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Effect of a 24-Week Integrated Yoga Programme on Body Mass Index and Fasting Blood Glucose in College Women With Polycystic Ovary Syndrome: A Randomised Controlled Trial

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Abstract

Introduction: Women with polycystic ovary syndrome (PCOS) are prone to obesity and dysglycaemia, and in Asian Indian women, this risk appears at lower body weights. This trial tested whether 24 weeks of integrated yoga reduces fasting blood glucose (FBG) and body mass index (BMI) in college students with PCOS and examined how changes in the two measures relate.

Methods: Students aged 18-25 years with PCOS (n = 131) at a university in Andhra Pradesh, India, took part in this pragmatic, assessor-blinded randomised controlled trial (CTRI/2024/10/075924). They were allocated to usual activity or to supervised yoga combining āsana, prāṇāyāma and meditation, delivered for one hour on five days each week over 24 weeks. BMI and FBG, 2 of 13 registered primary outcomes, were measured at baseline and Week 24 by assessors who did not know the allocation. The main analysis, in women retained to Week 24, was analysis of covariance adjusted for the baseline value; Week-24, change-score, non-parametric, robust, intention-to-treat (multiple imputation) and Bayesian analyses, clinical category analyses and within-group correlations supplemented it.

Results: All 120 women who completed the trial (60 per arm) were analysed. Compared with usual activity, yoga was followed by an adjusted Week-24 BMI 2.74 kg/m² lower (95% confidence interval (CI): -2.97 to -2.51) and FBG 9.41 mg/dL lower (95% CI: -12.31 to -6.52; $p < 0.001$ for both), and every sensitivity analysis, including an intention-to-treat analysis of all 131 randomised women with multiple imputation, gave similar estimates. A BMI reduction of 5% or more occurred in 58 of 60 women given yoga (96.7%) and in none of the controls, whose mean BMI increased by 0.41 kg/m². Of the women whose baseline FBG was 100 mg/dL or higher, 42.9% in the yoga arm and 14% in the control arm were below 100 mg/dL at Week 24 (number needed to treat (NNT): 3.5). Within each group, the correlation between change in FBG and change in BMI was small and non-significant (-0.06 and 0.11).

Conclusions: In young women with PCOS who completed 24 weeks of follow-up, sustained integrated yoga was accompanied by lower BMI and FBG than usual activity. The two changes did not correlate significantly, but this exploratory observation cannot show whether weight change mediated the fall in glucose. Because the effects were large and glycaemia rested on one fasting sample, the findings need independent confirmation with fuller glycaemic and body composition measures.

Categories: Therapeutics, Integrative/Complementary Medicine, Endocrinology/Diabetes/Metabolism
Keywords: body mass index, fasting blood glucose, impaired fasting glucose, lifestyle intervention, polycystic ovary syndrome, pranayama, randomised controlled, yoga

Introduction

Polycystic ovary syndrome (PCOS) is thought to occur in 10%-13% of women during their reproductive years, and its metabolic consequences are now considered as important as its reproductive features [1]. Its metabolic core is insulin resistance. Pooled studies using the euglycaemic-hyperinsulinaemic clamp, the reference technique for measuring insulin action, show that insulin sensitivity in PCOS averages 27% below that of control women and falls further as body mass index (BMI) rises [2]. The deficit matters clinically: compared with unaffected women, those with PCOS carry raised odds of impaired glucose tolerance (odds ratio (OR): 2.48) and type 2 diabetes (OR: 4.43), and the excess remains in studies matched for BMI [3]. South Asia warrants particular attention because cardiometabolic risk climbs at lower adiposity among South Asians than Europeans; Asian Indian adults are therefore classed as overweight from a BMI of 23 kg/m² and obese from 25 kg/m², rather than from 25 and 30 kg/m² [4].

Overall adiposity and fasting glycaemia are linked yet distinct therapeutic targets. BMI, the usual index of general adiposity, scales body mass to height. On the other hand, fasting blood glucose (FBG) reflects mainly the liver, through its basal output of glucose and its responsiveness to insulin, while skeletal muscle handles

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most glucose disposal after meals [5]. Because insulin resistance in PCOS exceeds what excess weight alone accounts for [2,3], an intervention need not shift the two measures in parallel. Glycaemia can improve before any appreciable weight change, and weight can fall with no glycaemic gain, so a study that records only one measure says little about the other. An earlier report from this trial, which covered total cholesterol together with waist-hip ratio, rested on the same reasoning that body size and blood markers capture different aspects of cardiometabolic risk [6].

