Cureus
Part of SPRINGER NATURE

Open Access Review Article

Role of Vitamin D as an Adjunctive Therapy in the Treatment of Type 2 Diabetes Mellitus: An Evidence-Based Review

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Review began 09/22/2026
Review ended 10/16/2026
Published 10/29/2026

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DOI: 10.7759/cureus.118769

Abstract

The increasing global prevalence of type 2 diabetes mellitus (T2DM) requires comprehensive and multifactorial approaches to its management. Vitamin D has attracted interest due to its potential role in regulating insulin secretion and sensitivity. This study aimed to evaluate the effectiveness of vitamin D supplementation as an adjunct therapy for glycaemic control in individuals with T2DM. An evidence-based review was conducted through searches of the PubMed, Cochrane Library, ECRI Guidelines Trust®, and SciELO databases, including articles published between October 2015 and October 2025 in Portuguese, English, and Spanish. Of the 162 articles identified, 20 were selected based on their relevance to the predefined outcomes. The results suggest a trend towards improved glycaemic control with vitamin D supplementation. However, the heterogeneity among studies limits the consistency of the findings. Current evidence remains insufficient to recommend routine vitamin D supplementation for all individuals with T2DM.

Categories: Family/General Practice, Medical Education, Endocrinology/Diabetes/Metabolism
Keywords: cholecalciferol, diabetes mellitus, ergocalciferol, type 2, vitamin d

Introduction And Background

Diabetes mellitus is a chronic metabolic disease characterised by persistent hyperglycaemia resulting from alterations in insulin secretion, peripheral insulin action, or both [1]. Among its different types, type 2 diabetes mellitus (T2DM) is the most prevalent and currently represents one of the major global public health challenges. Its progression is associated with multiple microvascular and macrovascular complications, including retinopathy, nephropathy, cardiovascular disease, and stroke, with a significant impact on patients' quality of life and healthcare systems [2,3].

From an epidemiological perspective, a progressive and consistent increase in the incidence and prevalence of T2DM has been observed over recent decades, largely explained by population ageing and the adoption of sedentary lifestyles [4,5]. It is estimated that the disease affects hundreds of millions of individuals worldwide, resulting in a substantial burden of morbidity and mortality [5].

Despite the therapeutic advances achieved in recent years, adequate glycaemic control remains a challenge for the vast majority of patients, reinforcing the need to explore complementary approaches. In this context, vitamin D has attracted increasing interest, not only because of its classical role in bone metabolism, but also because of its effects beyond the musculoskeletal system. Vitamin D is an essential micronutrient that, following hepatic and renal metabolism, is converted into calcitriol ($1,25(OH)_2D_3$), its biologically active form, which exerts hormonal actions in several tissues [6].

Consequently, in addition to its role in regulating calcium and phosphate metabolism, vitamin D also contributes to the modulation of immune responses, inflammation, and cellular processes such as differentiation and proliferation [7].

Vitamin D deficiency has been widely described in different populations and has been associated with several chronic conditions, including obesity, metabolic syndrome, and T2DM [8,9]. Observational studies have demonstrated lower serum concentrations of 25-hydroxyvitamin D in individuals with impaired glucose tolerance or diabetes, suggesting a possible association between hypovitaminosis D and metabolic dysfunction [10,11].

From a pathophysiological perspective, several mechanisms have been proposed to explain the potential role of vitamin D in the regulation of glucose metabolism. These include the direct modulation of insulin secretion through the action of vitamin D receptors present in pancreatic β-cells, its influence on peripheral insulin sensitivity, and systemic anti-inflammatory effects, which may contribute to reducing insulin

resistance [12,13]. Additionally, vitamin D may play a role in the regulation of intracellular calcium, which is essential for insulin secretion [14,15].

Despite biological plausibility and consistent observational evidence, the results of randomised clinical trials evaluating the effect of vitamin D supplementation on glycaemic control in patients with T2DM remain heterogeneous. Some meta-analyses suggest benefits in parameters such as fasting glucose, glycated haemoglobin, and insulin resistance, whereas others have not demonstrated significant effects [16-19]. These discrepancies may reflect methodological differences between studies, particularly regarding the dose, duration, and form of supplementation, as well as the characteristics of the populations included [20,21].

