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**Alternative and Complementary Therapies Used in the
Diabetes Management**

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Abstract

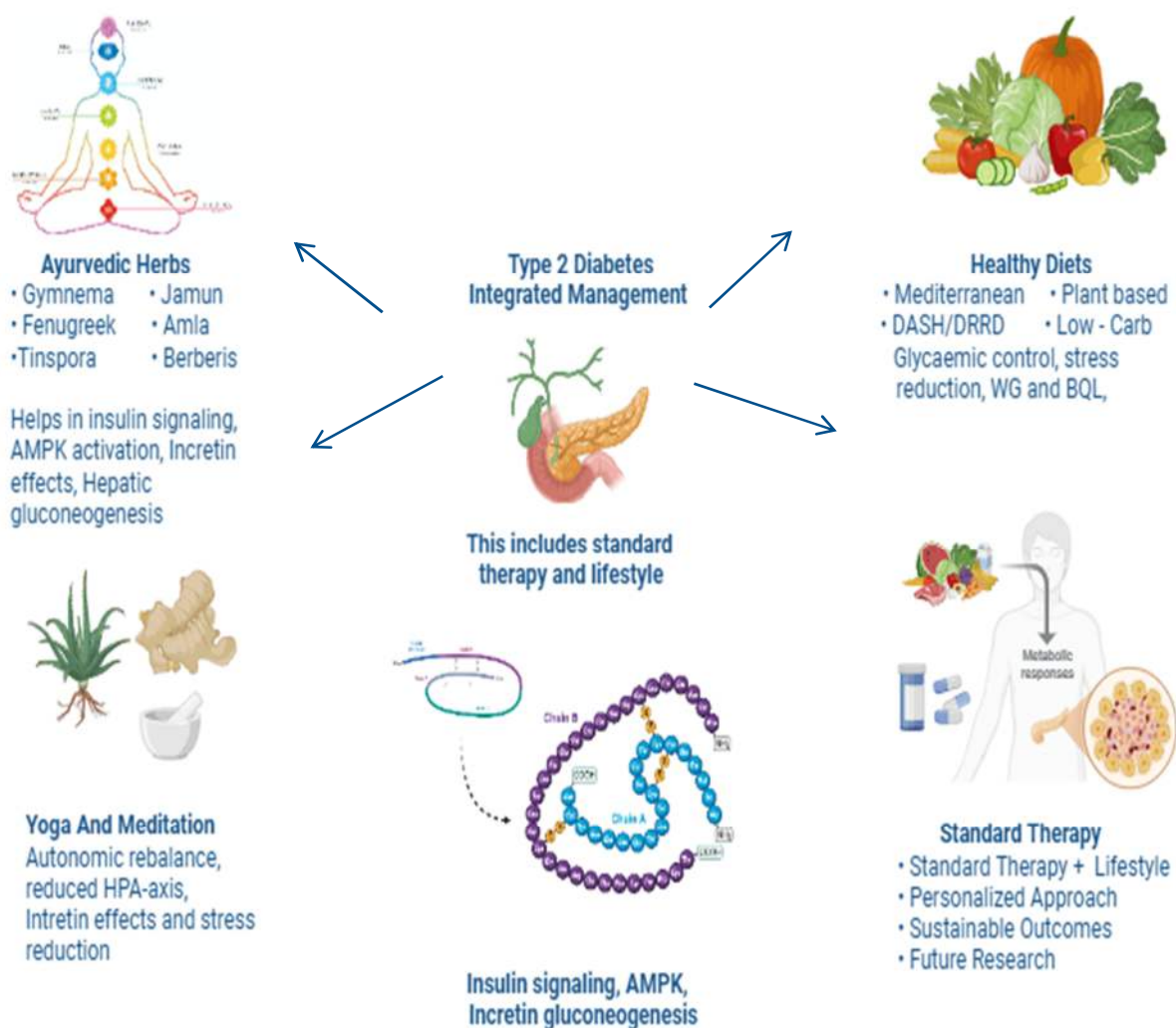
Type 2 diabetes mellitus (T2D) is a complex metabolic disorder driven by hyperglycaemia, insulin resistance, inflammation, and cardiometabolic dysfunction, necessitating multi targeted and integrative management strategies. This review synthesizes evidence up to 2025 on Ayurvedic polyherbal formulations, individual botanicals, yoga and meditation, and major dietary patterns as adjuncts to standard diabetes care. Recent clinical data indicate a cautiously positive but incremental role for well standardized Ayurvedic formulations. Randomized and real-world studies of polyherbals (e.g., combinations containing *Gymnema sylvestre*, *Syzygium cumini*, *Trigonella foenum-graecum*, *Emblica officinalis*, *Tinospora cordifolia*, *Berberis aristata*, and cinnamon) demonstrate modest but clinically meaningful reductions in fasting glucose and HbA_{1c} (0.3–0.8%) over 8–24 weeks when used alongside conventional therapy, with generally good short-term tolerability. Mechanistic and network-pharmacology studies explain these effects through poly-target actions on insulin signaling (IRS–PI3K–Akt), AMPK activation, incretin modulation, hepatic gluconeogenesis, oxidative stress, adipose inflammation, and gut-level carbohydrate handling.

Focused sections detail fenugreek, jamun, and gurmar, highlighting their bioactive constituents, mechanistic plausibility, formulation advances, dosing considerations, and safety caveats.

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Meta-analyses and recent randomized trials show that yoga and mindfulness-based interventions yield small-to-moderate improvements in glycaemic indices, stress, and quality of life, mediated by autonomic rebalance, reduced HPA-axis activation, improved muscle glucose uptake, and better self-care behaviors. Dietary pattern evidence from 2025 confirms that Mediterranean, plant-based, DASH/DRRD, and low-carbohydrate or ketogenic diets can improve glycaemic control, weight, and cardiometabolic risk, with differences in sustainability and patient suitability. Overall, the integrated evidence supports a personalized, adjunctive approach: combining evidence-guided Ayurvedic nutraceuticals, structured mind–body practices, and sustainable dietary patterns with standard pharmacotherapy. Future priorities include longer, double-blind trials with standardized products, mechanistic human studies, and pragmatic implementation research to clarify long-term efficacy, safety, and generalizability.

Graphical Abstract



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1.1 Introduction

Ayurvedic medicine has long proposed multi-herb (“polyherbal”) formulations often in tablet, churna or decoction form to address both hyperglycaemia and its cardiometabolic context. In 2025, the clinical evidence base nudged forward: a randomized trial of a branded polyherbal mix, new real-world prospective data from South Asia, and several mechanistic/preclinical papers that help explain why certain classics (e.g., Gymnema, Jamun, Amla, Tinospora, Berberis/berberine, Cinnamon) might matter clinically. Overall, the signal is cautiously positive especially for adjunctive use alongside standard care while the usual caveats around product quality, standardization, and trial rigor still apply. A multi-arm, open-label randomized trial evaluated an Indian polyherbal formulation (“DiabetEaze”) in adults with type 2 diabetes across four sites. Compared with usual care arms, the formulation achieved statistically significant reductions in fasting plasma glucose and HbA1c over 12 weeks, with no serious adverse events reported(1).

1.2 Importance

A narrative synthesis in Protoplasma mapped how classic Ayurvedic botanicals influence insulin signaling (IRS/PI3K-Akt), incretin pathways (DPP-IV inhibition), hepatic gluconeogenesis, AMPK activation, mitochondrial function, and low-grade inflammation the web of mechanisms you would want to target in T2D. Second, focused trials on individual botanicals or combinations kept arriving. A small randomized crossover study in Appetite showed Gymnema sylvestre reduced high-sugar food desire and intake, a behavioral lever that can indirectly improve glycaemia when paired with dietetic support In the European Journal of Nutrition, a double-blind randomized controlled trial of berberine combined with cinnamon, two bioactive compounds well-integrated into South Asian practices, demonstrated a reduction in HbA1c and HOMA-IR. Though berberine is often discussed in TCM contexts, its principal plant source *Berberis aristata* (daruharidra) is classical Ayurveda; cinnamon (dalchini) is likewise canonical. In 2025, the case for Ayurvedic formulations in T2D is stronger than a decade ago especially for well-standardized polyherbals used as adjuncts to standard care. High-quality, longer double-blind trials across diverse brands remain the next priority. Until then, clinicians and patients can make careful, evidence-guided choices by privileging formulations with contemporary human data, mechanistic plausibility, and rigorous quality control(1,2).

1.3 Work of these Ayurvedic formulation

Polyherbals seem to act “poly-target”: modest effects across multiple nodes (gut hormone modulation, delayed carbohydrate absorption, insulin secretion/sensitivity, hepatic glucose output, adipose inflammation, oxidative stress). Animal work in 2025 on an established Tamil Nadu polyherbal (MD-1: *Phyllanthus amarus*, *Tinospora cordifolia*, *Emblica officinalis*, *Syzygium cumini*, *Gymnema sylvestre*, *Cassia auriculata*) showed lowered fasting glucose and triglycerides, improved adipose tissue inflammation ($\downarrow$ TNF- α , $\uparrow$ PPAR- γ /GLUT4 expression), and better oxidative stress markers mechanistic changes aligned with human metabolic outcomes. Network pharmacology for *Syzygium cumini* (Jamun) further tied seed constituents to PI3K-Akt, AMPK, and PPAR signaling hubs relevant to insulin action. A 2025 scoping review on Ayurvedic therapeutics for T2D likewise emphasizes AMPK and mitochondrial bioenergetics as recurring targets(3).

Table 1: Nutraceuticals involved in diabetes management

Nutraceutical	Key Bioactives	Proposed Mechanisms	Typical Studied Dose (2025)	Evidence Summary (2025)	Safety Notes	References
Cinnamon (Cinnamomum spp.)	Cinnamaldehyde, procyanidins	Improves insulin signaling, AMPK activation, delays gastric emptying, α -glucosidase inhibition	1–3 g/day powder or extract	Systematic review/meta-analysis (2025) shows modest HbA1c & fasting glucose reductions, but heterogeneous results	Generally safe; Cassia species maximum in coumarin → prevent large quantity of doses	(4)
Bitter melon (Momordica charantia)	Charantin, polypeptide-p, cucurbitane triterpenoids	Insulin-mimetic/secretagogue, GLUT4 translocation, α -glucosidase inhibition	2.4–3.6 g/day capsule for 4–12 weeks	RCTs in prediabetes showed improved fasting glucose, insulin, and smaller HbA1c rise vs. control	Occasional mild GI upset; effect depends on preparation	(5,6)
Berberine (as Berberine-UDCA, HTD1801)	Berberine + ursodeoxycholic acid	AMPK activation, bile acid signaling, NLRP3 inflammasome inhibition	500–1000 mg twice daily, 12 weeks	Phase-2 RCT in T2D: HbA1c ↓ 0.4–0.7%, improved FPG, lipids, liver enzymes	GI upset; potential drug–drug interactions; investigational in some countries	(7)

1.4 Clinical effects

Pooled estimates across Ayurvedic medicines vary with formulation and trial quality, but the center of gravity sits around small to moderate HbA1c reductions (0.3 to 0.8% over 8 to 24

weeks) when used as add ons to diet and standard therapy comparable to other nutraceutical adjuncts. The 2025 DiabetEaze RCT reported meaningful glucose and HbA1c improvements within 12 weeks , while the real-world LG cohort sustained glycaemic and quality-of-life gains to 24 weeks. The berberine cinnamon RCT saw HbA1c and insulin resistance metrics improve vs. placebo. That said, product-to-product variability is high; effects seen with one branded formula shouldn't be assumed for another without equivalent standardization and dose.

1.5 Safety and standardization

Short-term tolerability in 2025 trials was generally good, with GI symptoms (bloating, mild nausea) the most common. Still, quality control matters. Case reports (pre-2025) have associated *Tinospora cordifolia* with idiosyncratic liver injury; while causality remains debated, it underscores the need for authenticated botanicals and batch testing. Encouragingly, the 2025 MD-1 preclinical work documented absence of heavy metals, aflatoxins and pesticides in tested batches and used HPTLC to quantify marker compounds (phyllanthin, berberine, gallic acid, gymnemic acids). Clinically, prudent use means: check LFTs if formulations include *Tinospora*; watch for additive hypoglycaemia when combined with insulin/sulfonylureas; review interactions (e.g., berberine can inhibit CYP3A4/P-gp at higher exposures)(8).

1.6 Ayurvedic formulations fit in care

As adjuncts. For people already on metformin and other agents, a standardized, studied polyherbal can be trialed for 8 to 12 weeks with objective monitoring: CGM metrics (time in range, mean glucose), fasting glucose, and HbA1c at 3 months. Clinicians should choose products backed by named-batch standardization and published clinical data (ideally RCTs, at minimum prospective cohorts). Pairing “metabolic-active” herbs with lifestyle coaching may compound benefits: e.g., *Gymnema* to blunt sugar cravings while a dietitian helps reduce refined carbohydrates. For weight-forward phenotypes, anti-inflammatory/adipose-targeted blends (MD-1-like compositions) are mechanistically appealing. For individuals with insulin resistance and dyslipidaemia, the combination of berberine and cinnamon is backed by supportive RCT data(9).

