A comparative study to assess the effect of a vitamin d-supplemented diet versus vitamin d supplementation on hba1c and lipid profile among patients with type 2 diabetes mellitus

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DOI: 10.5281/zenodo.23556081

Abstract:
Background: Type 2 diabetes mellitus (T2DM) is commonly accompanied by dyslipidaemia and a high burden of cardiovascular risk. Vitamin D has biological roles beyond bone metabolism, and observational research has linked lower circulating 25-hydroxyvitamin D [25(OH)D] concentrations with insulin resistance and adverse metabolic profiles. However, trials of vitamin D supplementation have produced inconsistent effects on glycated haemoglobin (HbA1c) and lipid fractions. It is therefore important to distinguish a food-based strategy from oral supplementation and to avoid presenting vitamin D as a replacement for established diabetes care.
Aim: To compare changes in HbA1c and lipid profile among adults with T2DM receiving a structured vitamin D-rich or vitamin D-fortified dietary intervention and those receiving clinician-directed oral vitamin D supplementation, in addition to usual diabetes care, at DH, Mohali.
Methods: The proposed study is a prospective, two-group comparative intervention study with baseline and 12-week assessments. A target of 100 participants (approximately 50 per group) is proposed as a planning estimate. Participants will undergo clinical assessment, medication review, dietary assessment, HbA1c and fasting lipid testing, and measurement of serum 25(OH)D where feasible. Group allocation procedures, dose decisions, and eligibility for a diet-only strategy must be approved before recruitment. Clinically indicated vitamin D treatment will not be withheld.
Primary outcome: Between-group difference in change in HbA1c from baseline to week 12. Secondary outcomes: changes in total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, serum 25(OH)D, body weight, dietary adherence, and safety events. Analysis will use an intention-to-treat principle where random allocation is approved and performed, with baseline-adjusted comparisons and confidence intervals.
Expected contribution: The study is intended to generate locally relevant comparative evidence about practical vitamin D strategies in routine T2DM care. No numerical effect is predicted as an observed finding. Vitamin D supplementation will be interpreted as an adjunct for appropriate clinical indications, not as a substitute for glucose-lowering medicines, lipid-lowering therapy, medical nutrition therapy, physical activity, or follow-up.
Protocol transparency: The final report should follow the applicable current trial-reporting guidance, provide the ethics approval and registration details, and report all prespecified outcomes regardless of statistical significance (Chan et al., 2025; Hopewell et al., 2025).

Keywords:
Type 2 diabetes mellitus; vitamin D; cholecalciferol; diet; HbA1c; lipid profile; dyslipidaemia; comparative study; Mohali.

1. Introduction:

Type 2 diabetes mellitus is a chronic metabolic disorder characterised by elevated blood glucose resulting from progressive impairment of insulin secretion, insulin action, or both. Its clinical impact extends beyond hyperglycaemia: many people also have central adiposity, hypertension, raised triglycerides, low high-density lipoprotein cholesterol, and other features that increase cardiovascular risk. Diabetes care therefore requires coordinated attention to glycaemic control, lipid management, renal function, blood pressure, nutrition, activity, medicine adherence, and prevention of complications. The World Health Organization describes diabetes as a major global public-health challenge, while the International Diabetes Federation has documented a growing global burden and substantial geographic variation in prevalence (World Health Organization, 2006, 2016; International Diabetes Federation, 2021).

In T2DM, HbA1c is a central marker of average glycaemia over approximately the preceding two to three months. It reflects glycation of haemoglobin during the lifespan of circulating erythrocytes and complements fasting or postprandial glucose measurements. HbA1c is useful for monitoring response to treatment, but interpretation may be affected by conditions that alter red-cell turnover, certain haemoglobin variants, anaemia, recent blood loss or transfusion, pregnancy, and some renal or haematological disorders. These caveats are relevant when comparing groups because an apparent HbA1c difference may not always reflect a true difference in glucose exposure. Standardised laboratory methods and documentation of potential confounders are therefore essential (Nathan et al., 2008).

Dyslipidaemia in T2DM commonly includes elevated triglycerides, reduced HDL cholesterol, and qualitative changes in LDL particles. LDL cholesterol remains an important therapeutic target, but lipid values are influenced by fasting status, diet, acute illness, alcohol use, thyroid disease, renal function, weight change, and lipid-lowering medicines. In a clinical study, a change in the lipid profile must be interpreted in context: a small mean difference after a nutritional intervention is not automatically clinically important, and no supplemental strategy should delay statin therapy or other indicated cardiovascular risk management.

Vitamin D is a fat-soluble nutrient involved in calcium and phosphate homeostasis, skeletal health, and signalling in multiple tissues. The circulating marker used to assess vitamin D status is 25(OH)D, which integrates vitamin D derived from cutaneous synthesis, food, and supplements. Vitamin D receptors and vitamin D-metabolising enzymes have been identified in several tissues relevant to metabolism, which has prompted research into possible links with insulin secretion, insulin sensitivity, inflammation, and lipid handling. Biological plausibility, however, does not itself demonstrate that supplementation causes a clinically meaningful improvement in diabetes outcomes (Holick, 2007; DeLuca, 2004; Bikle, 2014).

Observational studies have reported associations between low 25(OH)D levels and insulin resistance, impaired glucose tolerance, and metabolic syndrome. Such relationships may be partly explained by common determinants: people with obesity may have lower measured 25(OH)D; people with chronic disease may spend less time outdoors; dietary quality and socioeconomic circumstances may influence both vitamin D intake and diabetes risk; and kidney or liver disease may affect vitamin D metabolism. Reverse causation and residual confounding are therefore plausible. Intervention trials are needed to test causality, and their results have not been fully consistent (Chiu et al., 2004; Mitri et al., 2011).

The present proposed comparative study focuses on a pragmatic question for DH, Mohali: among adults with established T2DM, do changes in HbA1c and lipid profile differ between a structured food-based vitamin D strategy and oral vitamin D supplementation when both are considered alongside usual clinical care? The emphasis is not to assume that either strategy is superior. Instead, the study should measure baseline vitamin D status, keep usual diabetes treatment documented, track adherence, assess adverse events, and report uncertainty. A local study may help clinicians

understand feasibility and acceptability even if it does not establish whether vitamin D independently modifies long-term cardiovascular events.

2. Scientific background:

Vitamin D physiology and assessment:

Vitamin D can be obtained through ultraviolet-B-mediated synthesis in the skin, from naturally occurring foods, from fortified products, and from supplements. The liver converts vitamin D to 25(OH)D, the principal circulating marker, while the kidney and certain extra-renal tissues can convert it to the active hormone 1,25-dihydroxyvitamin D. These metabolites have different functions and are not interchangeable laboratory markers. Because 1,25-dihydroxyvitamin D is tightly regulated by calcium, phosphate, parathyroid hormone, and renal function, it is not generally the preferred initial test for routine vitamin D status. The study should use a validated assay for serum 25(OH)D and should record the laboratory method and units.

Vitamin D status is affected by season, time spent outdoors, clothing, skin pigmentation, age, body composition, dietary pattern, malabsorption, liver disease, kidney disease, and medicines that influence vitamin D metabolism. In India, a sunny climate does not guarantee adequate vitamin D status in every individual. Urban activity patterns, heat avoidance, air pollution, clothing, skin pigmentation, low intake of fortified foods, and inconsistent supplement use may all contribute. A review of Indian literature highlighted that vitamin D deficiency can remain common despite abundant sunlight, while also emphasising differences in study populations and thresholds (Ritu and Gupta, 2014).