Therefore, considering the high prevalence of T2DM and the need for additional therapeutic strategies, it is relevant to clarify the role of vitamin D in this context. The aim of this evidence-based review is to evaluate the impact of vitamin D supplementation on glycaemic control in adults with T2DM through the analysis of parameters such as fasting glucose, glycated haemoglobin, and insulin resistance, as well as potential modifying factors, including dose, intervention duration, and baseline vitamin D levels.

This work was presented as an oral presentation at the Fifth Porto Ocidental Conference, held on May 22, 2026.

Review

Methods

A search for randomised controlled trials (RCTs), meta-analyses (MAs), and systematic reviews (SRs) published between October 2015 and October 2025 and written in Portuguese, English, or Spanish was conducted. The following MeSH terms were used: “Vitamin D”, “Cholecalciferol”, “Ergocalciferol”, and “Diabetes Mellitus, Type 2”. The search was performed in PubMed, the Cochrane Library, ECRI Guidelines Trust®, and SciELO, yielding a total of 162 articles. The complete search strategy is presented in Table *1*.

Database	Search terms	Study types	Publication period	Languages
PubMed	“Vitamin D” OR “Cholecalciferol” OR “Ergocalciferol” AND “Diabetes Mellitus, Type 2”	RCTs, meta-analyses, systematic reviews	October 2015–October 2025	English, Portuguese, Spanish
The Cochrane Library	“Vitamin D” OR “Cholecalciferol” OR “Ergocalciferol” AND “Diabetes Mellitus, Type 2”	RCTs, meta-analyses, systematic reviews	October 2015–October 2025	English, Portuguese, Spanish
ECRI Guidelines Trust®	“Vitamin D” OR “Cholecalciferol” OR “Ergocalciferol” AND “Diabetes Mellitus, Type 2”	RCTs, meta-analyses, systematic reviews	October 2015–October 2025	English, Portuguese, Spanish
SciELO	“Vitamin D” OR “Cholecalciferol” OR “Ergocalciferol” AND “Diabetes Mellitus, Type 2”	RCTs, meta-analyses, systematic reviews	October 2015–October 2025	English, Portuguese, Spanish

TABLE 1: Search strategy

RCTs, randomised controlled trials.

The inclusion criteria were organised according to the PICO framework and were as follows: population, non-pregnant adults (⩾18 years) with a diagnosis of T2DM; intervention, vitamin D supplementation; comparison, placebo or no intervention; and outcome, improvement in glycaemic control, assessed through measurement of blood glucose, glycated haemoglobin (HbA1c), and/or a reduction in insulin resistance (HOMA-IR).

The exclusion criteria included duplicate articles, opinion articles, narrative reviews, clinical practice guidelines, and articles that did not meet the inclusion criteria regarding population, intervention, comparison, and outcomes. The inclusion and exclusion criteria are presented in Table *2*.

Domain	Inclusion criteria	Exclusion criteria
Population	Adults (≥18 years) with a diagnosis of type 2 diabetes mellitus	Studies involving participants who did not meet the predefined population criteria
Pregnancy	Non-pregnant adults	Studies involving pregnant participants
Intervention	Vitamin D supplementation	Studies not evaluating vitamin D supplementation
Comparison	Placebo or no intervention	Studies without a placebo or no-intervention comparison
Outcomes	Improvement in glycaemic control assessed by capillary blood glucose, HbA1c, and/or HOMA-IR	Studies not assessing the predefined glycaemic outcomes
Study design	Randomised controlled trials, meta-analyses, and systematic reviews	Opinion articles, narrative reviews, clinical practice guidelines, and other study designs
Publication period	Published between October 2015 and October 2025	Articles published outside the predefined period
Language	English, Portuguese, or Spanish	Articles published in other languages
Duplicate records	—	Duplicate articles
Relevance	Studies addressing the research question	Studies whose objectives were not relevant to the research question
Full-text availability	Full-text available	Full-text article unavailable

TABLE 2: Inclusion and exclusion criteria

HbA1c: glycated hemoglobin; HOMA-IR: Homeostatic Model Assessment for Insulin Resistance.

To determine the levels of evidence (LOE) and strength of recommendations (SOR), the Strength of Recommendation Taxonomy (SORT) scale developed by the American Academy of Family Physicians was used [22].