International guidance advises lifestyle change for every woman with PCOS and links weight management to clinically meaningful benefit in those with excess weight [1]. In India, yoga provides such change in a familiar cultural form, uniting bodily, breathing and attentional practice in one discipline. Patanjali's Yoga Sutras list posture (āsana), breath regulation (prāṇāyāma) and meditation (dhyāna) among the limbs of yoga [7], and according to the Hatha Yoga Pradipika, āsana confers steadiness, health and lightness of body (1.17) [8]. Both texts stress abhyāsa: Patanjali teaches that practice takes firm root only when it is continued for a long time, without break and with commitment (Yoga Sutra 1.14) [7], and the Hatha Yoga Pradipika credits success to practice, not to theory (1.65-1.66) [8]. Physiologically, such a programme brings together three components that could plausibly affect metabolism: repeated muscle work, slow controlled breathing and the training of attention and reactivity to stress.

Most research on yoga and glycaemia comes from type 2 diabetes, where pooled data from 23 studies and 2,473 participants show better FBG (standardised effect: 0.57) and glycated haemoglobin [9]. Evidence in PCOS is sparser. Of 11 studies (515 participants) identified by a systematic review, only 2 randomised trials were suitable for pooling; together, they showed FBG 0.22 mmol/L lower with yoga (95% confidence interval (CI): 0.01 to 0.44 lower), with low certainty [10]. The main randomised trial enrolled 90 adolescent girls and found that, over 12 weeks, yoga improved FBG, fasting insulin and insulin resistance more than conventional exercise did [11]. Effects on weight are small: lifestyle programmes in PCOS lower BMI by only 0.34 kg/m² on average [12], and pooled yoga trials show no overall BMI effect, with a benefit in overweight and obese participants that proved sensitive to selection bias [13]. Three gaps therefore remain. Randomised trials in PCOS are few, small and brief; young adult women outside infertility clinics are seldom studied; and few trials report outcomes that clinicians can readily interpret, such as crossing glycaemic thresholds, or relate glycaemic change to weight change.

In this randomised controlled trial, college women with PCOS attended supervised yoga five days a week for 24 weeks without changing the rest of their daily routine. The primary objective of this report was to estimate the effect of the programme on BMI and FBG at Week 24, adjusted for the baseline value, in women who completed follow-up, with an intention-to-treat analysis of all randomised women as a check. The secondary objectives were to describe shifts across Asian Indian BMI categories and American Diabetes Association glycaemic categories, including the proportion of women whose BMI fell by 5% or more. The exploratory objective was to relate the change in FBG to the change in BMI.

Materials And Methods

Study design, setting and ethics

This pragmatic trial randomised participants to two parallel arms, with outcome assessors blinded to allocation. All recruitment, screening, yoga sessions and measurements took place on the campus of KL Deemed to be University in Vaddeswaram (Guntur District, Andhra Pradesh, India), and academic oversight came from the Meenakshi Academy of Higher Education and Research (MAHER), Chennai. Enrolment ran from July 8-21, 2025, sessions began on July 28, 2025, and the last Week-24 assessments took place in January 2026.

MAHER's Institutional Human Ethics Committee gave approval on April 23, 2024, under study reference MAHER/IEC/PHD/15/JAN 24 (committee registrations: DHR, EC/NEW/INST/2023/3553; CDSCO, ECR/1906/INST/TN/2023), and written permission from KL Deemed to be University preceded recruitment. Registration with the Clinical Trials Registry-India (CTRI/2024/10/075924) was completed on October 25, 2024, ahead of the first enrolment. The study conformed to the Declaration of Helsinki, and this report follows the CONSORT 2025 [14]. Signed informed consent was obtained from every participant at the start of the study, before enrolment and before any measurement.