For a research protocol, the threshold used to classify deficiency or insufficiency must be declared before recruitment and aligned with the institutional laboratory and the guideline used by the treating team. The 2024 Endocrine Society guideline cautions that outcome-specific serum thresholds for many disease-prevention purposes remain uncertain, and it does not support indiscriminate testing or empiric high-dose supplementation for all healthy adults (Demay et al., 2024). This guidance does not remove the need to diagnose and treat a clinically recognised deficiency when indicated. The protocol must distinguish treatment of deficiency from experimental use of vitamin D as a glucose-lowering adjunct.

Assay consistency matters. If baseline samples are tested using one platform and follow-up samples by another, measurement differences may be mistaken for biological change. Ideally, all samples should be analysed in the same accredited laboratory using a standardised assay with internal quality control. The study record should specify whether the result is reported in ng/mL or nmol/L and use one unit consistently. Conversion should be checked carefully because 1 ng/mL equals 2.5 nmol/L.

Potential links with glucose metabolism:

Proposed mechanisms connecting vitamin D with glucose metabolism include effects on pancreatic beta-cell function, calcium-dependent insulin secretion, insulin receptor signalling, inflammatory pathways, and systemic metabolic regulation. The vitamin D receptor is expressed in tissues relevant to glucose homeostasis, and experimental studies suggest that active vitamin D signalling may influence insulin-related processes. The mechanistic evidence supports investigation, but it should not be translated directly into a clinical claim that oral vitamin D lowers HbA1c in every patient with T2DM. Physiological pathways can be context-dependent, and the effect of correcting deficiency may differ from the effect of raising already adequate levels.

Chiu and colleagues reported associations between lower 25(OH)D concentrations and insulin sensitivity in a study of glucose-tolerant adults, suggesting a relationship between vitamin D status and metabolic function. The study was observational and not a test of supplementation among patients with established T2DM. This distinction is important: an association can identify a potential mechanism or target population, but it cannot establish treatment benefit. A comparative clinical study should therefore measure rather than presume the baseline vitamin D status of enrolled participants.

Vitamin D could plausibly influence glycaemia indirectly through changes in inflammation, muscle function, body composition, or calcium regulation. However, these pathways may be weaker than established determinants of HbA1c, including the potency and adherence of glucose-lowering medicines, baseline HbA1c, duration of diabetes, weight change, food intake, and physical activity. The protocol will record these factors and avoid interpreting an unadjusted before-and-after comparison as evidence that vitamin D caused all observed changes.

Variation in trial duration is another consideration. HbA1c reflects glucose exposure over several weeks, so very short supplementation periods may fail to capture the full effect of a stable intervention. Conversely, changes in glycaemic medication during follow-up can affect HbA1c independently of vitamin D. A 12-week assessment is a practical minimum period for comparing short-term biomarker changes, while a longer follow-up may be required to evaluate durability, adherence over time, and clinical outcomes.

Potential links with lipid metabolism:

The proposed relationship between vitamin D and lipids is less straightforward than a simple direct lipid-lowering effect. Vitamin D status is correlated with body fat, diet quality, physical activity, and metabolic health, all of which also influence triglycerides, HDL cholesterol, and LDL cholesterol. Some studies propose that vitamin D signalling may modulate lipid synthesis, adipocyte function, inflammatory responses, or insulin-mediated regulation of triglyceride-rich lipoproteins. These mechanisms remain hypotheses for the purposes of the proposed local study and do not replace standard lipid treatment.

A meta-analysis of randomised controlled trials in people with T2DM found small pooled changes in total cholesterol and LDL cholesterol, while triglyceride and HDL effects were less certain; the authors also reported that baseline 25(OH)D, dose, treatment duration, and route could influence results (Jafari et al., 2016). An umbrella review subsequently found modest average effects across a broad evidence base but highlighted variation among underlying meta-analyses and outcomes (Radkhah et al., 2023). These results justify including a full lipid panel as a secondary outcome, without assuming that every fraction will improve.

Fasting status should be standardised for lipid testing if the clinical laboratory and local practice require fasting samples, especially where triglycerides are a primary concern. The same laboratory should use consistent methods at baseline and follow-up. LDL cholesterol may be calculated using a formula or measured directly, depending on laboratory practice; the method should be documented, and a calculated value may be less reliable when triglycerides are very high (Friedewald et al., 1972). Changes in statin dose, fibrate use, weight, alcohol intake, and recent illness should be captured because each may materially alter lipids.

In patients with T2DM, lipid changes may occur because usual treatment is initiated or intensified during study follow-up. This should not be discouraged merely to preserve research purity. Clinical care takes priority, and medically indicated lipid-lowering treatment must continue. The analysis should report medication changes and use sensitivity analyses where feasible. A transparent report should acknowledge that a short-term nutritional study cannot determine whether a small lipid difference translates into fewer myocardial infarctions, strokes, or deaths.

Dietary sources and oral supplementation:

Food-based vitamin D strategies may include foods that naturally contain vitamin D, such as oily fish and egg yolk, as well as products fortified with vitamin D where reliably available. The contribution from individual foods varies substantially by species, serving size, preparation method, and fortification practice. Dietary counselling should not assume that all milk, yoghurt, paneer, cereals, or plant-based drinks are fortified; the product label or local food-composition data should be checked. The dietary strategy should be practical for local food habits, affordability, vegetarian preferences, cultural practices, and comorbidities.

A food-based strategy may bring wider nutritional benefits when it is embedded in a balanced diabetes eating pattern that supports appropriate energy intake, fibre, high-quality protein, and unsaturated fats. It may also be easier for some people to sustain than a supplement, though foods alone may not correct established deficiency. Conversely, food-derived vitamin D exposure can be

difficult to quantify, and the availability of fortified foods is inconsistent. The protocol should therefore measure adherence with repeated dietary recalls and, where available, serum 25(OH)D rather than assuming that counselling equates to a known dose.

Oral vitamin D supplements offer more predictable intake, but the dose and duration must be determined by a qualified clinician in light of the participant's 25(OH)D status, calcium level where appropriate, renal function, history of stones, malabsorption, granulomatous disease, medicines, and existing supplements. The protocol will not prescribe a universal high dose for every participant. Therapeutic regimens used to correct deficiency should follow the approved institutional guideline, and participants at risk of hypercalcaemia or other complications need individual review. The 2024 Endocrine Society guideline underscores the difference between disease-specific treatment and empiric supplementation in people without a recognised indication (Demay et al., 2024).

The American Diabetes Association's 2026 Standards of Care emphasise individualised medical nutrition therapy, nutrient-dense dietary patterns, and evidence-based diabetes self-management. They do not recommend routine micronutrient supplementation for glycaemic benefit in the absence of an identified need, and they describe the evidence for vitamin D as mixed rather than definitive (American Diabetes Association Professional Practice Committee for Diabetes, 2026). Consequently, the proposed study will position vitamin D as the intervention under evaluation, not as an established alternative to guideline-directed pharmacological treatment.

Review of relevant clinical evidence:

Early reviews described a biologically plausible relationship between vitamin D and diabetes risk but concluded that observational associations did not establish that supplementation prevents or treats diabetes. Mitri and colleagues reviewed evidence concerning vitamin D and type 2 diabetes and noted the need for better randomised trials, particularly trials that considered baseline vitamin D status and achieved a meaningful change in exposure (Mitri et al., 2011). This remains relevant for the proposed study because any estimated effect may differ between participants with deficiency and participants whose baseline levels are already adequate.