1.7 Practical checklist for 2025

1. **Evidence:** Prefer formulations with 2025-era human data (e.g., DiabetEaze RCT, LG cohort) individual botanicals with RCT support (*Gymnema* for cravings; berberine+cinnamon for glycaemia/IR).
2. **Product quality:** Demand third-party testing and marker assay data (e.g., HPTLC/LC-MS content of gymnemic acids, gallic acid, berberine, cinnamaldehyde).
3. **Safety:** Baseline meds review; counsel on hypoglycaemia risk with insulin/secretagogues; consider LFTs if *Tinospora* is included; monitor GI tolerance.
4. **Follow-up:** Reassess at 12 weeks; continue only if HbA1c, fasting glucose, or CGM time-in-range improve without side effects.

5. **Define expectations:** Benefits are usually incremental and moderate; they are most effective when combined with nutrition, physical activity, adequate sleep, and stress management.

1.8 Types of Ayurvedic formulation

1.8.1 Methi (Fenugreek)

1) Introduction

Fenugreek (methi) has been used for a long time as both a culinary ingredient and a traditional remedy in South Asia, the Middle East, and North Africa. Modern clinical interest centers on seed preparations (powder, soaked seeds, extracts) and isolated compounds especially soluble fiber, the amino acid 4-hydroxyisoleucine, saponins (e.g., diosgenin derivatives) and alkaloids such as trigonelline which together are thought to modify carbohydrate handling and insulin physiology. For motivated patients interested in complementary approaches, small-to-moderate amounts of fenugreek (either standardized extract at trial doses or culinary seed preparations) can be considered as an adjunct to lifestyle measures and prescribed therapy, with clear counseling about monitoring glucose (and medication adjustment if hypoglycemia occurs). Prefer products with published clinical evaluation or standardized extracts, and avoid high-dose unmonitored use in pregnancy or in patients at high hypoglycemia risk. Clinicians should view fenugreek as supportive not substitutive of evidence based pharmacotherapy(10).

2) Mechanisms that matter clinically

Multiple mechanistic lines, from in-vitro and animal work reviewed in clinical overviews, help explain why fenugreek could lower blood glucose in humans. Soluble gum/galactomannan slows gastric emptying and reduces post-prandial glucose excursions by delaying carbohydrate absorption. 4-Hydroxyisoleucine exhibits the ability to stimulate insulin secretion in isolated islets and enhances insulin signaling in muscle and adipose tissues through the PI3K/Akt pathways, facilitating the translocation of GLUT-4 and promoting glucose uptake. Other pathways reported include mild inhibition of carbohydrate digesting enzymes (α amylase/ α -glucosidase), modulation of hepatic gluconeogenesis ($\downarrow$ glucose-6-phosphatase activity), anti-inflammatory effects and favorable changes in lipid metabolism all biologically plausible adjuncts to glycemic control. These mechanisms are repeatedly summarized in narrative reviews of preclinical and clinical literature(9,11).

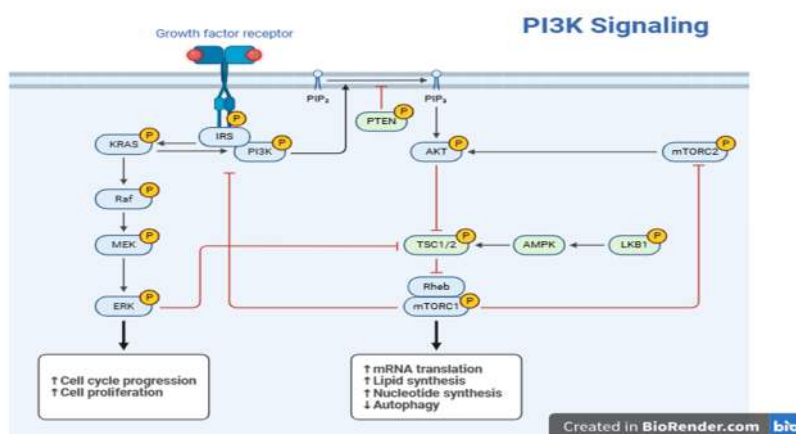


Figure 1: PI3K Signaling

The above figure, (figure 1) shows the PI3K–Akt signaling cascade which is the central “workhorse” that converts insulin’s arrival at the cell surface into the metabolic actions people care about: moving glucose from blood into muscle and fat, storing it as glycogen, and telling the liver to stop making more glucose. Think of insulin as a key, the insulin receptor as the lock, and the PI3K→Akt pathway as the chain of gears that turns when the key is pushed in when the gears turn smoothly, glucose is handled well; if one cog slips, blood sugar rises.

Mechanistically, insulin binding triggers phosphorylation of insulin-receptor substrates (IRS proteins), which recruit class IA PI3K to the membrane. PI3K converts PIP₂ into PIP₃, creating membrane docking sites that draw in PDK1 and Akt. PDK1 (and mTORC2) phosphorylate Akt at two key residues, fully activating it. Active Akt then phosphorylates multiple effectors: it promotes GLUT4 translocation in muscle and adipose (raising glucose uptake), inhibits GSK-3 (activating glycogen synthase for glucose storage), and phosphorylates FOXO transcription factors (repressing gluconeogenic gene expression in liver). Together these actions lower circulating glucose and restore metabolic balance(12,13).

Table 2: (Nutraceuticals in fenugreek (methi) formulations for T2D)

Nutraceutical (class)	Seed part / source	Primary antidiabetic mechanisms	Evidence level (to 2025)	Formulation / dose notes
Galactomannan (soluble fibre)	Gum fraction of seed	Forms viscous gel in gut → delays gastric emptying, slows carbohydrate absorption → lowers post prandial glucose. Also binds cholesterol.	Strong mechanistic + human support; featured in reviews & RCTs.	Present in whole seed powder; effective at food doses (several g/day) or as concentrated gum fractions.
4-Hydroxyisoleucine (4-HIL)	Seed protein fraction	Stimulates insulin secretion in a glucose-dependent manner, enhances β-cell sensitivity and insulin signaling (PI3K/Akt) based on preclinical studies.	Good preclinical evidence; supported by some clinical formulations and mechanistic reviews.	Often present in standardized extracts; clinical formulations report benefits at mg hundreds mg daily equivalent(14).

Nutraceutical (class)	Seed part / source	Primary antidiabetic mechanisms	Evidence level (to 2025)	Formulation / dose notes
Steroidal saponins / sapogenins (diosgenin, furostanolic saponins)	Seed saponin fraction	Improve insulin sensitivity, modulate lipid metabolism, antioxidant and anti-inflammatory effects; may protect liver and kidney in animal models.	Strong preclinical, included in clinically tested extracts (e.g., Fenfuro™).	Many proprietary extracts enrich saponins; typical clinical extract doses 500 mg bid in trials(15).
Trigonelline alkaloids	& Seed alkaloid fraction	May influence glucose metabolism and lipid profiles; potential insulin sensitizing via multiple pathways.	Moderate evidence (preclinical + small human data / included in meta-analyses).	Occurs in whole seed; content varies with processing standardization improves reproducibility.

3) Clinical trial evidence from 2025

Clinical trials have been heterogeneous in dose, formulation, duration and quality a point emphasized by systematic reviewers. Large multicenter add-on trials of a proprietary fenugreek extract (Fenfuro) reported significant reductions in fasting and post-prandial plasma glucose when used alongside standard therapy, and improved some insulin-sensitivity markers in secondary analyses. Practical, food-based interventions (for example, adding soaked fenugreek seed powder or incorporating seeds into bread) have also produced short-term reductions in fasting and post-prandial glucose in randomized or crossover trials, often over 4–12 weeks, albeit with variable HbA_{1c} change depending on study length and baseline control. A 2025 randomized, double blind, placebo controlled clinical trial explored co supplementation of low-dose berberine and fenugreek seed powder in people with established T2DM and found clinically meaningful improvements in Fasting blood glucose, HbA_{1c}, and inflammatory markers were measured over a 12-week period and compared to a placebo, indicating that fenugreek may play a positive role in a combined nutraceutical approach. Importantly, many smaller randomized trials included in systematic reviews showed reductions in postprandial glucose and, in longer studies, modest HbA_{1c} lowering; however, effect sizes varied and several trials had methodological limitations (small samples, incomplete blinding, heterogeneous doses)(16).

4) Benefits

Meta-analytic syntheses that pooled randomized trials available up to late 2025 report a pattern: consistent reductions in post-prandial glucose and modest improvements in HbA_{1c} when fenugreek is taken for weeks to months, but high heterogeneity across trials limits precise effect estimates. One pooled analysis that included trials through November 2022 reported a

statistically significant decrease in HbA_{1c} (mean difference approximately 0.8% in pooled estimates from available randomized data), while results for fasting glucose were mixed owing to study heterogeneity and variable dosing regimens. In short, fenugreek shows promise for improving glycemic parameters, especially post-prandial glucose; it is not a substitute for standard glucose-lowering drugs in people who need them, but may be a useful adjunct in some patients(17,18).

5) Dosing, formulations and safety

Clinical studies used a broad range of doses: from small extract doses (e.g., 0.5–1 g/day of standardized extract) up to food-equivalent seed intakes of 10–30 g/day. Proprietary extracts (e.g., Fenfuro) are standardized for certain saponins and were typically dosed twice daily in trials. Side effects reported across trials were generally mild and gastrointestinal (bloating, flatulence), with rare adverse events. Co-supplementation studies used lower doses of fenugreek alongside other agents (e.g., berberine) to mitigate GI tolerability and still showed benefit. Contraindications: because fenugreek can lower blood glucose and increase insulin secretion, there is a real risk of additive hypoglycemia when combined with insulin or sulfonylureas — dose adjustments and monitoring are prudent. There are anecdotal reports of allergic reactions in pasterns with legume allergy and theoretical interactions with anticoagulants and pregnancy (fenugreek has oxytocic properties at high doses), so counseling is needed(19,20).

6) Clinical research gaps

The main limitations of the 2022 evidence are heterogeneity (dose, product, duration), variable trial quality, and small sample sizes in many studies. Long-term safety data beyond months are limited. The standardization of active components (such as furostanolic saponins and 4-hydroxyisoleucine levels) is frequently lacking in studies, which complicates replication and dose equivalency. Head-to-head comparisons between standardized extract formulations and practical culinary uses (soaked seeds, powders) are sparse. Finally, most trials enrolled adults with T2DM generalizability to elderly patients with frailty, those on intensive insulin regimens, or those with advanced renal disease remains uncertain(9).

1.8.2 Jamun (*Syzygium cumini*)

1) Introduction

Jamun, commonly called jambolan or Indian blackberry, is a deep-purple tropical fruit long used in South Asian traditional medicine for “sweet blood.” Over the past decade scientific work has moved this from folklore into a multidisciplinary evidence base: phytochemistry, mechanism studies, food technology to improve delivery, and small human trials. By 2025 the balance of evidence positions Jamun as a multitarget adjunct for metabolic health especially for reducing post-meal glucose spikes and oxidative stress but not as a replacement for proven glucose-lowering drugs(21).

Jamun (*Syzygium cumini*) is a phytochemically rich fruit whose seed, pulp and leaf extracts have shown multi-layered activity against metabolic disturbances central to type 2 diabetes (T2D). A modern Jamun formulation does more than one thing at a time it nudges glucose handling at the gut, protects insulin-producing cells, calms inflammation, and supports insulin signalling in peripheral tissues. Below I unpack those mechanisms in a practical, step-by-step way and explain how contemporary formulation work (microencapsulation, standardized extracts) improves these actions(12,13).

2) Mechanisms of Jamun

Slowing carbohydrate digestion and blunting post-meal spikes.

