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EFFECT OF VITAMIN D SUPPLEMENTATION ON GLYCAEMIC CONTROL IN PREDIABETIC ADULTS: A RANDOMISED TRIAL

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1. INTRODUCTION

Prediabetes describes blood glucose levels above normal but below the diabetes threshold. By American Diabetes Association criteria it covers a fasting plasma glucose of 5.6 to 6.9 mmol/L, a two-hour value of 7.8 to 11.0 mmol/L during an oral glucose tolerance test, or an HbA1c of 5.7 to 6.4 per cent [1]. It matters because it marks a high-risk state, with a substantial proportion of affected adults progressing to type 2 diabetes over several years [2]. Lifestyle change and metformin can reduce that progression, as the Diabetes Prevention Program showed, but

Abstract

Vitamin D deficiency is common in adults with prediabetes, and observational studies link low serum 25-hydroxyvitamin D to higher glucose and insulin resistance. This narrative review summarises what randomised trials of vitamin D supplementation report for glycaemic control and progression to diabetes in prediabetic adults, together with the biological rationale, trial design issues, dosing and safety. Large prevention trials show a small, statistically uncertain benefit overall, while pooled analyses and smaller studies suggest advantage mainly in those who are deficient at baseline. Heterogeneity in dose, duration and outcomes limits firm conclusions. No new trial is reported; the article synthesises published evidence only.

sustaining lifestyle change at population scale has proved difficult, which keeps interest alive in simpler adjuncts [3].

Vitamin D is one such candidate. Low circulating 25-hydroxyvitamin D (25(OH)D)

is widespread, especially at higher latitudes, in people with darker skin, in older adults and in those with obesity, and these groups overlap with the people at greatest risk of diabetes [4]. Cohort studies have repeatedly found that lower 25(OH)D is associated with higher fasting glucose, greater insulin resistance and more incident diabetes [5, 6]. An association of this kind could reflect cause, or it could reflect confounding, because adiposity and inactivity lower vitamin D status and raise diabetes risk independently. Only randomised trials can decide between the two explanations.

This review looks at those trials. It addresses what has been tested in adults with prediabetes, what glycaemic outcomes have been reported, and why results differ between studies. It is a narrative synthesis of published randomised trials, meta-analyses and guidance, and no new participants were recruited and no new data are presented. The aim is to give a pharmacist or clinician a balanced picture: what the evidence supports, where it is thin, and what can reasonably be said to a patient who asks whether a vitamin D supplement will lower their diabetes risk.

2. BIOLOGICAL RATIONALE

A plausible biological case supports the hypothesis. Pancreatic β cells express the vitamin D receptor and the enzyme that converts 25(OH)D to the active hormone, calcitriol. Early animal work showed that vitamin D-deficient rats secreted less insulin in response to glucose, and that secretion improved with repletion [7]. Calcitriol also affects intracellular calcium handling, which is central to insulin release. The effect is not simply one of supply, since the response to a glucose load in deficient animals recovered only partly with supplementation, suggesting contributions from other mechanisms [7, 8]. These findings come from tissue studies.

Insulin resistance offers a second route. A vitamin D response element has been identified in the promoter of the human insulin receptor gene, and calcitriol increases receptor expression in cell models [9]. Vitamin D may also influence insulin sensitivity through calcium flux in muscle and fat, through effects on inflammatory

cytokines, and through the renin–angiotensin system [8]. In humans, studies using clamp techniques or fasting indices have linked low 25(OH)D to reduced insulin sensitivity and impaired β -cell function [10]. The mechanisms are biologically credible but come largely from cell and animal work, and their size in people at ordinary vitamin D intakes is unknown.

A counter-argument deserves equal space. If vitamin D acted on glucose metabolism by a threshold mechanism, then benefit would be expected only in people whose levels fall below the threshold, and supplementing people who are already replete would add nothing. Recommendations about adequacy are themselves contested. The Endocrine Society suggested that a serum 25(OH)D above 75 nmol/L is desirable, whereas the Institute of Medicine concluded that about 50 nmol/L covers the needs of nearly all people [11, 12]. These positions imply different definitions of who is deficient, and the choice affects how a trial's baseline population is interpreted, as well as how its results should be read.

