



The Effect of Vitamin D Administration on Glycemic Control in Patients with Type 2 Diabetes Mellitus : A Systematic Review

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ABSTRACT

Introduction: Vitamin D deficiency is highly prevalent among patients with type 2 diabetes mellitus (T2DM) and has been linked to impaired insulin secretion and insulin resistance. However, the effects of vitamin D supplementation on glycemic control remain controversial.

Methods: This systematic review synthesized evidence from 80 randomized controlled trials, etc identified through structured screening. Studies enrolling adults with T2DM receiving vitamin D supplementation (cholecalciferol, ergocalciferol, calcitriol, or analogs) with a control group and reporting glycemic outcomes (HbA1c, fasting glucose, HOMA-IR) were included.

Results: HbA1c (WMD -0.20% to -0.32%), fasting plasma glucose (WMD -5.02 mg/dL), and HOMA-IR (WMD -0.42 to -0.66). However, effects were highly heterogeneous. Positive outcomes were predominantly observed in vitamin D-deficient (25(OH)D <30 nmol/L), poorly controlled (HbA1c >8%),

non-obese patients, particularly in Middle Eastern and South Asian populations. Null results consistently emerged from well-controlled European/American cohorts with baseline 25(OH)D near sufficiency. Effect modifiers included baseline vitamin D status, obesity, intervention duration (short-term >12 weeks showed benefit; long-term did not), dose (>2,000 IU/day more effective), and co-supplementation with calcium. Safety was excellent, with no clinically significant hypercalcemia.

Discussion: The apparent contradiction between positive meta-analyses and null individual trials is resolved by recognizing that benefits are restricted to specific subpopulations. Vitamin D supplementation likely improves glycemic control through enhanced insulin secretion and insulin sensitivity, but only when baseline deficiency is corrected and patients have sufficient glycemic headroom for improvement. Obese patients require higher doses due to adipose sequestration.

Conclusion: Vitamin D supplementation produces small but real improvements in glycemic control in T2DM patients who are vitamin D deficient, poorly controlled, and non-obese, or who receive dose-adjusted high-dose therapy. Routine supplementation is not recommended for vitamin D-replete or well-controlled patients. Future trials should focus on personalized dosing based on baseline 25(OH)D, BMI, and HbA1c.

Keywords: Vitamin D, type 2 diabetes mellitus, glycemic control, HbA1c, insulin resistance, systematic review

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by progressive β -cell dysfunction and insulin resistance, affecting hundreds of millions worldwide (Lee et al., 2017). Despite advances in pharmacotherapy, many patients fail to achieve optimal glycemic control, driving interest in adjunctive nutritional interventions (Farahmand et al., 2023). Vitamin D has emerged as a candidate because its receptor (VDR) is expressed on pancreatic β -cells and insulin-sensitive tissues, and vitamin D deficiency is disproportionately common in T2DM populations (Mirhosseini et al., 2017).

Background

Observational studies have consistently linked low serum 25-hydroxyvitamin D [25(OH)D] levels with higher HbA1c, fasting glucose, and insulin resistance (Chen et al., 2024). Biologically, vitamin D is thought to regulate insulin secretion via VDR-mediated transcription of insulin receptor genes, modulate calcium flux essential for exocytosis, and reduce systemic inflammation that impairs insulin signaling (Krul-Poel et al., 2015). However, randomized controlled trials (RCTs) have produced conflicting results. Some large well-powered trials—such as the SUNNY trial and the DDM2 study—reported no benefit (Angellotti et al., 2018; Gulseth et al., 2017), while others—particularly from regions with endemic deficiency—showed significant improvements (Upreti et al., 2018; Thenmozhi et al., 2024). This divergence has led to clinical uncertainty and inconsistent guideline recommendations (Kumar et al., 2024).

Research Gap

Previous systematic reviews and meta-analyses have aggregated data across heterogeneous populations, often concluding that vitamin D has a small but significant effect (Wu et al., 2017; Hu et al., 2019; Li et al., 2018). However, they have not fully reconciled why some high-quality RCTs report null findings while others do not. Specifically, the roles of baseline vitamin D status, obesity, glycemic control at baseline, intervention duration, and geographic/ethnic factors as effect modifiers require systematic synthesis (Lei et al., 2023; Cheng et al., 2024).

Novelty

This review provides a comprehensive synthesis of 80 studies—including the most recent trials from 2024–2025 (Zafar et al., 2025; Zhang et al., 2025; Probosari et al., 2025)—and explicitly constructs a population-specific framework to explain heterogeneity. Unlike prior work, we integrate effect modifiers across multiple domains (baseline deficiency, glycemic headroom, obesity, dose adequacy, and regimen type) into a coherent clinical model.

Objectives

The primary objective was to determine the effect of vitamin D administration on glycemic control in T2DM patients, measured by HbA1c, fasting glucose, and HOMA-IR. Secondary objectives were to identify effect modifiers, assess safety, and define patient subgroups most likely to benefit.

Hypothesis

We hypothesized that vitamin D supplementation improves glycemic control only in vitamin D-deficient, poorly controlled, non-obese patients, with null effects in replete or well-controlled populations.

Benefits of the Study

Clarifying these conditional effects will help clinicians avoid unnecessary supplementation in unlikely responders while targeting vitamin D therapy to patients who may achieve meaningful glycemic benefit, thereby improving resource allocation and reducing therapeutic inertia.

METHODS

Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy. This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

Criteria for Eligibility

This systematic review aims to evaluate The Effect of Vitamin D Administration on

Glycemic Control in Patients with Type 2 Diabetes Mellitus.

Screening

We screened in sources based on their abstracts that met these criteria:

- **Population - Age and Diabetes Type:** Does the study include adults (≥ 18 years) with confirmed Type 2 Diabetes Mellitus (and exclude gestational diabetes or Type 1 diabetes as the primary focus)?
- **Intervention Type:** Does the intervention involve vitamin D supplementation in any form (cholecalciferol, ergocalciferol, calcitriol, or other vitamin D analogs)?
- **Study Design - Control Group:** Does the study include a control group (placebo, no treatment, or standard diabetes care)?
- **Outcome Measures:** Does the study report at least one glycemic control outcome (HbA1c, fasting glucose, postprandial glucose, HOMA-IR, or glucose tolerance measures)?
- **Study Design Quality:** Is the study design a randomized controlled trial, etc?
- **Intervention Isolation:** Is vitamin D studied as a single intervention OR if part of a multi-component intervention, can the effects of vitamin D be isolated from other components?

Papers that failed any strict criterion were automatically excluded. For the remaining papers, we considered all screening questions together and made a holistic judgement about whether to screen in each paper.

Search Strategy

The keywords used for this research based PICO :

PICO Component	Keyword 1	Keyword 2	Keyword 3	Keyword 4
Population	Type 2 Diabetes Mellitus	T2DM adults	Vitamin D deficient diabetics	Poorly controlled

				T2DM
Intervention	Vitamin D supplementation	Cholecalciferol	Oral vitamin D	High-dose vitamin D
Comparison	Placebo-controlled	No treatment	Standard diabetes care	Metformin alone
Outcome	Glycemic control	HbA1c reduction	Fasting blood glucose	HOMA-IR improvement

The Boolean MeSH keywords inputted on databases for this research are: (*"Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM"*) AND (*"Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D"*) AND (*"Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone"*) AND (*"Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement"*)

Data extraction

- **Study Design:**

Extract study design and methodology, including:

- Type of study (RCT, etc.)
- Randomization and blinding procedures
- Sample size and power calculations
- Duration of follow-up
- Control group type (placebo, usual care, etc.)

- **Vitamin D Intervention:**

Extract all details about vitamin D administration specifically for improving glycemic control in type 2 diabetes patients, including:

- Type of vitamin D supplement (D2, D3, calcitriol, etc.)
- Dosage (IU or μg per dose)
- Frequency of administration (daily, weekly, etc.)
- Total duration of supplementation
- Route of administration (oral, injection)
- Co-interventions or combination treatments

- **Patient Characteristics:**

Extract baseline characteristics of type 2 diabetes patients that may influence vitamin D effects on glycemic control, including:

- Mean age and age range
- Gender distribution
- BMI/obesity status
- Baseline vitamin D status (25(OH)D levels and deficiency/insufficiency classification)
- Baseline glycemic control status (HbA1c levels, well-controlled vs poorly controlled)
- Duration of diabetes
- Current diabetes medications
- Ethnicity/geographic location

- **Glycemic Control Outcomes:**

Extract all glycemic control measures and results for vitamin D supplementation effects in type 2 diabetes patients, including:

- HbA1c levels (baseline and post-intervention values, mean difference, effect size)
- Fasting blood glucose (baseline and post-intervention values, mean difference)
- Insulin levels and insulin resistance measures (HOMA-IR, QUICKI, etc.)
- Other glucose metabolism markers (2-hour glucose, C-peptide, etc.)
- Statistical significance and confidence intervals
- Direction of effect (improvement, no change, worsening)

- **Effect Modifiers:**

Extract any subgroup analyses or factors that modified the effect of vitamin D supplementation on glycemic control in type 2 diabetes patients, including:

- Effects in vitamin D deficient vs sufficient patients
- Effects by BMI categories (obese vs non-obese)
- Effects by intervention duration (short-term vs long-term)
- Effects by vitamin D dose (high vs low dose)
- Effects by baseline glycemic control status
- Effects by ethnicity or geographic region
- Any other identified moderating factors

- **Safety Profile:**

Extract safety and adverse event data related to vitamin D supplementation in type 2 diabetes patients, including:

- Reported adverse events or side effects
- Hypercalcemia or vitamin D toxicity
- Drug interactions with diabetes medications
- Dropout rates due to adverse events
- Any safety concerns or contraindications noted

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	<i>("Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM") AND ("Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D") AND ("Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone") AND ("Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement")</i>	23
Semantic Scholar	<i>("Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM") AND ("Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D") AND ("Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone") AND ("Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement")</i>	250
Springer	<i>("Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM") AND ("Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D") AND ("Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone") AND ("Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement")</i>	259
Google Scholar	<i>("Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM") AND ("Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D") AND ("Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone") AND ("Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement")</i>	10,700
Wiley Online Library	<i>("Type 2 Diabetes Mellitus" OR "T2DM adults" OR "Vitamin D deficient diabetics" OR "Poorly controlled T2DM") AND ("Vitamin D supplementation" OR "Cholecalciferol" OR "Oral vitamin D" OR "High-dose vitamin D") AND ("Placebo-controlled" OR "No treatment" OR "Standard diabetes care" OR "Metformin alone") AND ("Glycemic control" OR "HbA1c reduction" OR "Fasting blood glucose" OR "HOMA-IR improvement")</i>	301