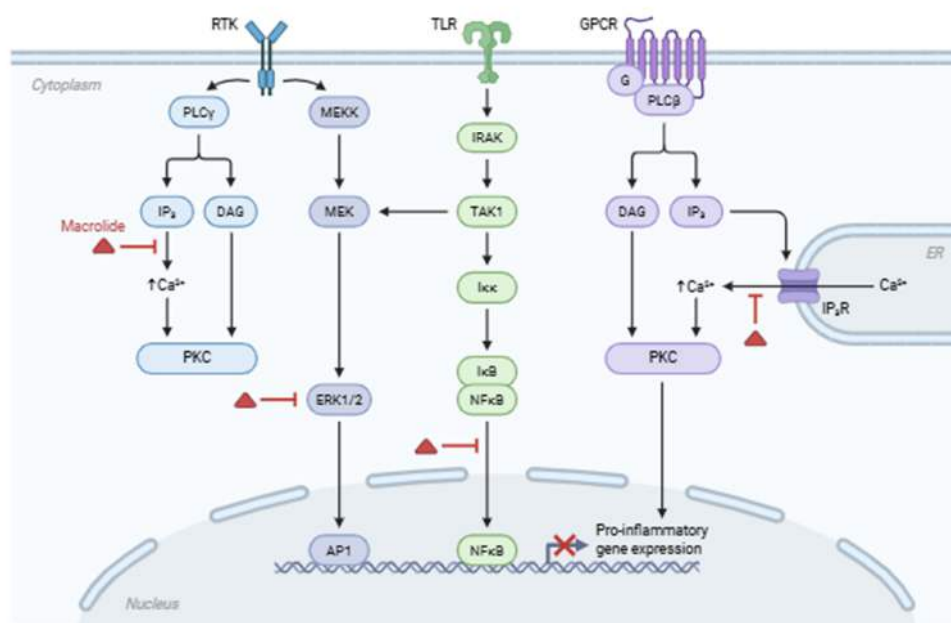
Jamun seed and pulp are rich in tannins and polyphenols that inhibit digestive enzymes such as α -amylase and α -glucosidase. By slowing starch breakdown in the intestinal lumen, less glucose is released at a fast rate into the blood after a meal, which reduces post-prandial glycemic excursions a major therapeutic target in T2D. This “natural enzyme braking” explains some of the clearest clinical effects reported with Jamun extracts(22).

Reducing intestinal glucose uptake and modulating transporters.

Beyond enzyme inhibition, specific Jamun phytochemicals interact with intestinal glucose transporters and reduce the permeability of the gut to rapid glucose absorption. This combined effect of slower digestion and decreased absorption helps to stabilize the glucose levels following meals and reduces the necessity for significant, quick insulin reactions. Network-level analyses predict multiple transporter and receptor interactions for identified Jamun constituents.

Antioxidant and anti-inflammatory protection of β -cells and insulin signalling. Chronic oxidative stress along with persistent inflammation play key roles in the development of insulin resistance and the impairment of β -cell function. Jamun anthocyanins, ellagic acid and related polyphenols are potent free-radical scavengers and down-regulators of pro-inflammatory pathways. By lowering reactive oxygen species and cytokine signalling in pancreas, liver and adipose tissue, Jamun preserves β -cell health and improves downstream insulin signalling cascades (for example, the PI3K/Akt axis), supporting better insulin secretion and peripheral glucose uptake.

Direct support of insulin action (insulinotropic and insulin-sensitizing effects). Preclinical models report that certain seed glycosides and terpenoids in Jamun can modestly stimulate insulin release from islets (insulinotropic effect) and enhance insulin signalling in muscle via increased phosphorylation of components in the PI3K/Akt pathway. These effects are usually modest and synergize with the gut-level mechanisms to improve overall glycemic control. Network pharmacology work from 2025 maps these compounds to targets involved in glucose uptake and glycogen synthesis.



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Figure 2 : Immunomodulation (Functions Of Macrolides), Synthesising NF- κB

As shown in the Fig 2, Mitigating advanced glycation and organ injury. Jamun constituents inhibit formation of advanced glycation end-products (AGEs) and modulate pathways (e.g., NF-κB, Nrf2) linked to oxidative stress and mitochondrial dysfunction. Recent in-vivo work showed Jamun preparations attenuate markers of diabetic nephropathy through Nrf2-dependent mitochondrial protection, suggesting benefits beyond immediate glycemia namely, protection of kidneys and vasculature from hyperglycemia-driven damage(22).

Modifying lipid metabolism and lipotoxic stress.

Some triterpenoids and saponins in Jamun reduce intestinal cholesterol absorption and improve lipid handling, lowering circulating lipotoxic intermediates (e.g., DAGs, ceramides) that otherwise interfere with insulin signalling. Lower lipotoxic stress feeds back to better insulin receptor and IRS function in muscle and liver.

Microbiome and metabolite mediation (emerging).

There is growing evidence that Jamun polyphenols are metabolized by gut microbes into bioactive metabolites that reach systemic circulation and influence insulin sensitivity. Although human microbiome-mechanism data are still emerging, formulation strategies that protect polyphenols until they reach the lower gut (see below) are likely to amplify these beneficial microbe-mediated effects.

From seeds to standardized, bioavailable extracts.

Raw jamun preparations vary widely. Modern formulations use standardized extracts (defined marker compounds), microencapsulation (protects anthocyanins from stomach acid and enzymes), and controlled-release matrices to improve bioavailability and reproducibility of clinical effects. Microencapsulation (ionic gelation, nanoparticles) preserves active polyphenols, slows their release, and can target delivery to the gut region where microbiome conversion or local enzyme inhibition is most effective. These technological advances are central to converting promising lab mechanisms into reliable clinical benefits(21,23).

Table 3: (Key nutraceuticals, source & principal diabetic mechanisms)

Nutraceutical	Primary source (Jamun part)	Principal anti-diabetic mechanisms	Evidence level (to 2025)
Anthocyanins (delphinidin, petunidin, malvidin)	Fruit pulp / peel	Antioxidant; inhibit α -glucosidase; improve endothelial/Nrf2 signalling; modulate gut microbiome metabolites.	Strong preclinical; formulation/encapsulation studies + small human signals.
Ellagic & gallic acids; tannins	Seeds, pulp	Protein glycation inhibition (anti-AGE); enzyme inhibition (α -amylase); antioxidant & anti-inflammatory effects.	Preclinical + small clinical trials / reviews(24).
Terpenoids (oleanolic/maslinic acids) & saponins	Seeds	Improve insulin signalling (PI3K/Akt markers in vitro); lipid metabolism modulation ($\downarrow$ lipotoxic intermediates).	Preclinical and mechanistic 2022–25 studies.
Seed glycosides & alkaloids (jambosine-like)	Seed	Mild insulinotropic effects (stimulate insulin release in islets); reduce gluconeogenesis in liver models.	Early preclinical, supported by network-pharmacology mapping.
Phenolic matrix (mixture)	Whole fruit/seed	Synergistic antioxidant/anti-inflammatory network; supports β -cell health and	Supported by systematic reviews and 2025 syntheses.

Nutraceutical	Primary source (Jamun part)	Principal anti-diabetic mechanisms	Evidence level (to 2025)
		reduces insulin resistance(24,25).	

3) Product form and standardization

A consistent theme in the literature to 2025 is product heterogeneity. Jamun preparations range from whole fruit juice and seed powders to methanolic extracts and modern microencapsulated formulations. Differences in extraction solvent, part of plant used (seed vs pulp vs leaf), and phytochemical standardization lead to variable bioavailability and clinical effects. Food technology research in 2022–2025 (e.g., microencapsulation, carrier matrices) has improved stability and controlled release of the active anthocyanins and terpenoids, offering a path to more reproducible clinical trials and products(26).

4) Safety and practical clinical advice

Jamun appears well tolerated in studied dose ranges; most adverse events are mild GI symptoms. The principal clinical caution is potential additive glucose lowering with insulin or sulfonylureas monitor glucose and consider medication adjustment. Long-term safety data are limited, and pregnant or lactating people were generally excluded from trials. Clinicians should encourage patients to use Jamun only as a supervised adjunct, prefer standardized extracts when available, and treat it as part of a lifestyle plus medicine plan rather than a substitute for prescribed therapy. Mechanistically, the combination of enzyme inhibition, antioxidant protection and modest insulin support provides a credible basis for Jamun's most consistent human signal: reduced post-prandial glucose, with occasional modest fasting glucose and HbA_{1c} improvements in trials. Clinicians should note the potential for additive hypoglycemia when Jamun is combined with insulin or insulin secretagogues and counsel patients to monitor glucose closely. Product choice matters prefer standardized, well-characterized formulations and avoid unverified extracts(26).

5) Research gaps and the next steps

By 2025 the research agenda is clear: conduct well powered randomized trials using standardized, bioavailable Jamun extracts (dose finding, which is less than equal to 6 month follow-up), include meaningful clinical endpoints (HbA_{1c}, TIR, cardio-renal markers), and evaluate long-term safety. Mechanistic human studies (e.g., on PI3K/Akt signalling, β -cell markers, microbiome mediated polyphenol metabolism) will help bridge preclinical promise and clinical practice. Network-pharmacology and omics approaches in 2024–2025 are generating testable hypotheses about precise molecular targets, which can guide rational formulation and trial design(26,27).

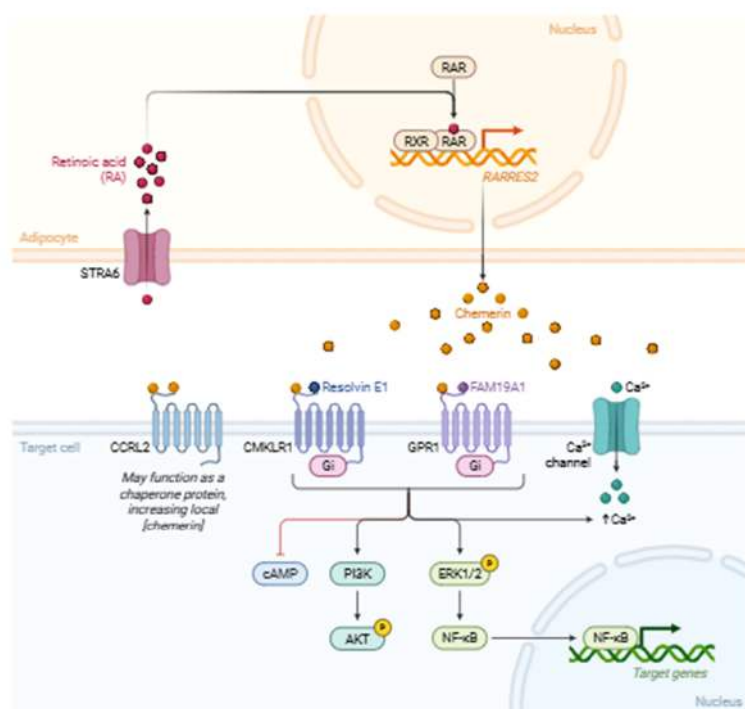
1.8.3 Gurmar (*Gymnema sylvestre*)

1) Introduction

Gymnema sylvestre (gurmar) leaves contain gymnemic acids (triterpene saponins), gurmardin peptides, and polyphenols that collectively target multiple points in glucose homeostasis. The best-supported human data suggest improvements in fasting and post-prandial glucose and HbA1c with standardized extracts as adjuncts to usual care, not stand alone replacements. A 2021 meta analysis of 10 studies (419 participants) reported statistically significant reductions in fasting glucose, post-prandial glucose, and HbA1c versus baseline, albeit with high heterogeneity and variable extract standardization important caveats when translating to practice (28). Classic trials with a standardized *Gymnema* extract (GS4) from the 1990s though small and pre CONSORT showed additive glycemic benefits when combined with conventional oral agents in T2D and with insulin in type 1 diabetes (T1D), supporting a plausible glucose-lowering signal (29,30). By 2025, *Gymnema sylvestre* remains a plausible, multi-mechanistic adjunct for adults with T2D most convincingly for post-prandial glucose and potentially for sugar-intake reduction provided that standardized extracts are used and therapy is integrated with lifestyle and evidence-based pharmacotherapy. Clinicians should set expectations for modest effect sizes, monitor for hypoglycemia with concurrent insulin/secretagogues, and emphasize product quality and consistency. Continued high-quality trials will determine how far gurmar can move from promising adjunct to routinely recommended component in comprehensive diabetes care(30).

2) Mechanisms

Pancreatic β -cell support and insulinotropic effects: One of the most important mechanisms by which *Gymnema sylvestre* (commonly known as Gurmar) exerts antidiabetic effects is through its influence on pancreatic β -cells, the insulin-producing cells of the islets of Langerhans. Type 2 diabetes is characterized by progressive β -cell dysfunction, resulting from chronic hyperglycemia, lipotoxicity, and oxidative stress. Gurmar has shown promising effects in both in-vitro and in-vivo studies, highlighting its role in enhancing insulin secretion, protecting β -cells, and sustaining endogenous insulin production. Gurmar enhances insulin secretion primarily when blood glucose is elevated, thereby reducing the risk of hypoglycemia. This effect resembles the action of incretin-based therapies, which also provide glucose regulation as it ensures safety while improving post-prandial glucose control.



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Figure : 3 Chemarin Synthesis and Signaling Pathway

As shown in the Fig 3 , Gurmar preparations have been shown to facilitate calcium influx into β -cells. When glucose enters the cell, it undergoes glycolysis and ATP production, leading to closure of ATP-sensitive potassium (K^+) channels. This depolarizes the β -cell membrane and opens voltage-gated calcium channels, allowing calcium entry. Gurmar appears to amplify this pathway, either by sensitizing calcium channels or enhancing downstream calcium signaling. Elevated intracellular calcium is the key trigger for insulin granule exocytosis, and thus Gurmar can potentiate glucose-stimulated insulin release through this mechanism. β -cell function is also regulated by intracellular kinases such as protein kinase A (PKA), protein kinase C (PKC), and phosphoinositide 3-kinase (PI3K)(30).

