

Glycaemic Trajectories Following a Long-Term Yoga and Lifestyle Intervention in Adults with Type 2 Diabetes Mellitus: A Single-Arm Observational Cohort Study with 21-Month Follow-Up

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Abstract: Background: Yoga-based lifestyle interventions are widely practised in India as a complementary strategy for type 2 diabetes mellitus (T2DM). While short-duration trial evidence supports modest glycaemic benefit, real-world data on the trajectories of fasting and postprandial glycaemia across extended follow-up are limited.

Aim: To describe the longitudinal trajectories of fasting blood glucose (FBG) and postprandial blood glucose (PPBG) in a cohort of adults with T2DM enrolled in a yoga-based lifestyle intervention with quarterly assessments over 21 months.

Methods: This is a prospective, single-arm, observational cohort study. Adults with T2DM ($n = 99$; 71 women, 28 men; mean age 45.6 ± 8.6 years) were enrolled and underwent eight quarterly assessments from August 2024 to April 2026. The intervention combined an intensive yoga camp with periodic reinforcement sessions and a structured low-carbohydrate, high-protein dietary plan. FBG and PPBG were measured at each visit. Statistical analyses included paired comparisons (paired t -test and Wilcoxon signed-rank test), linear mixed-effects models with random intercepts per participant, and pre-specified subgroup analyses by terminal medication status.

Results: Mean FBG declined from 160.9 ± 59.8 mg/dL at baseline to 102.9 ± 36.5 mg/dL at month 21 (mean change -58.0 mg/dL; paired $t = -21.5$, $p < 0.001$; Cohen's $d = -2.16$). Mean PPBG declined from 224.4 ± 90.6 mg/dL to 129.4 ± 47.3 mg/dL (mean change -95.0 mg/dL; paired $t = -18.7$, $p < 0.001$; Cohen's $d = -1.88$). Linear mixed-effects models estimated a fall in FBG of 7.94 mg/dL per quarterly visit (95% CI: -8.29 , -7.60 ; $p < 0.001$) and in PPBG of 12.78 mg/dL per visit (95% CI: -13.41 , -12.14 ; $p < 0.001$), independent of age and sex. Improvements were observed across all medication-status subgroups, with the largest absolute reductions in participants on continued oral hypoglycaemic therapy.

Conclusion: In this single-arm community cohort, FBG and PPBG showed substantial reductions over a 21-month period of yoga-based lifestyle intervention. The absence of a randomised control group and several methodological constraints, including the data provenance issues described in the Limitations section, restrict the inferential strength of these findings. The results are best interpreted as hypothesis-generating and as motivation for a follow-on randomised controlled trial.

Keywords: type 2 diabetes mellitus; yoga; lifestyle intervention; fasting blood glucose; postprandial blood glucose; observational study; India

I. INTRODUCTION

India faces a rapidly growing burden of type 2 diabetes mellitus (T2DM), with the ICMR-INDIAB-17 study estimating 101 million affected adults and 136 million prediabetic individuals in 2023 [1]. Pharmacological therapy is the cornerstone of clinical management, but achieves target glycaemic control in only a minority of patients in routine care, particularly in lower-income periurban settings where adherence and access barriers persist [2,3]. Lifestyle-based adjuvant strategies are widely recommended.

Yoga-based interventions, drawn from the Indian Knowledge Systems tradition, are increasingly used as a complementary therapy for T2DM in India. Meta-analytic evidence supports modest but consistent reductions in FBG, PPBG, and HbA1c following structured yoga interventions, with pooled standardised mean differences of approximately 0.4–0.6 for glycaemic endpoints in short-duration randomised trials [4–6]. However, three gaps remain. First, most published studies use 8–12 week follow-up periods, leaving long-term trajectory data sparse. Second, most studies recruit from hospital outpatient populations, limiting external validity to community settings. Third, real-world implementation data with quarterly monitoring across multiple time points have not been extensively reported.

