

Vitamin D supplementation in adults with pre-diabetes

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In this viewpoint, we recommend that general practitioners and other primary care providers in Aotearoa New Zealand (NZ) discuss vitamin D supplementation with patients at high risk of developing type 2 diabetes, as part of a broader strategy for diabetes prevention, including weight loss and other lifestyle changes. Pre-diabetes risk factors include obesity, physical inactivity, hypertension, dyslipidaemia, a history of gestational diabetes, and conditions associated with insulin resistance, such as metabolic dysfunction-associated steatotic liver disease.

This issue is relevant in NZ, where the number of adults with type 2 diabetes is projected to increase by 90% between 2020 and 2044, with Māori, Pasifika, and Asian populations, who are also more likely to have low vitamin D status, bearing a disproportionate share.¹

The evidence for our recommendation is below.

Vitamin D status in New Zealand

Vitamin D deficiency, generally defined as a blood 25-hydroxyvitamin D concentration (25(OH)D) less than 25 nmol/L, is common in large segments of the population – particularly for Pasifika (10.5%) and Māori (6.0%),² and in the South Asian community which has the lowest vitamin D levels in NZ with mean 25(OH)D levels consistently below 40 nmol/L,^{3,4} similar to those for Middle Eastern and African people but lower than those for Chinese and Southeast Asian.³

Vitamin D and type 2 diabetes in New Zealand

Two early NZ studies provided evidence suggesting that vitamin D offers protection against type 2 diabetes. A cross-sectional study of 5677 adults aged 40–64 years in worksites in Auckland and Tokoroa found lower mean blood 25(OH)D levels in individuals with prediabetes (70 nmol/L) and diabetes (67 nmol/L) than matched controls (76 nmol/L).⁵ In a randomised controlled trial of vitamin D supplementation in 81 South Asian women living in Auckland, aged 23–68 years with serum 25(OH)D levels below 50 nmol/L and insulin resistance, those randomised to receive 4000 IU of vitamin D₃ daily had a significant improvement in insulin sensitivity and a reduction in insulin resistance after 6 months, compared with placebo.⁶

Observational studies linking vitamin D status to type 2 diabetes

Over the past two decades, numerous longitudinal cohort studies have reported on the association between baseline 25(OH)D concentrations and incident type 2 diabetes. Their findings have been summarised in meta-analyses, which have consistently found significant inverse associations between baseline 25(OH)D levels and the risk of developing type 2 diabetes.⁷

Because observational studies are limited by confounding (eg adiposity, diet, physical activity, and socioeconomic status), randomised controlled trials have been conducted to test the hypothesis that vitamin D supplementation reduces the risk of developing diabetes.

Table 1. Characteristics and results of the three key randomised controlled trials of vitamin D and diabetes prevention in adults with prediabetes.

Variable	Tromsø study Jorde <i>et al.</i> (2016) ⁸	D2d study Pittas <i>et al.</i> (2019) ⁹	DPVD study Kawahara <i>et al.</i> (2022) ¹⁰	IPD meta-analysis Pittas <i>et al.</i> (2023) ¹¹
Country	Norway	United States	Japan	–
Participants (vitamin D/placebo)	511 (256/255)	2423 (1211/1212)	1256 (630/626)	4190 (2097/2093)
Mean age (years)	60.6	60.4	60.9	60.6
Men (%)	314 (61)	1337 (55)	685 (55)	2336 (56)
Baseline 25-hydroxyvitamin D (nmol/L)	60	70	52	63
Intervention	Vitamin D ₃ 20,000 IU/week	Vitamin D ₃ 4000 IU/day	Eldecalcitol 0.75 mcg/day	–
Follow-up period (years)	5	Median 2.5	Median 2.9	Median 3.0
New onset diabetes				
Vitamin D (events/N)	103/256	293/1211	79/630	475/2097
Placebo (events/N)	112/255	323/1212	89/626	524/2093
Hazard ratio (95% CI)	0.89 (0.68, 1.16)	0.88 (0.75, 1.03)	0.88 (0.65, 1.19)	0.85 (0.75, 0.96) ^A
Regression to normoglycaemia				
Vitamin D (events/N)	52/239	90/1037	129/605	271/1881
Placebo (events/N)	38/239	63/1050	108/600	209/1889
Hazard ratio (95% CI)	1.37 (0.90, 2.08)	1.45 (1.05, 2.00)	1.18 (0.92, 1.53)	1.30 (1.16, 1.46) ^A

^AThe hazard ratio was adjusted for race, sex, age, body mass index, and haemoglobin A1c.