Results

The initial search identified 162 articles. After applying the inclusion and exclusion criteria, 20 articles were selected, comprising seven randomised controlled trials [23-29], 12 meta-analyses [16,17,30-39], and one systematic review (Figure *1*) [40].

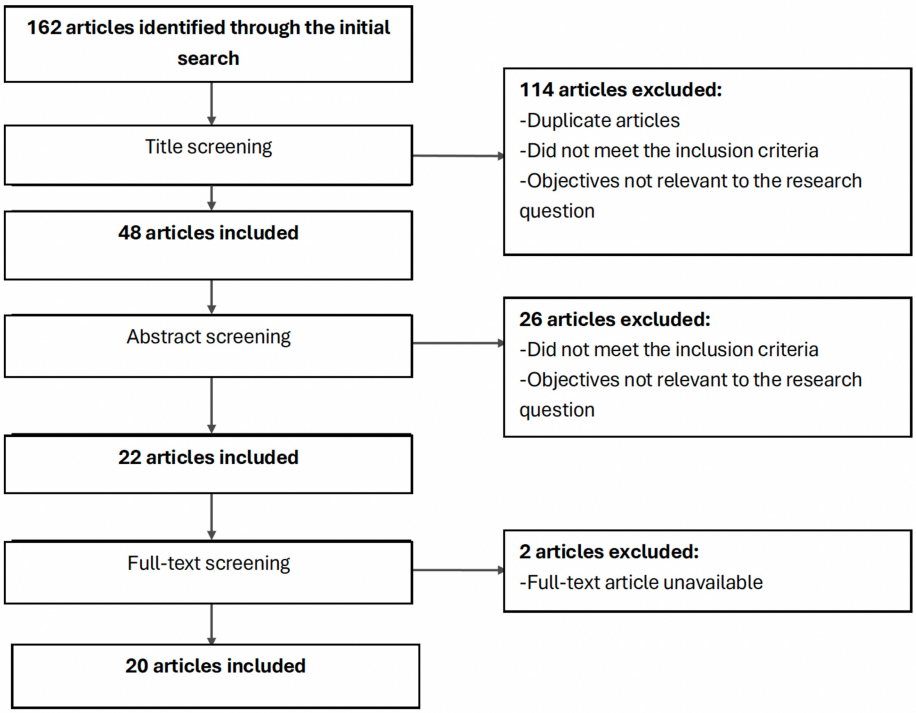


FIGURE 1: Flowchart of the article selection process

A summary of the selected articles included in the review is presented in Tables *3*-*5*.

Reference	Sample	Intervention	Outcomes	LOE/SOR
Cojic et al. (2021) [23]	Adults aged ≥ 30 years, treated with metformin and lifestyle measures, with HbA1c ≤ 7%; n = 130 (65 intervention, 65 control)	VitD3 + metformin vs. metformin alone. Oral; daily administration of Vigantol®. Dose: If 25(OH)D ≤ 50 nmol/L: 50,000 IU/week for 3 months + 14,000 IU/week for 3 months. If 25(OH)D > 50 nmol/L: 14,000 IU/week. Duration: 6 months.	Significant reduction in HbA1c at 3 months (p = 0.031) and 6 months (p = 0.017). No significant effect on fasting blood glucose or HOMA-IR.	2B
Upreti et al. (2018) [24]	Adults aged 21–60 years with T2DM receiving a stable dose of metformin for the previous 3 months, HbA1c ≤ 8.5% and VitD ≤ 30 ng/mL; n = 60 (30 intervention, 30 control)	VitD3 (Calcirol®) vs. placebo. Route: Oral. Dosing regimen: 60,000 IU/week for the first six weeks, followed by once-monthly administration until completion of the study. Duration: 6 months.	Significant reductions in HbA1c (-0.27%; p = 0.01) and fasting blood glucose (-28.2 mg/dL; p = 0.04) were observed at 6 months compared with baseline, whereas no significant changes were observed in the control groups.	2B
Derosa et al. (2022) [25]	Adults (> 18 years), Caucasian, with T2DM and VitD deficiency (< 30 ng/mL); n = 239 (122 intervention, 117 control)	VitD3 vs. placebo. Route: Oral. Dosing regimen: If 25(OH)D < 20 ng/mL: 50,000 IU/week for 12 weeks, followed by 25,000 IU every 2 weeks for 12 weeks. If 25(OH)D 20–30 ng/mL: 25,000 IU/week for 12 weeks, followed by	Significant reductions at 3 and 6 months within the intervention group over the course of the study and compared with the placebo group: HbA1c (7.6% → 7.3% → 7.1%; p = 0.045 vs. baseline; p = 0.046 vs. placebo); HOMA-IR (6.1 → 4.7 → 4.4; p = 0.0091 vs. baseline; p = 0.041 vs. placebo). A significant reduction in fasting blood glucose was observed at 3 and 6 months within the intervention group over the course of the study; however, no statistically significant difference was observed when	2B