The Vitamin D and Type 2 Diabetes (D2d) trial tested 4,000 IU/day of vitamin D3 against placebo among adults with high-risk prediabetes who were not selected for vitamin D deficiency. Over a median follow-up of 2.5 years, the intervention did not significantly reduce incident diabetes compared with placebo (Pittas et al., 2019). D2d addressed diabetes prevention rather than treatment of established T2DM, so its primary outcome is not directly interchangeable with HbA1c change in the current proposed study. Nonetheless, it provides an important caution against over-interpreting biological plausibility or observational associations.

The SUNNY trial evaluated vitamin D supplementation in people with T2DM and did not demonstrate a clear improvement in glycaemic control in the overall study population. Its findings suggested that routine supplementation cannot be assumed to lower HbA1c when patients are not selected on the basis of severe deficiency or another clear indication (Krul-Poel et al., 2015). Trial results are affected by sample size, intervention duration, achieved serum concentrations, adherence, and baseline therapy, which may explain some differences between individual trials.

Mohamad and colleagues studied vitamin D supplementation in people with T2DM and measured glycaemic and lipid markers before and after supplementation. Their work contributed to a literature in which some studies found improvements in selected biochemical outcomes, while others did not find consistent changes across the full range of lipid fractions. Because changes in a single-group or short-duration study can also reflect regression to the mean, concurrent care, or time trends, the design of the DH, Mohali study should use a defined comparator and baseline-adjusted between-group estimates (Mohamad et al., 2016).

Angellotti and colleagues examined vitamin D supplementation and cardiovascular risk factors in people with T2DM not selected for vitamin D deficiency. Their study did not find an overall meaningful change in the lipid profile or broad cardiovascular risk markers, apart from a subgroup observation involving triglycerides among participants not using cholesterol-lowering medication (Angellotti et al., 2019). Subgroup results should be interpreted cautiously because they are vulnerable to chance findings and multiple comparisons. The proposed study should prespecify subgroup analyses rather than selecting favourable subgroups after results are known.

Jafari and colleagues pooled randomised trial evidence on vitamin D and lipid outcomes in T2DM. They reported statistically significant but relatively small changes in total cholesterol and LDL cholesterol, while effects on triglycerides and HDL cholesterol were not uniform. Their analysis suggested that baseline vitamin D status, dose, route, and duration may be relevant moderators (Jafari et al., 2016). This supports recording these variables, but it does not identify a single optimal regimen for use in every clinical setting.

Farahmand and colleagues included 46 randomised controlled trials in a meta-analysis of vitamin D supplementation for glycaemic control among people with T2DM. They reported an average HbA1c reduction of approximately 0.20 percentage points, with substantial between-study heterogeneity and a possibility of publication bias; the apparent benefit was more evident in studies of vitamin D-deficient participants (Farahmand et al., 2023). A pooled mean effect can be useful for planning, but it should not be interpreted as a guaranteed response in an individual patient or a confirmed effect of a diet-based intervention.

Afraie and colleagues published an updated systematic review and meta-analysis that included studies reporting HbA1c, fasting glucose, LDL cholesterol, HDL cholesterol, and related risk factors. They found statistically significant pooled changes in several markers, while heterogeneity remained high for many outcomes (Afraie et al., 2024). The findings favour careful evaluation of baseline deficiency, dose, duration, body mass index, and disease characteristics. They also show why confidence intervals and magnitude of effect are more informative than a statement that a result was merely statistically significant.

Chen, Liu, and Hu conducted an updated meta-analysis of 39 randomised trials involving nearly 3,000 participants with T2DM. They reported a pooled HbA1c reduction of about 0.30 percentage points and improvements in fasting glucose and insulin-resistance measures, with stronger effects in certain subgroups such as people with deficiency or higher baseline HbA1c (Chen et al., 2024). The reported pooled effect differs from earlier syntheses, reflecting eligibility criteria, search periods, doses, and study populations. It supports ongoing research but does not resolve whether a structured dietary approach can match oral supplementation.

For lipid outcomes more broadly, Radkhah and colleagues synthesised meta-analyses of randomised trials and found modest pooled reductions in triglycerides and total cholesterol and a small increase in HDL cholesterol in standardised analyses. Their weighted mean difference analysis supported a clearer reduction only for triglycerides, illustrating that conclusions may differ according to the metric and evidence base (Radkhah et al., 2023). This reinforces the value of reporting each lipid fraction separately, along with effect sizes and confidence intervals.

Taken together, the literature provides a reasonable rationale for a comparative local study while also setting limits on the claims that can be made. Some meta-analyses suggest small average improvements in HbA1c or selected lipid parameters, especially where baseline vitamin D is low. Other trials report little or no benefit. The studies differ in baseline status, participant characteristics, supplementation regimen, co-interventions, duration, risk of bias, and outcome measurement. The proposed project should be designed to contribute a well-documented local comparison, not to settle a debate that would require larger and longer controlled trials.

Research problem and rationale:

Patients attending a tertiary-care centre such as DH, Mohali, may ask whether foods rich in vitamin D or an oral supplement can improve their diabetes control or cholesterol profile. Clinical teams need evidence that is relevant to local diet, affordability, medication use, patterns of vitamin D exposure, and feasibility of sustained follow-up. Advice based solely on observational associations can lead to unrealistic expectations, unnecessary purchases, or inappropriate changes to established treatment. A small, well-planned comparative study can help define feasibility and provide an estimate of short-term biomarker differences in this setting.

The research gap is not simply whether vitamin D can change a laboratory result. It is whether the strategy used to increase vitamin D exposure matters in a real-world group of adults with T2DM, and whether any observed change remains after accounting for baseline 25(OH)D, baseline HbA1c, diabetes duration, body mass index, medication changes, dietary adherence, and lipid-lowering

treatment. Direct comparative evidence between a food-based vitamin D strategy and oral vitamin D supplementation is less established than evidence comparing supplements with placebo. The proposed design should make this contrast explicit and measure the exposure as well as the outcome.

The study is also relevant because nutritional approaches vary in acceptability. A food-based intervention may be compatible with daily routines, but the actual vitamin D contribution may be modest and difficult to standardise. An oral supplement provides a more quantifiable exposure but introduces questions about indication, dose, adherence, safety, and whether it is appropriate for the participant. A robust protocol will keep these trade-offs visible and avoid treating the intervention groups as equivalent in dose when they are not.

The project should be undertaken only after institutional review confirms that the comparison is ethically acceptable. In particular, a participant with a clinically significant vitamin D deficiency or another accepted indication for treatment must not be assigned to a strategy that withholds necessary therapy. If clinical practice requires supplementation for such a participant, it must be provided, and the participant should either be managed under a pre-approved clinically appropriate pathway or excluded from a diet-only comparison. These safeguards may affect recruitment and generalisability, but they are essential to participant welfare.

Aim, objectives and hypotheses:

Overall aim: To compare the effect of a structured vitamin D-rich or vitamin D-fortified dietary strategy with clinician-directed oral vitamin D supplementation on HbA1c and lipid profile among adults with T2DM attending DH, Mohali, while maintaining standard diabetes care.