In brief, women aged 18-25 years with PCOS confirmed by the Rotterdam criteria were eligible. They were randomised to supervised integrated yoga (1 hour a day, 5 days a week for 24 weeks) or to their usual activity, and BMI and FBG were measured at baseline and at Week 24. The full eligibility criteria, intervention and outcome measures are given below and in the registered protocol, which the corresponding author can supply on request; a stand-alone statistical analysis plan was not written. The design, intervention and participant flow are also described in the two earlier reports from the trial [6,15]. Participants had no role in choosing the research question or designing the study, although their comments on session times helped fit the sessions around the academic timetable.

Trial outcomes and the scope of this report

The trial registered 13 primary outcomes, all measured at baseline and Week 24. This article reports only BMI and FBG. Waist-hip ratio with total cholesterol [6] and the high-density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol fractions [15] have been published separately, and the remaining outcomes will be reported elsewhere.

Participants

Participants were drawn from roughly 8,000 women studying at the university. Students first attended PCOS awareness seminars, where they completed screening questionnaires; those interested then joined an orientation session that demonstrated the practices and set out the time commitment, timetable and safety arrangements; finally, each candidate's earlier medical records were reviewed. On the basis of these questionnaires and records, the first author (VVS) decided eligibility and enrolled eligible students.

Each prior PCOS diagnosis was checked against the Rotterdam criteria, which call for at least two of ovulatory dysfunction, androgen excess (clinical or biochemical) and polycystic ovaries on ultrasound once other explanations are ruled out [16]. Oligomenorrhoea meant cycles lasting over 35 days or a total of under 8 cycles a year. Androgen excess was accepted if it appeared in earlier medical notes, and polycystic morphology required a transabdominal ultrasound report from the treating gynaecologist.

Students aged 18-25 years with a verified PCOS diagnosis were eligible if they could do the practices safely and gave written consent. Reasons for exclusion were other endocrine disease (thyroid disorder, Cushing's syndrome or congenital adrenal hyperplasia); type 1 diabetes or a known cardiovascular diagnosis; current use, or use in the previous three months, of insulin-sensitising, lipid-lowering, anti-androgen or hormonal contraceptive medication; regular structured exercise or yoga at any time in the previous six months; and any medical reason to avoid exercise. In keeping with the pragmatic design, participants received no instructions about diet, sleep, daily routine, study load or activity outside the sessions.

Randomisation, allocation concealment and blinding

Allocation took place only after each woman's baseline measurements were taken. A research assistant with no role in recruitment, the sessions or the assessments produced the random sequence in R (version 4.2.1), using 1:1 allocation in blocks of four with no stratification. With 131 participants, the final block was incomplete, so the Yoga group received 65 women and the Control group 66. This assistant also kept the sequence in opaque, sealed, consecutively numbered envelopes and opened the next one only when a participant had finished her baseline assessment; the first author, who enrolled participants, therefore had no advance knowledge of allocation. Blinding was impossible for the participants and the yoga instructor, but the staff who took anthropometric measurements, drew blood, ran the laboratory tests and analysed the data did not know group membership.

Intervention

The programme was designed as integrated yoga, not as an exercise routine given yogic names. Its dose applies abhyāsa, the principle of long and uninterrupted practice (Yoga Sutra 1.12-1.14) [7]: 120 supervised 1-hour sessions, held on 5 days a week across 24 weeks. Every session progressed from posture (āsana) to breath regulation (prāṇāyāma) and then meditation (dhyāna), three of Patanjali's eight limbs [7].

The first author taught every session in person, indoors on campus, and brings a master's degree in yoga, formal instructor training and over two decades of teaching. The order of practices, their duration, repetitions, holds and breath counts were fixed for all 24 weeks by a written protocol with a checklist for each session, and within those limits, the instructor tailored intensity and progression to each woman. Attendance was taken at each session, and a missed session could be made up in an extra slot after hours or close to the scheduled time. Attendance of 95% or more (114 of the 120 sessions) served as a monitoring target only; nobody was excluded for attending less. The written protocol, including the session checklist, is available from the corresponding author on request. Table *1* summarises the components, their roots in the classical texts and the physiological routes by which each might work.