		25,000 IU every 2 weeks for 12 weeks. Duration: 6 months.	compared with the placebo group. Fasting blood glucose (140.3 → 136.3 → 130.4 mg/dL; p = 0.042 vs. baseline; p = 0.067 vs. placebo).	
Hu et al. (2023) [26]	Adults aged 50–70 years with T2DM; n = 270 (135 intervention, 135 control)	VitD3 vs. no treatment. Route: Oral. Dosing regimen: 800 IU/day. Duration: 30 months.	No significant between-group difference was observed for HbA1c (−0.22 ± 1.19% vs. 0.06 ± 1.14%; p = 0.086). A significant reduction in HOMA-IR was observed in the VitD group (−0.99 ± 2.53; p = 0.001), with a significant difference compared with the control group (p = 0.012). Among participants who achieved 25(OH)VitD levels ≥ 30 ng/mL at the end of the study, a significant difference in fasting blood glucose was observed between the VitD group (−0.30 ± 2.52 mmol/L) and the control group (0.49 ± 1.78 mmol/L; p = 0.049). VitD levels, sex, and pre-existing obesity appear to modify the effects of VitD supplementation.	2B
Safarpour et al. (2020) [27]	Adults aged 25–65 years with T2DM, BMI ≥ 25 kg/m², VitD deficiency and HbA1c < 8.5%; clinically stable, with no recent changes in antidiabetic therapy and no use of vitamin or mineral supplements; n = 85 (42 intervention, 43 placebo)	VitD3, 50,000 IU/week vs. placebo. Route: Oral. Dosing regimen: 50,000 IU/week. Duration: 8 weeks.	Significant reduction in HbA1c in the intervention group (7.51 ± 0.87% → 6.76 ± 0.98%; p < 0.001), with a significant between-group difference after adjustment (p < 0.001). No significant between-group differences were observed in fasting blood glucose (p = 0.130) or HOMA-IR (p = 0.421).	2B
El Hajj et al. (2020) [28]	Non-obese adults with T2DM and VitD deficiency or insufficiency; n = 88 (45 intervention, 43 placebo)	VitD3 vs. placebo. Dosing regimen: 30,000 IU/week. Duration: 6 months.	No significant between-group differences were observed in fasting blood glucose (p = 0.84), HbA1c (p = 0.31), or HOMA-IR (p = 0.26) after 6 months of supplementation.	2B
Khan et al. (2018) [29]	Adults aged 40–70 years with T2DM, HbA1c 7.5–9.0% and VitD deficiency (25(OH)D < 20 ng/mL); n = 140 (70 intervention, 70 control)	VitD3 + metformin vs. metformin. Dosing regimen: 50,000 IU/week. Duration: 12 weeks.	Significant reduction in HbA1c after 12 weeks (5.93 vs. 7.02%; −1.09%, p < 0.05).	2B

TABLE 3: Summary analysis of the randomised controlled trials included in the evidence-based review (EBR)

HbA1c, glycated haemoglobin; VitD3, cholecalciferol; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; T2DM, Type 2 Diabetes Mellitus; VitD, Vitamin D; BMI, Body Mass Index; LOE, levels of evidence; SOR, strength of recommendations.

Reference	Sample	Intervention	Outcomes	LOE/SOR
Max et al. (2024) [40]	20 RCTs, n = 1204	VitD2/D3 vs. Placebo/no treatment. Route: Oral. Dose: ≥4,000 IU/day	Heterogeneous results, with a possible modest benefit in HbA1c.	2B

TABLE 4: Summary analysis of the systematic review included in the evidence-based review (EBR)

RCT, Randomised Controlled Trial; VitD2, ergocalciferol; VitD3, cholecalciferol; HbA1c, glycated haemoglobin; LOE, levels of evidence; SOR, strength of recommendations.