Specific objectives:

- To compare mean change in HbA1c from baseline to 12 weeks between the dietary strategy group and the oral supplementation group.
- To compare changes in total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides between groups.
- To assess changes in serum 25(OH)D, dietary vitamin D intake, and self-reported adherence over follow-up.
- To examine whether baseline vitamin D status, baseline HbA1c, body mass index, diabetes duration, or use of lipid-lowering medicines modifies the observed association.
- To document feasibility, acceptability, changes in concomitant treatment, and adverse events in both groups.

Primary hypothesis: The mean change in HbA1c from baseline to week 12 differs between the two intervention strategies. The protocol does not assume in advance which strategy will be superior. Secondary hypotheses concern potential between-group differences in each lipid fraction and serum 25(OH)D. These hypotheses should be finalised before enrolment and documented in a dated protocol and statistical analysis plan.

Null hypothesis: There is no between-group difference in mean change in HbA1c or in the prespecified lipid outcomes over the 12-week observation period. Statistical significance will not be interpreted as proof of clinical importance; the magnitude, precision, safety, feasibility, and consistency of the estimates will be considered.

3. Methods:

Study design:

The proposed design is a prospective, two-group comparative intervention study with measurements at baseline and week 12. If the institutional ethics committee and clinical team approve random allocation, a computer-generated allocation sequence with concealed assignment should be used, and the trial should be registered in an appropriate public registry before the first participant is enrolled. If random allocation is not feasible, a prospective non-randomised comparative cohort may be used, but the manuscript must then avoid describing the study as a randomised trial and should address confounding explicitly.

The study should follow the principles of prespecification and transparent reporting. SPIRIT 2025 provides guidance for reporting randomised trial protocols, while CONSORT 2025 provides a current framework for reporting randomised trial findings (Chan et al., 2025; Hopewell et al., 2025). These frameworks do not replace careful design, clinical oversight, or an appropriate analysis plan, but they help ensure that eligibility criteria, outcomes, harms, recruitment, and deviations are reported completely.

Study setting:

The study is proposed for the outpatient diabetes, medicine, or endocrinology services of DH, (Mohali), Punjab. The final protocol should confirm the exact participating department, laboratory service, recruitment pathway, and the institutional name as used by the ethics committee. Any additional clinic or laboratory involved should be named, and responsibilities for intervention delivery, sample handling, data management, and safety review should be allocated before recruitment.

Study duration and assessment schedule:

The proposed intervention period is 12 weeks for each participant, with recruitment and follow-up duration depending on clinic flow and staffing. A 12-week window permits a follow-up HbA1c assessment that reflects a meaningful period of glycaemic exposure. A shorter interval may be used for safety contacts, but the primary endpoint should remain tied to the prespecified timing. The full timeline from staff training and pilot testing to database lock and reporting should be specified in the final protocol, not inferred retrospectively.

Study population:

The target population is adults with established T2DM attending the participating DH, Mohali, clinical service. The target population should be sufficiently broad to represent routine care, while eligibility criteria should protect participants and support interpretability. Recruitment should not be restricted in ways that exclude common comorbidities without a scientific reason, but conditions that materially alter vitamin D metabolism, HbA1c reliability, or the safety of supplementation require careful clinical review.

Eligibility criteria:

Proposed inclusion criteria are: adults aged 30–70 years; an established diagnosis of T2DM documented in the medical record; usual glucose-lowering treatment that has been reasonably stable over the preceding 8–12 weeks where clinically feasible; ability to provide informed consent; and willingness to complete dietary assessment and the follow-up visit. The precise age range and stability window may be revised by the research team after feasibility assessment. Participants should have a clinical plan that permits ethical comparison of the strategies under evaluation.

Proposed exclusion criteria are: type 1 diabetes or gestational diabetes; current pregnancy or breastfeeding; acute severe illness or recent hospitalisation likely to affect glucose or lipid values; advanced kidney or liver disease if judged to compromise safety or interpretation; known hypercalcaemia; recurrent calcium kidney stones or a disorder associated with hypercalcaemia unless cleared by the treating specialist; malabsorption requiring therapeutic supplementation; recent high-dose vitamin D treatment; use of medicines that substantially alter vitamin D metabolism without a stable plan; and conditions that make HbA1c uninterpretable. The final criteria should be reviewed by clinicians rather than applied mechanically.

A participant who has a clinical indication for vitamin D correction must not be deprived of treatment for study purposes. The protocol should either define an ethically appropriate treatment pathway that applies to both groups or exclude that participant from a comparison where diet alone would otherwise be used. The ethics committee should approve the exact approach in advance. Exclusions and treatment crossovers must be documented in the participant flow diagram and reported transparently.

Sampling strategy and proposed sample size:

Consecutive sampling of eligible clinic attendees is a practical recruitment strategy for a single-centre study. Research staff should approach potentially eligible patients in a manner that does not interfere with care, explain the study in plain language, and allow sufficient time for questions. A

screening log may document numbers assessed, eligible, approached, declined, enrolled, and completing follow-up, without recording unnecessary identifiers. If random allocation is approved, allocation should occur only after eligibility and baseline data collection are complete.

A preliminary sample-size estimate is included for planning and must be verified by a biostatistician before submission. Assuming a clinically relevant between-group difference in HbA1c change of 0.5 percentage points, a common standard deviation of 0.8 percentage points, two-sided alpha of 0.05, and 80% power, a conventional two-independent-group calculation gives approximately 41 participants per group before attrition. Allowing around 15% loss to follow-up yields a target of about 48 participants per group; therefore, 100 participants, approximately 50 per group, is proposed. These values are planning assumptions rather than established properties of the DH population.

The calculation should be updated if pilot data indicate a different standard deviation, expected effect, correlation between baseline and follow-up, or attrition rate. If the primary analysis uses baseline-adjusted regression or repeated measures, the statistician may calculate a more efficient design-specific sample size. The final protocol should include the formula or software used, all assumptions, and a rationale for choosing the minimum difference considered clinically meaningful. If recruitment is constrained, the work should be described as exploratory and uncertainty emphasised.

4. Intervention procedures:

Dietary vitamin D strategy group:

Participants in the dietary strategy group will receive individualised counselling from a trained dietitian or research nutritionist. The counselling will explain locally available foods containing vitamin D and the use of reliably labelled fortified foods, while also reinforcing a balanced eating pattern appropriate for T2DM. Potential foods may include oily fish where consumed, egg yolk, and verified fortified dairy or plant-based products. Food choice will respect vegetarian preferences, affordability, allergies, cultural practices, renal or cardiovascular dietary restrictions, and the participant's usual meals.

The dietary plan should be designed to improve practical vitamin D intake without encouraging excessive calories, saturated fat, added sugar, or sodium. For example, a participant who chooses a fortified milk product should be guided to select an unsweetened option where suitable, and fish should be prepared without deep-frying or excessive salt. The intervention should not claim to deliver a fixed pharmacological-equivalent vitamin D dose unless food labels and portion records support that estimate. Dietary exposure will be measured through repeated 24-hour recalls or a validated food-frequency instrument and checked at follow-up.

Counselling should be standardised by a brief manual, sample meal plans, food-source handouts, and a record of each session. Initial counselling may occur at baseline, followed by a check-in during the intervention and a review at week 12. The number and duration of contacts should be the same across participants as far as possible. Staff should record any barriers, substitutions, seasonal changes, and reported adherence. The dietitian should not recommend unverified products or make claims that vitamin D foods can replace prescribed diabetes therapy.

