with reported values around 99 %, meaning the test has a low false-positive rate and effectively identifies those without deficiency.[21]
- CLIA (Chemiluminescent Immunoassay): Similar to ELISA but uses light-emitting chemical reactions for detection. It offers high sensitivity and specificity, though it may be susceptible to inter-assay variability and matrix effects.[22,23] CLIA sensitivity for vitamin D typically ranges from 90 % to 95 %, effectively detecting most deficiency cases. Specificity is also high (~95 %), resulting in a low false-positive rate.

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Reference Methods

- HPLC (High-Performance Liquid Chromatography): Allows separation and quantification of different vitamin D forms but requires specialized equipment and trained personnel. It is considered one of the most accurate methods, though less common in clinical labs due to complexity.[19,22] HPLC methods are highly sensitive (>90 %) and specific (~95 %), making them effective for deficiency detection with minimal false positives.
- LC-MS/MS (Liquid Chromatography–Tandem Mass Spectrometry): Currently the reference method for 25(OH)D measurement. It provides highly precise results and quantifies multiple vitamin D metabolites, though its use is limited by infrastructure and personnel requirements.[19,22] LC-MS/MS exhibits very high sensitivity (>95 %) and specificity (95–98 %), ensuring accurate identification of both deficient and non-deficient individuals.

Measurement precision varies significantly across methods. Techniques like LC-MS/MS have shown reduced imprecision over time, with a coefficient of variation (CV) of 5–10 % and a bias of ~5 %, making them more consistent than immunoassays, which often suffer from poor standardization.[19] Additionally, 25(OH)D results can vary considerably between methods. For example, a comparative study between Roche and Siemens assays revealed significant differences in reported 25(OH)D concentrations, highlighting the critical need for assay standardization.[22] Table 1 summarizes the advantages and disadvantages of each analytical method.

Table 1. Advantages and disadvantages of different analytical tests.

Method	Sensitivity / Specificity [Cost]	Advantages	Disadvantages
ELISA	~90–99% / ~95% [Low]	Widely used, good precision	Kit-dependent variability, longer processing time
CLIA	~90–95% / ~95% [Medium]	High automation, suitable for high-volume labs	Requires specialized equipment
HPLC	>90% / ~95% [High]	Precise, separates D2 and D3 metabolites	Complex, requires specialized infrastructure
LC-MS/MS	>95% / 95–98% [High]	Reference standard, high sensitivity and specificity	Very costly, requires highly trained personnel

Factors affecting vitamin D level measurement

Measurement of vitamin D levels—specifically 25-hydroxyvitamin D (25(OH)D)—can be influenced by several factors that must be considered to ensure result accuracy and clinical relevance:[4,23]

- Seasonal variation: Vitamin D levels fluctuate seasonally due to changes in sun exposure. Levels tend to be lower in winter. One study found that 25(OH)D concentrations are significantly lower in winter compared to summer, which may affect results if samples are collected at different times of the year.
- Time of day: Blood collection at different times may influence vitamin D levels. Morning sampling is recommended to minimize diurnal variability, as levels can fluctuate throughout the day.
- Sample storage conditions: 25(OH)D stability may be compromised if samples are not stored at appropriate temperatures (typically –20°C or –80°C) to prevent vitamin D degradation prior to analysis.

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Effects on glycemic control

Two meta-analyses published in 2020 evaluated the effect of vitamin D_3 supplementation on glycemic control in T2DM patients. Results were inconsistent: some studies showed improvements in insulin sensitivity and beta-cell function, while others found no significant benefits.(24,25)

Vitamin D has gained relevance in T2DM due to its potential role in glucose metabolism regulation and insulin sensitivity. Recent studies suggest that vitamin D deficiency may be a significant risk factor for T2DM development, as low levels are associated with greater insulin resistance and impaired glycemic control. This underscores the importance of investigating how optimizing vitamin D status may improve metabolic health and prevent diabetes-related complications.(26)

Vitamin D receptors (VDR) are present in various tissues, including pancreatic beta cells, suggesting that vitamin D may influence insulin secretion and overall metabolic function. Additionally, its anti-inflammatory properties may mitigate chronic inflammation linked to obesity and diabetes. Understanding these mechanisms could lead to novel diabetes management strategies and improved patient quality of life. However, despite growing evidence supporting this relationship, further research is needed to establish clear recommendations on vitamin D supplementation and monitoring in the general population and in those at risk of developing diabetes—potentially enabling more effective preventive and therapeutic measures in clinical practice.(27,28)

CONCLUSIONS

The relationship between vitamin D and T2DM is promising, as studies suggest that adequate vitamin D levels may improve insulin sensitivity and glycemic control through direct effects on pancreatic beta cells and anti-inflammatory actions. However, current evidence remains inconclusive regarding the efficacy of vitamin D supplementation in preventing or treating T2DM, due to inconsistent findings across studies. Factors such as variability in study design and measurement techniques may influence outcomes, highlighting the need for methodological standardization and further research to establish clear guidelines for vitamin D supplementation in populations at risk of developing T2DM.

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