Reference	Sample	Intervention	Outcomes	LOE/SOR
Li et al. (2018) [30]	20 RCTs; n = 2703	VitD2/D3 vs. placebo/no supplementation; Route: Oral; Duration: ≥8 weeks	Significant reduction in HOMA-IR (−0.57; p = 0.03). No consistent overall effect on HbA1c or fasting blood glucose. Benefits in fasting blood glucose were more evident in subgroups with baseline characteristics or interventions including Baseline VitD deficiency (<50 nmol/L; −5.77, p = 0.017), shorter interventions (<3 months; −0.44, p < 0.001), and higher doses (>2,000 IU/day; −8.70, p < 0.001).	2B
Chen et al. (2024) [16]	39 RCTs; n = 2982	VitD2/D3 vs. placebo; Route: Oral; Dosing regimen: Daily or weekly; Duration: ≥2 months	Significant reductions in fasting blood glucose (−0.49 mmol/L, p < 0.001), HbA1c (−0.30%, p < 0.001), and HOMA-IR (−0.39; p = 0.002). More pronounced effects with doses ≤2,000 IU/day, duration ≤3 months, baseline VitD insufficiency, overweight, and HbA1c ≥8%.	2B
Afraie et al. (2024) [31]	61 RCTs; n = 5600	VitD2/D3 vs. placebo/no supplementation	Significant reduction in HbA1c (−0.15; p < 0.001) and fasting blood glucose (−0.28; p < 0.001). Weight gain, higher BMI, and longer disease duration may reduce the effect of VitD supplementation on DM control.	2B
Wu et al. (2017) [32]	24 RCTs; n = 1874	VitD2/D3 vs. placebo/no supplementation; Route: Oral or intramuscular	Significant overall reduction in HbA1c (−0.25; p = 0.001). No significant overall effect on fasting blood glucose (−0.14; p = 0.10). Subgroup analyses showed more pronounced results in specific populations: Patients with baseline VitD deficiency: significant reductions in both HbA1c (−0.38, p = 0.009) and fasting blood glucose (−0.27, p = 0.007).; Non-obese patients (BMI <30 kg/m²): significant reduction in HbA1c (−0.30, p = 0.01).	2B
Farahmand et al. (2023) [33]	46 RCTs; n = 4313	VitD2/D3 vs. placebo or no supplementation; Route: Oral or intramuscular; Dosing regimen: Various; Duration: 8–48 weeks	Significant reduction in fasting blood glucose (−5.02 mg/dL; p < 0.001). Significant reduction in HbA1c (−0.20%; p < 0.001). Significant reduction in HOMA-IR (−0.42; p = 0.019). In subgroup analysis, the effects were more pronounced in individuals with VitD deficiency and with higher doses/shorter treatment duration.	2B
Zhang et al. (2025) [34]	40 RCTs; n = 4181	VitD2/D3 vs. placebo; Route: Oral; Dose categories: low (<1,000 IU/day, LDS), medium (1,000–2,000 IU/day, MDS), high (2,000–4,000 IU/day, HDS), and extremely high (≥4,000 IU/day, EHDS); Dosing regimen: Daily, every 2 weeks, weekly, or one or two single doses.; Duration: ≥3 months	VitD2 significantly reduced fasting blood glucose compared with placebo (−0.81; [−1.47, −0.16]) and no treatment (−1.13 [−1.92, −0.33]). EHDS significantly reduced fasting blood glucose compared with no treatment (−0.56 [−0.99, −0.13]). LDS significantly reduced HbA1c (−0.41 [−0.77, −0.04]). No statistically significant results were obtained for HOMA-IR. The data suggest that higher doses of VitD may be useful for shorter treatment durations, whereas lower doses may be more suitable for longer treatment durations.	2B
Probosari et al. (2025) [35]	9 RCTs; n = 913	VitD3 vs. placebo/no treatment; Dose: 1,000 IU/day to 100,000 IU as a bolus followed by daily administration; Duration: 12–24 weeks	Fasting blood glucose: no statistically significant differences were found between the intervention and control groups. Reduction in HbA1c at 12 weeks (−0.30, p = 0.02). Reduction in HOMA-IR at 24 weeks (−0.38; p < 0.00001).	2B
			Reduction in HbA1c (−0.32%; p = 0.022), with more pronounced	