Usual diabetes care will continue. If the clinical team identifies a need for vitamin D supplementation or another change in medication during the follow-up period, that care must not be withheld. The change will be recorded, and a prespecified approach will be used for the main analysis and sensitivity analysis. Participants who require rescue treatment should be managed clinically and retained in follow-up whenever appropriate, rather than disappearing from the dataset.

Oral vitamin D supplementation group:

Participants in the oral supplementation group will receive vitamin D under a clinician-approved prescription pathway. Cholecalciferol (vitamin D3) may be the preferred preparation if consistent with local formulary and clinical practice, but the dose, frequency, and duration must be set by the treating clinician according to baseline 25(OH)D, renal function, calcium status where indicated, existing supplements, comorbidities, and the institutional guideline. The protocol must not impose a

high-dose regimen solely to maximise the chance of a laboratory response. The prescribed preparation, brand or formulation where relevant, dose, route, start date, planned duration, dispensing source, missed doses, and any dose adjustment will be recorded. Participants will be instructed not to add other vitamin D or calcium supplements without discussing them with the clinical team. Medication reconciliation should be repeated at each contact because over-the-counter products and traditional preparations may contain vitamin D or calcium that are not initially disclosed. The study will monitor symptoms or laboratory findings that could suggest adverse effects, including hypercalcaemia or renal complications when clinically relevant. Safety tests will follow the treating clinician's judgment and approved protocol. Particular caution is required in participants with kidney disease, a history of nephrolithiasis, hyperparathyroidism, granulomatous disease, or concurrent medications that affect calcium balance. Any suspected serious adverse event should be assessed promptly and reported according to institutional policy.

To preserve interpretability, the oral supplement group will receive the same standard diabetes education and usual medical care as the dietary strategy group. Differences in contact time should be minimised. If the study intends to compare the strategies as they are delivered in practice rather than equalised doses, that pragmatic purpose must be explicit. Serum 25(OH)D change and adherence data will help show whether the groups experienced different vitamin D exposure.

Common care and co-intervention control:

Both groups will continue evidence-based diabetes management as determined by their treating clinicians. The study must not require the withdrawal, delay, or reduction of glucose-lowering or lipid-lowering medicines to preserve an intervention contrast. When clinically safe, major changes to glucose-lowering therapy should be recorded with date and reason, but treatment escalation must occur whenever needed. Usual care includes dietary counselling, physical activity guidance, foot and eye care, renal assessment, blood pressure management, and cardiovascular risk reduction as clinically indicated. The study team should record prescriptions for metformin, insulin, sulfonylureas, GLP-1 receptor agonists, SGLT2 inhibitors, DPP-4 inhibitors, statins, fibrates, antihypertensive drugs, glucocorticoids, anticonvulsants, and other relevant medicines where present. This list is not intended to imply that all participants use these drugs. The goal is to capture changes and identify strong confounders. Medication adherence should be assessed consistently, and participants should receive the same level of routine clinical follow-up irrespective of group.

Outcome measures and operational definitions:

The primary outcome is the between-group difference in mean change in HbA1c from baseline to week 12. HbA1c will be measured in percentage units by the participating laboratory using a method with appropriate quality control. The same laboratory method should be used at baseline and follow-up whenever possible. A percentage-point change will be reported as follow-up minus baseline; a negative value indicates a decrease in HbA1c. The analysis will report adjusted group differences with 95% confidence intervals and exact P values rather than relying on significance labels alone.

Secondary lipid outcomes are changes in total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides, reported in mg/dL or mmol/L consistently. The protocol should specify whether LDL is directly measured or calculated by the laboratory. Secondary outcomes will also include serum 25(OH)D, body weight, body mass index, dietary vitamin D intake, self-reported adherence, changes in diabetes medication, and adverse events. The secondary outcome list should be finalised before enrolment; exploratory outcomes will be labelled as such.

Vitamin D status will be assessed with serum 25(OH)D using a validated assay if resource and clinical relevance permit. Categories for deficiency, insufficiency, and sufficiency will be prespecified according to the institutional standard and the cited guidance. The protocol should not switch cut-points after observing the data. Where feasible, baseline calcium and renal function will be reviewed from routine clinical assessment or tested when indicated by the treating clinician and intervention safety plan.

Table 1. Main study variables, definitions and proposed sources

Variable	Operational definition	Timing / source
HbA1c (primary outcome)	Laboratory HbA1c, percentage; change = week 12 minus baseline	Baseline and week 12; accredited laboratory
Lipid profile	Total cholesterol, LDL-C, HDL-C and triglycerides in a consistent unit	Baseline and week 12; same laboratory where feasible
Vitamin D status	Serum 25(OH)D and prespecified status category	Baseline and week 12 if approved and available
Dietary vitamin D exposure	Food sources, fortified products, portion frequency and estimated intake	Baseline and follow-up recall / food-frequency tool
Adherence	Counselling attendance, diet checklist and/or supplement use record	Interim contact and week 12
Potential confounders	Age, sex, BMI, diabetes duration, medicines, smoking, diet, activity, renal/liver history	Baseline; key changes during follow-up
Safety outcomes	Adverse symptoms, clinically relevant laboratory abnormalities, treatment interruption	At each contact and as clinically indicated

Data collection procedures:

Trained study staff will use a structured case-report form. At screening, staff will verify age, diagnosis, relevant medical history, current medications, and consent eligibility. At baseline, participants will undergo height and weight measurement, body mass index calculation, blood pressure measurement if part of routine assessment, dietary and activity history, and laboratory testing as specified. Measurements will follow a short standard operating procedure to reduce between-observer variation. Instruments should be calibrated, and staff should receive training before first recruitment. Weight should be measured with a calibrated scale on a level surface, with participants wearing light clothing and no shoes where possible. Height should be measured using a stadiometer. BMI will be calculated as weight in kilograms divided by height in metres squared. These measures can help interpret the relationship between vitamin D status and metabolic outcomes, but changes in body weight over 12 weeks may also reflect altered caloric intake or medication effects rather than a direct vitamin D mechanism.

Dietary data should be collected in a way that reflects typical eating rather than only the day immediately before assessment. A trained interviewer can use multiple-pass 24-hour recalls on non-consecutive days, or a locally validated food-frequency questionnaire, depending on staff capacity. Intake should capture fortified product brands where possible, fish, egg, dairy and alternatives, portion sizes, cooking practices, and supplement use. Since dietary recall can be affected by memory and social desirability, the tool and limitations should be stated in the final manuscript.

Laboratory samples should be collected and processed following the laboratory's clinical protocol. Fasting instructions must be consistent where fasting lipid measurements are required. The laboratory should report quality-control procedures and the assay used for HbA1c and 25(OH)D. Samples should be labelled with a study code rather than participant names on research worksheets. Access to results that may require clinical action must remain available to the treating team.

At the interim contact, staff will assess adherence, adverse symptoms, changes in medication, additional supplement use, hospitalisation, and barriers to following the assigned strategy. At week 12, the baseline assessments relevant to the outcomes will be repeated. If a participant misses the planned window, the actual date will be recorded. Staff should make reasonable attempts to obtain

outcome measures for participants who discontinue the intervention, provided consent remains in place and collection is ethically appropriate.