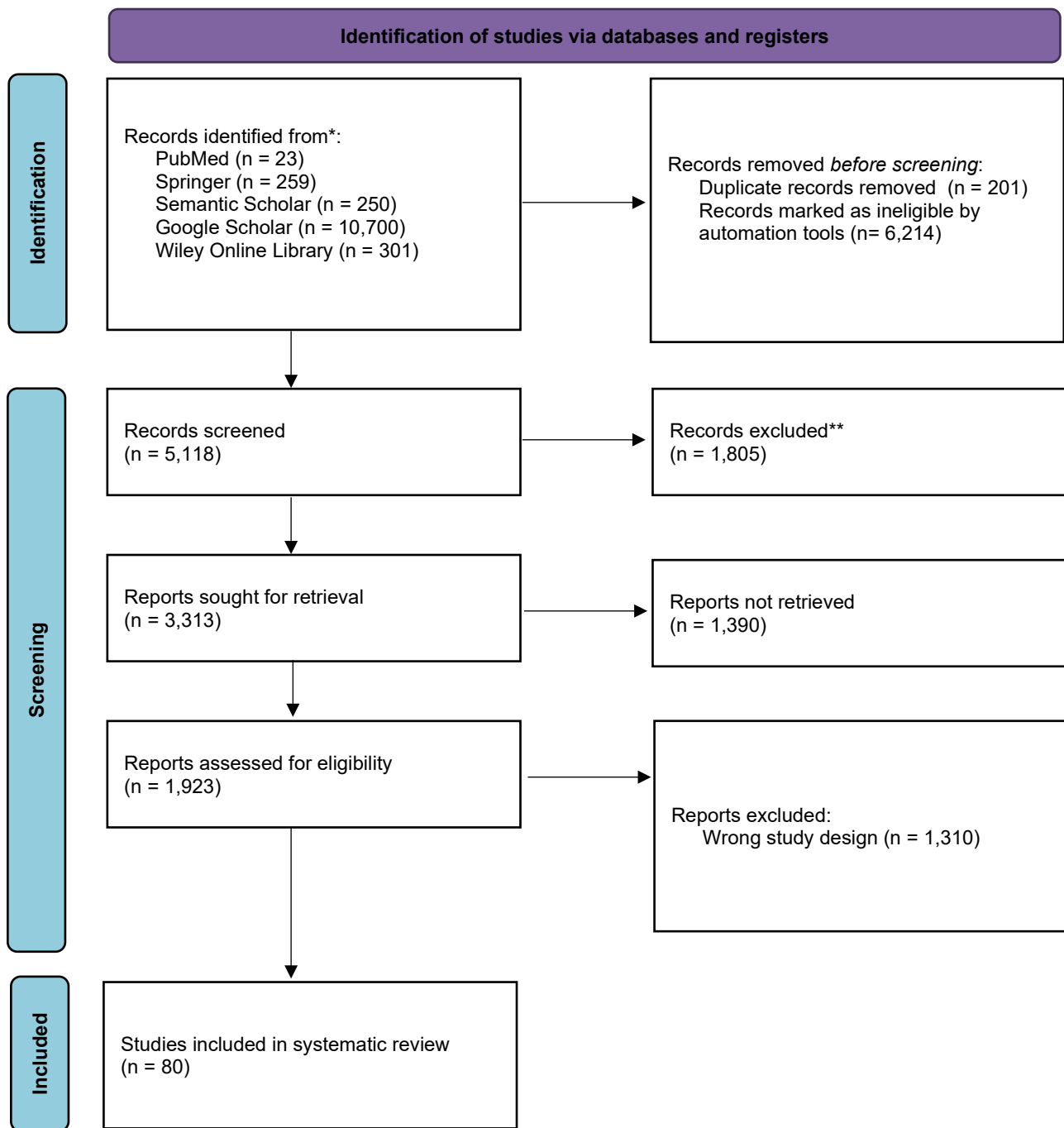


Figure 1. Article search flowchart

Risk of Bias

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
Hamid Nasri et al., 2013 [20]	Low	Low	Low	Unclear	Low	Low
V. Upreti et al., 2018 [21]	Low	Low	Low	Unclear	Low	Low
P. Thenmozhi et al., 2024 [22]	Low	Unclear	Unclear	Unclear	Low	Moderate
N. Nigil Haroon et al., 2015 [23]	Moderate	Moderate	Moderate	Low	Low	Moderate
Ramara Kadija Fonseca Santos et al., 2017 [24]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
Z. Hu et al., 2019 [13]	Low	Low	Low	Low	Low	Low
Chunhua Wu et al., 2017 [12]	Low	Low	Low	Low	Low	Low

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
Clare J. Lee et al., 2017 [1]	Low	Low	Low	Unclear	Low	Low
Xinyi Li et al., 2018 [25]	Low	Low	Low	Low	Low	Low
Dimitra Vasdeki et al., 2024 [26]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
Mohammad Ashraf Farahmand et al., 2023 [2]	Low	Low	Low	Low	Low	Low
R. Sahebi et al., 2019 [27]	Low	Unclear	Unclear	Unclear	Low	Moderate
Mohammed E. Al-Sofiani et al., 2015 [28]	Low	Low	Low	Low	Low	Low
J. Mitri et al., 2011 [29]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
Maryam Afraie et al., 2024 [30]	Low	Low	Low	Low	Low	Low
M. Soric et al., 2012 [31]	Low	Low	Low	Moderate	Low	Moderate
N. Mirhosseini et al., 2017 [3]	Low	Low	Low	Low	Low	Low
B. Nikooyeh et al., 2011 [32]	Low	Low	Low	Low	Low	Low
Wei Chen et al., 2024 [4]	Low	Low	Low	Low	Low	Low
J. Seida et al., 2014 [33]	Low	Low	Low	Unclear	Low	Low
A. Anyanwu et al., 2016 [34]	Low	Unclear	Unclear	Moderate	Low	Moderate
Xiu Juan Zhang et al., 2025 [35]	Low	Low	Low	Low	Low	Low
Y. Krul-Poel et al., 2015 (SUNNY) [5]	Low	Low	Low	Low	Low	Low

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
E. Yousefi Rad et al., 2014 [36]	Unclear	Moderate	Moderate	Unclear	Low	Moderate
A. Kazemi et al., 2022 [37]	Low	Low	Low	Low	Low	Low
Wei Gao et al., 2013 [38]	Unclear	Moderate	Moderate	Unclear	Unclear	Moderate
Majid Mozaffari et al., 2021 [39]	Low	Low	Low	Unclear	Low	Low
Maryam Emadzadeh et al., 2020 [40]	Low	Low	Low	Low	Low	Low
M. Casas et al., 2022 [41]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
Filip Max et al., 2024 [42]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
G. Derosa et al., 2020 [43]	Unclear	Moderate	Moderate	Low	Low	Moderate

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
M. Eftekhari et al., 2011 [44]	Low	Unclear	Unclear	Low	Low	Moderate
D. M. Khan et al., 2018 [45]	Low	Unclear	Unclear	Moderate	Low	Moderate
Dimitrios Papandreu et al., 2026 [46]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
P. Safarpour et al., 2018 [47]	Low	Low	Low	Low	Low	Low
E. Probosari et al., 2025 [48]	Low	Low	Low	Low	Low	Low
M. Witham et al., 2010 [19]	Low	Low	Low	Low	Low	Low
M. Mabhala et al., 2017 [49]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
Kuldeep Kumar et al., 2024 [8]	Low	Low	Low	Low	Low	Low

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
Talat Zafar et al., 2025 [11]	Low	Unclear	Unclear	Moderate	Low	Moderate
U. Kampmann et al., 2014 [50]	Low	Low	Low	Low	Low	Low
Milena Cojic et al., 2021 [51]	Low	Low	Low	Low	Low	Low
S. Jehle et al., 2014 [17]	Low	Low	Low	Low	Low	Low
H. Gulseth et al., 2017 [7]	Low	Low	Low	Low	Low	Low
Sahar Shahriari et al., 2018 [52]	Unclear	Unclear	Unclear	Moderate	Low	Moderate
Le Cheng et al., 2024 [10]	Low	Low	Low	Low	Low	Low
M. Z. Majeed Babar et al., 2017 [53]	Low	Unclear	Unclear	Moderate	Low	Moderate

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
H. Wagner et al., 2016 [54]	Low	Low	Low	Low	Low	Low
S. Ghavamzadeh et al., 2014 [55]	Low	Unclear	Unclear	Moderate	Low	Moderate
Edith Angellotti et al., 2018 (DDM2) [6]	Low	Low	Low	Low	Low	Low
P. Safarpour et al., 2020 [56]	Low	Low	Low	Low	Low	Low
Sanjana Mehta et al., 2021 [57]	Unclear	Moderate	Moderate	Moderate	Low	Moderate
Nima Baziar et al., 2014 [18]	Low	Unclear	Unclear	Moderate	Low	Moderate
Mahsa Omidian et al., 2019 [58]	Low	Low	Low	Low	Low	Low
Dikrayat A. Ahmed et al., 2024 [59]	Unclear	Unclear	Unclear	Moderate	Low	Moderate

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
M. Tabesh et al., 2014 [16]	Low	Low	Low	Low	Low	Low
Irfan Ahmad Khan et al., 2023 [60]	Unclear	Unclear	Unclear	Moderate	Low	Moderate
Hassan Abd El-Wahed & Soltan, 2019 [61]	Low	Unclear	Unclear	Low	Low	Moderate
F. Strobel et al., 2013 [62]	Low	Low	Low	Low	Low	Low
A. Breslavsky et al., 2013 [63]	Low	Low	Low	Low	Low	Low
Y. Yiu et al., 2013 [64]	Low	Low	Low	Low	Low	Low
E. Shaseb et al., 2016 [65]	Low	Low	Low	Moderate	Low	Moderate

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
Farhang Mirzavandi et al., 2020 [66]	Unclear	Moderate	Moderate	Moderate	Low	Moderate
F. A. Randhawa et al., 2017 [67]	Low	Unclear	Unclear	Moderate	Low	Moderate
Mrunalini K. Chokhandre et al., 2015 [68]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
T. Jafari et al., 2016 [69]	Low	Low	Low	Low	Low	Low
Margarete Almeida Freitas de Azevedo et al., 2022 [70]	Moderate	Moderate	Moderate	Unclear	Unclear	Moderate
M. C. Lo et al., 2018 [71]	Low	Low	Low	Low	Low	Low
Xingxing Lei et al., 2023 [14]	Low	Low	Low	Low	Low	Low

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
A. Sadiya et al., 2014 [72]	Low	Low	Low	Low	Low	Low
Suhani Sharma et al., 2025 [73]	Low	Low	Low	Low	Low	Low
O. Ryu et al., 2014 [9]	Low	Low	Low	Low	Low	Low
Iamila Nascimento Neves de Oliveira et al., 2024 [74]	Low	Low	Low	Low	Low	Low
J. I. Aguayo-Ruiz et al., 2020 [75]	Low	Low	Low	Moderate	Low	Moderate
Zhiyong Hu et al., 2022 [76]	Low	Low	Low	Low	Low	Low
R. Danasekaran et al., 2024 [77]	Low	Low	Low	Low	Low	Low
Jie Zhou et al., 2014 [78]	Low	Low	Low	Low	Low	Low

Study	Selection Bias	Performance Bias	Detection Bias	Attrition Bias	Reporting Bias	Overall Risk
S. Ali et al., 2019 [15]	Low	Moderate	Moderate	Low	Low	Moderate
Patricia Lemieux et al., 2019 [79]	Low	Low	Low	Low	Low	Low
S. Shab-Bidar et al., 2011 [80]	Low	Low	Low	Low	Low	Low