Intestinal carbohydrate handling: These compounds are structurally similar to glucose molecules. Because of this similarity, they can bind competitively to intestinal glucose transporters and receptors, thereby reducing the absorption of glucose into the bloodstream. This "molecular mimicry" effectively slows the rate of carbohydrate uptake without completely blocking it, making it a gentle but effective regulator of postprandial glycemia. Glucose absorption across the small intestinal epithelium is mainly mediated by transporters such as SGLT1 (sodium-glucose linked transporter 1) and GLUT2 (glucose transporter 2). Studies suggest that gymnemic acids may compete with glucose for binding to these transporters, leading to partial inhibition of glucose entry into enterocytes (intestinal cells). By limiting SGLT1 activity, Gurmar reduces the active transport of glucose with sodium across the brush-border membrane. By attenuating GLUT2 translocation to the apical surface (a process

triggered under high-glucose conditions), it can further limit facilitated glucose diffusion. This dual interaction can meaningfully reduce the magnitude of glucose spikes after a carbohydrate-rich meal. In addition to affecting transporters, *Gymnema* exerts mild inhibitory activity on digestive enzymes: α -Amylase, responsible for breaking down starch into maltose and oligosaccharides. α -Glucosidase, which hydrolyzes disaccharides into absorbable monosaccharides like glucose. By slowing these enzymatic processes, Gurmar delays the release of free glucose from complex carbohydrates. This effect prolongs digestion and results in a gradual rise in blood sugar rather than an abrupt surge.

Central and sensory modulation of sweetness and intake:

Gymnemic acids transiently blunt sweet taste at the T1R2/T1R3 receptor on the tongue, lowering the hedonic response to sugar and, in behavioral studies, reducing intake of sweet foods (31). While this is not a direct glycemic mechanism, sustained reductions in discretionary sugar can reduce glycemic variability and caloric load, indirectly supporting T2D control. Sweet taste on the tongue is primarily detected through heterodimeric G-protein-coupled receptors T1R2/T1R3 located on taste buds. Gymnemic acids, due to their structural similarity with glucose, bind to these receptors and act as competitive antagonists. When gymnemic acids occupy T1R2/T1R3, they prevent activation by sugar molecules which results in a temporary suppression of sweet taste perception, often lasting for 30 minutes to 2 hours after exposure. By reducing the sensory input from taste receptors, the reward signals normally triggered by sugar in the dopaminergic pathways of the brain may be blunted. This reduces the reinforcement loop that drives cravings and habitual sugar consumption. Functional imaging studies on taste suppression agents suggest a measurable decline in activity in brain regions associated with food reward, such as the orbitofrontal cortex. The sensory impact of *Gymnema* offers a distinct supplementary approach that enhances its metabolic advantages: In contrast to medications aimed at stimulating insulin production or glucose absorption, *Gymnema* tackles the behavioral cause of excessive sugar consumption. Its use as a lozenge, mouth rinse, or extract before meals may help individuals avoid overeating desserts or sugary snacks(32).

Anti-inflammatory and metabolic signaling:

In addition to its pancreatic, intestinal, and sensory effects, *Gymnema sylvestre* exerts systemic metabolic benefits through its anti-inflammatory and antioxidant activities, as well as modulation of lipid metabolism. These properties add another layer of biological plausibility for its role in type 2 diabetes (T2D) prevention and management. Chronic hyperglycemia in T2D drives the overproduction of reactive oxygen species (ROS), impairing insulin signaling, damaging β -cells, and promoting vascular complications. Preclinical studies indicate that gymnemic acids and associated polyphenols in *Gymnema* enhance endogenous antioxidant defenses, including superoxide dismutase, catalase, and glutathione peroxidase. This results in reduced oxidative injury to pancreatic β -cells and an improved oxidative balance supports insulin sensitivity in peripheral tissues such as muscle and adipose tissue. T2D is strongly associated with chronic low-grade inflammation, driven by cytokines like TNF- α , IL-6, and CRP. Gymnemic acids have been reported to downregulate pro-inflammatory mediators by inhibiting nuclear factor kappa B (NF- κ B) and mitogen-activated protein kinase (MAPK) pathways. This leads to reduced secretion of inflammatory cytokines in adipose and liver

tissues and lower inflammation correlates with improved insulin receptor sensitivity, reducing insulin resistance(33).

Table 4: Key nutraceuticals, plant source & principal antidiabetic mechanisms

Nutraceutical	Primary source (leaf/seed)	Principal mechanisms	Evidence level (to 2025)
Gymnemic acids (triterpene saponins)	Leaves	Compete with glucose at sweet receptors and intestinal transporters; inhibit α -glucosidase → reduce post-prandial glucose.	Strong preclinical + human behavioral and PPG studies.
Gurmarin (peptides)	Leaves	Modulate sweet taste perception; possible neuromodulatory effects on feeding behavior.	Behavioral human data and sensory studies.
Polyphenols / flavonoids (quercetin, kaempferol etc.)	Leaves	Antioxidant, anti-inflammatory; protect β -cells and improve insulin signalling (PI3K/Akt).	Robust preclinical; biomarker changes in small human trials.
Triterpenoids / minor saponins	Leaves	Improve lipid handling, reduce hepatic lipogenesis, support mitochondrial function.	Preclinical and network-pharmacology mapping (2025).
Phenolic metabolites (microbiome-derived)	Gut metabolism of leaf polyphenols	Systemic metabolites that modulate insulin sensitivity and inflammation.	Emerging human/microbiome data; mechanistic plausibility(34,35).

3) What 2025 adds

The most recent clinically oriented publications do not overturn earlier conclusions but refine use cases and safety framing: Nutraceutical positioning and taste-based behavior change. A 2025 narrative review in *Nutrients* describes how gymnemic acids' sweet-taste antagonism can be harnessed to reduce sugar cravings and caloric sugar intake an appealing adjunct for people with T2D who struggle with post-meal spikes or late evening snacking. While not a substitute for structured nutrition counseling, this sensory route may be particularly helpful for patients working on reducing added sugars. Mechanistic granularity. Ongoing molecular reports in 2025 (e.g., *J Cell Mol Med*) detail gene network and protein interactions consistent with the multiple small to moderate effects observed clinically, including nodes tied to insulin signaling,

oxidative stress, and lipid handling. These papers bolster plausibility but are not themselves practice changing without large clinical corroboration. Related clinical contexts. A 2024 randomized study in *Nutrients* compared *Gymnema* with berberine in adults with obesity and showed metabolic improvements with both agents, indirectly supporting broader cardiometabolic relevance of *Gymnema* beyond glucose alone. Although not a T2D-only cohort, the directionality aligns with earlier diabetes-focused work(36,37).

4) Practical use: extract, dose, and integration

Standardization and product selection: Not all *Gymnema* products are equivalent. Clinical trials that produced measurable, reproducible effects almost always used standardized leaf extracts that declare a gymnemic acid content (for example, GS type extracts). When advising patients or designing a trial, prefer products that list: (a) the percentage of gymnemic acids, (b) batch level certificates of analysis from an accredited laboratory, and (c) Good Manufacturing Practice (GMP) production and third party testing for contaminants (heavy metals, pesticides, microbial load). This minimizes discrepancies in exposure and enhances the dependability of clinical results and safety assessments(38–40).

Starting dose and titration strategy : Because trials vary, anchor dosing to the specific product. Typical studied ranges for standardized extracts are 200–500 mg/day, often split into two doses; traditional seed/leaf powders in food contexts are generally 5–10 g/day. A conservative clinician-led approach is: Initiate at the lower end (e.g., 200 mg/day standardized extract or 2–3 g seed powder divided with meals). Observe glucose patterns and tolerance for 7–14 days, focusing on fasting glucose and 1–2 hour post-prandial measurements. Titrate upward every 1–2 weeks as needed and tolerated, toward the mid-range studied dose (e.g., 400–500 mg/day extract or up to 10 g powder), with re-assessment at 4–12 weeks. Document the exact product and lot(41–43).

Timing relative to meals and therapy: For intestinal effects (enzyme inhibition and transporter modulation), taking a dose 10–30 minutes before a carbohydrate-rich meal is logical and supported by mechanistic data and clinical practice, for taste suppression (lozenge/chewable forms), administer immediately before foods or snacks that trigger cravings, emphasize that *Gymnema* is adjunctive: continue evidence-based glucose-lowering medications and lifestyle measures. It is not a substitute for indicated pharmacotherapy(44).

Documentation and shared decision-making: Record baseline glycemic control (fasting glucose, 2-hour post-prandial, recent HbA_{1c}), concurrent medications, and patient goals. Obtain informed consent that includes: expected magnitude of benefit (modest), timeframe (weeks to months), monitoring plan, and a plan for medication adjustment if glucose falls. This creates a safe, traceable pathway for use(45,46).

5) Safety and monitoring

The main clinical safety concern is additive glucose-lowering when *Gymnema* is combined with insulin or insulin secretagogues (e.g., sulfonylureas). Implement a proactive monitoring plan that is If a patient uses insulin or a sulfonylurea, instruct them to check fasting and post-prandial glucose daily for the first 2–4 weeks after initiation. Consider more frequent checks (pre-meal, bedtime) for intensive insulin regimens. If symptomatic or confirmed glucose values

fall below target (clinically defined thresholds per local protocols), reduce the hypoglycaemic agent dose incrementally (e.g., 10–20% depending on severity) and reassess daily until stable. Coordinate closely with the prescribing clinician/endocrinologist(47,48). Gastrointestinal effects (bloating, mild nausea, constipation) are the most commonly reported adverse events; advise patients to take doses with food if they occur. Drug herb interactions: although major CYP-mediated interactions are uncommon for Gymnema, review concurrent medications for metabolic or hepatic interactions and err on the side of caution for narrow-therapeutic-index drugs. Stop supplements before major surgery per standard perioperative guidance. Pregnancy and lactation: insufficient safety data avoid unless supervised by a specialist(49,50).

2. Role of yoga and meditation in glycemic control

2.1 Introduction

Yoga and meditation: how mind body practices help blood sugar and what the evidence (to 2025) actually says that for many people with diabetes, the phrase “manage your blood sugar” has long meant pills, diet and exercise. Over the last decade, however, yoga and meditation have moved from fringe complementary options into interventions tested in randomized trials and pooled in meta-analyses. By 2025 a reasonably consistent picture has emerged: regular yoga (postures + breath + relaxation) and structured mindfulness/meditation programs can produce modest but meaningful improvements in glycemic indices, plus clearer benefits for stress, mood and self care all of which matter for long-term diabetes outcomes(51).

2.2 Importance of trials and meta-analyses

Meta analytic summaries that pooled randomized controlled trials report small to moderate reductions in HbA1c and fasting/post prandial glucose after short to-medium term yoga or mindfulness programs. For example, a systematic review and meta analysis of RCTs comparing yoga (or walking) against usual care found that yoga reduced fasting glucose, post-prandial glucose and HbA1c (pooled HbA1c reduction $\approx 0.7\%$) and improved insulin resistance markers versus no regular exercise. That same analysis suggested yoga produced greater glycemic benefit than walking alone. A separate Frontiers meta analysis focusing on uncomplicated type 2 diabetes also reported significant improvements in HbA1c, fasting and post-prandial glucose and triglycerides after 10 to 24 weeks of yoga based interventions. The effect sizes varied across studies, and heterogeneity was substantial, but the direction of change favored yoga. Mindfulness and meditation trials (MBSR/MBCT and related programs) give a more nuanced picture. Umbrella reviews and meta-analyses of mindfulness interventions suggest average HbA1c reductions are smaller (typically ~ 0.2 to 0.4%) and evidence quality is mixed; however, psychological outcomes (stress, depression, diabetes distress) show more consistent improvement. Because stress responses, sleep and coping affect glucose via hormonal (HPA axis) and behavioral pathways, these mental health gains are clinically relevant even when HbA1c changes are modest(52).

2.3 Mechanisms

- 1) Autonomic rebalance and stress hormones. Yoga and meditation enhance parasympathetic tone (vagal activity) and blunt sympathetic overactivity. Lower sympathetic drive and reduced cortisol spikes lead to less stress-mediated gluconeogenesis and insulin resistance a plausible physiological pathway connecting

mind body practice to improved glucose metrics. Several trials documented improved heart rate variability and lower cortisol correlating with glycemic benefits.

- 2) Increased physical activity and muscle glucose uptake. Many yoga styles include sustained isometric holds, flowing sequences and breathing coordinated movements (e.g., Surya Namaskar) that raise muscle glucose use and insulin sensitivity an effect similar in direction (though smaller in magnitude) to conventional moderate exercise. That helps explain improvements in fasting and post-prandial glucose seen in trials.
- 3) Behavioural pathways: better self-care and dietary choices. Meditation, mindful eating and the reflective habits developed during yoga foster awareness of hunger, cravings and medication routines. Several studies showed improved adherence to diet, medication, and activity after mindfulness training a likely mediator of longer-term glycemic change.
- 4) Anti-inflammatory and metabolic modulation. Small trials indicate reductions in inflammatory markers (CRP, IL-6) and oxidative stress after yoga programs; since chronic inflammation contributes to insulin resistance, this provides a biologically plausible secondary mechanism(53).

