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Mechanistic Basis of Yoga-Based Lifestyle Intervention in the Management of Type 2 Diabetes Mellitus: A Narrative Review of Psycho-Neuro-Endocrine and Metabolic Pathways

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Abstract: *Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder with rapidly escalating prevalence in India, where more than 101 million adults are currently estimated to live with diabetes and another 136 million with prediabetes. Pharmacological management addresses hyperglycaemia but does not modify the underlying lifestyle drivers, and is constrained by cost, adherence, and long-term side effects. Yoga-based lifestyle interventions have emerged as a complementary therapeutic strategy with reproducible glycaemic benefit, although the biological mechanisms linking practice to outcome remain incompletely integrated.*

Aim: To synthesise the current mechanistic evidence on how yoga-based interventions influence the pathophysiology of T2DM, focusing on psycho-neuro-endocrine, autonomic, inflammatory, and metabolic pathways.

Methods: A narrative review of peer-reviewed literature published between January 2000 and February 2026 was conducted using PubMed, Google Scholar, and the Cochrane Library. Search terms combined "yoga", "pranayama", "meditation", "type 2 diabetes", "insulin resistance", "HPA axis", "cortisol", "heart rate variability", "inflammation", and "oxidative stress". Studies were prioritised by methodological strength (randomised controlled trials, systematic reviews, meta-analyses) and biological mechanism specificity.

Results: Six interlinked mechanism families emerge from the literature: (i) recalibration of the hypothalamic-pituitary-adrenal (HPA) axis with reduction in circulating cortisol; (ii) shift in sympathovagal balance toward parasympathetic dominance, reflected in improved heart rate variability; (iii) attenuation of chronic low-grade inflammation, with reductions in C-reactive protein, interleukin-6, and tumour necrosis factor-α; (iv) reduction of systemic oxidative stress; (v) improvement in peripheral insulin sensitivity and skeletal-muscle glucose uptake; and (vi) behavioural changes including mindful eating, treatment adherence, and stress-coping. These pathways are not independent: they converge on stress physiology and metabolic flexibility, providing a coherent mechanistic basis for the glycaemic effects observed in meta-analyses of yoga in T2DM.

Conclusion: Yoga acts on T2DM through several parallel and partially overlapping biological pathways rather than a single mechanism. Future research should prioritise integrated, multi-biomarker studies in well-defined cohorts, with attention to dose, intervention components, and the relative contribution of asana, pranayama, and meditation.

Keywords: type 2 diabetes mellitus; yoga; pranayama; HPA axis; heart rate variability; insulin resistance; inflammation; oxidative stress; mind-body intervention

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I. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is among the most pressing non-communicable disease challenges of the twenty-first century. According to the 2023 ICMR-INDIAB-17 national cross-sectional study, the prevalence of diabetes in India has reached 11.4% of adults aged 20 years and older, with prediabetes at 15.3%; these proportions translate into approximately 101 million people living with diabetes and 136 million with prediabetes [1]. The disease burden is unevenly distributed across states, but the underlying driver is consistent: a shift toward sedentary lifestyles, energy-dense diets, and chronic psychosocial stress against a backdrop of the so-called Asian-Indian phenotype of early central adiposity and insulin resistance [2,3].

Standard pharmacological care, including metformin, sulphonylureas, dipeptidyl peptidase-4 inhibitors, sodium-glucose cotransporter-2 inhibitors, and insulin, is effective at controlling hyperglycaemia but does not modify the upstream behavioural and stress-related drivers of the disease. Long-term medication carries cost, adherence, and adverse-event burdens that are particularly relevant in resource-constrained settings [4]. There is a recognised need for adjunctive, lifestyle-based strategies that target the metabolic, autonomic, and psycho-emotional substrates of T2DM simultaneously.