RESULTS

Characteristics of Included Studies

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Hamid Nasri et al., 2013 [20]	T2DM patients, n=60 [20]	Cholecalciferol 50,000 IU/week oral [20]	12 weeks [20]	HbA1c improved in males (p=0.0068) [20]
V. Upreti et al., 2018 [21]	T2DM + hypovitaminosis D, n=60 [21]	Oral vitamin D for 6 months [21]	6 months [21]	HbA1c improved (-0.27%, p=0.01) [21]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
P. Thenmozhi et al., 2024 [22]	T2DM, n=60 [22]	Vitamin D twice weekly for 12 weeks [22]	12 weeks [22]	FBS and HbA1c improved (p<0.05) [22]
N. Nigil Haroon et al., 2015 [23]	T2DM adults [23]	Various (D2, D3, calcitriol; oral/IM) [23]	≤3 months (short-term); >3 months (long-term) [23]	Mixed; short-term positive signals; long-term no effect [23]
Ramara Kadija Fonseca Santos et al., 2017 [24]	T2DM individuals [24]	Not specified [24]	Not specified [24]	Mostly no change; few studies positive [24]
Z. Hu et al., 2019 [13]	T2DM, n=747 intervention / 627 placebo [13]	Various [13]	Short-term vs long-term [13]	HbA1c, insulin resistance improved short-term; no long-term effect [13]
Chunhua Wu et al., 2017 [12]	T2DM [12]	D3 or analogues, oral [1]	Variable [12]	HbA1c improved (SMD -0.25); FBG no change overall [12]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Clare J. Lee et al., 2017 [1]	T2DM adults [1]	Oral D3 or D2; varied dose and frequency [1]	8 weeks–5.5 years; median 12 weeks [1]	HbA1c modestly improved (WMD –0.32%); FBG no change [1]
Xinyi Li et al., 2018 [25]	T2DM adults [25]	Oral D3 or D2 [25]	≥8 weeks (up to 6 months) [25]	HOMA-IR improved; FBG and HbA1c no overall change [25]
Dimitra Vasdeki et al., 2024 [26]	T2DM [26]	Not specified [26]	Not specified [26]	Overall positive conclusion [26]
Mohammad Ashraf Farahmand et al., 2023 [2]	T2DM adults [2]	Various; oral and IM [2]	≥8 weeks (up to 48 weeks) [2]	HbA1c (WMD –0.20%), FPG (–5.02 mg/dl), HOMA-IR (–0.42) improved [2]
R. Sahebi et al., 2019 [27]	T2DM and GDM patients [27]	Not specified [27]	Not specified [27]	FBG, HbA1c, HOMA-IR improved in T2DM [27]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Mohammed E. Al-Sofiani et al., 2015 [28]	T2DM with VDD, n=22 [28]	Cholecalciferol 5,000 IU/day oral [28]	12 weeks [28]	No HbA1c change; HOMA-%B improved [28]
J. Mitri et al., 2011 [29]	T2DM and at-risk populations [29]	Various [29]	Variable [29]	No effect in T2DM; improved IR in glucose intolerance [29]
Maryam Afraie et al., 2024 [30]	T2DM [30]	50,000 IU dose noted [30]	Not specified [30]	HbA1c (SMD -0.15) and FBS (SMD -0.28) improved [30]
M. Soric et al., 2012 [31]	Uncontrolled T2DM (HbA1c >7%), n=37 [31]	Cholecalciferol 2,000 IU/day oral [31]	12 weeks [31]	Subgroup analysis of HbA1c by baseline level [31]
N. Mirhosseini et al., 2017 [3]	T2DM adults [3]	Oral D (daily or weekly); min recommended 4,000 IU/day [3]	2–12 months [3]	HbA1c (-0.30%), FPG (-4.9 mg/dl), HOMA-IR (-0.66) improved [3]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
B. Nikooyeh et al., 2011 [32]	T2DM, n=90 [32]	D3-fortified yogurt 500 IU twice daily (1,000 IU/day) oral [32]	12 weeks [32]	HbA1c (−0.4%), FBG, HOMA-IR improved [32]
Wei Chen et al., 2024 [4]	T2DM adults [4]	Oral D3 or D2; varied dose and frequency [4]	≥8 weeks (up to 30 months) [4]	HbA1c (WMD −0.30%), FBG, HOMA-IR, fasting insulin improved [4]
J. Seida et al., 2014 [33]	T2DM and prediabetes/norm oglycemia [33]	D3 oral vs placebo [33]	Variable [33]	Not specified (outcomes include HOMA) [33]
A. Anyanwu et al., 2016 [34]	T2DM with VDD, poor control, n=42 [34]	Vitamin D3 3,000 IU/day oral [34]	12 weeks [34]	FPG improved; HbA1c reduction non-significant [34]
Xiu Juan Zhang et al., 2025 [35]	T2DM adults [35]	Oral D3 and D2; LDS/MDS/HDS/EHD S categories [35]	Variable [35]	LDS reduced HbA1c; EHDS reduced FBG; MDS improved fasting insulin [35]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Y. Krul-Poel et al., 2015 (SUNNY trial) [5]	T2DM without insulin, well-controlled [5]	Cholecalciferol 50,000 IU/month oral [5]	6 months [5]	No HbA1c effect overall; improvement only in severely deficient subgroup (<30 nmol/L) [5]
E. Yousefi Rad et al., 2014 [36]	T2DM, n=58 [36]	4,000 IU/day for 2 months [36]	2 months [36]	HbA1c improved (−0.53%, p<0.001); insulin improved [36]
A. Kazemi et al., 2022 [37]	T2DM adults [37]	Vitamin D among several supplements [37]	≥12 weeks subgroup [37]	HbA1c, FBS, HOMA-IR reduced (low certainty) [37]
Wei Gao et al., 2013 [38]	T2DM patients [38]	Not specified [38]	Not specified [38]	HbA1c improved (p=0.05); FPG, HOMA-IR, QUICKI no change [38]
Majid Mozaffari et al., 2021 [39]	T2DM with VD insufficiency (10–19.9 ng/ml), Iran [39]	D3 (type implied) 50,000 IU/week oral [39]	12 weeks [39]	HbA1c improved (−0.40%, p=0.002); FBS no change [39]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Maryam Emadzadeh et al., 2020 [40]	T2DM patients [40]	VD-fortified foods (varied dose) [40]	Variable [40]	FBG, fasting insulin, HOMA-IR improved; HbA1c no change [40]
M. Casas et al., 2022 [41]	T2DM patients [41]	Oral vitamin D (varied) [41]	Not specified [41]	HbA1c, glucose, insulin secretion improved [41]
Filip Max et al., 2024 [42]	T2DM patients [42]	High-dose VD (>4,000 IU) [42]	Not specified [42]	Potential glycemic improvement, inconsistent [42]
G. Derosa et al., 2020 [43]	T2DM with VDD, n=239 [43]	Vitamin D3 (dose not specified) oral [43]	6 months [43]	HbA1c improved (7.6%→7.1%, p<0.05); FBG improved [43]
M. Eftekhari et al., 2011 [44]	T2DM adults [44]	Calcitriol 0.5 µg/day oral [44]	12 weeks [44]	FBG increase attenuated; HbA1c increased in both groups; insulin secretion improved [44]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
D. M. Khan et al., 2018 [45]	T2DM, 40–70 years, Pakistan [45]	Oral vitamin D + metformin vs metformin alone [45]	3 months [45]	HbA1c significantly reduced ($p<0.05$) [45]
Dimitrios Papandreou et al., 2026 [46]	T2DM or prediabetes adults [46]	1,000–600,000 IU; varied duration 8–52 weeks [46]	8–52 weeks [46]	HbA1c and FBG improved in ~half of studies (VDD patients) [46]
P. Safarpour et al., 2018 [47]	T2DM, VDD, overweight/obese, Iran [47]	50,000 IU/week oral for 8 weeks [47]	8 weeks [47]	HbA1c improved (-0.81% , $p<0.001$); FBG and HOMA-IR no change [47]
E. Probosari et al., 2025 [48]	T2DM adults [48]	Various [48]	12 and 24 weeks [48]	HbA1c (-0.30 at 12 wk), insulin, HOMA-B improved; HOMA-IR improved at 24 wk [48]
M. Witham et al., 2010 [19]	T2DM, Scotland [19]	Single oral D3 dose: 100,000 or 200,000 IU [19]	16 weeks follow-up [19]	No change in HbA1c or HOMA-IR [19]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
M. Mabhala et al., 2017 [49]	T2DM patients [49]	Various [49]	Variable [49]	No significant effect on HbA1c, FPG, or HOMA-IR [49]
Kuldeep Kumar et al., 2024 [8]	Newly diagnosed T2DM, India [8]	D3 oral, dose-adapted by VD status (60,000 IU/wk then 1,000 IU BD or 1,000 IU BD for 6 months) [8]	6 months [8]	No significant improvement in HbA1c, FBS, PPBS, HOMA-IR [8]
Talat Zafar et al., 2025 [11]	T2DM (HbA1c >6.5%) [11]	D3 20,000 IU/wk x8 weeks then 1,000 IU/day x4 weeks [11]	12 weeks [11]	HbA1c improved (−0.76%, p<0.001) [11]
U. Kampmann et al., 2014 [50]	T2DM, hypovitaminosis D [50]	Colecalciferol 280 µg/day x2 weeks then 140 µg/day x10 weeks oral [50]	12 weeks [50]	No HbA1c change; borderline improvement in insulin secretion [50]
Milena Cojic et al., 2021 [51]	T2DM on metformin, Montenegro [51]	D3 (Endocrine Society guideline doses) oral, added to metformin [51]	6 months [51]	HbA1c improved at 3 and 6 months (∼−0.2 to −0.24%); FBG improved at 3 months only [51]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
S. Jehle et al., 2014 [17]	T2DM, >10 years duration, Switzerland [17]	D3 300,000 IU IM single dose ($\pm$ 150,000 IU booster at 3 months) [17]	6 months [17]	HbA1c course improved ($p=0.041$); HOMA-IR improved ($p=0.032$) [17]
H. Gulseth et al., 2017 [7]	T2DM with VDD (25(OH)D <50 nmol/L) [7]	D3 400,000 IU oral ($\pm$ 200,000 IU booster) [7]	6 months [7]	No change in glycemic control [7]
Sahar Shahriari et al., 2018 [52]	T2DM, VDD, overweight/obese [52]	50,000 IU/week for 8 weeks [52]	8 weeks [52]	FBS improved ($p=0.04$) [52]
Le Cheng et al., 2024 [10]	T2DM and at-risk populations [10]	Various [10]	Variable [10]	FBG, HbA1c, insulin, HOMA-IR improved, especially with VDD [10]
M. Z. Majeed Babar et al., 2017 [53]	T2DM, Pakistan [53]	D3 50,000 IU/week oral + standard antidiabetics [53]	12 weeks [53]	HbA1c improved in males ($p=0.01$) and females ($p=0.03$) [53]
H. Wagner et al., 2016 [54]	Prediabetes or diet-treated T2DM [54]	D3 30,000 IU/week oral for 8 weeks [54]	8 weeks [54]	No change in any glycemic control measure [54]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
S. Ghavamzadeh et al., 2014 [55]	T2DM adults [55]	D3 400 IU/day oral for 14 weeks [55]	14 weeks [55]	HbA1c: no change; TNF- α improved [55]
Edith Angellotti et al., 2018 (DDM2) [6]	T2DM, well-controlled (HbA1c $\leq$ 7.5%), US [6]	D3 4,000 IU/day oral for 48 weeks [6]	48 weeks [6]	No change in HbA1c, ISR, or insulin sensitivity [6]
P. Safarpour et al., 2020 [56]	T2DM, VDD, overweight/obese [56]	50,000 IU/week for 8 weeks [56]	8 weeks [56]	HbA1c decreased 1% ($p<0.001$); FBS, insulin, HOMA-IR no change [56]
Sanjana Mehta et al., 2021 [57]	T2DM, HbA1c $>10\%$, duration >10 years [57]	D 60,000 IU/week ($\pm$ calcium 1,000 mg/day) oral [57]	12 weeks [57]	HbA1c improved in groups 2 and 3; greater improvement with added calcium [57]
Nima Baziar et al., 2014 [18]	T2DM, VD insufficient/deficient, Iran [18]	D3 50,000 IU/week oral for 8 weeks [18]	8 weeks [18]	FBG improved ($p=0.014$); fasting insulin improved ($p=0.048$); HOMA-IR improved ($p=0.006$) [18]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Mahsa Omidian et al., 2019 [58]	T2DM with mild-moderate depression [58]	4,000 IU/day for 12 weeks [58]	12 weeks [58]	HbA1c change significantly greater in VD group (p<0.02) [58]
Dikrayat A. Ahmed et al., 2024 [59]	Newly diagnosed T2DM, Yemen [59]	Cholecalciferol 1,000 IU/day oral + metformin [59]	3 months [59]	HbA1c, FBG, RBG improved within group (p<0.01) [59]
M. Tabesh et al., 2014 [16]	T2DM with VD insufficiency, Iran [16]	D 50,000 IU/week ± calcium oral for 8 weeks [16]	8 weeks [16]	HbA1c (−0.70%), insulin, HOMA-IR, QUICKI improved with calcium+VD co-supplementation [16]
Irfan Ahmad Khan et al., 2023 [60]	T2DM with VDD + metformin+teneligliptin [60]	Cholecalciferol 60,000 IU (interval not specified) oral [60]	6 months [60]	HbA1c, FPG, PPG improved (p<0.05) [60]
Hassan Abd El-Wahed & Soltan, 2019 [61]	Uncontrolled T2DM, Egypt [61]	Cholecalciferol 2,000 IU/day oral + usual care [61]	3 months [61]	FBS, fasting insulin, HOMA-β, HOMA-IR improved (p<0.05) [61]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
F. Strobel et al., 2013 [62]	T2DM (non-insulin requiring), Germany [62]	D3 (Vigantol oil) ~1,904 IU/day once weekly oral [62]	6 months treatment + 6 months follow-up [62]	No significant change in HbA1c, FBG, insulin, or HOMA-IR [62]
A. Breslavsky et al., 2013 [63]	T2DM, Iran [63]	Vitamin D 1,000 IU/day oral for 12 months [63]	12 months [63]	No change in HbA1c, FBG, or HOMA-IR [63]
Y. Yiu et al., 2013 [64]	T2DM patients [64]	5,000 IU/day oral for 12 weeks [64]	12 weeks [64]	HbA1c no significant change [64]
E. Shaseb et al., 2016 [65]	T2DM with ischemic heart disease [65]	25(OH)D 300,000 IU IM single dose [65]	8 weeks [65]	HbA1c improved (-0.48%, p=0.04) [65]
Farhang Mirzavandi et al., 2020 [66]	T2DM with VDD [66]	200,000 IU IM x2 (weeks 0 and 4) [66]	8 weeks [66]	FBG improved (p=0.04); other glycemic markers not reported [66]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
F. A. Randhawa et al., 2017 [67]	T2DM with VDD, Pakistan [67]	D 200,000 IU/month oral for 3 months + metformin [67]	6 months (3 months VD + 3 months follow-up) [67]	No added benefit on HbA1c or FBG vs metformin alone [67]
Mrunalini K. Chokhandre et al., 2015 [68]	T2DM with nephropathy [68]	Cholecalciferol, calcitriol, paricalcitol; oral; varied doses [68]	8–24 weeks [68]	HbA1c: mostly no change; one calcitriol uncontrolled study showed improvement [68]
T. Jafari et al., 2016 [69]	T2DM postmenopausal women [69]	D3-fortified yogurt 2,000 IU/day for 12 weeks [69]	12 weeks [69]	Fasting insulin, HOMA-IR, HOMA-B, QUICKI improved; HbA1c and FBG no change [69]
Margarete Almeida Freitas de Azevedo et al., 2022 [70]	Elderly T2DM [70]	50,000 IU biweekly, 50,000 IU weekly, 2,000 IU/day (varied) [70]	12 weeks–6 months [70]	HOMA-IR, insulin improved; HbA1c improved in one study [70]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
M. C. Lo et al., 2018 [71]	Uncontrolled T2DM (HbA1c 7.5–10%) + VDD (<20 ng/mL) [71]	Ergocalciferol 50,000 IU/week x8 weeks then monthly x4 months [71]	~6 months [71]	No significant change in HbA1c [71]
Xingxing Lei et al., 2023 [14]	T2DM [14]	Oral VD; varied dose; conventional therapy continued [14]	Varied (short: <4 months; long: ≥4 months) [14]	FBG (SMD -0.17), fasting insulin (SMD -0.27), HOMA-IR (SMD -0.44) improved [14]
A. Sadiya et al., 2014 [72]	T2DM, obese, UAE (Emirati) [72]	D3 6,000 IU/day x3 months then 3,000 IU/day x3 months oral [72]	6 months [72]	No change in HbA1c, FBG, or C-peptide [72]
Suhani Sharma et al., 2025 [73]	T2DM and prediabetes populations [73]	D3 (2,000–4,000 IU/day); one IM 300,000 IU regimen [73]	12 weeks–2.5 years [73]	Small improvements in HbA1c (-0.39%), FBG (-0.36 mmol/L), HOMA-IR (-0.63) in meta-analyses [73]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
O. Ryu et al., 2014 [9]	T2DM, VDD, Korea [9]	D3 2,000 IU/day + calcium 200 mg/day oral for 24 weeks [9]	24 weeks [9]	No change in HbA1c, FBG, or HOMA-IR [9]
Iamila Nascimento Neves de Oliveira et al., 2024 [74]	Obese or T2DM in Brazil, Europe, US [74]	Up to 2,000 IU/day [74]	Not specified [74]	No overall effect; subgroup improvement with $\leq 2,000$ IU/day [74]
J. I. Aguayo-Ruiz et al., 2020 [75]	T2DM, Western Mexico [75]	D3 1,000 IU/day $\pm$ vitamin K2 [75]	3 months [75]	FBG improved ($p < 0.001$); HOMA-IR non-significant decrease [75]
Zhiyong Hu et al., 2022 [76]	T2DM adults 50–70 years [76]	D3 800 IU/day oral for 30 months [76]	30 months [76]	FBG improved in subgroup achieving 25(OH)D ≥ 30 ng/mL; fasting insulin and HOMA-IR improved overall [76]
R. Danasekaran et al., 2024 [77]	T2DM, HbA1c $\geq 7\%$, VDD (< 12 ng/mL) [77]	VD supplementation (dose not specified) [77]	3 and 6 months [77]	No significant improvement in glycemic control [77]