2.4 Clinical evidence from recent controlled trials

Recent RCTs continue to support short term glycemic improvements and quality of life gains. A 2025 randomized trial of a three month, standardized yoga module in adolescents with type 1 diabetes reported significant HbA1c and average blood glucose reductions plus decreased insulin requirements and improved QoL measures demonstrating feasibility and metabolic signal even in insulin treated youth. While encouraging, the study's attrition and short follow up underline the need for larger, longer trials. Pooled reductions in HbA1c for yoga interventions commonly cluster around 0.4–0.7% in meta-analyses; for mindfulness interventions reductions tend to be smaller (≈ 0.2 –0.4%) and less consistent. A 0.5–0.7% HbA1c fall is clinically relevant for many patients (it lowers complication risk), but heterogeneity across trials differing yoga styles, dose, supervision, baseline HbA1c and concurrent pharmacotherapy complicates universal claims. Importantly, people with higher baseline HbA1c and poor stress control tend to show larger benefits(54).

2.5 Practical recommendations for clinicians and patients

1) Treat yoga/meditation as adjuncts

Yoga (postures, breathing, relaxation) and meditation (e.g., mindfulness based programs) can improve glycemic control and self management, but they should complement guideline-directed therapy (nutrition counseling, aerobic/resistance exercise, and medications), not supplant it. The American Diabetes Association (ADA) Standards of Care 2025 explicitly emphasize structured behavior change supports and psychological well being alongside medical therapy, urging clinicians to integrate mind body practices where appropriate while maintaining evidence based pharmacotherapy and medical nutrition therapy. A 2025 randomized trial in adolescents with type 1 diabetes found that adding a structured 3 month yoga program to usual care reduced HbA1c and insulin requirements and improved quality of life; benefits accrued *in addition to* standard insulin therapy rather than replacing it. For adults with type 2 diabetes (T2D), meta analytic syntheses show yoga and mindful movement programs yield modest but clinically meaningful HbA1c improvements and stress reduction,

with effects similar to other low to moderate intensity behavioral interventions. Mindfulness-based programs also improve self-regulation and health behaviors, which translate indirectly into better glycemia over time. The clinical takeaways are Position yoga/meditation as structured “add-ons” that can help patients meet ADA self-care targets (nutrition, physical activity, coping skills) without disrupting essential pharmacologic plans(55,56).

2) Dose matters: program length and practice frequency

The strongest signals for glycemic and psychosocial benefits appear with 8–12 weeks of structured practice, ideally with 3–5 guided sessions per week or a daily home practice supported by instruction and logs. In the 2025 RCT (three months of supervised sessions plus home practice), HbA1c fell and insulin needs declined, suggesting that consistent exposure and skill acquisition are key. A 2022 meta-analysis of yoga trials in adults underscores that programs spanning ≥ 8 weeks with regular sessions produce more reliable metabolic and cardiometabolic benefits than brief, sporadic offerings. In the mindfulness domain, a 2023 systematic review likewise found that multi week, manualized programs (e.g., MBSR, MBCT) yield stronger improvements in stress, depression, and health behaviors than minimalist formats. The program design tips are, start with 60–75 minute sessions (mixed asana, breathing, relaxation) 3 times weekly, plus short daily breathing/meditation (10–20 minutes), use progressive goals (e.g., add one home session per week) and simple tracking (paper or app) to reinforce adherence, pair practice blocks with outcomes monitoring (fasting glucose, CGM time-in-range, mood/sleep scales) to connect effort with results and maintain motivation(57).

3) Safety first—and individualized

Mind body programs are low-risk for most adults, but diabetes specific considerations matter. The ADA 2025 Standards advise matching activity to comorbidities, screening for complications, and incorporating behavioral health supports. Practical cautions include: Retinopathy: Avoid or modify head down/inverted postures and forceful breath holds in people with proliferative diabetic retinopathy or recent ocular procedures; favor gentle postures and breath pacing instead. While yoga specific diabetes guidance is evolving, standard ophthalmology care and ADA activity precautions support this conservative approach. Peripheral neuropathy or balance issues: Prefer chair-supported or floor based sequences; emphasize slow transitions. Cardiovascular disease: Begin with low-to-moderate intensity; prioritize breath led, non straining practices and gradual progression. Insulin or sulfonylurea use: Check glucose before/after sessions during initiation; carry hypoglycemia treatment; coordinate exercise timing relative to meals/boluses per ADA advice. Autonomic dysfunction: Start with breath regulation and restorative postures; monitor for dizziness or hypotension. These precautions help by yoga and meditative breathing can increase parasympathetic tone and improve heart-rate variability (HRV) while lowering sympathetic arousal, which is desirable but dose and posture intensity should be titrated for those with complications(58).

4) Behavioral framing: mindful eating, habit formation, and teamwork

Meditation and yoga become most effective when tied to *concrete self management behaviors*. ADA 2025 recommends routine assessment of behavioral outcomes and psychological well being and encourages strategies that build sustainable habits. Practically speaking, mindful eating practices (focusing on hunger and fullness signals, enjoying food, and managing

triggers) can decrease snacking and the consumption of added sugars key factors influencing fluctuations in blood sugar levels. Observational and interventional work links mindful eating practice with improved diet quality and weight trajectories in people with elevated metabolic risk. Implementation intentions (“If it’s 7pm, I do 10 minutes of breathing before dinner”) and habit stacking (short meditation after tooth brushing) convert intentions into routines that persist beyond the class period. Digital supports (simple logs, timers, prompts) sustain frequency. Many programs pair yoga/meditation with CGM feedback: patients see how pre meal breathing or a short restorative sequence blunts post-prandial spikes, which reinforces practice through visible biofeedback. Team based care: Health coaches, diabetes educators, and behavioral health professionals can guide patients in adapting sequences, troubleshooting barriers, and aligning practice with nutrition and medication plans an approach explicitly encouraged in ADA 2025 Section 5. Mechanistic points include down-regulating hypothalamic pituitary adrenal (HPA) axis activity and sympathetically mediated hepatic glucose output via breathing/relaxation likely complements improvements in diet and activity. Trials and reviews show yoga and mindfulness can enhance HRV, modestly lower blood pressure, and improve stress biomarkers (e.g., cortisol) in cardiometabolic populations, all of which contribute to glycemic stability over time(59,60).

2.6 Putting it together: a sample 12-week protocol

Weeks 1–2: Two supervised sessions/week plus one guided home session (20–30 min). Elements: gentle mobility, diaphragmatic breathing (5–6 breaths/min), brief body scan. Safety screen; set CGM alerts and a simple practice log. Weeks 3–6: Three supervised sessions/week (45–60 min) + three home practices (10–20 min). Add moderate sequences (e.g., dynamic chair squats, wall dog, supine leg work), extend breathing/relaxation to 10–15 min. Cue mindful eating at one meal/day. Weeks 7–12: Two supervised + four home practices weekly. Introduce light resistance (bands/body-weight), longer relaxation (15–20 min). Personalize with patient selected goals (e.g., pre-meal 10 minute breathing before largest meal, or an evening restorative practice for sleep). Metrics: HbA1c (baseline/12 weeks), CGM time in range or 7 point SMBG, perceived stress (PSS-10), sleep quality, and adherence (sessions/week). Adjust medications in usual care as glycemia improves. Expected outcomes are small to moderate reductions in HbA1c and glycemic variability, improved stress/sleep, and better self efficacy particularly when practice is frequent and integrated with diet, activity, and medication adherence [1–5]. For insulin-treated patients, monitor for reduced requirements as stress and post prandial excursions soften (as seen in the 2025 RCT)(60).

2.7 Research gaps (2025)

The literature still needs larger, multi site RCTs with standardized, reproducible yoga/mindfulness protocols; longer follow-up (≥ 12 months); robust attention matched controls; and mechanistic end points (HPA axis markers, autonomic function, inflammatory cytokines). Studies should also report interactions with modern glucose lowering agents (GLP-1 RAs, SGLT2 inhibitors) and use continuous glucose monitoring to capture variability and time in range(95,97,98).

3. Dietary Patterns in Diabetes Management

Dietary patterns are integral to the management and prevention of diabetes, influencing glycemic control, insulin sensitivity, and overall metabolic health. Recent studies from 2025 have provided deeper insights into how specific dietary patterns impact diabetes outcomes. This article reviews the latest evidence on dietary patterns and their effects on diabetes management, focusing on findings from systematic reviews and meta analyses published in 2025(64)

3.1 Mediterranean Diet: A Cornerstone in Diabetes Management

3.1.1 Introduction

The Mediterranean diet is widely recognized as one of the most effective dietary patterns for preventing and managing type 2 diabetes (T2D). It is rich in fruits, vegetables, whole grains, legumes, nuts, and olive oil, all of which contribute to a nutrient dense eating pattern with anti inflammatory and antioxidant properties. A 2025 longitudinal study tracked individuals with impaired fasting glucose over 20 years and found that those who consistently adhered to the Mediterranean diet had a 59% lower risk of developing T2D compared to those who did not follow this diet pattern. These findings underscore the diet's potential to modulate insulin sensitivity, reduce systemic inflammation, and improve metabolic markers.

Another pivotal study involved individuals with both T2D and hypertension. Over 12 months, participants following the Mediterranean-DASH diet an approach that combines Mediterranean dietary principles with elements of the DASH (Dietary Approaches to Stop Hypertension) diet experienced improvements in body composition, lipid profile, and blood pressure. The inclusion of lean proteins, fiber rich foods, and healthy fats was found to enhance satiety and regulate glucose excursions after meals, leading to better long-term adherence and health outcomes(65).

3.1.2 Plant-Based Diets: Promising but Inconclusive

In recent years, plant-based diets have emerged as a promising nutritional strategy for reducing the risk and managing type 2 diabetes (T2D). By focusing on foods derived predominantly from plants such as vegetables, fruits, whole grains, legumes, nuts, and seeds while minimizing or eliminating animal products, these diets offer potential benefits for metabolic health, weight management, and glycemic control. However, as of 2025, the evidence supporting their effectiveness remains promising but not yet definitive, particularly because the quality and composition of plant-based diets vary considerably. Plant based diets broadly include vegetarian diets, which may allow dairy and eggs, and vegan diets, which exclude all animal products. Additionally, some individuals follow flexitarian or semi vegetarian diets, reducing animal protein intake without fully eliminating it. Importantly, adherence to these diets can range from highly structured, nutrient-dense plans rich in whole foods, to less planned approaches reliant on processed plant-based alternatives high in added sugars and refined carbohydrates. The potential benefits of plant-based diets in diabetes management are rooted in several physiological mechanisms: Diets rich in fiber and phytonutrients enhance insulin action by reducing inflammation and oxidative stress in the body. Whole grains, legumes, and leafy vegetables are particularly beneficial for maintaining healthy blood glucose levels.

Enhanced Satiety and Glycemic Control High fiber content slows gastric emptying and delays glucose absorption into the bloodstream, leading to smoother post-meal glucose curves and lower spikes. This steady absorption pattern reduces the demand on pancreatic β -cells. Diets rich in fruits, vegetables, and unsaturated fats have been shown to lower circulating markers of inflammation, such as C-reactive protein (CRP), which are associated with insulin resistance and vascular complications. Plant-based diets help lower LDL cholesterol and triglyceride levels while improving HDL cholesterol, contributing to overall cardiovascular health, which is closely linked to glycemic outcomes(66).

3.1.3 Low-Carbohydrate Diets: Short-Term Efficacy

The use of very low carbohydrate, high-protein, and calorie-restricted diets has been extensively investigated for their role in managing type 2 diabetes (T2D). These diets, which emphasize minimal intake of carbohydrates and often pair with moderate to high protein consumption while restricting overall calories, have shown promising results, particularly in the short term. However, as dietary science progresses, researchers are paying closer attention not only to efficacy but also to the sustainability, safety, and personalized applicability of such interventions. High-protein diets, on the other hand, ensure that 20% ,30% or more of daily energy comes from protein sources such as lean meats, dairy, eggs, legumes, or plant-based alternatives. Calorie restriction often complements these diets by reducing total energy intake by 20% to 40% of usual A systematic review and network meta-analysis published in 2025 examined more than 40 randomized controlled trials involving over 6,000 participants with T2D. The findings were striking: HbA_{1c} Reduction: Participants on very low-carbohydrate diets saw reductions of 0.8% to 1.2% in HbA_{1c} at 6 months, which is considered clinically significant in managing long-term glycemic control. Fasting Plasma Glucose: Improvements ranged from 15 mg/dL to 30 mg/dL lower fasting glucose levels at 6 months, contributing to better glucose regulation throughout the day. Body Weight: Calorie restriction combined with macronutrient adjustments resulted in average weight loss of 6–9 kg, further enhancing insulin sensitivity and reducing metabolic strain. These benefits were more pronounced during the initial phases of the intervention, with patients reporting increased energy levels, improved mood, and decreased dependence on diabetes medications.