Lee et al. (2017) [36]	29 RCTs; n = 3324	VitD2/D3 vs. placebo; Route: Oral	results in patients with Baseline VitD deficiency (−0.45%, p < 0.001), shorter treatment duration (−0.30%; p < 0.001), and lower doses (−0.49%; p < 0.001) No statistically significant difference in fasting blood glucose. The results showed high heterogeneity between studies.	2B
Krul-Poel et al. (2017) [17]	23 RCTs; n = 1797	VitD2/D3 vs. placebo.	Benefit in HbA1c only in the subgroup of patients with HbA1c ≥9% (−1.4%; p = 0.01). Increase in fasting blood glucose only in the subgroup of patients with HbA1c ≥8% (0.36; p = 0.003). No significant effect of supplementation on HOMA-IR.	2B
Hu et al. (2019) [37]	19 RCTs; n = 1374	VitD2/D3 vs. placebo	Modest reduction in HbA1c (−0.17; p = 0.007) and HOMA-IR (−0.75; p < 0.001) only in shorter-duration interventions. No significant effect on fasting blood glucose.	2B
Sahebi et al. (2019) [38]	37 RCTs; n = 1673	VitD (including fortified food) vs. placebo or VitD and calcium supplementation	Significant reductions in HbA1c (p = 0.003), fasting blood glucose (p = 0.001), and HOMA-IR (p = 0.005).	2B
Mirhosseini et al. (2017) [39]	24 RCTs; n = 1528	VitD with or without calcium vs. placebo; Dose: ≥4,000 IU/day; Dosing regimen: Daily or weekly; Duration: ≥2 months	HbA1c: Significant reduction (−0.30%; p < 0.001), particularly in non-obese patients (−0.34; p < 0.001). Fasting blood glucose: Significant reduction (−4.9; p = 0.003), particularly in non-obese patients (−8.1; p = 0.003 vs. −5.0; p = 0.009 in obese patients) and in patients with VitD levels between 20 and 50 nmol/dL (−8.6, p = 0.001). HOMA-IR: Significant reduction (−0.66; p = 0.001), particularly in patients with VitD levels between 20 and 50 nmol/dL (−0.82; p = 0.01). Combined calcium supplementation appears to provide greater reductions in fasting blood glucose compared with VitD alone (−9.5; p = 0.004 vs. −6.7; p = 0.003).	2B

TABLE 5: Summary analysis of the meta-analyses included in the evidence-based review (EBR)

RCT, randomised controlled trial; VitD2, ergocalciferol; VitD3, cholecalciferol; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; HbA1c, glycated haemoglobin; VitD, vitamin D; BMI, body mass index; DM, diabetes mellitus; LOE, levels of evidence; SOR, strength of recommendations.

Discussion

The results of this evidence-based review indicate potential beneficial effects of vitamin D supplementation in adults with T2DM, although these effects remain inconsistent. Most of the included systematic reviews and meta-analyses reported statistically significant reductions in fasting glucose, HbA1c, and insulin resistance (HOMA-IR). However, these reviews overlap substantially in the primary trials they pooled, so their agreement should not be interpreted as independent confirmation. Moreover, the reported effects were generally small, particularly for HbA1c, and their clinical relevance remains uncertain.

Among the included clinical trials (Table *1*), most demonstrated a significant reduction in HbA1c following vitamin D supplementation, particularly the studies by Cojic et al. [23], Upreti et al. [24], Derosa et al. [25], Safarpour et al. [27], and Khan et al. [29], suggesting a potential benefit of this intervention in glycaemic control among individuals with T2DM. However, the results were not consistent, as Hu et al. [26] and El Hajj et al. [28] did not observe significant improvements, highlighting the heterogeneity of the available evidence.