Measurement adds uncertainty at the very first step. Serum 25(OH)D can be measured by immunoassay or by liquid chromatography–tandem mass spectrometry, and results from different methods and laboratories can disagree by clinically relevant amounts, which complicates comparing trials and defining deficiency [13]. Vitamin D status also varies with season, so a trial that recruited in winter and measured outcomes in summer, or the reverse, may see changes in 25(OH)D unrelated to the intervention. These problems are well recognised, yet many trial reports are vague about assay type and season, which limits what the reader can conclude from a stated baseline concentration.

3. EVIDENCE FROM RANDOMISED TRIALS

The largest trial is the Vitamin D and Type 2 Diabetes (D2d) study, which randomised about 2,400 adults with prediabetes and overweight or obesity to 4,000 IU of vitamin D₃ daily or placebo, and followed them for a median of roughly two and a half years [14].

Diabetes developed in a slightly lower proportion of the vitamin D group, with a hazard ratio of about 0.88, but the confidence interval included no effect, so the trial did not meet its primary aim. The result was neutral in the conventional sense. It also showed that supplementation raised 25(OH)D substantially and was safe in terms of calcium, kidney stones and other adverse events. Study results of this type are summarised in table 1, and fig. 1 shows how they divide by type of evidence.

Table 1: Randomised and pooled evidence on vitamin D supplementation and glycaemia in prediabetes

Study design	Reported finding	Ref.
D2d trial about 2,400 adults, 4,000 IU daily	Slightly lower diabetes incidence; interval included no effect; supplement was safe.	[14]
Tromsø trial 20,000 IU weekly, 5 years	No difference in progression to diabetes despite higher 25(OH)D.	[15]
Individual participant data meta-analysis three trials	Roughly 15% lower risk of diabetes; absolute benefit about three points over three years.	[16]
Secondary analysis of D2d exposure by level	Greater benefit with low baseline or high on-treatment 25(OH)D; exploratory.	[17]
Systematic reviews glycaemic outcomes	No overall effect on fasting glucose or HbA1c; possible benefit if glucose intolerant.	[18, 19]
CaDDM trial vitamin D plus calcium, 4 months	Small gain in β -cell function and smaller rise in HbA1c.	[20]
South Asian women trial insulin resistant, deficient	Insulin resistance fell after six months of supplementation.	[21]
High-dose trial prediabetes with low 25(OH)D	25(OH)D rose; no improvement in glucose or insulin secretion over a year.	[22]
Six-month placebo-controlled trial prediabetes	No improvement in insulin sensitivity or secretion.	[23]
Cohort studies and reviews observational	Low 25(OH)D associated with higher glucose and incident diabetes.	[5, 6]
Cell and animal studies β cell and insulin receptor	Vitamin D receptor in β cells; response element in insulin receptor gene.	[7, 9]

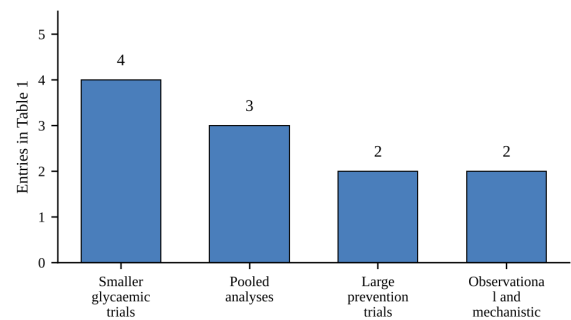


Fig. 1: Number of entries in table 1 by type of evidence

The Tromsø trial in Norway reached a similar conclusion in a different population. Adults with prediabetes were given 20,000 IU of vitamin D₃ weekly or placebo for five years, and progression to diabetes did not differ between groups, even though 25(OH)D roughly doubled in the treated arm [15]. A Japanese trial using an active vitamin D analogue in adults with impaired glucose tolerance was the third large study of diabetes prevention. Taken individually, none of these trials was able to exclude a modest benefit, and each was powered for a bigger effect than might be expected from a nutrient supplement.