Yoga, a mind-body discipline originating in the Indian Knowledge Systems and now practised globally, is one such candidate intervention. Several randomised controlled trials and meta-analyses have documented improvements in fasting blood glucose (FBG), postprandial blood glucose (PPBG), glycated haemoglobin (HbA1c), and lipid profile following structured yoga-based interventions in adults with T2DM [5–7]. Cui and colleagues' meta-analysis of 12 randomised trials with 864 participants reported a pooled weighted mean difference of −23.72 mg/dL (95% CI: −37.78, −9.65) for FBG and −0.47% (95% CI: −0.87, −0.07) for HbA1c in favour of yoga [6]. Thind and colleagues' meta-analysis of 23 trials similarly found pooled standardised mean differences of 0.36 for HbA1c, 0.58 for FBG, and 0.40 for PPBG [7]. These effect sizes, while modest compared to the most aggressive pharmacological or surgical interventions, are clinically meaningful when achieved through a low-cost, scalable, community-based modality.

However, while the clinical efficacy of yoga in T2DM is increasingly accepted, the question of how it works has received less integrated attention. Most mechanistic discussions focus on a single pathway, such as cortisol reduction or heart rate variability improvement, without addressing how these effects converge on the disease process. The present narrative review aims to address this gap by synthesising current evidence on six interlinked mechanism families: HPA-axis recalibration, autonomic balance, inflammation, oxidative stress, insulin signalling, and behavioural pathways. We then propose an integrated framework and identify priority areas for future mechanistic research.

II. METHODS

This is a narrative (non-systematic) review. PubMed, Google Scholar, and the Cochrane Library were searched between October 2025 and February 2026 for publications between January 2000 and February 2026. Search strings combined free-text terms and MeSH headings including "yoga", "pranayama", "meditation", "type 2 diabetes mellitus", "insulin resistance", "HPA axis", "cortisol", "heart rate variability", "sympathetic", "parasympathetic", "inflammation", "oxidative stress", "adiponectin", "BDNF", and "mindfulness". Reference lists of identified reviews and meta-analyses were hand-searched. Articles were selected for inclusion based on relevance, methodological quality (with priority to randomised controlled trials and systematic reviews), mechanistic specificity, and citation impact. We did not apply a formal risk-of-bias assessment, consistent with narrative-review methodology. Given the wide variability in yoga protocols, no attempt at quantitative synthesis was made; the review is integrative rather than confirmatory.

III. PATHOPHYSIOLOGY OF TYPE 2 DIABETES: A BRIEF MECHANISTIC MAP

T2DM is not a single disease but a syndrome arising from the interaction of peripheral insulin resistance, progressive beta-cell dysfunction, chronic low-grade inflammation, and dysregulation of stress physiology [8]. In skeletal muscle, the dominant glucose-disposal tissue, insulin resistance is characterised by impaired insulin-receptor signalling, reduced GLUT4 translocation, and decreased postprandial glucose uptake. In the liver, insulin resistance manifests as a failure to suppress hepatic glucose output, contributing to fasting hyperglycaemia. In adipose tissue, the expansion of visceral fat depots is accompanied by macrophage infiltration and the release of pro-inflammatory cytokines including tumour

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necrosis factor-α (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1, which act in an endocrine fashion to worsen muscle and hepatic insulin resistance [9].

Superimposed on this metabolic substrate is a dysregulated stress response. Chronic activation of the HPA axis leads to sustained cortisol elevation, which antagonises insulin action through gluconeogenesis stimulation, glycogenolysis, and lipolysis. Sympathetic nervous system overactivity, mirrored by parasympathetic withdrawal, accelerates resting heart rate, blood pressure, and catecholamine drive, and contributes to insulin resistance via direct effects on skeletal muscle and adipose tissue [10,11]. The convergence of these pathways produces what Björntorp and others have termed a "stress-related metabolic syndrome" [12]. It is precisely on this convergence that yoga is hypothesised to act.

IV. MECHANISM 1: RECALIBRATION OF THE HPA AXIS AND CORTISOL REDUCTION

The HPA axis is the principal neuroendocrine system mediating the response to psychological and physical stressors. In healthy individuals, acute stress produces a transient rise in corticotropin-releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus, adrenocorticotropic hormone (ACTH) from the anterior pituitary, and cortisol from the adrenal cortex, followed by negative feedback and return to baseline. In chronic psychosocial stress, this regulatory loop becomes dysregulated, producing sustained hypercortisolism with flattened diurnal variation [13].