Component	Duration (minutes)	Practices	Traditional basis	Proposed physiological pathway (hypothesised; not measured)
Loosening and centring	12	Sukshma Vyayama; Om chanting	Recitation of Om (*praṇava*), Yoga Sutra 1.27–1.28 [7]	Joint mobilisation; a prolonged exhalation that begins the shift towards slow breathing
Dynamic warm-up	8	Surya Namaskar	Devotional sequence of the later hatha tradition; not described in the classical texts	Moderate-to-vigorous whole-body muscular work; contraction-stimulated glucose uptake by skeletal muscle
Targeted postures	20	Prasarita Padottanasana, Utkata Konasana, Malasana, Vakrasana, Mandukasana (classical seated form), Baddha Konasana, Upavistha Konasana, Janu Sirsasana, Setu Bandhasana, Supta Konasana, Bhujangasana, Dhanurasana	Āsana brings steadiness, health and lightness of body (Hatha Yoga Pradipika 1.17) [8]; Bhujangasana is said to increase the bodily fire (Gheranda Samhita, chapter 2) [17]; Vakrasana is a simplified form of Matsyendrasana (Hatha Yoga Pradipika 1.26-1.27) [8]	Sustained isometric holds of large lower-limb muscle groups (standing and squatting postures); prone postures with abdominal contact and a seated twist that change intra-abdominal pressure
Mudras	5	Viparita Karani; Yoni Mudra	Viparita Karani is among the mudras of the Hatha Yoga Pradipika (chapter 3) [8]; Yoni Mudra is described in the Gheranda Samhita (chapter 3) [17]	Inverted, passive posture; relaxation
Prāṇāyāma	8	Bhramari; Nadi Shodhana	Breath regulation, Yoga Sutra 2.49 [7]; alternate-nostril breathing purifies the channels (*nāḍī*) and correct practice wards off disease (Hatha Yoga Pradipika 2.7-2.10, 2.16-2.17) [8]	Slow breathing with a prolonged exhalation; greater vagal and lower sympathetic activity
Meditation and rest	7	Breath-watching meditation; Quick Relaxation Technique	Meditation (*dhyāna*), Yoga Sutra 3.2 [7]	Lower stress reactivity, with less cortisol and catecholamine output

TABLE 1: Components of the yoga programme, their traditional basis and proposed physiological pathways

Women in the Control group carried on with their normal daily and academic lives and started no medication, and the study offered them nothing further: no exercise sessions, no advice specific to PCOS and no counselling.

Any injury to muscles or joints, fainting, cardiovascular event or other physical or psychological harm that occurred in, or could be traced to, a session counted as an adverse event. Participants reported such events themselves and through questionnaires throughout the 24 weeks.

Outcome measures

Body Mass Index

At baseline and Week 24, trained anthropometrists who did not know the allocation recorded height and weight. Participants were weighed barefoot in light clothes on a standard hospital scale (precision: 0.2 kg), and standing height was read to 0.1 cm from a tape fixed to the wall. BMI equals body weight (kg) over height (m) squared. Categories followed the Asian Indian consensus cut-offs [4]: below 23.0 kg/m², normal; 23.0-24.9 kg/m², overweight; and 25.0 kg/m² or more, obese. We also report values of 30 kg/m² or more, and no participant was underweight (below 18.5 kg/m²). A weight loss response was defined as a fall in BMI of 5% or more from baseline.

Fasting Blood Glucose

Venous samples were drawn on campus at the baseline visit and at the end-of-study visit (Week 24), after participants had fasted overnight for 12 hours. Lalitha Super Speciality Hospital in Guntur, an external clinical laboratory with ISO certification, measured glucose enzymatically by the glucose oxidase-peroxidase (GOD-POD) method, using an identical sampling procedure, laboratory and assay for both groups and both visits. Glucose is given in mg/dL (dividing by 18.016 gives mmol/L). Following the American Diabetes Association [18], values under 100 mg/dL were classed as normal, 100-125 mg/dL as impaired fasting glucose and 126 mg/dL or more as in the diabetes range. Here, impaired fasting glucose means 100-125 mg/dL alone, whereas elevated FBG means any value of 100 mg/dL or more. One fasting result of 126 mg/dL or more is not enough to diagnose diabetes without a confirmatory test [18]; the 5 women with such a baseline result (Yoga: 2, Control: 3) stayed in the analysis, and a sensitivity analysis without them is also reported.