Pooling the three trials at participant level gave more precision. An individual participant data meta-analysis of the large prevention trials reported a reduction in diabetes risk of about 15 per cent in the vitamin D arms, with an absolute risk reduction of around three percentage points over three years, and benefit was seen across age, sex and weight categories [16]. A reduction of this size is statistically significant, but is smaller than that produced by lifestyle change or metformin [3]. The authors noted that the meta-analysis was prespecified and used consistent definitions, which strengthens it, though the number of trials is small and two of the three trials gave only a neutral result.

Earlier trials with calcium and vitamin D together deserve a place in the account. A three-year randomised study in older adults without diabetes, given 700 IU of vitamin D and 500 mg of calcium daily, found a smaller rise in fasting glucose and a smaller increase in insulin resistance in the subgroup who began with impaired fasting glucose, while those with normal glucose showed no

difference [24]. The trial was designed around bone and not glucose outcomes, so the glycaemic findings were secondary, and the sample was modest. Even so, it anticipated the idea that people with some degree of dysglycaemia, and not the general population, are those most likely to respond.

Secondary and subgroup analyses add nuance. In D2d, participants who had very low baseline 25(OH)D, below about 30 nmol/L, appeared to have a larger reduction in risk, and participants achieving higher on-treatment levels also did better, but these are exploratory findings that cannot establish cause [17]. People who reach high 25(OH)D concentrations on supplementation may differ from others in adherence and lifestyle, and subgroup comparisons carry a risk of false positives. The pattern is nevertheless consistent with the threshold idea described earlier, and it has encouraged the view that trials in deficient populations would be more informative than further studies in replete ones.

Smaller trials on glycaemic and insulin-related outcomes give a mixed picture. In South Asian women who were insulin resistant and deficient, six months of vitamin D reduced insulin resistance by indices derived from fasting values [21]. The CaDDM trial in adults at high risk found a small benefit on β -cell function and a smaller rise in HbA1c with vitamin D plus calcium over four months [20]. In contrast, high-dose vitamin D in adults with prediabetes and low 25(OH)D raised blood levels but did not improve glucose or insulin secretion over a year [22], and a six-month placebo-controlled trial found no improvement in insulin sensitivity or secretion [23]. Selection of participants, dose and outcome measure differ across these studies.

Body size modifies exposure and deserves explicit mention. Adults with obesity, who make up most of the prediabetic population, typically reach lower 25(OH)D concentrations than lean adults on the same dose, because vitamin D is distributed into a larger fat compartment, and clinical guidance suggests that they may require two to three times the usual intake to correct deficiency [4, 11]. A fixed dose in a heterogeneous trial

population therefore leads to varied attained levels. This is one reason why analyses by on-treatment concentration, as in D2d, are more informative than comparisons based only on assigned dose [17]. It also complicates comparison with trials in leaner populations.

4. TRIAL DESIGN AND MEASUREMENT ISSUES

Differences in design explain much of the apparent disagreement between trials. Doses range from around 1,000 IU a day to the equivalent of several thousand, given daily, weekly or as occasional bolus doses, and the pharmacokinetics of these schedules differ [14, 15, 22]. Duration runs from a few weeks to more than five years, which matters since changes in insulin sensitivity might appear quickly while changes in the diabetes diagnosis take years to accumulate. The form of vitamin D also varies, with D₃ being the usual choice, but D₂ and active analogues appearing in some trials. Combining these studies without regard to such differences risks obscuring true effects or inventing false ones.

The choice of outcome carries equal weight. HbA1c is convenient and clinically meaningful, but it reflects average glycaemia over about three months and can be influenced by anaemia, haemoglobin variants and kidney disease. Fasting glucose is cheap but variable from one day to the next. Oral glucose tolerance tests are more sensitive to early abnormality, though less reproducible and harder to run at scale [25]. Clamp studies and model-based indices measure insulin sensitivity and secretion with greater precision, but only small trials can afford them. A trial may therefore find a change on one measure and none on another, without any contradiction.