A similar pattern was observed in the included systematic review without meta-analysis (Table *2*), which presented heterogeneous results, although it suggested a possible modest benefit in reducing HbA1c. Similarly, some of the included meta-analyses (Table *3*) identified modest but statistically significant reductions in HbA1c, generally ranging from 0.15% to 0.32% [16,31-33,37-39]. Although the magnitude of these effects is lower than that observed with the main antidiabetic therapies currently available, the findings suggest that vitamin D may play a complementary role in metabolic control, particularly among individuals with vitamin D deficiency or insufficiency. In this context, supplementation represents a relatively simple, safe, and low-cost strategy that may contribute to optimising glycaemic control when integrated into a multifactorial therapeutic approach.

One of the main obstacles to generalising these results is the high degree of methodological heterogeneity. Substantial differences can be observed in supplementation regimens, including variations in dosage (400 IU to >100,000 IU), pharmaceutical form (cholecalciferol vs. ergocalciferol), route of administration (oral vs.

intramuscular), frequency, and duration of the intervention. Additionally, the populations studied varied considerably with regard to age, body mass index, duration of diabetes, metabolic profile, and baseline glycaemic control. This aspect is particularly important, as the evidence suggests that the benefits were more evident in individuals with pre-existing vitamin D deficiency. This observation reinforces the hypothesis that the observed effects may result from the correction of a nutritional deficiency rather than from a direct pharmacological effect of vitamin D. This finding is consistent with the results of the meta-analyses by Chen et al. [16], Wu et al. [32], Farahmand et al. [33], and Lee et al. [36], which identified more pronounced effects among participants with lower baseline 25-hydroxyvitamin D concentrations. Some studies have also suggested a dose- and duration-dependent effect of supplementation, with better results observed in shorter interventions (particularly those lasting less than three months) and with the use of higher doses. However, these findings are not consistent across studies, preventing the establishment of standardised therapeutic recommendations regarding these parameters. Conversely, the considerable variability in therapeutic regimens, including daily, weekly, monthly, or bolus dosing schedules, makes it difficult to define an optimal supplementation protocol. The analysis by Zhang et al. [34] further suggests that higher doses may be more effective in short-term interventions, whereas lower doses appear to be appropriate for long-term maintenance strategies. These findings may reflect a more pronounced initial metabolic response following correction of vitamin D deficiency, followed by a plateau effect over time.

From a pathophysiological perspective, the results are biologically plausible. Vitamin D acts directly on receptors in pancreatic beta cells, influences calcium influx, which is essential for insulin secretion, and exerts anti-inflammatory effects that appear to optimise peripheral insulin sensitivity [12-15]. However, the translation of these mechanisms into consistent clinical benefits remains to be demonstrated.

The strength of the evidence is further limited by the variable quality of the primary studies. Although all reviews presented a level of evidence of 2 according to the SORT taxonomy [22], the strength of the recommendation remained classified as B, reflecting the persistent inconsistency of the available data.

Finally, it is important to consider the intrinsic limitations of this review. The evidence is affected by the potential overlap of populations among meta-analyses and by the predominant focus on intermediate outcomes rather than major clinical outcomes, such as microvascular or macrovascular complications. Additionally, the robustness of the conclusions is limited by the absence of an independent quantitative analysis and by the potential risk of publication bias, factors that should be considered when applying these findings to clinical practice.

Conclusions

Based on the evidence analysed, vitamin D supplementation in adults with T2DM shows heterogeneous results regarding its impact on glycaemic control. Although the intervention is biologically plausible and some studies demonstrate modest improvements in parameters such as fasting glucose, HbA1c, and insulin resistance (HOMA-IR), most systematic reviews and meta-analyses do not support a universal recommendation for the use of vitamin D as an adjunct therapy in the treatment of T2DM (SORT B). The benefits appear to be more consistent in subgroups with pre-existing vitamin D deficiency, suggesting that the observed effects are mainly related to the correction of a nutritional deficiency. Therefore, the available evidence does not support the existence of a generalisable benefit across the entire population with T2DM.

Current evidence does not support routine vitamin D supplementation in adults with T2DM solely for the purpose of optimising glycaemic control, and the decision to prescribe supplementation should be individualised and considered on a case-by-case basis. Larger randomised controlled trials of longer duration and greater methodological standardisation are required to clarify the role of vitamin D supplementation as an adjunct therapy in the treatment of T2DM in clinical practice.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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