Genetic Influences: Genetic markers related to insulin sensitivity, glucose metabolism, and lipid processing may help predict which individuals respond best to carbohydrate restriction. Microbiome Composition: Variations in gut microbiota affect nutrient absorption and inflammation. Personalized diets tailored to microbiome profiles could optimize glycemic outcomes. Behavioral and Cultural Context: Food preferences, eating habits, and cultural dietary patterns influence adherence. Personalized plans that accommodate taste and lifestyle preferences increase long-term success. Medication Interactions: Individuals on glucose-lowering medications such as GLP-1 receptor agonists or SGLT2 inhibitors require careful dietary adjustments to prevent hypoglycemia and ensure medication compatibility. The emerging field of nutrigenomics and digital health monitoring tools, such as continuous glucose monitoring (CGM) systems, provides clinicians with real-time data to tailor diets dynamically, adjusting macronutrient ratios based on glucose trends and physical activity(67).

3.1.4 Balanced Diets: A Causal Perspective

A balanced diet typically includes Fruits and vegetables: Rich in fiber, vitamins, and antioxidants. Whole grains: Sources of complex carbohydrates and micronutrients. Legumes and nuts: Provide plant-based proteins, healthy fats, and minerals. Lean proteins: Such as poultry, fish, and plant proteins like tofu and lentils. Healthy fats: From sources like olive oil, seeds, and nuts. The key principle is variety, ensuring that nutrient requirements are met while avoiding excesses that can lead to metabolic strain. This pattern supports steady energy supply, reduces inflammation, and aids in weight management all critical components in diabetes prevention. The study identified four proteins AGR2, DBI, IL17RA, and SERPINH1 that were consistently downregulated in individuals with higher adherence to balanced diets. These proteins are implicated in pathways related to inflammation, insulin signaling, and cellular stress: AGR2 (Anterior Gradient 2): Associated with endoplasmic reticulum stress and cellular repair mechanisms. Lower expression suggests reduced metabolic stress, which is a known contributor to insulin resistance. DBI (Diazepam Binding Inhibitor): Involved in lipid metabolism and energy homeostasis. Its downregulation indicates improved fat utilization and reduced adiposity both protective against T2D. IL17RA (Interleukin 17 Receptor A): A key component in inflammatory signaling. Reduced levels suggest lower systemic inflammation, which is closely linked to insulin resistance and β -cell dysfunction. SERPINH1 (Serpin Family H Member 1): Plays a role in collagen synthesis and tissue repair. Its modulation points toward improved structural integrity and metabolic function. The downregulation of these proteins in individuals adhering to balanced diets supports the hypothesis that such eating patterns contribute to reduced inflammation, better metabolic resilience, and improved glucose homeostasis all pathways implicated in the development and progression of T2D.

3.1.5 Dietary Risk Reduction Diet (DRRD): Broader Health Implications

The Diabetes Risk Reduction Diet has emerged as a promising nutritional approach for preventing and managing chronic diseases, particularly type 2 diabetes (T2D). Initially designed to target factors contributing to insulin resistance and impaired glucose metabolism, the DRRD has also shown benefits beyond diabetes, extending to cancer prevention and overall survival. A comprehensive meta-analysis published in 2025 reviewed data from multiple observational and interventional studies that assessed dietary adherence to the DRRD pattern. This diet emphasizes foods that are rich in nutrients and low in processed sugars and unhealthy fats. Specifically, it encourages high consumption of whole grains, vegetables, fruits, legumes, and healthy fats, while reducing refined carbohydrates, red and processed meats, and sugary beverages. The results were striking. Individuals with the highest adherence to the DRRD experienced a 47% lower risk of developing T2D compared to those with minimal adherence. The diet's effectiveness likely stems from its ability to improve insulin sensitivity, reduce systemic inflammation, and support healthy lipid profiles, all of which are key mechanisms in diabetes prevention. Beyond diabetes, the meta-analysis also revealed a 36% reduction in cancer incidence among high adherents. Although the exact mechanisms are still under investigation, researchers believe that the DRRD's anti-inflammatory properties, antioxidant-rich foods, and improved metabolic balance contribute to this protective effect. Furthermore, the diet was associated with a 24% decrease in all-cause mortality, suggesting that its benefits extend to overall health and longevity. Balanced nutrition, improved metabolic markers, and

reduced oxidative stress may collectively enhance the body's resilience against various diseases.

Importantly, the meta-analysis emphasized that dietary patterns—not isolated nutrients play a significant role in long-term health. It also underscored that consistency and adherence are crucial for achieving sustained benefits. Lifestyle interventions that incorporate behavioral counseling, meal planning, and community support can further help individuals stay aligned with the DRRD. The Diabetes Risk Reduction Diet stands out as a holistic approach to chronic disease prevention. Its association with reduced risks of T2D, cancer, and mortality reinforces the value of dietary patterns in promoting health. Moving forward, clinicians and researchers are encouraged to incorporate DRRD principles into public health recommendations while continuing to explore how individual variability impacts dietary effectiveness(67).

3.1.6 DASH Diet and Sodium Reduction: Cardiovascular Benefits

The DASH diet (Dietary Approaches to Stop Hypertension), initially developed to lower high blood pressure, has gained recognition as an effective dietary strategy for individuals with type 2 diabetes (T2D), especially because hypertension is a common comorbidity in this population. In 2025, a randomized clinical trial investigated the combined effects of the DASH diet with reduced sodium intake referred to as the DASH4D diet on cardiovascular health in adults living with T2D. The trial involved participants who followed a structured meal plan rich in fruits, vegetables, whole grains, lean proteins, and low-fat dairy while limiting sodium consumption to recommended levels. Over a period of several months, researchers observed a significant reduction in both systolic and diastolic blood pressure readings compared to a control group receiving standard care. One of the key findings was that the blood pressure-lowering effects were primarily driven by sodium restriction, which is particularly important in diabetes care. Elevated sodium levels are known to worsen fluid retention and increase vascular resistance, both of which strain the cardiovascular system and exacerbate complications associated with diabetes. By reducing sodium intake, participants experienced improvements in blood vessel elasticity and lowered arterial pressure, ultimately decreasing the risk of heart disease and stroke. The trial underscored that adherence to dietary patterns rich in natural, minimally processed foods plays a central role in mitigating the adverse effects of hypertension in diabetes management. It also highlighted the importance of combining sodium reduction with other heart-healthy components, such as nutrient-dense foods and balanced macronutrients, to achieve optimal results. Importantly, the DASH4D diet offers a practical and sustainable approach for patients and healthcare providers. Unlike strict pharmacologic interventions, dietary changes can empower individuals to take active control of their health. Educational support, meal planning, and regular follow ups helped participants maintain compliance and adapt the diet to their cultural and personal preferences(68,69).

3.1.7 High-Fiber Diets: Beneficial for Type 1 Diabetes

A meta-analysis, which pooled results from several interventional and observational studies, concluded that individuals with T1D who increased their intake of dietary fiber while minimizing other rapidly digestible carbohydrates achieved improved glucose regulation. Specifically, participants adhering to such diets showed reduced post prandial glucose excursions and lower HbA1c levels over the course of the interventions. The beneficial effects of a high-fiber diet in T1D can be attributed to multiple mechanisms: Fiber forms a viscous

matrix in the gastrointestinal tract, slowing gastric emptying and reducing the rate at which glucose enters the bloodstream. This mechanism helps prevent sudden glucose fluctuations, which can be challenging to manage in insulin-dependent diabetes. Some studies suggest that higher fiber intake may influence insulin receptor function, potentially improving the body's response to insulin. This is particularly important in T1D where even minor improvements in insulin efficiency can lead to better glucose control and reduced insulin requirements. Fiber serves as a prebiotic, nourishing beneficial bacteria that contribute to improved metabolic regulation. Short-chain fatty acids produced by bacterial fermentation of fiber may reduce inflammation and improve insulin sensitivity, further enhancing glycemic control. High-fiber foods promote a feeling of fullness, helping individuals regulate portion sizes and reduce caloric intake. Managing weight is a critical component of overall diabetes management, as excess body fat can exacerbate insulin resistance even in individuals with T1D(69).

3.2 Ketogenic Diet in Diabetes Management

3.2.1 Introduction

The ketogenic diet (KD) is a very-low-carbohydrate, high-fat, moderate-protein dietary pattern designed to induce nutritional ketosis a metabolic state in which ketone bodies (β -hydroxybutyrate, acetoacetate) supply a meaningful share of energetic needs. Typical KD prescriptions limit digestible carbohydrate to roughly 20–50 g/day (often <10% of total energy), raise fat to ~65–75% of energy, and provide moderate protein. For people with diabetes, KD's primary therapeutic rationale is blunt: fewer available carbohydrates :smaller post-prandial glucose excursions: less need for insulin secretion and exogenous insulin dose. In 2025, evidence supports that ketogenic or very low-carbohydrate diets can be effective adjuncts to standard care in type 2 diabetes, particularly for: Rapid improvements in HbA_{1c} and fasting glucose during the first 3-6 months. Significant weight loss and reductions in visceral adiposity. Improvement in triglycerides and HDL, with mixed effects on LDL but the following caveats apply: Benefits attenuate over longer periods (≥ 12 months) unless adherence is maintained. Risks around nutrient deficiencies, potential adverse lipid shifts (especially LDL), and patient burden must be weighed. Definition inconsistencies limit portability of results into guidelines. For suitable patients, KD can be trialed under close supervision, with built-in reviews at 3, 6, 12 months, and tailoring according to metabolic response and patient preference. It should be one tool among many (diet, exercise, medication) rather than a universal solution(70).

3.2.2 Short-term metabolic benefits (what the trials show)

Systematic reviews and recent meta-analyses through 2024–2025 show that carbohydrate-restricted diets including ketogenic and very-low-carbohydrate approaches produce consistent short-term improvements in glycemia and weight in people with type 2 diabetes (T2D). Pooled randomized controlled trial data demonstrate clinically meaningful reductions in HbA_{1c} (typical short-term effect sizes ≈ -0.3 to -0.6% at 3–6 months) and modest falls in fasting glucose and insulin resistance markers, accompanied by greater early weight loss versus comparator diets. Triglycerides fall and HDL cholesterol typically rises on KD; LDL-cholesterol results are heterogeneous and appear sensitive to the types of dietary fat consumed. Importantly, most meta-analyses report that these gains are largest in the first 3 to 6 months

and attenuate thereafter if strict carbohydrate restriction is not maintained. These findings are robust across several 2024–25 meta analyses and systematic reviews(70).

3.2.3 Head-to-head and randomized evidence

Large pragmatic trials in 2024–2025 have compared calorie-restricted ketogenic protocols against Mediterranean or other guideline diets. Some trials report superior short-term weight loss and greater improvements in insulin sensitivity on calorie-matched KD arms, while others find no long term glycemic superiority after 12–24 months once adherence declines. A multi-arm randomized clinical trial that compared a calorie restricted KD, time-restricted eating and Mediterranean diet found KD produced greater early weight loss; however, long-term differences narrowed as adherence waned [3]. Thus, KD can be a powerful short term tool for rapid metabolic improvement, especially when weight loss is an objective, but its long term advantage over flexible, sustainable diets is not settled(71).

3.2.4 Mechanisms that explain the effects

1) Reduced carbohydrate load

When carbohydrate intake is restricted to ~20–50 g/day, the amount of glucose entering the bloodstream after meals is dramatically reduced. Normally, carbohydrates are rapidly digested to glucose, producing postprandial (after-meal) glucose spikes. In T2D, impaired insulin secretion and insulin resistance make these spikes more pronounced, worsening HbA_{1c} and glycemic variability. By sharply cutting carbohydrate intake, KD minimizes these excursions, resulting in: Lower postprandial glucose levels, reducing the immediate need for insulin. Flattened glycemic variability, which is important since wide swings in glucose contribute to oxidative stress, endothelial dysfunction, and cardiovascular risk. The KD led to reductions in fasting glucose and HbA_{1c} at 3–6 months largely attributable to this mechanism (72).

2) Lower insulin demand

Because fewer carbohydrates enter the system, the body needs less insulin to maintain glucose balance. This has two implications: Endogenous insulin: In people with insulin resistance, lower carbohydrate load reduces the chronic hyperinsulinemia that is often present. Over time, this improves insulin receptor sensitivity, meaning the body can respond more effectively to smaller amounts of insulin. Exogenous insulin: For patients on insulin therapy, KD often allows dose reduction. This not only lowers the risk of hypoglycemia but also reduces insulin-induced weight gain, a common problem in diabetes management. Clinical data from 2025 showed that KD interventions consistently reduced total daily insulin requirements in insulin-treated T2D patients, in some cases by 30–50% within months(72).