In people with T2DM, this dysregulation is reflected in elevated morning cortisol, blunted cortisol awakening response, and increased 24-hour urinary free cortisol. Cortisol opposes insulin in three principal ways: by stimulating hepatic gluconeogenesis, by promoting lipolysis with release of free fatty acids that worsen muscle insulin resistance, and by impairing pancreatic beta-cell insulin secretion [14]. The reduction of HPA axis tone is therefore mechanistically plausible as a therapeutic target.

A substantial body of evidence demonstrates that yoga practices, particularly those incorporating slow-breathing pranayama and meditative components, reduce circulating cortisol. The meta-analysis of Thind and colleagues, drawing on randomised trials with cortisol endpoints, reported significant reductions following yoga compared to control conditions [7]. Mechanistically, the cortisol-lowering effect is attributed to engagement of the parasympathetic nervous system through controlled respiration, modulation of amygdalar reactivity through interoceptive attention training in meditation, and reduction of perceived stress with downstream effects on CRH release [15,16]. The clinical implication for T2DM is that sustained reduction of cortisol over months may translate into improved hepatic insulin sensitivity, reduced gluconeogenic drive, and improved fasting glycaemia, providing one mechanistic explanation for the FBG reductions observed in trials.

V. MECHANISM 2: SYMPATHOVAGAL REBALANCING AND HEART RATE VARIABILITY

Autonomic dysfunction in T2DM is well-characterised, and is associated with cardiovascular mortality, silent ischaemia, gastroparesis, and impaired hypoglycaemia awareness [17]. The standard non-invasive index of autonomic balance is heart rate variability (HRV), with high-frequency power reflecting parasympathetic (vagal) influence and the low-frequency-to-high-frequency ratio approximating sympathovagal balance. In adults with T2DM, total HRV is reduced, with disproportionate loss of vagal tone.

Yoga, and pranayama in particular, has consistently been shown to improve HRV indices. The Streeter Vagal-GABA hypothesis [18] proposes that slow-breathing practices stimulate vagal afferents via thoracic and oropharyngeal stretch receptors, increasing efferent parasympathetic outflow and brainstem GABAergic activity. This in turn down-regulates sympathetic drive, reduces allostatic load, and produces measurable shifts in HRV, mood, and cardiovascular reactivity. Direct empirical support comes from controlled trials of pranayama and yoga-asana in T2DM, which demonstrate increased high-frequency HRV power, reduced low-frequency-to-high-frequency ratio, and improved baroreflex sensitivity [19,20].

Two clinically important corollaries follow. First, the cholinergic anti-inflammatory pathway, demonstrated by Tracey and colleagues, links vagal activity to suppression of macrophage TNF-α release via the alpha-7 nicotinic acetylcholine receptor [21]. Improved vagal tone may therefore reduce chronic low-grade inflammation, an upstream driver of insulin resistance. Second, autonomic rebalancing may reduce catecholamine-driven hepatic glucose output and improve insulin secretion under stress conditions, contributing to both fasting and postprandial glycaemia.

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VI. MECHANISM 3: ANTI-INFLAMMATORY AND ANTIOXIDANT EFFECTS

6.1 Chronic low-grade inflammation

T2DM is increasingly conceptualised as a chronic inflammatory state. Elevated CRP, IL-6, and TNF-α are not just markers but active contributors to insulin resistance, acting through serine phosphorylation of insulin receptor substrate-1, suppression of GLUT4, and induction of suppressor of cytokine signalling-3 (SOCS3) [22]. Reduction of these mediators is therefore a legitimate mechanistic target.

Several randomised controlled trials have documented reductions in CRP, IL-6, and TNF-α following structured yoga interventions of 8–12 weeks' duration [23,24]. The proposed pathways include vagal-mediated cholinergic anti-inflammatory signalling, reduction of cortisol-driven glucocorticoid resistance in immune cells, reduced adiposity with consequent reduction in adipokine-mediated inflammation, and direct effects of moderate physical activity on circulating cytokines.