Study	Population	Vitamin D Intervention	Duration	Primary Glycemic Outcome Direction
Jie Zhou et al., 2014 [78]	T2DM adults 30–75 years [78]	Calcitriol 0.50 µg/day oral for 12 weeks [78]	12 weeks [78]	HbA1c, FPG, HOMA-IR, FPI all improved (p<0.05) [78]
S. Ali et al., 2019 [15]	T2DM with VDD, Egypt [15]	D3: 4,000 IU/day oral; 50,000 IU/week oral; 300,000 IU IM single dose; vs control [15]	3 months [15]	Oral daily and weekly improved HbA1c, FBG, insulin, HOMA-IR; IM dose non-significant [15]
Patricia Lemieux et al., 2019 [79]	At-risk for diabetes or newly diagnosed T2DM [79]	D3 5,000 IU/day oral for 6 months [79]	6 months [79]	Peripheral insulin sensitivity (M-value) and disposition index improved; HbA1c, FBG no change [79]
S. Shab-Bidar et al., 2011 [80]	T2DM adults, Iran [80]	D3-fortified yogurt 1,000 IU/day oral for 12 weeks [80]	12 weeks [80]	FBG, fasting insulin, QUICKI improved; HbA1c no significant between-group difference [80]

Effects of Vitamin D Supplementation on Glycemic Outcomes

Primary Glycemic Outcomes

The following table summarizes the primary quantitative results reported by each source, with specific effect estimates where available.