Clinical trials of vitamin D supplementation in prediabetes

Several randomised clinical trials of vitamin D supplementation for diabetes prevention have been carried out in adults with prediabetes, because of their increased risk of diabetes, with progression to diabetes or regression to normoglycemia as key outcomes. Three of these trials were specifically designed and conducted to test whether vitamin D reduces the risk of developing diabetes in adults with prediabetes: the Tromsø study in Norway;⁸ the vitamin D and type 2 diabetes (D2d) study in the U.S.;⁹ and the Diabetes Prevention with active Vitamin D (DPVD) study in Japan. The Tromsø and D2d studies tested cholecalciferol; the DPVD tested eldecalcitol, which is relevant because its physiological endpoint – calcitriol – is the same as for cholecalciferol.¹⁰ The relative risk reduction for diabetes in the DPVD trial was similar to trials using cholecalciferol, supporting a common mechanism (*P*-value for interaction = 0.99). Details and results of these three trials, along with an individual participant data (IPD) meta-analysis combining their results,¹¹ are summarised in Table 1.

Progression to diabetes

The individual participant data meta-analysis that combined data from the three trials showed that vitamin D reduces the

risk of progression from prediabetes to diabetes by 15% (hazard ratio 0.85; 95% CI 0.75–0.96).¹¹ Based on an absolute risk reduction of 3.3% over the median follow-up period, the number needed to treat (NNT) to prevent one new case of diabetes was 30 over 3 years.¹¹ A major advantage of IPD analyses is the ability to do subgroup analyses. In participants with a baseline serum level of 25(OH)D <30 nmol/L, the hazard ratio for the risk of progression to diabetes in those receiving vitamin D was 0.58 (95% CI 0.35, 0.97), suggesting that people with a lower blood 25(OH)D concentration have a greater benefit from vitamin D supplementation. Further, among the Tromsø and D2d trial participants receiving vitamin D₃, which raises the blood 25(OH)D concentration, those who maintained their 25(OH)D levels in the range of 100–124 nmol/L, or ≥125 nmol/L, had absolute risk reductions at 3 years of 11.4% and 18.1% respectively, compared to participants who maintained their 25(OH)D levels in the range of 50–74 nmol/L – indicating that the long-term maintenance of high serum vitamin D levels has the greatest benefit in preventing diabetes. These results are consistent with other effective interventions, such as statins and incretin-based therapies, where dose escalation is associated with greater efficacy.

Regression to normoglycaemia

In the IPD meta-analysis of the three clinical trials (Table 1), participants in the vitamin D group (cholecalciferol or

eldecalcitol combined) were 30% more likely to have regressed to normal glucose regulation at the last study visit than those in the placebo group.¹¹ The NNT to reverse one case of prediabetes to normoglycaemia is 30 over 3 years.

Clinical significance

In summary, the scientific evidence supporting a role for vitamin D in diabetes prevention meets all of the Bradford Hill criteria for causation. Although the effect of vitamin D is smaller than the risk reduction observed with intensive lifestyle interventions in clinical trials,¹² it is important to note that the effect of vitamin D was observed on top of lifestyle recommendations provided to all participants in the vitamin D diabetes prevention trials. Based on the individual participant data meta-analysis, the NNT to prevent one new diabetes case over 3 years is 30 with vitamin D,¹¹ compared to seven for the intensive lifestyle intervention in the Diabetes Prevention Program study.¹³ However, the latter involves individualised, resource-intensive counselling sessions aimed at weight loss through improved diet and physical activity, an approach that is costly and not scalable. In contrast, vitamin D is an inexpensive and safe intervention (see Supplementary material).

For comparison, a recently published NZ study of two intravenous zoledronate infusions (5 years apart) had an NNT of 22 over 10 years to prevent one new fracture.¹⁴ The NNT of 30 to prevent one new case of diabetes over 3 years (from the IPD meta-analysis¹¹) is lower if vitamin D was taken for a 10-year period. For example, using the incidence rates from the IPD meta-analysis,¹¹ the risk difference over 10 years in the intention-to-treat analysis gives an NNT of 23.

Conclusion

Given the very low cost, minimal patient burden, and favourable safety profile of vitamin D, we recommend that primary healthcare practitioners consider vitamin D supplementation for adult patients with pre-diabetes, as part of a broader strategy for diabetes prevention.

Because patients who will benefit most from vitamin D supplementation are those with lower 25(OH)D levels, clinicians should understand the risk factors for vitamin D deficiency to prioritise patients at the highest risk. For such patients, consistent with the US Endocrine Society Guideline,¹⁵ we recommend empiric vitamin D supplementation, without 25(OH)D testing. In the trials, daily doses equivalent to approximately 3500 IU/day were used, providing a pragmatic dosing range of 2000–5000 IU/day that can be readily applied in clinical practice. This approach allows dosing to be individualised based on patient risk and

context, not unlike intensive lifestyle interventions, in which a recommended range is tailored to the patient's needs.

The determinants of vitamin D status, strategies to increase vitamin D levels, and the safety of vitamin D supplements are discussed in the Supplementary material.

Supplementary material

Supplementary material is available online.

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