6.2 Oxidative stress

Hyperglycaemia drives the generation of reactive oxygen species through mitochondrial overload, glucose auto-oxidation, and advanced glycation end-product formation. Reduced antioxidant defence, reflected in low superoxide dismutase (SOD) and glutathione levels and high malondialdehyde (MDA), is documented in T2DM and contributes to endothelial dysfunction and microvascular complications [25]. Hegde and colleagues' controlled trial of three-month yoga in T2DM patients with and without complications demonstrated significant reductions in MDA and increases in SOD compared to standard care alone [26]. The yoga effect on oxidative stress is plausibly mediated by improved glycaemic control itself, by improved mitochondrial efficiency with moderate exercise, and by reduction of catecholamine-driven oxidative bursts.

VII. MECHANISM 4: IMPROVED INSULIN SIGNALLING AND SKELETAL MUSCLE METABOLISM

Yoga asana, while frequently classified as a low-to-moderate-intensity activity, engages skeletal muscle through isometric and dynamic postures and through specific abdominal contractions. The energy cost of a typical hatha-style yoga session is estimated at 2.5–4.5 metabolic equivalents (METs), placing it in the moderate-intensity range used by physical activity guidelines for diabetes prevention [27]. Beyond energy expenditure, however, asana exerts effects on muscle insulin sensitivity through several mechanisms.

First, the contractile activity of yoga postures triggers AMP-activated protein kinase (AMPK)-mediated GLUT4 translocation in an insulin-independent manner, allowing glucose entry into muscle even in the presence of insulin resistance. Second, regular asana over months produces modest reductions in visceral and ectopic fat, with consequent improvements in hepatic and muscle insulin sensitivity. Third, specific postures incorporating sustained abdominal compression and twists are hypothesised to influence pancreatic perfusion and beta-cell function, though direct evidence in humans is limited and this proposal should be treated as suggestive rather than established [28]. Singh and colleagues, in a controlled study of pranayama and asana in T2DM, documented significant reductions in serum insulin and glucose, with parallel improvement in homeostatic model assessment of insulin resistance (HOMA-IR) [29].

VIII. MECHANISM 5: BEHAVIOURAL AND LIFESTYLE PATHWAYS

Beyond direct physiological effects, yoga influences T2DM through behavioural pathways that are often under-emphasised in mechanistic discussions. Three are particularly relevant.

First, mindful eating: the meditative attention training that accompanies serious yoga practice extends to interoception of hunger and satiety cues. Several trials of mindfulness-based interventions in T2DM and prediabetes have documented modest weight loss, reduced binge-eating behaviour, and improved dietary quality [30]. The yogic principle of mitāhāra (moderation in eating) provides a culturally familiar framing for portion control and slow eating, which is increasingly supported by metabolic evidence.

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Second, treatment adherence: T2DM is a self-managed condition; long-term outcomes depend on consistent medication use, dietary discipline, and physical activity. The structure of regular yoga practice, particularly when delivered in group settings, can produce indirect benefits on overall lifestyle adherence by reinforcing routine, providing social support, and improving self-efficacy. Sreedevi and colleagues' randomised feasibility study of yoga and peer support in women with T2DM demonstrated incremental benefit on glycaemic outcomes when both components were combined, suggesting a behavioural-physiological synergy [31].

Third, sleep and circadian rhythm: poor sleep quality and short sleep duration are risk factors for T2DM and insulin resistance. Meditation-based components of yoga, including Yoga Nidra and related relaxation practices, improve sleep quality in clinical populations, with downstream effects on glucose metabolism via leptin, ghrelin, and growth hormone rhythms [32].

IX. AN INTEGRATED MECHANISTIC FRAMEWORK

The mechanisms reviewed above are not independent. The most parsimonious framework treats yoga as a multi-component intervention whose effects converge on three integrative nodes: the HPA-autonomic stress axis, peripheral metabolic signalling, and behavioural self-regulation. Stress-axis recalibration reduces cortisol-driven gluconeogenesis and catecholamine-driven hepatic glucose output, while concurrently lowering the inflammatory tone that drives insulin resistance. Improved vagal activity recruits the cholinergic anti-inflammatory pathway, completing a feedback loop that connects respiration, autonomic function, and immunity. Physical components of asana contribute insulin-independent muscle glucose uptake and modest reductions in adiposity. Behavioural components reinforce these effects through dietary self-regulation and adherence.