3) Weight and visceral fat loss

KD promotes fat loss through several overlapping mechanisms: Early glycogen depletion: Each gram of stored glycogen binds ~3–4 g of water. Within the first week, glycogen depletion leads to rapid water loss and early weight reduction. Sustained fat oxidation: Low carbohydrate availability shifts metabolism toward fatty acid oxidation and ketone production, mobilizing adipose stores. Appetite suppression: Ketones such as β -hydroxybutyrate may blunt appetite by affecting hunger hormones (ghrelin, leptin). This helps people spontaneously reduce caloric

intake without conscious restriction. This preferential loss of visceral (abdominal) fat is particularly important because visceral adiposity is strongly linked to insulin resistance, inflammation, and hepatic steatosis. Several 2025 imaging-based studies confirmed that KD led to significant reductions in visceral fat and liver fat within 12–24 weeks(73).

4) Changes in lipid handling and ketone signaling

KD alters lipid metabolism in ways that affect glucose regulation and systemic health: Triglyceride-rich lipoproteins: KD often lowers circulating triglycerides, likely due to reduced hepatic very-low-density lipoprotein (VLDL) synthesis and greater fat oxidation. HDL increase: High-density lipoprotein (HDL) typically rises, improving lipid ratios and potentially reducing cardiovascular risk. LDL variability: While some patients experience a rise in LDL cholesterol, others see stable or reduced LDL depending on the balance of saturated vs. unsaturated fats in their KD. Beyond lipids, ketone bodies act as metabolic signals. β -hydroxybutyrate has been shown to: Enhance mitochondrial efficiency and reduce oxidative stress. Act as a signaling molecule to suppress inflammation. Potentially influence appetite-regulating neurocircuits(73).

5) Integration of mechanisms: impact on β -cells

For people with T2D, pancreatic β -cells are under chronic stress they must secrete large amounts of insulin to overcome insulin resistance. Over time, this contributes to β -cell exhaustion and progressive decline in insulin secretion. Lowering glucose excursions, reducing insulin demand, decreasing visceral adiposity, and improving lipid and ketone signaling, KD reduces the “physiologic workload” on β -cells. This may help preserve residual β -cell function and slow disease progression. While long-term proof is still lacking, animal models and short-term human studies support this concept (73).

3.2.5 Safety, adverse effects and special populations

KD is not risk-free and requires tailored clinical oversight in people with diabetes: Hypoglycemia risk: when glucose lowering drugs (insulin or secretagogues) are continued unchanged, strict carbohydrate restriction can precipitate hypoglycemia; medication down titration is often needed during KD initiation. Continuous glucose monitoring and staged insulin adjustments mitigate this risk. Lipid changes: although triglycerides and HDL often improve, LDL-C can rise in some individuals particularly if saturated fat intake is high. Clinicians should monitor lipids and emphasize unsaturated fat sources where possible. Renal, hepatic and micronutrient issues: KD can unmask or worsen kidney stones, gout, or hepatic steatosis in susceptible people; fiber and micronutrient intake may fall if green vegetables and fortified foods are not prioritized. Supplementation and dietitian supervision are recommended. Type 1 diabetes (T1D): evidence is limited and mixed. Small series and case reports show some people with T1D reduce insulin doses and smooth glycemic excursions, but the diet increases practical risks notably hypoglycemia and (rarely) euglycemic diabetic ketoacidosis. Professional societies advise extreme caution, close monitoring (CGM), and specialist oversight before recommending KD in T1D(74).

5.2.6 Adherence and sustainability — the practical reality

One consistent message from 2024–25 data is that KD's metabolic benefits are tightly linked to adherence. The very restrictiveness of KD food choices, social constraints, taste fatigue leads many people to relax carbohydrate targets over time. As adherence drifts, the advantages over less restrictive, calorie conscious patterns (e.g., Mediterranean, DASH) decline. Therefore, successful KD programs usually include structured education, meal planning, behavioral support, and medical oversight. Long-term maintenance plans often involve transitioning to a less restrictive, lower-carbohydrate Mediterranean-style pattern that preserves gains while being easier to sustain. KD is a reasonable, evidence-based option for selected people with T2D who need rapid weight loss or glucose lowering, are motivated and can access structured support. It may be particularly useful for people with marked post-prandial hyperglycaemia or those who have failed other diets. KD is less attractive for patients with a history of severe dyslipidemia, chronic kidney disease, pregnancy, or those unable to engage with close monitoring. For T1D, KD can be considered only with specialist teams and frequent monitoring(75).

3.2.6 Clinical implementation: pragmatic steps

If clinicians and patients choose KD: Pre-screening: baseline lipid panel, kidney and liver function, medication review. Medication plan: proactive insulin/secretagogue dose reductions with daily glucose (or CGM) review. Diet design: prioritize unsaturated fats, non starchy vegetables, adequate protein, and fiber supplementation when needed. Monitoring: HbA_{1c}, lipids, renal function, and ketone checks as indicated; CGM strongly recommended for insulin users. Behavioral support: dietary education, follow up, and a step down plan to a sustainable maintenance diet.

3.2.7 Key Efficacy Findings

1) Glycemic Control & HbA_{1c}

A meta-analysis surveyed 17 RCTs (1,197 overweight or obese participants with T2D) comparing low-carbohydrate diets (LCDs), many approaching or overlapping with KD, to control diets. The findings indicated significant decreases in HbA_{1c} (mean difference (−0.36%, 95% CI −0.44 to −0.29) and fasting glucose (−10.7 mg/dL, CI −14.4 to −7.03) after ~3–6 months. However, effects tend to peak within 6–12 months. After this period, HbA_{1c} reductions often diminish or converge toward outcomes from less restrictive, balanced diets. The greatest gains in glycemic control are typically observed early.

2) Weight Loss & Body Composition

The same meta-analysis noted that participants on KD/LCDs lost more body weight and BMI relative to control diets, particularly in the short term (first few months). Mean weight loss ranges differ by study, but appreciable reductions (e.g., several kilograms) were commonly reported. Beyond weight, there were improvements in waist circumference and body fat percentage (BFP) in many trials, though fat-free mass (i.e. lean tissue) preservation depends heavily on protein content and physical activity in the diet plan.

3) Lipid Profile and Cardiometabolic Markers

KD/LCD interventions improved lipid profiles: triglycerides dropped, HDL cholesterol increased in a consistent way. Effects on total cholesterol and LDL cholesterol are more mixed: some studies found mild worsening or negligible change in LDL; others observed modest improvement. The type of fat (saturated vs unsaturated), baseline lipid profile, and individual metabolic response seem to influence these outcomes. Secondary benefits included reductions in blood pressure (especially diastolic), improvements in insulin sensitivity, and favorable effects on inflammatory markers in some studies(76).

3.2.8 Limitations & Sustainability

Adherence declines over time. Many studies report strong adherence and benefits in the first 3-6 months, but a drop in compliance thereafter. Taste fatigue, social eating constraints, dietary monotony, and restrictive nature contribute. Nutrient intake concerns: Very low carbohydrate intake often inadvertently reduces fiber, certain micronutrients (e.g., magnesium, potassium), and may increase saturated fat intake. Long-term safety data are limited. Heterogeneity in definitions: “Low-carbohydrate” covers a wide spectrum from moderate restriction (e.g. 100g/day) to rigorously ketogenic (<30g/day). Different studies use different thresholds and macronutrient mixes. This inconsistency complicates interpretation and guideline formulation. Lack of very long-term RCTs (beyond 1-2 years) that examine outcomes like cardiovascular events, renal function, bone density, and overall mortality. Most evidence focuses on intermediate markers (HbA_{1c}, lipids, blood pressure)(77).

3.2.9 Patient & Clinical Considerations

Patient selection: KD may be more beneficial for overweight or obese individuals with T2D who struggle with post-prandial glucose spikes, insulin resistance, or challenging weight loss with standard diets. Those with dyslipidemia may need careful fat-type planning (favoring unsaturated fats). Monitoring: Frequent glucose monitoring (including post-prandial), lipid panels, kidney function, and possibly ketone levels are advisable. Adjustments to medications (insulin, secretagogues) needed to avoid hypoglycemia. Diet design: To maximize benefit and reduce risk, diets should ensure adequate protein, include sources of fiber from non-starchy vegetables and nuts/seeds, ensure adequate micronutrients (via food or supplements), and involve behavioral support to sustain long-term adherence. Behavioral & social factors: Support structures (dietitian guidance, peer groups, digital tools) enhance sustainability. Also cultural preferences and food environment matter heavily(77).

3.3 Plant-based dietary patterns

3.3.1 Introduction

Plant based diets ranging from whole-food, predominantly plant diets to vegetarian and vegan patterns have become a major focus of diabetes research and clinical practice. By emphasizing vegetables, fruits, whole grains, legumes, nuts and seeds and de-emphasizing or excluding animal products, these patterns act on multiple metabolic pathways relevant to type 2 diabetes (T2D): post-prandial glycemia, insulin sensitivity, body composition, inflammation and gut microbiota(78).

3.3.2 Clinical efficacy: trials and meta-analyses

Randomized controlled data are particularly persuasive. A large 24 week randomized trial of an intensive whole-food, plant-based lifestyle intervention that combined diet, structured exercise and group support reported strong improvements in glycemic control and medication use: the intervention lowered HbA_{1c} by 1.3% at 12 weeks and 0.7% at 24 weeks versus standard care, and 63% of medicated participants reduced their glucose lowering drugs (versus 24% with usual care). The trial also documented weight loss, lower CRP, and some cases of diabetes remission, demonstrating that an organized plant based program can produce clinically meaningful, rapid gains in people with T2D. Systematic reviews and meta-analyses of randomized trials reinforce these findings. A 2025 meta-analysis of vegetarian and vegan RCTs found pooled HbA_{1c} reductions (weighted mean difference 0.36%) and modest decreases in BMI and LDL-cholesterol compared with omnivorous control diets, with stronger signals when interventions lasted >12 weeks and when diets emphasized whole, minimally processed plant foods. These pooled data support the idea that plant-predominant diets can improve glycemic and cardiometabolic risk factors when properly implemented(78,79).

3.3.3 Mechanisms that link plant foods to better glucose control

Several, partly overlapping mechanisms explain the metabolic benefits: Fiber and glycemic buffering in which whole plant foods are rich in soluble and insoluble fiber; fiber slows gastric emptying, flattens post-prandial glucose curves and supports satiety, which helps both immediate glucose control and weight management. Weight loss and adiposity shifts which includes plant based interventions often reduce energy density and promote modest weight loss, particularly visceral fat reduction a key driver of insulin resistance. Improved insulin sensitivity in which plant diets rich in polyphenols and unsaturated fats improve endothelial function and insulin signaling pathways; observational and short-term trial data show better HOMA-IR and fasting insulin metrics. Anti-inflammatory and antioxidant effects which includes phytochemicals and higher antioxidant intake lower systemic markers of inflammation (e.g., CRP) that contribute to insulin resistance and β -cell stress. Gut microbiome modulation which includes increased prebiotic substrates (fiber) change microbiota composition and short-chain fatty acid production pathways increasingly implicated in glucose regulation.

These mechanisms combine to reduce glycaemic variability and the physiologic demand on pancreatic β -cells, explaining why structured plant-based programs can improve HbA_{1c} and permit medication reductions in many people(79).

3.3.4 Practical outcomes

Outcomes across studies vary by intensity and support. Intensive programs (meal provision, regular classes, behavior coaching) show the largest short-term HbA_{1c} falls and medication reductions; less-structured advice yields smaller but still meaningful effects. Benefits appear within weeks for post prandial measures and over months for HbA_{1c}. Major real-world advantages include low cost of many plant foods, cardio-protective effects, and favorable effects on weight and inflammation.

However, challenges exist: adherence, nutrient planning (B12, iron, vitamin D, omega-3s), and cultural acceptability. Clinicians should ensure B12 supplementation for strict vegans, counsel

on iron and protein sources, and emphasize whole foods over processed “plant” substitutes. Importantly, replacing animal products with refined carbs or ultra-processed plant foods can abolish benefits quality of plant foods is the decisive factor.

Plant-based strategies suit adults with T2D who seek to reduce medication burdens, lose weight, or lower cardiovascular risk. Practical implementation steps include individualized meal plans emphasizing legumes, whole grains, vegetables, nuts and modest amounts of fruit; staged medication review to avoid hypoglycaemia; B12 and, where indicated, iron/vitamin D assessment; and behavioral support (group classes, dietitian follow-up). Low intensity versions (flexitarian or increased hPDI) can be effective and are often easier to sustain(80).

3.3.5 Research gaps

Despite promising evidence, significant research gaps remain in evaluating plant based dietary patterns for type 2 diabetes (T2D). Most trials to date are of short duration (≤ 6 –12 months), limiting understanding of long term adherence, durability of glycemic benefits, and sustainability across populations. A 2025 systematic review highlighted that while whole food, plant based interventions improved HbA_{1c} and reduced medication use, only a few studies extended beyond one year, making it unclear whether benefits persist or plateau. Another key gap involves mechanistic insights, particularly the role of the gut microbiome. Emerging studies suggest that microbiome diversity and short chain fatty acid (SCFA) production may mediate improved insulin sensitivity in plant based eaters, but causal links remain underexplored. Precision nutrition approaches integrating microbiome, metabolomic, and genetic profiling could help explain individual variability in dietary response. Finally, pragmatic implementation studies are needed. Most RCTs occur in controlled environments, yet real-world translation into diverse healthcare systems, cultures, and socioeconomic settings remains under-studied. Addressing barriers such as food access, cost, and cultural acceptability is critical to scaling plant-based interventions globally. Comparative effectiveness trials versus other diets (e.g., Mediterranean, low-carb) are also lacking(80).