Baseline vitamin D status is the design factor with the clearest implications. Trials that recruited unselected adults with prediabetes enrolled many participants with adequate 25(OH)D, in whom further supplementation would be expected to do little [14, 15]. Those that selected deficient or insulin-resistant participants more often reported improvements in insulin resistance [21].

Future trials could enrich for deficiency, although this raises an ethical point, since withholding supplementation from deficient participants for years in a placebo arm is hard to justify. Short trials with a deficient population, or trials with rescue supplementation, are possible compromises.

The population studied may matter as much as the design. A trial in people who already had diabetes but normal vitamin D status found that cholecalciferol did not change glycaemic control, which is consistent with the view that benefit depends on baseline deficiency [26]. The converse, a trial of deficient people with diabetes, would tell more, but few have been reported, and those that exist are small. For prediabetes, the available evidence comes mainly from people with a wide range of baseline levels, and the authors' choice of subgroup cut-offs (30, 50 or 75 nmol/L) varies, so that the comparison of subgroup results across publications is not straightforward [11, 12, 17].

Trial quality should be assessed rather than assumed. Reporting standards such as CONSORT set out what a trial report should contain, and the RoB 2 tool provides a structured assessment of bias arising from randomisation, deviations from intended interventions, missing outcome data, outcome measurement and selective reporting [27, 28]. Applied to vitamin D trials, these frameworks often show adequate randomisation and blinding but incomplete reporting of adherence, background supplementation and assay methods. Many trials also allowed participants to take their own vitamin D outside the study, which dilutes contrast between arms. Such issues tend to bias results toward no effect, a point worth keeping in mind when reading neutral findings.

5. DOSE, SAFETY AND PRACTICE

Safety evidence from these trials is reassuring within the dose ranges used. In D2d, daily doses of 4,000 IU did not increase the rate of hypercalcaemia, hypercalciuria or kidney stones to any marked degree, and serious adverse events were similar to placebo [14]. Other large trials of 2,000 IU daily in the general population also found no excess harm [29]. The Institute of Medicine set the

tolerable upper intake at 4,000 IU daily for adults, while the Endocrine Society allowed up to 10,000 IU daily in some circumstances [11, 12]. Toxicity from supplements is uncommon and mostly follows excessive or erroneous dosing, but sustained intakes above recommended limits have little to justify them.

What should a clinician say to an adult with prediabetes? The evidence does not support vitamin D as a stand-alone intervention for preventing diabetes, since the effect is small and uncertain, and it cannot replace dietary change, physical activity or, where indicated, metformin [1, 3, 16]. Where a patient has documented deficiency, correcting it is reasonable for skeletal health, and any metabolic benefit would be a bonus. Routine testing of everyone with prediabetes to find deficiency is harder to justify. The dose to correct deficiency is typically 1,000 to 2,000 IU daily, adjusted according to follow-up level, and this is well within safe limits [11, 12].

Monitoring after starting treatment can be kept simple. For a patient with prediabetes who begins vitamin D because of documented deficiency, repeating 25(OH)D after about three months confirms that the intended level has been reached and flags poor adherence or malabsorption, while glycaemic status continues to be reviewed on the usual schedule, at least annually, using fasting glucose or HbA1c [1, 25]. Sampling timing and assay method should be consistent between measurements, since a change of laboratory can produce an apparent shift unrelated to the patient [13]. Patients should be told that any change in glucose measures is unlikely to be dramatic.

Pharmacists are well placed to guide these conversations. Many patients already buy vitamin D, often in doses that vary widely between products, and the same patients may take calcium, multivitamins or fortified foods that add to the total. A medicines review can identify duplication, check for interacting drugs such as thiazide diuretics (which raise calcium), and reinforce that supplementation complements and does not substitute for glucose-lowering lifestyle measures. Discussing the realistic size of any benefit

helps to counter claims made in marketing, which often cite observational data and describe vitamin D as protective against diabetes in terms that the trials do not support [30, 31].