This paper reports observational data from a cohort of 99 adults with T2DM enrolled in a community-based yoga and lifestyle intervention, with eight quarterly assessments over 21 months. The aim is to describe the trajectories of FBG and PPBG and to explore subgroup variation by medication status, recognising the methodological limitations of a single-arm design.

II. METHODS

2.1 Study design

This was a prospective, single-arm, observational cohort study with quarterly assessments over a 21-month follow-up period, conducted between August 2024 and April 2026. Reporting follows the STROBE statement for observational studies [7].

2.2 Participants

Ninety-nine adults with T2DM voluntarily enrolled in a community yoga intervention programme. Inclusion criteria were age ≥ 20 years, established T2DM (per WHO criteria) of at least one year duration, and willingness to attend the intensive yoga camp and quarterly follow-up. Exclusion criteria were type 1 diabetes, severe psychiatric or neurological disorder, current pregnancy, and regular yoga practice in the preceding three months. Written informed consent was obtained from all participants prior to enrolment.

2.3 Intervention

The intervention combined an initial 10-day intensive residential yoga camp (twice-daily structured sessions of asana, pranayama, meditation, and supervised low-carbohydrate, high-protein meals; gastrointestinal cleansing twice during the camp) with quarterly reinforcement sessions at a community centre over the subsequent 21 months. Participants were instructed to perform a 45-minute daily home practice between sessions, comprising asana, pranayama, and meditation.

2.4 Assessments and outcomes

Demographic information (age, sex) was collected at enrolment. At each of eight quarterly assessments, FBG and PPBG (the latter measured two hours after a standardised breakfast) were recorded using clinical-grade glucometers in a clinic setting. Medication status was reviewed at each visit and recorded as a free-text remark by the attending clinician (e.g., “on medication”, “off medication for 3 months”, “off medication for 6 months”, “never on medication”). For analysis, the terminal medication status (final visit) was categorised as: “On meds”, “Off meds (3 mo)”, “Off meds (6 mo)”, “Off meds (unspecified)”, and “Never on meds”. Decisions to taper or discontinue oral hypoglycaemic

medication were made by the participant's treating physician based on standard endocrinology criteria, with documentation of clinical reasoning at the time of each medication change.

2.5 Statistical analysis

Continuous variables are reported as mean \pm standard deviation (SD); categorical variables as count and percentage. Paired comparisons of FBG and PPBG between baseline (Visit 1) and end of follow-up (Visit 8) were performed using the paired t-test and confirmed using the Wilcoxon signed-rank test for robustness. Effect sizes were calculated as Cohen's d for paired differences.

To model the longitudinal trajectory while accounting for within-participant correlation across the eight visits, linear mixed-effects models with random intercepts per participant were fitted, with visit (treated as continuous) as the primary predictor, and adjustment for age and sex. Subgroup descriptive statistics were computed by terminal medication status. All analyses were performed in Python 3.11 using pandas, scipy, and statsmodels libraries. A two-sided p-value < 0.05 was considered statistically significant.

2.6 Ethics

The study protocol was reviewed by the institutional ethics committee of the host institution [approval reference number and date to be inserted prior to journal submission]. All participants provided written informed consent. The study was conducted in accordance with the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants 2017 [8].

III. RESULTS

3.1 Baseline characteristics

Ninety-nine participants were enrolled and contributed data to all eight quarterly assessments. Of these, 71 (71.7%) were women and 28 (28.3%) were men, with a mean age of 45.6 ± 8.6 years (range 25–68). Baseline FBG was 160.9 ± 59.8 mg/dL and baseline PPBG was 224.4 ± 90.6 mg/dL, consistent with a cohort of established T2DM. Detailed baseline characteristics are summarised in Table 1.