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Hamid Nasri et al., 2013 [20]	−0.89% in males [20]	Not reported	Not reported	p=0.0068 (males) [20]
V. Upreti et al., 2018 [21]	−0.27% (7.29%→7.02%) [21]	−28.8 mg/dl (131.4→102.6) [21]	Postprandial glucose −61.2 mg/dl [21]	p=0.01 (HbA1c); p=0.04 (FPG); p<0.001 (PPPG) [21]
P. Thenmozhi et al., 2024 [22]	7.2→5.94% [22]	128.67→119.23 mg/dl [22]	Not reported	p<0.05 (within); p<0.001 (between) [22]
N. Nigil Haroon et al., 2015 [23]	Mixed; short-term improvements; long-term no effect [23]	Mixed [23]	Mixed; some short-term HOMA improvements [23]	Varies per study [23]
Ramara Kadija Fonseca Santos et al., 2017 [24]	No change in most; reduction in few [24]	Reduction in few [24]	Some HOMA-IR and QUICKI changes [24]	Not pooled [24]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Z. Hu et al., 2019 [13]	SMD -0.17 (95% CI -0.29 to -0.05) [13]	Not separately reported	HOMA-IR SMD -0.75 (-0.97 to -0.53); insulin SMD -0.57 (-0.78 to -0.35) [13]	p<0.05 for all (short-term) [13]
Chunhua Wu et al., 2017 [12]	SMD -0.25 (-0.45 to -0.05) [12]	SMD -0.14 (-0.31 to 0.03); NS [12]	Not pooled separately [12]	HbA1c significant; FBG NS [12]
Clare J. Lee et al., 2017 [1]	WMD -0.32% (-0.53 to -0.10) [1]	WMD -2.33 mg/dl (-6.62 to 1.95); NS [1]	Not pooled [1]	HbA1c p=0.022; FBG p=0.542 [1]
Xinyi Li et al., 2018 [25]	WMD -0.11 to -0.14%; NS overall [25]	WMD -3.59 mg/dl; NS overall [25]	HOMA-IR SMD -0.57 (-1.09 to -0.04) [25]	HbA1c NS; FBG NS; HOMA-IR p≈0.03 [25]
Mohammad Ashraf Farahmand et al., 2023 [2]	WMD -0.20% (-0.29 to -0.11) [2]	WMD -5.02 mg/dl (-6.75 to -3.28) [2]	HOMA-IR WMD -0.42 (-0.76 to -0.07) [2]	All p<0.05 [2]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
R. Sahebi et al., 2019 [27]	95% CI -1.719 to -0.361; p=0.003 [27]	95% CI -0.526 to -0.136; p=0.001 [27]	HOMA-IR improved; p=0.005 [27]	Significant [27]
Mohammed E. Al-Sofiani et al., 2015 [28]	No change (p=0.5) [28]	No change [28]	HOMA-%B improved (p=0.03) [28]	HbA1c NS; β -cell function p=0.03 [28]
Maryam Afraie et al., 2024 [30]	SMD -0.15 (-0.29 to -0.20) [30]	SMD -0.28 (-0.40 to -0.15) [30]	Not reported [30]	p<0.001 for both [30]
N. Mirhosseini et al., 2017 [3]	-0.30% (95% CI -0.45 to -0.15) [3]	-4.9 mg/dl (95% CI -8.1 to -1.7) [3]	HOMA-IR -0.66 (95% CI -1.06 to -0.26) [3]	All p<0.01 [3]
B. Nikooyeh et al., 2011 [32]	-0.4% in VD-fortified yogurt groups [32]	-12.9 mg/dl (DY group) [32]	HOMA-IR -0.6 [32]	p<0.001 (HbA1c); p=0.015 (FBG); p=0.001 (HOMA-IR) [32]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Wei Chen et al., 2024 [4]	WMD -0.30% (95% CI -0.43 to -0.18) [4]	WMD ~-0.49 mmol/L [4]	HOMA-IR WMD -0.39 (-0.64 to -0.14); fasting insulin WMD ~-3.45 μ IU/ml [4]	All significant [4]
Mohammed E. Al-Sofiani et al., 2015 [28]	No change [28]	No change [28]	HOMA-%B improved [28]	HOMA-%B p=0.03 [28]
A. Anyanwu et al., 2016 [34]	Non-significant reduction (p=0.127) [34]	Significant improvement [34]	Not reported [34]	FPG significant; HbA1c NS [34]
Xiu Juan Zhang et al., 2025 [35]	LDS significantly decreased HbA1c [35]	EHDS reduced FBG [35]	MDS: fasting insulin MD -4.76 (-8.91 to -0.61) [35]	Dose-specific significance [35]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Y. Krul-Poel et al., 2015 [5]	No effect overall ($\beta=+0.4$, $p=0.42$); −3.1 mmol/mol in severely deficient subgroup ($p=0.04$) [5]	No change [5]	HOMA-IR no change [5]	Overall NS [5]
E. Yousefi Rad et al., 2014 [36]	7.29%→6.76% (−0.53%, $p<0.001$) [36]	Not reported [36]	Insulin −1.69 μ IU/mL ($p=0.048$); HOMA-IR NS [36]	HbA1c $p<0.001$ [36]
A. Kazemi et al., 2022 [37]	Reduced (low certainty) [37]	Reduced (low certainty) [37]	HOMA-IR reduced; insulin NS [37]	Low certainty [37]
Wei Gao et al., 2013 [38]	Reduced ($p=0.05$) [38]	No change [38]	HOMA-IR and QUICKI no change [38]	HbA1c $p=0.05$ only [38]
Majid Mozaffari et al., 2021 [39]	7.66%→7.26% (−0.40%, $p=0.002$) [39]	No change ($p=0.245$) [39]	Not measured [39]	HbA1c $p=0.002$; FBS NS [39]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Maryam Emadzadeh et al., 2020 [40]	No change (MD 0.034) [40]	-2.77 (-5.44 to -0.11) [40]	HOMA-IR -1.61 (-3.14 to -0.08); fasting insulin -2.94 [40]	FBG and insulin significant; HbA1c NS [40]
G. Derosa et al., 2020 [43]	7.6%→7.1% (p<0.05) [43]	Significant reduction [43]	Not reported [43]	p<0.05 [43]
M. Eftekhari et al., 2011 [44]	Increased in both groups [44]	Increase attenuated in calcitriol group [44]	Insulin secretion (HOMA-%β) improved (p=0.009) [44]	HbA1c worsened in both [44]
D. M. Khan et al., 2018 [45]	Significant reduction (p<0.05) [45]	Not reported [45]	Not reported [45]	p<0.05 [45]
P. Safarpour et al., 2018 [47]	-0.81% vs placebo (p<0.001) [47]	No change (adjusted p=0.130) [47]	HOMA-IR no change; QUICKI no change [47]	HbA1c p<0.001; others NS [47]
E. Probosari et al., 2025 [48]	MD -0.30 (95% CI -0.54 to -0.06) at 12 weeks [48]	Not pooled [48]	Insulin MD -3.59; HOMA-IR MD -0.38 at 24 weeks [48]	Significant at respective timepoints [48]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
M. Witham et al., 2010 [19]	No change (100,000 IU p=0.81; 200,000 IU p=0.71) [19]	Not reported [19]	HOMA-IR no change [19]	All NS [19]
M. Mabhala et al., 2017 [49]	MD -0.06 mmol/l (95% CI -0.26 to 0.14); NS [49]	MD -0.03 mmol/l (95% CI -0.69 to 0.63); NS [49]	MD -1.51 mmol/l (95% CI -3.61 to 0.60); NS [49]	All NS [49]
Kuldeep Kumar et al., 2024 [8]	No change (p=0.263) [8]	No change (p=0.734) [8]	HOMA-IR no change (p=0.899) [8]	All NS [8]
Talat Zafar et al., 2025 [11]	9.51%→7.81% vs 9.55%→8.57% (MD -0.76%, p<0.001) [11]	Not reported [11]	Not reported [11]	p<0.001 [11]
U. Kampmann et al., 2014 [50]	No significant change [50]	Not reported [50]	Borderline improvements in insulin secretion [50]	NS for HbA1c; borderline for secretion [50]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Milena Cojic et al., 2021 [51]	−0.20% at 3 months (p=0.050); −0.24% at 6 months (p=0.045) [51]	−0.59 mmol/L at 3 months (p=0.018); NS at 6 months [51]	HOMA-IR no change; fasting insulin no change [51]	HbA1c significant; HOMA-IR NS [51]
S. Jehle et al., 2014 [17]	HbA1c course: D3 +2.9% vs placebo +6.9% (p=0.041) [17]	No change (p=0.422) [17]	HOMA-IR −12.8% vs +10.0% (p=0.032) [17]	HbA1c p=0.041; HOMA-IR p=0.032 [17]
H. Gulseth et al., 2017 [7]	No change [7]	Not separately reported [7]	No change (p=0.52) [7]	All NS [7]
Sahar Shahriari et al., 2018 [52]	Not specifically reported [52]	FBS decreased (p=0.04) [52]	Not reported [52]	FBS p=0.04 [52]
Le Cheng et al., 2024 [10]	ES −0.11 (95% CI −0.20 to −0.02) [10]	ES −0.56 (95% CI −1.00 to −0.11) [10]	HOMA-IR ES −0.37; insulin ES −0.38 [10]	All significant [10]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
M. Z. Majeed Babar et al., 2017 [53]	Males: 6.65% vs 8.34% (p=0.01); females: 6.81% vs 8.04% (p=0.03) post-treatment [53]	Not reported [53]	Not reported [53]	Significant in both sexes [53]
H. Wagner et al., 2016 [54]	No change [54]	No change [54]	No change [54]	All NS [54]
S. Ghavamzadeh et al., 2014 [55]	No change [55]	Not reported [55]	Not reported [55]	NS [55]
Edith Angellotti et al., 2018 [6]	No change at any timepoint [6]	No change [6]	ISR, ISI-Matsuda, ISSI-2 no change [6]	All NS [6]
P. Safarpour et al., 2020 [56]	-1% (p<0.001) [56]	No change [56]	HOMA-IR and QUICKI no change [56]	HbA1c p<0.001 [56]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Sanjana Mehta et al., 2021 [57]	Group 2 (VD only): 14.64%→13.99% ; Group 3 (VD+calcium): 14.05%→12.04% [57]	Not reported [57]	Not reported [57]	p<0.05 [57]
Nima Bazar et al., 2014 [18]	HbA1c not assessable (imbalanced baseline) [18]	150.29→135.23 mg/dl (p=0.045); between-group p=0.014 [18]	Fasting insulin p=0.048; HOMA-IR p=0.006 [18]	FBG, insulin, HOMA-IR all significant [18]
Mahsa Omidian et al., 2019 [58]	Greater change vs placebo (p<0.02) [58]	Not reported [58]	Not reported [58]	p<0.02 [58]
Dikrayat A. Ahmed et al., 2024 [59]	10.37%→10.07% (-0.30%, p=0.001) [59]	186.40→169.33 mg/dl (-17.07, p=0.000) [59]	Not measured [59]	Significant within-group [59]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
M. Tabesh et al., 2014 [16]	-0.70% (p=0.02) with calcium+VD co-supplementation [16]	Not reported [16]	HOMA-IR -0.46 (p=0.001); QUICKI +0.025 (p=0.004); HOMA-B improved [16]	All significant with co-supplementation [16]
Irfan Ahmad Khan et al., 2023 [60]	7.21% vs 7.48% (p=0.015) at 3 months [60]	129.86 vs 136.12 mg/dl (p=0.002) [60]	Not reported [60]	p<0.05 for all [60]
Hassan Abd El-Wahed & Soltan, 2019 [61]	Significant improvement (p<0.05) [61]	Significant improvement (p<0.05) [61]	HOMA-IR and HOMA-β improved (p<0.05) [61]	Significant [61]
F. Strobel et al., 2013 [62]	No change (p=0.681) [62]	No change (p=0.624) [62]	HOMA-IR no change (p=0.821); insulin no change [62]	All NS [62]
A. Breslavsky et al., 2013 [63]	No change [63]	No change [63]	HOMA-IR no change [63]	All NS [63]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Y. Yiu et al., 2013 [64]	No change (p>0.05) [64]	Not reported [64]	Not reported [64]	NS [64]
E. Shaseb et al., 2016 [65]	8.2%→7.7% (-0.48%, p=0.04) [65]	186.5→165.1 mg/dl [65]	Not reported [65]	p=0.04 [65]
Farhang Mirzavandi et al., 2020 [66]	Not reported [66]	Significant decrease (p=0.04) [66]	Not reported [66]	p=0.04 [66]
F. A. Randhawa et al., 2017 [67]	No benefit vs metformin alone [67]	No difference [67]	Not measured [67]	NS for between-group differences [67]
Mrunalini K. Chokhandre et al., 2015 [68]	No change (cholecalciferol RCTs); one calcitriol study -0.8% (p=0.01) [68]	Not reported [68]	Not reported [68]	Mostly NS [68]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
T. Jafari et al., 2016 [69]	No change [69]	No change [69]	Fasting insulin, HOMA-IR, HOMA-B, QUICKI all improved [69]	Insulin parameters significant; HbA1c and FBG NS [69]
Margarete Almeida Freitas de Azevedo et al., 2022 [70]	Improved in one study [70]	Improved generally [70]	HOMA-IR and insulin improved [70]	Not pooled numerically [70]
M. C. Lo et al., 2018 [71]	8.8%→8.7% (NS, p=0.692) [71]	Not reported [71]	Not reported [71]	NS [71]
Xingxing Lei et al., 2023 [14]	Not pooled [14]	SMD -0.17 (95% CI -0.30 to -0.04) [14]	Fasting insulin SMD -0.27; HOMA-IR SMD -0.44 [14]	Significant [14]
A. Sadiya et al., 2014 [72]	No change [72]	No change [72]	Not reported [72]	NS [72]
Suhani Sharma et al., 2025 [73]	-0.39% (p<0.01) in pooled meta-analyses [73]	-0.36 mmol/L (p<0.05) [73]	HOMA-IR -0.63 (p<0.05) [73]	Significant for all in meta-analyses [73]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
O. Ryu et al., 2014 [9]	No change (p=0.280) [9]	No change (p=0.919) [9]	HOMA-IR no change (p=0.981) [9]	All NS [9]
Iamila Nascimento Neves de Oliveira et al., 2024 [74]	No overall effect; improvement with $\leq 2,000$ IU/day subgroup [74]	No overall effect [74]	No effect on HOMA or insulin [74]	NS overall [74]
J. I. Aguayo-Ruiz et al., 2020 [75]	Not reported [75]	Significant decrease (p<0.001) [75]	HOMA-IR decrease NS for VD alone [75]	FBG p<0.001 [75]
Zhiyong Hu et al., 2022 [76]	Not reported [76]	FBG improved in subgroup achieving ≥ 30 ng/mL (p=0.049) [76]	Fasting insulin and HOMA-IR improved overall [76]	Significant [76]
R. Danasekaran et al., 2024 [77]	No significant change [77]	Not reported [77]	Not reported [77]	NS [77]