Calcium is a point of detail with practical importance. Several of the earlier trials gave calcium together with vitamin D, so a benefit or lack of benefit cannot be assigned to one component alone [20, 24]. Calcium supplements carry their own considerations, including gastrointestinal effects and a debated cardiovascular signal, and the combined products on pharmacy shelves are often chosen for bone health. A patient looking for a glucose effect from such a product should understand that the evidence for calcium is even less developed than for vitamin D, and that the doses used in trials were generally modest.

6. LIMITATIONS AND FUTURE DIRECTIONS

The evidence discussed here has several limitations. The number of large trials is small, and their populations were mainly overweight adults who were not deficient on average, so they cannot answer the question for severely deficient people or for those of different ancestry and diet. Subgroup findings are exploratory. Follow-up in most studies was short compared with the natural history of diabetes. Smaller trials differ in outcome and quality, and the pooled estimates from meta-analyses depend on the studies included [16, 18, 19]. Publication bias towards positive findings cannot be excluded, though the many neutral reports suggest that it is not overwhelming.

Several questions remain open. Does supplementation help specific groups, such as people with very low 25(OH)D or particular ancestries in whom the relationship between total and bioavailable vitamin D differs? Is there a threshold of 25(OH)D above which more vitamin D does nothing? Would earlier intervention, in people with normal glucose but high risk, behave differently? Answering these requires trials enriched for deficiency, long enough to detect diabetes, and with standardised assays and outcomes [13, 17]. Mechanistic sub-studies measuring insulin

secretion and sensitivity directly would help to separate effects on the two arms of glucose regulation.

Interpretation should stay modest. Vitamin D is not a proven strategy for preventing diabetes in prediabetes, but the data are not entirely neutral either, since pooled analyses leave open a small benefit that could matter at population level given how common the condition is [16]. Accurate communication matters here: neither overstated enthusiasm nor flat dismissal reflects the literature. Systematic reporting of baseline status, assay, dose, adherence and outcomes would make the next generation of trials easier to combine and the next review easier to write. Reviews in this area should also be updated as new pooled analyses and ongoing trials report, since a single large study can shift the balance of evidence considerably.

Cost-effectiveness has received little attention. Vitamin D is inexpensive, and even a small reduction in diabetes risk could be worthwhile at population level if safe, which is one of the arguments made by those who favour continued investigation [16]. Against this, widespread supplementation without testing could divert attention from interventions with larger effects. Economic modelling using the pooled risk estimate, with realistic adherence, has not been widely published, and its absence leaves policy makers without a clear case in either direction. Until it is available, statements about public-health value should be framed as possibilities.

7. CONCLUSION

Randomised trials of vitamin D in adults with prediabetes do not show a clear, large effect on glycaemic control or on progression to diabetes. The biggest prevention studies were neutral on their own terms, a pooled analysis points to a modest reduction in risk, and smaller trials of insulin sensitivity and glucose measures are inconsistent [14, 15, 16, 22, 23]. Benefit, where it appears, is concentrated among people who are deficient or insulin resistant at the start. Supplementation at usual doses was safe, so there is little reason for concern about harm in the dose ranges examined [11, 12, 14].

For practice, vitamin D should be viewed as a correction for deficiency and a possible minor adjunct, not a substitute for lifestyle change or metformin in people at risk [1, 3]. For research, the priorities are trials that enrol deficient participants, report assay methods and adherence, use consistent outcomes, and run long enough to capture diabetes diagnosis [13, 17, 27]. Until such work is available, honest advice to patients is that the supplement is cheap and generally safe, but the evidence that it prevents diabetes is limited. Further pooled analyses may refine this view.

8. ABBREVIATIONS

25(OH)D-25-Hydroxyvitamin D, ADA-American Diabetes Association, CaDDM-Calcium and Vitamin D for Diabetes Mellitus (trial), D2d-Vitamin D and Type 2 Diabetes (trial), HbA1c-Glycated haemoglobin, IU-International units, OGTT-Oral glucose tolerance test, RoB 2-Risk of bias tool, version 2, CONSORT-Consolidated Standards of Reporting Trials

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10. AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

11. CONFLICT OF INTERESTS

Declared none

12. REFERENCES

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