This integration also explains the modest pooled effect sizes seen in meta-analyses: any single mechanism, considered in isolation, has limited magnitude. The clinical effect emerges from the convergence of several small effects acting on related pathways. It also explains why studies using narrowly defined yoga protocols (asana alone, or pranayama alone) tend to show smaller effects than comprehensive interventions including diet, breathing, asana, and meditation [5–7].

Table 1. Summary of Proposed Mechanisms of Yoga in T2DM

Mechanism family	Proposed biological pathway	Main biomarkers / endpoints	Key references
HPA axis recalibration	Reduction of CRH-ACTH-cortisol drive via parasympathetic activation and meditative attention	Serum cortisol, salivary cortisol, FBG, hepatic glucose output	[7,13–16]
Autonomic rebalancing	Vagal stimulation via slow breathing and stretch receptors; cholinergic anti-inflammatory pathway	HRV (HF, LF/HF), baroreflex sensitivity, resting HR	[18–21]
Anti-inflammatory effect	Cytokine suppression via vagal pathway and cortisol normalisation	CRP, IL-6, TNF-α	[22–24]
Antioxidant effect	Reduced oxidative stress through improved glycaemic control and reduced catecholamine drive	MDA, SOD, glutathione	[25,26]
Insulin signalling and muscle metabolism	AMPK-mediated GLUT4 translocation; reduced visceral adiposity; possible pancreatic effects (suggestive)	HOMA-IR, FBG, PPBG, BMI, waist circumference	[27–29]

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Behavioural pathways	Mindful eating, treatment adherence, sleep, social support	Dietary recall, adherence scores, sleep quality, QoL	[30–32]

X. CLINICAL TRANSLATION AND EVIDENCE GAPS

Despite three decades of clinical evidence, integrated mechanistic studies in yoga and T2DM remain rare. Most published trials use a single biomarker domain (often HbA1c plus a lipid panel) and do not measure cortisol, HRV, and cytokines together. As a result, the relative contribution of each pathway, the dose-response of individual components (asana versus pranayama versus meditation), and the duration required for stable mechanistic change cannot yet be specified with confidence.

Three priority areas for future research follow from this review. First, multi-biomarker trials in well-defined cohorts that simultaneously measure stress, autonomic, inflammatory, and metabolic endpoints. Second, factorial designs that isolate the contribution of asana, pranayama, and meditation components. Third, mechanistic neuroimaging studies, including functional MRI of the prefrontal-amygdalar circuit during stress challenges in yoga practitioners versus controls, to test the hypothesis that yoga modifies central stress-regulatory networks in a measurable, durable way.

XI. LIMITATIONS OF THE REVIEW

This is a narrative review and does not apply systematic review methodology. We did not perform formal risk-of-bias assessment, and our selection of studies, while broad, is inevitably influenced by citation impact and language (English-only). The literature on yoga in T2DM is heterogeneous in protocol, dose, and outcome measures, which limits the strength of mechanistic inference. Some of the proposed mechanisms, particularly pancreatic effects of specific asana, are based on theoretical reasoning and animal data and require human confirmation. We have attempted to distinguish established from suggestive mechanisms in the text.

XII. CONCLUSION

Yoga-based lifestyle interventions act on T2DM through a convergent set of psycho-neuro-endocrine, autonomic, inflammatory, and metabolic pathways, rather than through any single mechanism. The strongest mechanistic evidence supports HPA-axis recalibration, autonomic rebalancing with vagal stimulation, and anti-inflammatory effects. Insulin sensitivity, behavioural change, and reduced oxidative stress contribute additionally. Integrated, multi-biomarker mechanistic studies are needed to specify the relative contribution of each pathway and to guide protocol design for clinical practice. As India confronts a growing diabetes epidemic, mechanistic clarity about how yoga works will strengthen both clinical credibility and rational protocol design.

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Author contributions: SSG conceived the review and prepared the initial draft; AKJ supervised, contributed to interpretation, and revised the manuscript. Both authors approved the final version.

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