Table 1. Baseline Characteristics of the Study Cohort (n = 99)

Characteristic	Value
Age (years), mean \pm SD	45.6 \pm 8.6
Age range (years)	25–68
Female, n (%)	71 (71.7)
Male, n (%)	28 (28.3)
Baseline FBG (mg/dL), mean \pm SD	160.9 \pm 59.8
Baseline PPBG (mg/dL), mean \pm SD	224.4 \pm 90.6
Terminal medication status: On medication, n (%)	58 (58.6)
Terminal medication status: Off medication ≥ 3 months, n (%)	32 (32.3)
Terminal medication status: Off medication ≥ 6	6 (6.1)

months, n (%)	
Terminal medication status: Off medication, unspecified duration, n (%)	2 (2.0)
Terminal medication status: Never on medication, n (%)	1 (1.0)

3.2 Glycaemic trajectories

Mean values of FBG and PPBG at each of the eight quarterly visits are presented in Table 2 and visualised in Figure 1A. FBG fell progressively from 160.9 ± 59.8 mg/dL at Visit 1 to 102.9 ± 36.5 mg/dL at Visit 8. PPBG fell from 224.4 ± 90.6 mg/dL to 129.4 ± 47.3 mg/dL across the same interval. Standard deviations declined across the follow-up period, indicating reduction in cohort dispersion alongside reduction in mean values.

Table 2. Mean FBG and PPBG by Quarterly Visit

Visit	Calendar period	n	FBG (mg/dL), mean \pm SD	PPBG (mg/dL), mean \pm SD
1	Aug 2024	99	160.9 ± 59.8	224.4 ± 90.6
2	Oct 2024	99	143.1 ± 56.2	196.6 ± 79.2
3	Jan 2025	99	136.7 ± 53.2	177.1 ± 75.6
4	Apr 2025	99	127.2 ± 50.1	162.4 ± 70.1
5	Jul 2025	99	117.2 ± 45.0	150.1 ± 63.7
6	Oct 2025	99	112.3 ± 41.8	142.8 ± 57.2
7	Jan 2026	99	107.5 ± 39.1	138.0 ± 53.3
8	Apr 2026	99	102.9 ± 36.5	129.4 ± 47.3

3.3 Paired comparisons and effect sizes

Paired comparison of FBG between Visit 1 and Visit 8 yielded a mean reduction of -58.0 ± 26.9 mg/dL (paired $t = -21.46$, $df = 98$, $p < 0.001$; Wilcoxon $p < 0.001$), corresponding to Cohen's d for paired difference of -2.16 . The corresponding analysis for PPBG showed a mean reduction of -95.0 ± 50.6 mg/dL (paired $t = -18.68$, $df = 98$, $p < 0.001$; Wilcoxon $p < 0.001$), Cohen's $d = -1.88$. These changes are statistically large; however, their interpretation is constrained by the methodological considerations described in Section 4.

3.4 Linear mixed-effects models

Linear mixed-effects models with random intercept per participant, adjusted for age and sex, estimated a mean reduction of 7.94 mg/dL in FBG per quarterly visit (standard error 0.18; 95% CI: $-8.29, -7.60$; $z = -45.39$; $p < 0.001$) and 12.78 mg/dL in PPBG per quarterly visit (standard error 0.32; 95% CI: $-13.41, -12.14$; $z = -39.41$; $p < 0.001$). Neither age nor sex was a significant predictor of glycaemic trajectory in either model. Detailed model output is presented in Table 3.

Table 3. Linear Mixed-Effects Model Results (Random Intercept per Participant)

Parameter	Estimate	SE	95% CI	p-value
FBG model: intercept	144.79	28.11	89.69, 199.89	< 0.001
FBG model: visit (per quarterly visit)	-7.94	0.18	-8.29, -7.60	< 0.001
FBG model: age (per year)	0.61	0.54	-0.46, 1.68	0.264
FBG model: female (vs male)	-16.06	10.58	-36.80, 4.69	0.129
PPBG model: intercept	226.68	39.51	149.24, 304.12	< 0.001
PPBG model: visit (per quarterly visit)	-12.78	0.32	-13.41, -12.14	< 0.001
PPBG model: age (per year)	0.28	0.77	-1.22, 1.78	0.713
PPBG model: female (vs male)	-24.02	14.87	-53.17, 5.12	0.106