3.3.6 Outcomes of Dietary Patterns in Diabetes Management

Dietary patterns are a cornerstone in diabetes management, offering benefits that extend beyond glycemic control to cardiovascular risk, body weight, and overall metabolic health. The accumulating evidence from randomized controlled trials (RCTs), meta-analyses, and cohort studies has clarified how different diets influence outcomes in both type 1 and type 2 diabetes (T2D).

- 1) **Glycemic Control** :Multiple studies demonstrate that structured dietary approaches significantly lower glycated hemoglobin (HbA_{1c}) and fasting glucose levels. For example, a 2025 meta analysis comparing low carbohydrate, plant-based, Mediterranean, and DASH diets found that all patterns improved HbA_{1c} by 0.5–1.2% over 6–12 months, though the sustainability of low-carbohydrate effects declined after 12 months. Plant-based and Mediterranean diets consistently showed moderate but durable improvements, suggesting they may be better suited for long-term glycemic stability.
- 2) **Weight and Body Composition** :Obesity remains a major driver of insulin resistance, and dietary interventions targeting weight reduction are strongly beneficial. Very-low-

carbohydrate and ketogenic-style diets produced rapid reductions in body weight at 6 months; however, adherence issues often led to weight regain by 12–24 months. In contrast, Mediterranean and plant-based diets promoted steady, sustainable weight loss and reductions in visceral adiposity, with associated improvements in insulin sensitivity.

- 3) **Lipid and Cardiovascular Outcomes:** Beyond glucose control, dietary patterns strongly influence cardiovascular risk a key concern since cardiovascular disease is the leading cause of morbidity and mortality in diabetes. A 2025 cohort study confirmed that adherence to a Mediterranean diet was associated with a 59% lower risk of progression from prediabetes to T2D, and also reduced cardiovascular events over two decades. Similarly, the Diabetes Risk Reduction Diet (DRRD) was linked not only to a 47% reduction in T2D incidence but also to a 36% reduction in cancer risk and a 24% decline in all-cause mortality, underscoring the systemic benefits of dietary quality.
- 4) **Blood Pressure and Renal Outcomes:** Hypertension is a common comorbidity in T2D, and diets rich in fruits, vegetables, and whole grains play a role in blood pressure regulation. A randomized clinical trial in 2025 showed that the DASH4D diet, particularly when combined with sodium reduction, significantly decreased both systolic and diastolic blood pressure in individuals with T2D and hypertension. These effects highlight the importance of considering cardiovascular and renal health alongside glycemic control in dietary recommendations.
- 5) **Quality of Life and Behavioral Outcomes :** Dietary interventions also impact psychological well being and health behaviors. Plant based diets, for instance, were associated with improvements in energy, reduced sugar cravings, and better dietary adherence when integrated into culturally tailored programs. Similarly, dietary patterns that emphasize mindful eating, variety, and satiety such as the Mediterranean diet may encourage sustained adherence compared to restrictive regimens like ketogenic diets(81).

Table 5: Clinical outcomes from plant-based trials and meta-analyses

Outcome	Typical magnitude (when reported)	Evidence source / notes
HbA_{1c} (short term, 8–24 wk)	↓ 0.7–1.3% in intensive whole-food plant-based (WFPB) trials at 12 weeks; sustained 0.4–0.8% at 24 weeks in some studies.	RCT (WFPB intensive program): substantial early fall in HbA _{1c} and medication reduction.
HbA_{1c} (meta-analyses)	Weighted mean difference 0.3 to –0.4% across vegetarian/vegan RCTs vs control.	Systematic review / meta analysis (Nutr Rev 2025).
Weight / BMI	Modest moderate reductions; larger losses when program includes caloric focus and behavioral support.	RCTs and pooled analyses; weight loss contributes to glycemic improvements.

Outcome	Typical magnitude (when reported)	Evidence source / notes
Lipid profile	↓ LDL modestly in many trials; triglycerides and HDL improvements variable depending on food quality.	Meta analyses show cardiometabolic benefits when diets emphasize whole foods.
Medication use / remission	In intensive programs many participants reduced meds; a subset achieved remission (per trial definitions).	Diabetologia RCT reported medication reductions and some remissions.
Microbiome metabolites	Increased fibre → ↑ SCFA producers (e.g., Faecalibacterium) and improved microbial diversity in short medium term; links to insulin sensitivity under study(81).	Systematic reviews and cohort microbiome studies.

Table 6 : Practical points: mechanisms, pros/cons, implementation

Topic	Key points
Primary mechanisms	High fibre slows glucose absorption; lower energy density supports weight loss; polyphenols/unsaturated fats reduce inflammation; microbiome shifts (↑ SCFA) modulate insulin sensitivity.
Who most benefits	People with T2D who can adopt whole-food patterns, want medication reductions, or need cardiometabolic risk lowering; motivated patients in structured programs show greatest gains.
Risks monitoring	/ Possible B12, iron, vitamin D, omega-3 shortfalls in strict vegans monitor and supplement as needed; watch hypoglycaemia when drugs are reduced.
Sustainability	Intensive, supported programs show biggest early gains; pragmatic, culturally adapted hPDI (healthy Plant-Based Diet Index) approaches improve long term adherence.
Implementation tips	Prioritize legumes, whole grains, vegetables, nuts; provide B12 supplement guidance; schedule medication review; use behavior coaching and follow up(82,83).

Intensive whole food, plant based (WFPB) programs with meal guidance and behavioral support produced large early reductions in HbA_{1c} (1.3% at 12 weeks in a published RCT) and allowed many participants to lower or stop glucose lowering medications; some achieved trial defined remission. These clinical changes were linked to weight loss, reduced post prandial glycemic load, and improved insulin sensitivity. Larger meta-analyses across vegetarian/vegan RCTs show moderate pooled HbA_{1c} benefits (0.3 to −0.4%) and favorable shifts in LDL and

BMI when diets emphasize whole foods rather than processed plant products. Cohort dose-response analyses differentiate a healthy plant-based index (hPDI) high in whole grains, fruits, vegetables, legumes which is consistently associated with lower incident T2D risk, from an unhealthy plant index (uPDI) high in refined grains and sugary plant foods which does not confer protection and may increase risk. This demonstrates that *plant-based* $\neq$ *automatically healthy*; food quality is decisive. Mechanistically, abundant dietary fibre slows intestinal glucose uptake and feeds beneficial microbes that produce short-chain fatty acids (SCFAs). SCFAs have been linked to improved insulin sensitivity and lower inflammation plausible mediators of the metabolic benefits seen after adopting plant-rich diets. Systematic reviews and large cohort microbiome studies through 2024–2025 show consistent microbiome changes with plant-rich diets, though causal chains linking specific microbes to glycemic outcomes need stronger interventional evidence. From a pragmatic standpoint, outcomes are best when plant-based prescriptions are structured, culturally adapted and supported (meal plans, supplement advice, medication review, behavioral counselling). Key research gaps for 2025 include longer pragmatic RCTs comparing scalable plant-based programs to other evidence-based diets, mechanistic trials integrating microbiome/metabolomics, and implementation science in diverse health-care settings(84).

Conclusion

The growing burden of type 2 diabetes mellitus (T2D) highlights the need for integrative, multi-targeted management strategies that extend beyond pharmacotherapy alone. Evidence available up to 2025 supports a cautiously positive role for Ayurvedic formulations, yoga and meditation, and structured dietary patterns as adjuncts to standard diabetes care. Recent randomized trials and real-world studies suggest that well-standardized Ayurvedic polyherbal formulations and selected botanicals such as *Gymnema sylvestre*, *Syzygium cumini*, *Trigonella foenum-graecum*, berberine, and cinnamon can produce modest but clinically meaningful reductions in glycaemic indices ($HbA_{1c} = 0.3\text{--}0.8\%$) when used alongside conventional therapy. Mechanistic studies provide biological plausibility through coordinated effects on insulin signaling, AMPK activation, inflammation, oxidative stress, and gut-level carbohydrate handling.

Mind–body interventions, particularly yoga and meditation, demonstrate additional benefits through autonomic rebalancing, stress reduction, improved insulin sensitivity, and enhanced self-care behaviors, contributing to small yet relevant improvements in glycaemic control and quality of life. Dietary pattern research from 2025 further confirms that Mediterranean, plant-based, DASH/DRRD, and low-carbohydrate approaches can improve glycaemia, weight, and cardiometabolic risk, with long-term success largely dependent on diet quality and adherence rather than strict macronutrient restriction.

Overall, the evidence favors a personalized, evidence-guided integrative approach in which standardized Ayurvedic nutraceuticals and lifestyle interventions complement guideline-directed pharmacotherapy. Future priorities include longer-duration, high-quality randomized trials, improved standardization, and pragmatic implementation research to clarify long-term efficacy, safety, and generalizability in diverse populations.

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List of abbreviations

Abbreviation	Full Form
ADA	American Diabetes Association
AGE	Advanced Glycation End-products
Akt (PKB)	Protein Kinase B
AMPK	Adenosine Monophosphate-Activated Protein Kinase
BFP	Body Fat Percentage
BMI	Body Mass Index
CGM	Continuous Glucose Monitoring
CKD	Chronic Kidney Disease
CRP	C-Reactive Protein
CYP	Cytochrome P450
DAG	Diacylglycerol
DASH	Dietary Approaches to Stop Hypertension
DASH4D	DASH Diet for Diabetes
DBI	Diazepam Binding Inhibitor
DKA	Diabetic Ketoacidosis
DPP-IV	Dipeptidyl Peptidase-IV
DRRD	Diabetes Risk Reduction Diet
ER	Endoplasmic Reticulum
FOXO	Forkhead Box O Transcription Factors
FPG	Fasting Plasma Glucose
GIP	Glucose-Dependent Insulinotropic Polypeptide
GI	Gastrointestinal
GLP-1 RA	Glucagon-Like Peptide-1 Receptor Agonist
GLUT	Glucose Transporter
GLUT4	Glucose Transporter Type-4
GMP	Good Manufacturing Practice
GSK-3	Glycogen Synthase Kinase-3
HbA _{1c}	Glycated Hemoglobin
HDL	High-Density Lipoprotein
HOMA-IR	Homeostatic Model Assessment of Insulin Resistance

Abbreviation	Full Form
HPA Axis	Hypothalamic–Pituitary–Adrenal Axis
HPTLC	High-Performance Thin-Layer Chromatography
HRV	Heart Rate Variability
IL-6	Interleukin-6
IL17RA	Interleukin-17 Receptor A
IRS	Insulin Receptor Substrate
KD	Ketogenic Diet
LC-MS	Liquid Chromatography–Mass Spectrometry
LDL	Low-Density Lipoprotein
LG	Longitudinal Glycaemia (cohort context)
MAPK	Mitogen-Activated Protein Kinase
MBSR	Mindfulness-Based Stress Reduction
MBCT	Mindfulness-Based Cognitive Therapy
MD-1	Multi-Drug Polyherbal Formulation-1
mTORC2	Mammalian Target of Rapamycin Complex-2
NF-κB	Nuclear Factor Kappa-Light-Chain-Enhancer of Activated B Cells
NLRP3	NOD-Like Receptor Protein-3 Inflammasome
Nrf2	Nuclear Factor Erythroid-2-Related Factor-2
OGTT	Oral Glucose Tolerance Test
PDK1	3-Phosphoinositide-Dependent Protein Kinase-1
PI3K	Phosphoinositide 3-Kinase
PIP ₂	Phosphatidylinositol-4,5-Bisphosphate
PIP ₃	Phosphatidylinositol-3,4,5-Trisphosphate
PPAR-γ	Peroxisome Proliferator-Activated Receptor-Gamma
PPG	Post-Prandial Glucose
QoL	Quality of Life
RCT	Randomized Controlled Trial
ROS	Reactive Oxygen Species
SCFA	Short-Chain Fatty Acid
SGLT1	Sodium-Glucose Linked Transporter-1
SMBG	Self-Monitoring of Blood Glucose
T1D	Type 1 Diabetes Mellitus
T2D	Type 2 Diabetes Mellitus
TIR	Time in Range
TNF-α	Tumor Necrosis Factor-Alpha
UDCA	Ursodeoxycholic Acid

Abbreviation	Full Form
VLDL	Very-Low-Density Lipoprotein
WFPB	Whole-Food Plant-Based
hPDI	Healthy Plant-Based Diet Index
uPDI	Unhealthy Plant-Based Diet Index

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