Table 2. Proposed schedule of enrolment, intervention and assessments

Assessment / activity	**Screening**	**Baseline**	**Interim contact**	**Week 12**
Eligibility, consent and demographic details	Yes	Confirm	—	—
Medical history and medicines review	Yes	Yes	Update	Update
HbA1c	—	Yes	—	Yes
Lipid profile	—	Yes	—	Yes
Serum 25(OH)D	Clinical indication / per protocol	Yes if approved	As indicated	Yes if approved
Anthropometry and dietary assessment	—	Yes	Brief adherence review	Yes
Intervention delivery and adherence	—	Start	Review	Final review
Adverse-event assessment	—	Baseline review	Yes	Yes

Quality assurance and bias reduction:

To reduce selection bias, eligibility criteria and recruitment procedures will be applied consistently. If randomisation is used, the sequence should be generated independently, allocation concealed until after baseline data collection, and deviations documented. If allocation is based on patient preference or clinic workflow, reasons for group assignment must be recorded and statistical adjustment cannot fully eliminate unmeasured confounding. The design label and limitations must match what was actually implemented9 (Mohammed AS, Nadeem, Kumar M, et al., 2026).

Performance bias is difficult to avoid because participants will generally know whether they are receiving food-based counselling or tablets. However, laboratory staff and the primary analyst can be blinded to group assignment if practical. The protocol should use identical timing for measurements, consistent communication, and standardised instructions. The primary outcome and direction of change should be prespecified to prevent selective emphasis on a favourable endpoint.

Missing data should be minimised through appointment reminders, flexible scheduling, and respectful follow-up. The team should document why each measurement is missing. A complete-case analysis alone may be biased if participants who fail to return differ systematically from those who complete follow-up. The analysis plan should specify how missing values will be handled and include sensitivity analysis under reasonable assumptions. Missing outcome values must not be silently treated as no change.

Statistical analysis plan:

Baseline characteristics will be summarised by group. Continuous variables will be described using means and standard deviations when distributions are reasonably symmetric, or medians and interquartile ranges when skewed. Categorical variables will be reported as counts and percentages. Baseline differences will be described to understand comparability; they should not be used as a mechanical test of whether randomisation "worked." The magnitude of baseline imbalance and clinical relevance will guide adjustment decisions.

The primary analysis will estimate the difference between groups in week-12 HbA1c adjusted for baseline HbA1c, using linear regression or analysis of covariance if assumptions are acceptable. Group assignment will be the primary explanatory variable. Prespecified covariates may include

age, sex, baseline 25(OH)D, BMI, diabetes duration, baseline medication class, and changes in glucose-lowering therapy, but the number of covariates must be appropriate for the sample size. Adjusted mean differences, 95% confidence intervals, exact P values, and unadjusted descriptive changes will be reported.

For lipid outcomes, similar baseline-adjusted models will be used for total cholesterol, LDL-C, HDL-C, and triglycerides. Triglycerides may require log transformation or a robust approach if markedly skewed. Results should include units and make clear whether a change is expressed as follow-up minus baseline. Multiple secondary outcomes increase the probability of chance-positive findings; therefore, secondary analyses should be interpreted with appropriate caution and, if the research team chooses, a multiplicity strategy should be prespecified.

If randomised allocation is implemented, the main analysis should follow the intention-to-treat principle, analysing participants according to assigned group regardless of adherence, while documenting clinical rescue treatment. A per-protocol analysis may be presented as secondary only with explicit criteria for adherence and a clear warning that it may introduce bias. If randomisation is not used, the analysis must be described as an adjusted observational comparison, with covariate selection justified on clinical grounds and residual confounding acknowledged. Potential effect modification by baseline vitamin D status may be investigated using a prespecified interaction term, provided the sample size supports it. Other subgroup analyses such as HbA1c category, BMI, diabetes duration, or statin use should be limited and labelled exploratory. The study should not claim a subgroup benefit only because one subgroup has a statistically significant result while another does not; the interaction test is the appropriate comparison. Estimates with wide confidence intervals will be interpreted as imprecise rather than as proof of no effect.

No numerical results should be inserted until the data are collected, cleaned, and analysed. The final manuscript should report the number screened, excluded, enrolled, allocated, followed up, and analysed; baseline characteristics; the primary outcome estimate; secondary outcomes; adherence; medication changes; and adverse events. Results must be presented irrespective of direction or statistical significance, in accordance with transparent trial reporting guidance (Hopewell et al., 2025).

Ethical considerations and participant safety:

Prior approval from the competent Institutional Ethics Committee is mandatory before recruitment, access to research-only samples, or intervention delivery. The final protocol must include the committee name, approval number, approval date, and the approved consent documents. If the study is randomised or otherwise interventional, registration should be completed in a recognised public clinical trial registry before the first participant is enrolled, consistent with applicable Indian regulatory and institutional requirements. This document itself is not evidence of ethics approval or trial registration.

Each participant will receive a plain-language explanation of the purpose, procedures, anticipated benefits, potential risks, confidentiality arrangements, alternatives, and right to decline or withdraw without affecting medical care. Written informed consent should be obtained before research procedures. Participants should be told that improvement in HbA1c or lipids is not guaranteed and that vitamin D is not established as a substitute for diabetes or cholesterol medicines. Consent materials should be available in a language the participant understands, with interpreter assistance where approved.

The main foreseeable risks include inconvenience from dietary recording and follow-up, phlebotomy discomfort, incidental abnormal laboratory findings, and possible adverse effects associated with supplementation. Excess vitamin D can cause hypercalcaemia and related renal or systemic complications; the risk depends on dose, duration, concurrent calcium intake, underlying conditions, and individual susceptibility. The protocol should include a process for clinical review, dose interruption, testing, referral, and reporting of suspected adverse events. There should be no unsupervised high-dose supplementation.

Participants with vitamin D deficiency or another accepted treatment indication must receive appropriate clinical care. The allocation procedure cannot override clinical judgement. When the

clinician decides that supplementation, dose adjustment, or another treatment is necessary, the action should be taken promptly and documented. The investigator must not maintain a diet-only allocation at the expense of a participant's safety. The ethics committee should explicitly review this rescue-treatment pathway and its impact on the primary analysis.

Confidentiality will be protected through coded study identifiers, secure storage of paper forms, password-protected electronic files, restricted access, and retention/destruction according to institutional policy. Identifying data should be stored separately from analytical data where feasible. Publications and presentations will use aggregate results and will not identify individual participants. Any plan to share de-identified data should be described in the approved protocol and consent documents.

Expected findings and interpretation framework:

Because the study has not yet been conducted, expected findings must be treated as possibilities rather than results. One possibility is that oral supplementation produces a larger measurable increase in serum 25(OH)D because its dose is more quantifiable than food-based intake. That biochemical change may or may not be accompanied by a meaningful between-group difference in HbA1c or lipid profile. Another possibility is that a structured dietary strategy improves overall food quality and adherence, with metabolic changes related partly to energy balance, fibre intake, or other dietary substitutions rather than vitamin D alone.

If the supplement group shows a larger reduction in HbA1c, the interpretation should consider baseline 25(OH)D, achieved 25(OH)D, duration of exposure, adherence, baseline HbA1c, medication changes, and regression to the mean. A statistically significant adjusted difference can still be too small to matter clinically. If no difference is found, the study should not conclude that vitamin D has no biological role; a single-centre sample and 12-week follow-up may be insufficient to detect small effects, particularly when group exposure differs only modestly.