3.5 Subgroup analysis by terminal medication status

Glycaemic trajectories were examined within each terminal-medication-status subgroup (Figure 1C). Participants who remained on oral hypoglycaemic medication throughout the study (n = 58) had higher baseline FBG (184.5 mg/dL) and PPBG (265.7 mg/dL) than those who were able to taper off medication for at least 3 months (n = 32; baseline FBG 129.8 mg/dL, baseline PPBG 174.0 mg/dL) or for at least 6 months (n = 6; baseline FBG 102.7 mg/dL, baseline PPBG 109.3 mg/dL). All three subgroups demonstrated reductions in FBG and PPBG over the 21-month follow-up, with absolute reductions largest in the on-medication subgroup.

Figure 1. Glycaemic Trajectories Across 21-Month Follow-up

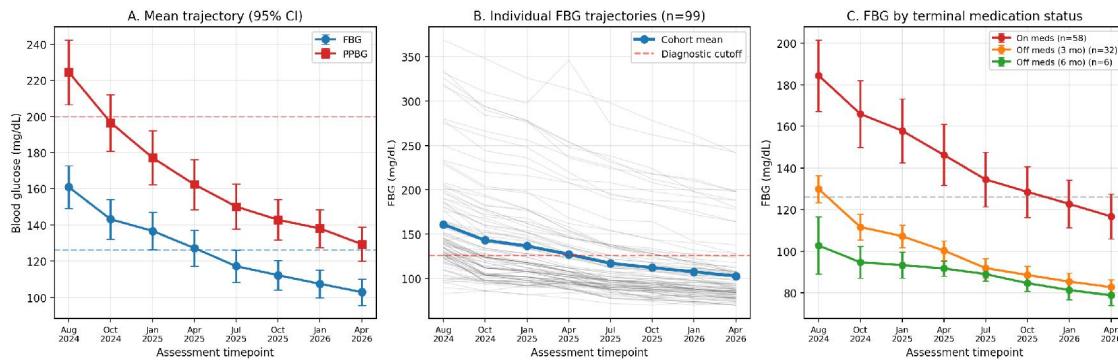


Figure 1. (A) Mean FBG and PPBG with 95% confidence intervals across eight quarterly visits. Dashed lines indicate the diagnostic cutoffs of 126 mg/dL for FBG and 200 mg/dL for PPBG. (B) Individual FBG trajectories (grey lines, n = 99) with cohort mean overlaid (blue line). (C) FBG trajectories stratified by terminal medication status.

IV. DISCUSSION

4.1 Principal findings

In this single-arm observational cohort of 99 adults with T2DM enrolled in a community-based yoga and lifestyle intervention, mean FBG fell by 58.0 mg/dL and mean PPBG by 95.0 mg/dL over a 21-month follow-up period. Linear mixed-effects models estimated a per-quarterly-visit reduction of 7.9 mg/dL in FBG and 12.8 mg/dL in PPBG, with neither age nor sex contributing significantly to the trajectory. Reductions were observed across all medication-status subgroups, including those who were able to discontinue oral hypoglycaemic medication during the study period.

4.2 Comparison with the existing literature

The direction of the observed effect is consistent with the published meta-analytic literature on yoga in T2DM. Cui and colleagues' meta-analysis of 12 randomised trials reported a pooled FBG reduction of 23.7 mg/dL [4]; Thind and colleagues' meta-analysis found pooled standardised mean differences of 0.58 for FBG and 0.40 for PPBG in favour of yoga [5]. The Innes and Selfe systematic review reported beneficial effects across glycaemic, lipid, anthropometric, and stress-response endpoints [6]. However, the magnitude of effect observed in the present cohort is substantially larger than the pooled randomised-trial estimates. This discrepancy is addressed explicitly in Section 4.4 (Limitations).