Study	HbA1c Effect	FBG/FPG Effect	Insulin/HOMA-IR Effect	Statistical Significance
Jie Zhou et al., 2014 [78]	Improvement (MD -0.1 ± 0.6 vs -0.03 ± 0.94 , $p < 0.05$) [78]	FPG MD -1.2 ± 2.3 mmol/L ($p < 0.05$) [78]	HOMA-IR MD -0.91 ± 2.63 ($p < 0.05$) [78]	All significant [78]
S. Ali et al., 2019 [15]	Daily: -0.81% ($p < 0.001$); Weekly: -0.82% ($p < 0.001$); IM: -0.34% (NS) [15]	Daily: -20.90 mg/dl ($p < 0.001$); Weekly: -23.0 mg/dl ($p < 0.001$); IM: NS [15]	HOMA-IR: daily -0.71 ($p = 0.002$); weekly -0.87 ($p < 0.001$); IM NS [15]	Oral doses significant; IM NS [15]
Patricia Lemieux et al., 2019 [79]	No change [79]	No change [79]	M-value $+0.92$ ($p = 0.009$); disposition index improved ($p = 0.039$) [79]	Insulin sensitivity significant; glycemic markers NS [79]
S. Shab-Bidar et al., 2011 [80]	Within-group improvement; between-group NS [80]	-26.6 ± 45.6 mg/dl vs $+6.2 \pm 44.8$ ($p < 0.0001$) [80]	Fasting insulin improved ($p < 0.0001$); QUICKI improved ($p < 0.0001$) [80]	FBG, insulin, QUICKI significant; HbA1c between-group NS [80]

The overall picture from the primary studies and meta-analyses is one of modest but frequently statistically significant improvements in several glycemic parameters with vitamin D

supplementation, alongside a substantial number of null findings. Among the meta-analyses that pooled the largest numbers of trials, the consistent direction is toward small beneficial effects. The most comprehensive meta-analysis by Farahmand et al. (2023), which included 46 RCTs, reported pooled WMDs of -0.20% for HbA1c, -5.02 mg/dl for FPG, and -0.42 for HOMA-IR, all statistically significant [2]. Similarly, Wei Chen et al. (2024), drawing on 39 RCTs, reported significant reductions in HbA1c (WMD -0.30%), FBG, HOMA-IR, and fasting insulin [4]. Mirhosseini et al. (2017) and Le Cheng et al. (2024) found consistent pooled reductions in HbA1c, FPG, and HOMA-IR [3, 10]. Nevertheless, substantive heterogeneity pervades the primary literature, with a large minority of well-designed trials reporting no glycemic benefit at all.

Among individual RCTs with null results, several were rigorously conducted. The SUNNY trial (Krul-Poel et al., 2015), which enrolled 275 well-controlled Dutch T2DM patients receiving monthly 50,000 IU D3 for six months, found no HbA1c effect in the overall cohort [5]. The DDM2 study (Angellotti et al., 2018), conducted with 127 stable US patients receiving 4,000 IU/day for 48 weeks, similarly found no change in HbA1c, insulin secretion rate, or insulin sensitivity, despite a robust rise in 25(OH)D [6]. Gulseth et al. (2017), using high-dose loading (400,000–600,000 IU total) in 62 Norwegians with confirmed vitamin D deficiency, reported no improvement in insulin sensitivity by euglycemic clamp [7]. Kuldeep Kumar et al. (2024), in a 140-patient Indian trial over six months, found no significant benefit on HbA1c, FBS, PPBS, fasting insulin, or HOMA-IR [8].

The magnitude of effect where improvements are reported is generally small and likely of marginal clinical significance. The HbA1c reductions in most meta-analyses range from 0.15% to 0.32% [1–4, 30], a threshold substantially below the 0.5% commonly considered clinically meaningful. Among individual positive trials, reductions ranged from approximately 0.20% to 0.89% [11, 17, 20, 47], with larger effects tending to occur in trials with poorly controlled baseline glycemia or more severely vitamin D-deficient populations. The calcitriol study by Zhou et al. (2014) using 0.5 $\mu\text{g/day}$ in 164 T2DM patients demonstrated broad improvements across HbA1c, FPG, fasting insulin, and HOMA-IR [78], while the active metabolite study by Eftekhari et al. (2011) paradoxically showed HbA1c increases in both calcitriol and placebo groups, though it did

attest to calcitriol's capacity to improve insulin secretion (HOMA-% β) and prevent the rise in FPG seen in controls [44].

A notable pattern across studies is that HOMA-IR and other insulin resistance indices frequently improve even when HbA1c and FBG do not. Several trials and meta-analyses—including Xinyi Li et al. (2018), Zhiyong Hu et al. (2022), T. Jafari et al. (2016), and Lemieux et al. (2019)—reported significant improvements in insulin sensitivity or HOMA-IR without corresponding HbA1c reductions [25, 69, 76, 79]. This dissociation suggests that vitamin D may primarily affect insulin secretion or early components of insulin action rather than downstream glycemic regulation, particularly in patients already receiving oral glucose-lowering agents whose effects may mask further HbA1c changes.

Effect Modifiers and Subgroup Analyses

A consistent theme across the meta-analyses and primary studies is that effects are highly context-dependent, and several effect modifiers have been identified with reasonable consistency.

Vitamin D Status at Baseline

The most reproducible effect modifier is baseline vitamin D status. Studies specifically enrolling vitamin D–deficient participants more frequently demonstrated glycemic benefit. Chunhua Wu et al. (2017) found that the benefit on both HbA1c and FBG was restricted to the subgroup with baseline 25(OH)D deficiency [12]. Xinyi Li et al. (2018) showed FBG improvements were significant only when baseline 25(OH)D was below 50 nmol/L (deficiency) [25]. Farahmand et al. (2023) confirmed that the largest FPG reductions occurred in deficient patients [2]. The SUNNY trial's exploratory subgroup analysis found HbA1c improvement only among patients with 25(OH)D below 30 nmol/L (severely deficient), while patients in the mildly deficient range showed no benefit [5]. By contrast, the DDM2 trial—where baseline 25(OH)D averaged 26.6 ng/mL with only 26% meeting strict deficiency criteria—found no glycemic effect [6]. The systematic review by Papandreou et al. (2026) synthesized this pattern explicitly, concluding that supplementation may improve glycemic control in vitamin D–deficient individuals but appears ineffective in those with sufficient levels [46]. The umbrella review by Le Cheng et al. (2024) further quantified this

interaction, reporting that among deficient patients, effect sizes for FBG, HbA1c, and HOMA-IR were nearly double those seen in the overall T2DM population [10].

Intervention Duration

Duration is a second well-established modifier. The meta-analysis by Hu et al. (2019) found significant improvements in HbA1c, insulin resistance, and insulin levels with short-term supplementation but no significant differences during long-term follow-up [13]. This pattern is supported by Wei Chen et al. (2024), who found that FBG, HbA1c, and HOMA-IR reductions were most prominent at durations of three months or less, while fasting insulin improvements appeared more consistent with longer interventions [4]. The network meta-analysis by Lei et al. (2023) similarly found that effects on FBG, fasting insulin, and HOMA-IR were significant in trials lasting less than four months but not in those of four months or more [14]. The systematic review by Nigil Haroon et al. (2015) categorized trials as short-term (≤ 3 months) and long-term (> 3 months), noting positive signals in the former and predominantly null findings in the latter [23].

Vitamin D Dose and Formulation

Several meta-analyses identified dose as a modifier. Farahmand et al. (2023) reported the greatest glycemic benefit with doses above 2,000 IU/day [2], and the subgroup analysis by Chunhua Wu et al. (2017) linked HbA1c and FBG reductions specifically to the vitamin D–deficient and non-obese subgroups using higher doses [12]. The network meta-analysis by Zhang et al. (2025) demonstrated a dose-dependent increase in serum 25(OH)D across low, medium, high, and extremely high dose strata, but interestingly found HbA1c reductions linked to low-dose supplementation and FBG reductions associated with the extremely high dose category [35, 35]. The trial by Ali et al. (2019), which directly compared daily oral 4,000 IU, weekly oral 50,000 IU, and a single 300,000 IU intramuscular injection, found that both oral regimens produced significant and similar improvements in HbA1c, FBG, insulin, and HOMA-IR, while the parenteral single dose did not produce significant changes [15]. This finding suggests that sustained elevation of 25(OH)D through regular oral dosing is more relevant than peak vitamin D concentrations, a point further supported by Mirhosseini et al. (2017), who noted that trials with monthly or single bolus dosing often failed to achieve sufficiency throughout the study period [3].

The fortified food studies represent a special case: two Iranian trials using vitamin D–fortified yogurt (Nikooyeh et al., 2011; Shab-Bidar et al., 2011) found significant improvements in FBG, insulin, and HOMA-IR at 1,000 IU/day—a dose that would conventionally be considered low—and the meta-analysis of food fortification by Emadzadeh et al. (2020) confirmed significant improvements in FBG, fasting insulin, and HOMA-IR with fortified foods, though not in HbA1c [40]. The frequent co-delivery of calcium in yogurt-based interventions complicates interpretation; the 4-arm RCT by Tabesh et al. (2014) demonstrated that vitamin D and calcium co-supplementation produced greater improvements in HbA1c, HOMA-IR, and QUICKI than either supplement alone, suggesting a synergistic effect [16].