If lipid outcomes improve in one group, the analysis should consider statin dose changes, fasting status, body weight, dietary fat composition, alcohol use, glycaemic improvement, thyroid status, and kidney function. Since multiple lipid fractions are examined, isolated positive results should be treated cautiously. The study should report the full pattern of results, including outcomes that do not improve, instead of selectively highlighting the largest effect.

A finding that a dietary strategy is feasible and acceptable may be useful even if its HbA1c or lipid effect is uncertain. Feasibility indicators include attendance, completion of follow-up tests, affordability of foods or supplements, reported adherence, ease of food substitution, and need for clinical rescue treatment. These measures can inform whether a larger multicentre trial is practical. They should not be mislabelled as proof of metabolic efficacy.

Interpretation should remain consistent with the broader evidence. Recent meta-analyses suggest potential modest effects on glycaemic markers in some T2DM subgroups but show substantial heterogeneity; other trials have not demonstrated consistent benefits. Current guidance supports individualised nutrition and evidence-based diabetes care rather than routine supplementation for glycaemic improvement in all patients (Farahmand et al., 2023; Afraie et al., 2024; Chen et al., 2024; American Diabetes Association Professional Practice Committee for Diabetes, 2026).

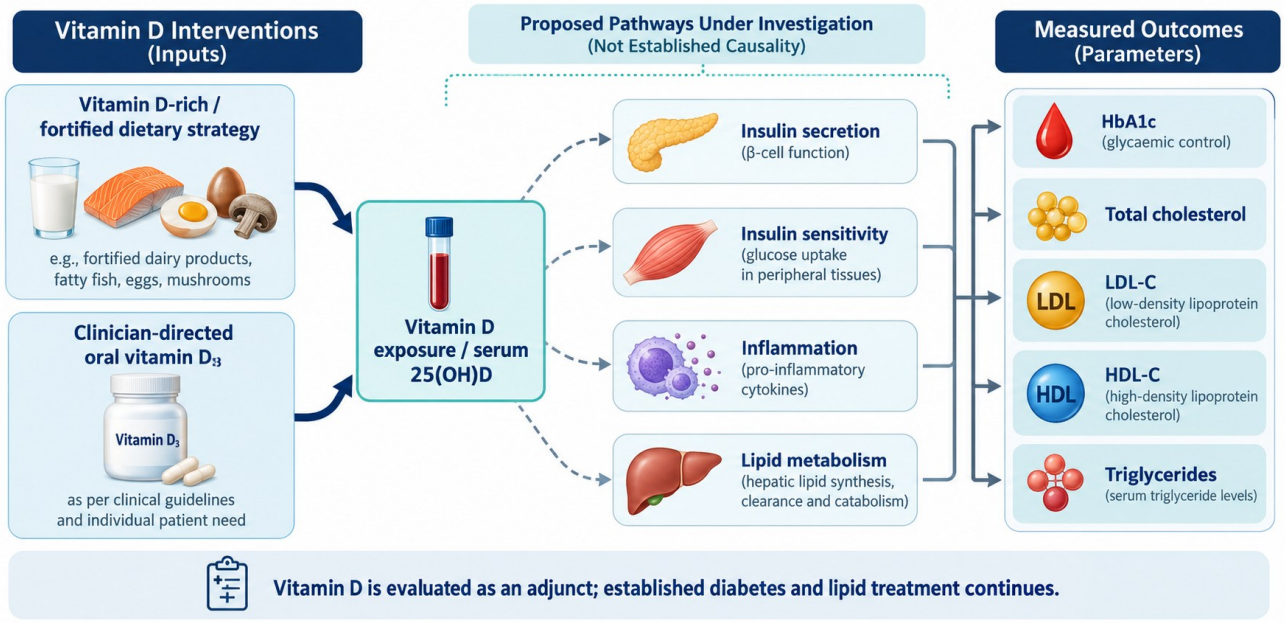


Figure 1. Proposed biological and clinical framework linking vitamin D exposure with glycaemic and lipid outcomes

5.Discussion:

The proposed study addresses a clinically relevant question at the intersection of micronutrient status, nutrition counselling, and routine T2DM management. Its potential value lies in direct comparison of two strategies that patients may encounter in practice. A strength of the proposal is the inclusion of both HbA1c and the complete lipid profile, alongside vitamin D status and adherence. This permits the research team to distinguish whether biochemical exposure changes occur and whether they accompany clinically monitored metabolic markers.

A central challenge is that a vitamin D-rich diet and a prescribed supplement are not equivalent interventions. Dietary exposure depends on actual foods, product fortification, portion size, absorption, and adherence; supplement exposure can be quantified more readily but may be tailored to clinical need. Any difference between groups therefore compares strategies, not equal doses of vitamin D. The manuscript should state this clearly and avoid attributing every observed effect specifically to vitamin D rather than to the broader dietary pattern.

The evidence base supports equipoise rather than a predetermined direction. Farahmand and colleagues and Chen and colleagues reported modest pooled reductions in HbA1c among people with T2DM, with stronger effects in certain subgroups, particularly where deficiency was present. Afraie and colleagues reported pooled changes in glycaemic and lipid markers but also substantial heterogeneity. Conversely, individual trials such as the SUNNY trial and the cardiovascular-risk study by Angellotti and colleagues did not demonstrate consistent broad benefit (Farahmand et al., 2023; Chen et al., 2024; Afraie et al., 2024; Krul-Poel et al., 2015; Angellotti et al., 2019). This discordance is a reason to measure outcomes rigorously, not to assume a positive result.

Baseline vitamin D status deserves special attention. If participants begin with low 25(OH)D, correcting deficiency may produce a larger change in serum concentration and may plausibly be associated with metabolic changes. If participants begin with adequate status, additional supplementation may have little effect on glycaemia, while the risk of unnecessary treatment remains. For this reason, baseline 25(OH)D should be measured where feasible, and the protocol should prespecify whether participants with deficiency are included under a common treatment pathway or are excluded from a diet-only comparison for safety.

The primary outcome should remain HbA1c because the title and clinical objective emphasise glycaemic control. The lipid profile should be treated as secondary, with each fraction reported. A 12-week outcome interval is long enough to observe an HbA1c response to changes in glucose

exposure, yet too short to determine cardiovascular event reduction or long-term sustainability. The study should avoid presenting biomarkers as surrogate proof of reduced cardiovascular morbidity or mortality.

Concomitant treatment is likely to complicate interpretation. A treating clinician may increase an antihyperglycaemic medicine because glucose remains above target, or may initiate a statin because cardiovascular risk has been reviewed. These actions are desirable clinical care and must not be restricted. The correct response is to record them carefully and choose an analysis plan that estimates the intervention contrast transparently. Sensitivity analysis can explore the effect of excluding or adjusting for major treatment changes, but such analysis must not replace an unbiased primary analysis.

A strength of using a hospital-based setting is the availability of laboratory testing and medical review, which can improve diagnostic accuracy and safety monitoring. However, a single-centre study may recruit a population whose education, access to healthcare, disease severity, dietary practices, and socioeconomic profile differ from those elsewhere. Consecutive recruitment and detailed description of the sample will support a more realistic assessment of generalisability. Future studies may need several centres and longer follow-up to assess durability and patient-centred outcomes.

The dietary intervention must be designed with local food availability in mind. A plan that relies on expensive fish, specialised imported products, or foods that participants do not usually consume will have limited acceptability. Counselling should prioritise familiar foods and verified fortification labels and should preserve the broader dietary principles used in diabetes care. Participant feedback may show that the most useful effect is improved nutrition literacy or more consistent food choices, even if the measured vitamin D exposure is modest.