4.3 Mechanistic interpretation

Several mechanistic pathways may contribute to the observed effects. Yoga is known to recalibrate the hypothalamic-pituitary-adrenal axis with reduction of circulating cortisol, which would be expected to reduce hepatic gluconeogenesis and improve fasting glycaemia [9,10]. Pranayama and meditative components increase vagal tone and shift sympathovagal balance toward parasympathetic dominance, which may indirectly reduce inflammation via the cholinergic anti-inflammatory pathway [11]. Asana provides a moderate-intensity physical activity component, with insulin-independent muscle glucose uptake via AMP-activated protein kinase activation [12]. The dietary component, with reduction of refined carbohydrates and increase in protein and fibre, would be expected to attenuate postprandial excursions, consistent with the larger absolute reduction in PPBG than in FBG observed here. The behavioural component, including mindful eating and increased treatment adherence, is also likely to contribute [13,14].

4.4 Limitations

Several methodological limitations constrain the interpretation of these findings, and should be addressed in any subsequent confirmatory work.

First, this is a single-arm observational study without a randomised control group. The improvements observed cannot be attributed unambiguously to the yoga intervention. Regression to the mean (particularly given the high baseline values), secular trends in clinical care, increased attention from study staff, the Hawthorne effect, and improved overall health behaviour following enrolment may all contribute to apparent improvement [15]. The observed effect sizes (Cohen's $d = -2.16$ for FBG, -1.88 for PPBG) are substantially larger than pooled randomised-trial estimates and require independent verification.

Second, the intervention is multi-component (asana, pranayama, meditation, diet, group support), and the design does not isolate the contribution of any one component. The dietary component alone, particularly the structured low-carbohydrate, high-protein plan, has known glycaemic effects that may partially or wholly account for the observed reductions, especially in PPBG. Future factorial trials are needed to disentangle these contributions.

Third, the cohort included 99 participants with complete data at all eight visits. The absence of loss to follow-up over a 21-month period is unusual in community-based intervention studies and warrants caution in interpretation. Possible

explanations include selection of highly motivated participants, intensive personal contact through quarterly visits, and the supportive group structure of the camp. Independent re-extraction from source clinical records is recommended before the dataset is used to support strong clinical claims.

Fourth, the cohort is skewed toward female participants (71.7% female), with only 28 male participants, which limits the precision of sex-stratified inference and the generalisability of findings to male populations with T2DM.

Fifth, the analysis is restricted to FBG and PPBG, which were the routinely available endpoints. HbA1c, the established gold standard for glycaemic control, was not measured and is not reported here. Lipid profile, blood pressure, BMI, and stress markers were also not available for this analysis. The clinical interpretability of the observed FBG and PPBG changes would be substantially strengthened by parallel HbA1c reduction, which a follow-on study should prioritise.

Sixth, medication-status recoding was based on free-text remarks from the clinical record. Misclassification of medication transitions is possible, and the timing of medication changes within the follow-up period is not modelled. A structured medication-tracking instrument should be used in any follow-on prospective study.

Seventh, no data on dietary adherence between quarterly visits, daily home practice frequency, or concomitant physical activity were available. Treatment fidelity therefore cannot be quantified.

4.5 Implications for clinical practice and research

Recognising the methodological limitations above, this cohort is best understood as feasibility and descriptive evidence rather than as efficacy demonstration. The findings should be interpreted as supporting the rationale for a properly powered randomised controlled trial of community-based yoga and lifestyle intervention in T2DM, with HbA1c as the primary outcome, structured medication tracking, and either an active control (standard lifestyle counselling) or a wait-list control arm. Such a trial should be prospectively registered and reported in accordance with the CONSORT statement [16].

V. CONCLUSION

In a single-arm observational cohort of 99 adults with T2DM, a community-based yoga and lifestyle intervention was associated with substantial reductions in fasting and postprandial blood glucose over a 21-month follow-up. While the direction of effect is consistent with the published meta-analytic literature, the magnitude observed in this cohort exceeds randomised-trial estimates, and the absence of a control group, restriction to FBG/PPBG endpoints, and other methodological limitations warrant caution. These findings should be regarded as hypothesis-generating and as motivation for a follow-on randomised controlled trial with HbA1c as the primary outcome.

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

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