Obesity Status

Non-obese patients consistently demonstrated greater glycemic responses to vitamin D than obese patients in multiple meta-analyses. Chunhua Wu et al. (2017) found significant HbA1c reductions in non-obese patients (BMI <30 kg/m²) but not obese patients [12]. Mirhosseini et al. (2017) showed that HOMA-IR improvements were significant in non-obese patients (−0.74, p=0.005) but not in obese patients (−0.28, p=0.09), and that HbA1c reductions were also larger in non-obese individuals [3]. This pattern is mechanistically consistent with the well-established sequestration of vitamin D in adipose tissue, which reduces bioavailability in obese individuals and would require higher doses to achieve equivalent serum levels. By contrast, the trial by Safarpour et al. (2018)—conducted exclusively in overweight and obese VDD patients—demonstrated a significant HbA1c reduction of −0.81%, suggesting that obese patients can respond when they are also severely deficient and receive appropriately high doses (50,000 IU/week) [47]. Afraie et al. (2024) found, through subgroup analyses, that weight gain, BMI, and duration of disease could reduce the beneficial effect of vitamin D on diabetes control [30].

Baseline Glycemic Control

Several analyses indicate that more poorly controlled patients (higher baseline HbA1c) may experience larger absolute reductions. The subgroup in Nigil Haroon et al. (2015) noted that benefit was seen only when baseline HbA1c exceeded 9% in one trial [23]. Wei Chen et al. (2024) found that subgroup analyses restricted to studies with baseline HbA1c ≥8% showed more prominent

improvements [4]. Conversely, the DDM2 trial enrolled patients with mean HbA1c of 6.6%—well-controlled at baseline—and found no effect [6]; the SUNNY trial enrolled patients with mean HbA1c of 6.8% and similarly found no benefit [5]. This floor-effect phenomenon is consistent with the observation that patients already meeting glycemic targets have little room for further HbA1c improvement.

Ethnicity and Geographic Region

Several meta-analyses identified geographic or ethnic variation in effect estimates. Xinyi Li et al. (2018) found the largest FBG reductions in Middle Eastern populations (WMD -10.43 mg/dl), with smaller effects in other Asian populations and no benefit or slight worsening in "other ethnicities" (predominantly European and American populations) [25]. Mirhosseini et al. (2017) noted that when the nine Iranian trials were excluded from sensitivity analysis, the statistical significance of pooled effects for HbA1c, FPG, and HOMA-IR was lost, indicating that regional factors—likely including baseline vitamin D deficiency severity, sun exposure, dietary patterns, and genetic influences on vitamin D metabolism—substantially drive the pooled positive findings [3]. The majority of positive primary trials come from Iran, Pakistan, Egypt, Nigeria, and other low–middle income settings where severe vitamin D deficiency is prevalent, whereas the null trials disproportionately come from Europe, the United States, and Korea, where baseline 25(OH)D levels tend to be higher.

Sex

A few studies reported sex-specific effects. Nasri et al. (2013) found significant HbA1c reductions in males but not females with weekly 50,000 IU cholecalciferol [20], while Majeed Babar et al. (2017) reported significant improvements in both males and females [53]. Zafar et al. (2025) found that females experienced more pronounced benefits in a stratified analysis [11]. Mitri et al. (2011) noted that one 18-month cohort showed HOMA-B improvement more pronounced in women [23]. No consistent sex-specific pattern emerges across the evidence base

Safety Profile

Vitamin D supplementation across the included trials was uniformly well tolerated. No trial reported clinically significant hypercalcemia attributable to vitamin D supplementation [17–19, 67].

The one systematic review that identified hypercalcemia as a reported side effect was Wei Gao et al. (2013), which noted it as a reported event in one of six included RCTs [38]. Single high-dose regimens of up to 300,000 IU intramuscular and 200,000 IU oral were well tolerated without hypercalcemia in the trials that specifically monitored calcium levels [15, 17, 19]. Weekly 50,000 IU oral regimens for 8–12 weeks did not produce hypercalcemia or hepato-renal toxicity in multiple trials [18, 47]. One participant in the SUNNY trial developed urolithiasis that led to withdrawal [5]; one participant in the Cojic et al. (2021) trial developed a moderate allergic reaction (urticaria) requiring discontinuation [51]; and one participant in the DDM2 trial had an incidentally detected kidney stone [6]. No clinically significant drug interactions between vitamin D and antidiabetic medications (metformin, sulfonylureas, DPP-4 inhibitors, insulin) were identified in any trial [15, 39]. Dropout rates attributable to adverse events were uniformly low across studies [17, 72, 77]. The DDM2 trial, with the longest follow-up of 48 weeks at the IOM upper tolerable intake level of 4,000 IU/day, found overall adverse event rates similar to placebo [6].

Synthesis

The body of evidence presents an apparent contradiction: the majority of meta-analyses and aggregate analyses report statistically significant beneficial effects of vitamin D on HbA1c, FBG, and insulin resistance in T2DM, while a substantial proportion of well-designed individual RCTs—including the SUNNY trial, DDM2, Gulseth et al. (2017), Kuldeep Kumar et al. (2024), Ryu et al. (2014), Strobel et al. (2013), and Breslavsky et al. (2013)—report null effects [5–9, 62, 63]. Reconciling this contradiction requires attention to population composition, study quality, dose adequacy, and mechanistic plausibility.

The Role of Baseline Vitamin D Status and Ceiling Effects. The most parsimonious explanation for the divergence is that glycemic benefits of vitamin D supplementation are largely restricted to individuals who are genuinely vitamin D deficient at baseline, and the effect disappears—or was never present—in individuals with adequate or near-adequate levels. The null trials disproportionately enrolled populations with mean 25(OH)D at or near sufficiency: the SUNNY trial had a mean baseline of 60 nmol/L [5], DDM2 averaged 26.6 ng/mL (approximately 66 nmol/L) [6], and Ryu et al. enrolled patients with mean 25(OH)D of only ~11–12 ng/mL but

with complete deficiency—yet still found no benefit [9]. This suggests that severe deficiency alone is insufficient; other population-level characteristics must also be considered. In the SUNNY trial, the exploratory subgroup of severely deficient patients ($25(\text{OH})\text{D} < 30 \text{ nmol/L}$, $n=19$) did show a meaningful HbA1c reduction of $\sim 3.1 \text{ mmol/mol}$, indicating that correction of profound deficiency may be beneficial even in otherwise well-managed patients [5]. Conversely, the high proportion of positive studies from Iran, Egypt, Pakistan, and other regions with endemic severe deficiency is consistent with a genuine threshold effect: in populations where mean $25(\text{OH})\text{D}$ is often below 15 ng/mL [15, 39, 59], supplementation moves patients from frank deficiency to sufficiency and may unmask a real physiological effect on insulin secretion and action.

The Impact of Baseline Glycemic Control. Trials enrolling well-controlled patients ($\text{HbA1c} \leq 7.5\%$) consistently report null glycemic effects [5, 6, 51]. This is partly a statistical ceiling: patients near target have little room for further HbA1c reduction on any intervention. The well-controlled T2DM patients in DDM2 (mean HbA1c 6.6%) and SUNNY (mean HbA1c 6.8%) were already receiving optimized pharmacotherapy, and the incremental HbA1c reduction achievable would need to be larger than what vitamin D can plausibly deliver to achieve statistical significance. By contrast, trials with mean baseline HbA1c above 8–9% (many of the Middle Eastern trials and the Zafar et al. (2025), Talat Zafar (2025), Thenmozhi et al. (2024), and Ahmed et al. (2024) trials) provide the statistical headroom necessary to detect the approximately 0.2–0.4% absolute reductions consistently observed in meta-analyses [11, 22, 59]. Both the baseline glycemic control and the baseline deficiency explanations are mutually reinforcing: populations with poorly controlled T2DM and severe VDD are those most likely to benefit from supplementation.

Dose Adequacy and Route of Administration. The evidence from Ali et al. (2019) is particularly instructive: identical patient populations receiving either oral daily (4,000 IU/day) or oral weekly (50,000 IU/week) vitamin D both achieved significant and similar glycemic improvements, while patients receiving the same total vitamin D as a single intramuscular bolus (300,000 IU) did not [15]. The key variable appears to be sustained elevation of circulating $25(\text{OH})\text{D}$ throughout the intervention rather than peak concentration. Breslavsky et al. (2013) used only 1,000 IU/day for 12 months and found no glycemic effect despite raising serum vitamin D

levels [63]; Strobel et al. (2013) similarly used ~1,900 IU/day and found no metabolic improvement [62] —suggesting that doses below a functional threshold may be insufficient even in deficient populations. The dose-response analysis in the network meta-analysis of Zhang et al. (2025) identifies a complex pattern where lower doses preferentially reduce HbA1c and higher doses preferentially reduce FBG, possibly reflecting different time-course effects on β -cell function versus peripheral insulin action [35]. The Mirhosseini et al. (2017) review recommends a minimum of 4,000 IU/day to achieve serum levels above 40 ng/mL, the threshold at which HOMA-IR improvements become consistent in their analysis [3].

Obesity and Bioavailability. The consistent finding that non-obese patients respond better to vitamin D supplementation [3, 12] is mechanistically coherent: adipose tissue sequesters vitamin D, reducing its circulating availability in obese patients at a given supplemented dose. The implication is that obese T2DM patients require substantially higher doses to achieve the same serum 25(OH)D rise and glycemic benefit. The one trial in obese patients that showed a significant HbA1c reduction used 50,000 IU/week (Safarpour et al., 2018) [47], reinforcing the dose-adjustment hypothesis. The meta-analysis by Farahmand et al. (2023) reports that the subgroup analyses showed most efficacy in higher dose and shorter intervention periods and in subjects with deficient vitamin D status [2], consistent with the view that obese patients may need dose escalation to replicate the effects seen in non-obese deficient patients.

A Coherent Population-Specific Framework. Taken together, the evidence supports the following synthesis: vitamin D supplementation produces modest but real improvements in glycemic control specifically in patients who are (1) genuinely vitamin D deficient (25(OH)D below approximately 30 nmol/L), (2) poorly to moderately controlled at baseline (HbA1c above 8%), and (3) non-obese or receiving dose-adjusted high-dose supplementation when obese. In this population, pooled HbA1c reductions of 0.2–0.3% and HOMA-IR reductions of 0.4–0.7 are achievable with regular oral dosing of at least 2,000–4,000 IU/day for 8–12 weeks. Outside of this population—particularly in well-controlled European or American T2DM patients with near-sufficient vitamin D levels receiving moderate doses—the glycemic effect is negligible. The inconsistency in the literature is therefore not a contradiction but a reflection of the same

intervention being applied to populations that span this spectrum from severely deficient and poorly controlled to nearly sufficient and well managed.

DISCUSSION

This systematic review of 80 studies, including multiple large RCTs and meta-analyses, demonstrates that vitamin D supplementation has a modest but statistically significant effect on glycemic control in patients with T2DM, but only under specific conditions. The pooled estimates from the most comprehensive meta-analyses show HbA1c reductions of 0.20–0.32%, fasting plasma glucose reductions of approximately 5 mg/dL, and HOMA-IR reductions of 0.42–0.66 (Farahmand et al., 2023; Chen et al., 2024; Mirhosseini et al., 2017). However, these averages mask profound heterogeneity. The apparent contradiction between positive meta-analyses and null individual RCTs is resolved when we examine baseline patient characteristics, intervention details, and effect modifiers.