The supplement arm requires a clear safety framework. Vitamin D is widely available, but unsupervised high-dose or prolonged dosing can be harmful in susceptible individuals. The prescription should be medically indicated or ethically justified, tailored to baseline status and clinical factors, and monitored according to local standards. Supplementation must never be treated as a universally benign intervention simply because it is a nutrient. Recording concurrent calcium use and non-prescription supplements is particularly important.

Strengths of the proposed study:

- It evaluates two practical vitamin D strategies in a defined clinical setting rather than relying only on an observational association.
- It prespecifies HbA1c as the primary outcome and measures each major lipid fraction separately.
- It includes baseline vitamin D status, dietary exposure, adherence and medication history to support better interpretation.
- It prioritises clinical safety and does not permit indicated treatment to be withheld for research purposes.
- It plans to report confidence intervals, missing data, adverse events and deviations rather than only statistically significant findings.

Limitations and potential sources of bias:

The anticipated sample size is modest and is based on an assumed HbA1c difference and standard deviation that require validation. The study may be underpowered for smaller effects, for separate lipid outcomes, and especially for subgroup analyses. Confidence intervals may be wide, and lack of statistical significance should not automatically be interpreted as equivalence. If the study is non-randomised, differences in baseline motivation, diet, socioeconomic position, clinical severity, and clinician prescribing may remain despite adjustment.

Participants and intervention staff cannot easily be blinded to food counselling versus oral supplements. Self-reported dietary intake and adherence are vulnerable to recall and social-desirability bias. Serum 25(OH)D testing may be costly and may not be available for every participant; if measurements are missing, the study may be less able to assess whether the interventions changed vitamin D exposure. Seasonal variation during recruitment may also affect cutaneous synthesis and therefore the difference between baseline and follow-up.

Changes in diabetes medicines, statins, body weight, physical activity, infection, renal function, and dietary quality may influence outcomes during the follow-up period. It is not ethical to prevent clinically indicated treatment changes, so complete isolation of a vitamin D effect may not be possible. The analysis can document and adjust for measured changes, but residual confounding is possible. Short-term changes in HbA1c or lipids also cannot establish longer-term cardiovascular benefit or safety.

The dietary and supplement strategies will differ in more than vitamin D dose. Food-based counselling may alter energy intake, fat quality, or other micronutrients, whereas a supplement has fewer direct dietary co-effects. Therefore, even a randomised comparison would estimate the effect of the strategy package, not necessarily the isolated effect of vitamin D. A future trial designed to isolate vitamin D specifically may need a placebo-controlled supplementation arm, identical dietary counselling, and stratification by baseline vitamin D status, while still providing standard treatment for deficiency.

The findings will be most applicable to patients similar to those recruited from DH, Mohali, and to the exact intervention implemented. Results may not generalise to people with advanced chronic kidney disease, severe liver disease, malabsorption, pregnancy, or other excluded conditions. The final paper should report these limits explicitly and avoid extrapolating from a single-centre short-term study to all people with T2DM.

Clinical and public-health implications:

For clinicians, the most important implication is to separate the management of vitamin D deficiency from the unproven use of vitamin D as a universal metabolic treatment. When deficiency is diagnosed or a clinical indication exists, it should be treated according to appropriate care standards. For a patient with adequate vitamin D status, prescribing additional vitamin D solely to lower HbA1c or cholesterol should not replace interventions with established benefit. The study could clarify how this distinction is understood and implemented in local practice.

For dietitians and diabetes educators, a structured food-based strategy may provide an opportunity to discuss fortified foods, protein sources, unsaturated fats, fibre, calorie balance, label reading, and affordable meal planning. The intervention should not focus narrowly on one nutrient at the expense of the wider eating pattern. Education should be practical, culturally acceptable, and adapted to the participant's preferences and medical needs. A successful programme should be measured by uptake and feasibility as well as laboratory change.

For policymakers and hospital administrators, locally relevant data can inform whether dietary counselling materials, staff training, or laboratory testing are feasible within routine diabetes services. If the study identifies barriers such as cost, limited fortified-food availability, difficulty returning for testing, or confusion about supplements, these issues may guide service improvement. Any policy recommendation should be proportionate to the strength of evidence and should not convert an exploratory biomarker result into a broad population supplement recommendation.

For future researchers, the study can inform a larger, randomised, well-powered trial with longer follow-up, stratification by baseline 25(OH)D, consistent dietary education in all arms, a clinically justified supplement dose, blinded laboratory assessment, and systematic adverse-event monitoring. Future work may also measure patient-reported outcomes, medication adherence, quality of life, and cost-effectiveness. Clinical endpoints and long-term cardiovascular outcomes would require substantially larger samples and longer observation.

Dissemination, authorship and data management:

The research team should agree before recruitment on authorship responsibilities, data access, investigator roles, and the process for resolving disagreements. Authorship should reflect substantial contributions to the design, conduct, analysis, or interpretation and to drafting or revising the manuscript, together with approval of the final version and accountability for the work. Contributors who do not meet authorship criteria may be acknowledged with permission. No author names should be inserted into the submission version until the investigators confirm contributions and affiliations.

The final results should be reported regardless of whether findings support the original hypothesis. A report may be submitted to a peer-reviewed journal, presented at a scientific meeting, and shared with the participating clinical service in plain language. Any secondary analyses should be described as prespecified or exploratory. The results should not be marketed as proof that vitamin D cures diabetes, reduces cardiovascular events, or allows reduction of prescribed medication unless appropriate evidence actually supports those claims.

Data management will include a protocol-defined data dictionary, secure coded database, controlled access, regular backups, and a record of any amendments. The data-sharing plan should state whether de-identified participant-level data and analysis code may be shared, under what conditions, and after which approvals. The consent form and institutional policy should align with the plan. Data should be retained for the period required by applicable institutional and regulatory guidance.

6. Conclusion:

This protocol proposes a prospective comparison of a vitamin D-rich or vitamin D-fortified dietary strategy and clinician-directed oral vitamin D supplementation among adults with T2DM attending DH, Mohali. The main outcome is the between-group change in HbA1c over 12 weeks, with changes in lipid fractions, serum 25(OH)D, adherence, and safety as secondary outcomes. Existing evidence suggests possible modest metabolic effects in some populations, particularly in those with low vitamin D status, but findings are heterogeneous and routine supplementation is not established as a universal glucose- or lipid-lowering treatment. A rigorous study must preserve usual diabetes care, treat clinically indicated deficiency, document concomitant medicines, prespecify analysis, and report results transparently. No study findings can be claimed until recruitment, follow-up, and analysis have actually been completed.

Declarations to be completed before journal submission:

Ethics approval: To be added after written approval from the appropriate Institutional Ethics Committee. Do not state that approval has been obtained until the approval letter is available.

Trial registration: To be added if applicable, including registry name, registration number, and registration date. Registration should occur before enrolment where required.

Informed consent: Written informed consent is planned; actual consent procedures must be reported after the study is conducted.

Funding: To be completed by the investigator team. State the funder and its role, or state that no external funding was received only if that is true.

Conflicts of interest: To be completed by all authors. Do not insert “none” without individual declarations.

Data availability: To be completed based on the approved data-sharing plan and actual dataset.

Author contributions: To be completed using the actual contributions of each author.

Acknowledgements: To be added only with permission from named individuals or services.

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