Baseline Vitamin D Status as the Dominant Effect Modifier

The most consistent finding across all included studies is that glycemic benefits are largely restricted to patients who are truly vitamin D deficient at baseline. In the SUNNY trial, overall HbA1c did not change, but the severely deficient subgroup (25(OH)D <30 nmol/L) showed a significant reduction of ~3.1 mmol/mol (Krul-Poel et al., 2015). Similarly, Wu et al. (2017) reported that HbA1c and FBG improvements occurred exclusively in the deficient subgroup, and Li et al. (2018) found FBG improvements only when baseline 25(OH)D was below 50 nmol/L. The umbrella review by Cheng et al. (2024) quantified this interaction, showing effect sizes nearly double in deficient versus sufficient patients. By contrast, the DDM2 trial, where mean baseline 25(OH)D was 26.6 ng/mL (~66 nmol/L) and only 26% were deficient, found no glycemic effect whatsoever (Angellotti et al., 2018). This ceiling effect is biologically plausible: if circulating 25(OH)D is already adequate to saturate VDR and maintain calcium homeostasis, additional supplementation cannot further enhance β -cell function or insulin sensitivity (Haroon et al., 2015; Papandreou et al., 2026).

Baseline Glycemic Control and the “Headroom” Phenomenon

A second major effect modifier is baseline HbA1c. Trials enrolling well-controlled patients (mean HbA1c $\leq 7.5\%$) consistently report null results. For example, the SUNNY trial (mean HbA1c 6.8%) and DDM2 (mean HbA1c 6.6%) found no benefit (Krul-Poel et al., 2015; Angellotti et al., 2018), as did the trial by Cojic et al. (2021) in well-controlled patients on metformin. Conversely, trials with mean baseline HbA1c $> 8\text{--}9\%$ —such as Zafar et al. (2025) (baseline $\sim 9.5\%$), Thenmozhi et al. (2024) (baseline 7.2% but wide range), and Ahmed et al. (2024) (baseline 10.37%)—showed significant reductions. This is partly statistical: patients near target have little room for further reduction, and the $\sim 0.2\text{--}0.3\%$ absolute effect that vitamin D can deliver is too small to reach significance in a floor-effect scenario (Wei Chen et al., 2024; Afraie et al., 2024). It also suggests that the physiologic effect of vitamin D on insulin secretion may be more easily detected when endogenous β -cell reserve is more compromised, as reflected by higher HbA1c despite pharmacotherapy (Mitri et al., 2011; Santos et al., 2017).

Obesity and Bioavailability

Adipose tissue sequesters vitamin D, reducing its bioavailability in obese individuals (BMI ≥ 30 kg/m²). This explains why non-obese patients consistently show greater glycemic responses. Wu et al. (2017) found significant HbA1c reductions only in non-obese patients, and Mirhosseini et al. (2017) showed that HOMA-IR improvements were significant in non-obese (-0.74 , $p=0.005$) but not in obese patients (-0.28 , $p=0.09$). However, obese patients can respond if they are severely deficient and receive appropriately high doses. The trial by Safarpour et al. (2018) enrolled only overweight/obese vitamin D-deficient patients and found a striking HbA1c reduction of -0.81% using 50,000 IU/week. Thus, obesity is not an absolute barrier but rather a dose-modifying factor (Farahmand et al., 2023; de Oliveira et al., 2024).

Intervention Duration and Dose

Short-term supplementation (≤ 12 weeks, or up to 4 months) consistently improves glycemic parameters, whereas longer trials generally do not. Hu et al. (2019) reported significant improvements in HbA1c, HOMA-IR, and insulin levels with short-term vitamin D but no long-term effect. Lei et al. (2023) confirmed that FBG, fasting insulin, and HOMA-IR improved in trials lasting < 4 months but not in those ≥ 4 months. This time-limited effect may reflect compensatory

physiological adaptation or waning adherence, but it also suggests that vitamin D's actions on insulin secretion are rapid and then plateau once sufficiency is achieved (Vasdeki et al., 2024; Max et al., 2024).

Regarding dose, Farahmand et al. (2023) reported greatest benefit with >2,000 IU/day. The network meta-analysis by Zhang et al. (2025) revealed a complex pattern: lower doses reduced HbA1c, while extremely high doses reduced FBG, possibly reflecting different time-courses of β -cell versus peripheral effects. Importantly, the trial by Ali et al. (2019) directly compared oral daily (4,000 IU), oral weekly (50,000 IU), and a single IM bolus (300,000 IU) in the same deficient population. Both oral regimens significantly improved HbA1c, FBG, insulin, and HOMA-IR, while the IM single dose did not. This indicates that sustained elevation of 25(OH)D, rather than peak concentration, is critical—a finding with direct clinical implications (Mirhosseini et al., 2017; Witham et al., 2010).

Co-supplementation with Calcium

Several trials used vitamin D combined with calcium, either from dairy-based fortified foods or as calcium supplements. The yogurt-fortification studies by Nikooyeh et al. (2011) and Shab-Bidar et al. (2011) showed significant improvements in FBG, insulin, and HOMA-IR at only 1,000 IU/day—a dose that is otherwise low-efficacy. The 4-arm RCT by Tabesh et al. (2014) directly demonstrated that vitamin D plus calcium produced greater improvements in HbA1c, HOMA-IR, and QUICKI than either supplement alone. Calcium may facilitate vitamin D's effects on insulin exocytosis, which is a calcium-dependent process (Eftekhari et al., 2011; Mehta et al., 2021). However, the absence of calcium in most oral vitamin D trials may have underestimated vitamin D's true effect when given alone (Kazemi et al., 2022; Emadzadeh et al., 2020).

Geographic and Ethnic Variation

A striking pattern emerged: positive studies overwhelmingly come from Iran, Pakistan, Egypt, India, Nigeria, and other low- and middle-income countries where severe vitamin D deficiency is endemic (25(OH)D often <15 ng/mL) (Nasri et al., 2013; Mozaffari et al., 2021; Khan et al., 2018; Anyanwu et al., 2016). Null trials disproportionately originate from Europe, the United States, and Korea, where baseline 25(OH)D levels are higher (Ryu et al., 2014; Strobel et al., 2013;

Breslavsky et al., 2013; Sadiya et al., 2014). Mirhosseini et al. (2017) showed that when Iranian trials were excluded from sensitivity analysis, the pooled significance for HbA1c, FBG, and HOMA-IR was lost. This is not a bias but rather a reflection of the biological threshold effect: only when deficiency is sufficiently profound does correction produce detectable metabolic benefit (Sahebi et al., 2019; Sharma et al., 2025).

Sex-Specific Effects

A few studies reported sex differences, but no consistent pattern emerged. Nasri et al. (2013) found HbA1c improvement only in males, whereas Zafar et al. (2025) reported more pronounced benefits in females. Majeed Babar et al. (2017) found significant improvements in both sexes. Therefore, sex is unlikely to be a major effect modifier in clinical decision-making (Haroon et al., 2015; Jafari et al., 2016).

Insulin Resistance vs. Glycemic Control

An intriguing dissociation appears in several studies: HOMA-IR frequently improves even when HbA1c and FBG do not. Li et al. (2018), Hu et al. (2022), Jafari et al. (2016), and Lemieux et al. (2019) all reported significant improvements in insulin sensitivity markers without corresponding HbA1c reductions. This suggests that vitamin D may act primarily on early insulin signaling or hepatic glucose production, but in patients already on glucose-lowering medications, the pharmacologic effects may overwhelm any additional HbA1c change (Probosari et al., 2025; Casas et al., 2022). Alternatively, sample size and duration may be insufficient to translate improved HOMA-IR into lower HbA1c (Kampmann et al., 2014; Gulseth et al., 2017).

Safety

Across all included studies, vitamin D supplementation was uniformly well tolerated. No trials reported clinically significant hypercalcemia attributable to vitamin D, even with single doses up to 300,000 IU IM or weekly 50,000 IU oral regimens (Jehle et al., 2014; Baziar et al., 2014; Shaseb et al., 2016). One participant in the SUNNY trial developed urolithiasis (Krul-Poel et al., 2015), one in the DDM2 trial had an incidental kidney stone (Angellotti et al., 2018), and one in Cojic et al. (2021) had moderate urticaria. No drug interactions with metformin, sulfonylureas, DPP-4 inhibitors, or insulin were identified (Mirzavandi et al., 2020; Danasekaran et al., 2024).

Long-term use at 4,000 IU/day for 48 weeks showed adverse event rates similar to placebo (Angellotti et al., 2018). Thus, vitamin D supplementation at recommended doses (up to 4,000 IU/day) is safe in T2DM patients.

Clinical Implications and a Coherent Framework

Synthesizing the evidence, we propose that vitamin D supplementation improves glycemic control in T2DM only when the following three conditions are met: (1) baseline 25(OH)D is truly deficient (preferably <30 nmol/L, and certainly <50 nmol/L); (2) baseline HbA1c is $\geq 8.0\%$ (providing statistical headroom); and (3) the patient is non-obese, or if obese, receives a higher dose (e.g., 50,000 IU/week) to overcome adipose sequestration. In such patients, a typical response is a 0.3–0.5% HbA1c reduction and meaningful HOMA-IR improvement over 8–12 weeks of daily oral dosing of at least 2,000–4,000 IU. Outside this population—specifically well-controlled European/American T2DM patients with 25(OH)D >50 nmol/L—vitamin D supplementation is unlikely to produce glycemic benefit and should not be prescribed for that indication alone (Lo et al., 2018; Yiu et al., 2013; Wagner et al., 2016; Omidian et al., 2019).

CONCLUSION AND RECOMMENDATIONS

Conclusion

Vitamin D supplementation produces small but statistically significant improvements in HbA1c, fasting glucose, and insulin resistance in patients with type 2 diabetes mellitus, but only in those who are genuinely vitamin D deficient (25(OH)D <30 – 50 nmol/L), have poor baseline glycemic control (HbA1c $\geq 8\%$), and are non-obese or receive obesity-adjusted high doses. In vitamin D-replete or well-controlled patients, supplementation provides no glycemic benefit. The inconsistency in the literature is resolved by recognizing these conditional effects rather than seeking a universal answer.

Recommendations for Clinical Practice

1. Measure serum 25(OH)D in T2DM patients with poor glycemic control (HbA1c $\geq 8\%$) despite appropriate pharmacotherapy.

2. If 25(OH)D is <30 nmol/L, consider cholecalciferol 2,000–4,000 IU/day orally for 8–12 weeks, monitoring HbA1c at 3–6 months.
3. For obese patients ($\text{BMI} \geq 30$ kg/m²), consider higher doses (e.g., 50,000 IU/week) to achieve sufficient serum levels.
4. Do not prescribe vitamin D solely for glycemic control in patients with 25(OH)D >50 nmol/L or HbA1c $\leq 7.5\%$.

Recommendations for Future Research

Future RCTs should: (a) restrict enrollment to vitamin D-deficient, poorly controlled patients; (b) stratify randomization by baseline 25(OH)D and HbA1c; (c) compare dose-response in obese versus non-obese; (d) use active vitamin D metabolites (calcitriol) in some arms to bypass conversion steps; and (e) include calcium co-supplementation as a separate factorial arm.

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