Sample size

A single sample size calculation (G*Power, version 3.1.9.7) served the whole trial. It used HDL cholesterol, the registered outcome expected to change least: detecting a 4.2 mg/dL difference (pooled SD: 6.8 mg/dL; Cohen's d = 0.62) with 85% power and a two-sided α of 0.05 required 49 women per group, or 98 in total. To allow for loss to follow-up, all 131 eligible women were randomised; after 11 withdrawals, 120 (60 per group) were analysed. With 60 women in each group, a standardised difference of 0.52 (α = 0.05) or 0.57 (α = 0.025) in BMI or FBG could be detected with 80% power.

Statistical analysis

All computations used Python 3.13 with the SciPy 1.18 and statsmodels 0.15 libraries, and Matplotlib 3.11 produced the figures. All statistical tests were two-tailed, and a p-value below 0.05 was considered statistically significant. Between-group differences are Yoga minus Control, and change is the Week-24 value minus the baseline value.

Analysis Population and Estimand

The 120 women (60 in each group) who stayed in the trial until Week 24 had complete data at both visits and formed the primary analysis set; none of the 11 who withdrew had Week-24 measurements. The quantity estimated was the baseline-adjusted difference in mean Week-24 BMI and FBG between women allocated to yoga and women allocated to usual activity, among those who completed follow-up. An intention-to-treat analysis, in which all 131 randomised women were analysed in the groups to which they were allocated, is described below.

Description and Distribution

We summarise continuous data as mean ± standard deviation (SD) with 95% CIs. Distributions were checked for normality with Q-Q plots, skewness and kurtosis, and formally with the Shapiro-Wilk test and the Lilliefors-corrected Kolmogorov-Smirnov test. BMI and FBG at both visits, and their changes, were normally distributed in both groups (Shapiro-Wilk, $p > 0.05$), except for the change in FBG in the Control group ($p = 0.023$); age was not normally distributed ($p = 0.003$ in each group). Parametric tests were therefore the main tests, and rank-based tests were run alongside them only as a sensitivity check on these departures from normality; they were not used to choose between results.

Baseline Comparison

At entry, groups were compared on age, BMI and FBG by independent-samples t tests and on categories by the χ^2 test. Since some women withdrew after allocation, these tests show only how alike the analysed groups were at the start, not whether randomisation worked.

Primary Analysis

For each outcome, an analysis of covariance (ANCOVA) modelled the Week-24 value on group, adjusting for the baseline value of the same outcome. We chose ANCOVA as the primary method for this report because it is the preferred approach when a trial measures an outcome both before and after treatment [19]; the methods that served as primary analyses in the trial's earlier reports appear here as consistency checks. We report the adjusted difference in means with its 95% CI, the F statistic and p-value, and partial η^2 with its 95% CI, and we standardised the adjusted difference by dividing it by the pooled SD at baseline. Model checks covered residual normality, equality of variances by Levene's test, parallel regression slopes (tested by adding a group × baseline term) and influence (Cook's distance and studentised deleted residuals).

Consistency Analyses

To see whether the conclusion depended on the analytic approach, we also compared groups on their Week-24 values (Student and Welch t tests), on their change scores (independent-samples t test) and in a 2 × 2

mixed-design analysis of variance (ANOVA) in which group varied between participants and time within them. Effect sizes are Cohen's d and Hedges' g with 95% CIs. Within each group, change was assessed by paired t tests, with dz (mean change over its SD) as the effect size; the Wilcoxon signed-rank test was added only as a sensitivity check, because the change in FBG in the Control group was not normally distributed. These within-group results are descriptive and were not used to judge the effect of the intervention.

Robustness and Sensitivity

The ANCOVA result was checked with heteroscedasticity-consistent (HC3) standard errors; a bias-corrected and accelerated (BCa) bootstrap CI (10,000 resamples, stratified by group); the Mann-Whitney U test on change scores, with the Hodges-Lehmann estimate of the shift; adjustment for age; exclusion of observations with a studentised deleted residual beyond ±3; and exclusion of the participants with baseline FBG of 126 mg/dL or above.

Intention-to-Treat Analysis and Missing Data

To analyse all 131 randomised participants in the groups to which they were allocated (intention to treat), the change from baseline of each of the 11 who withdrew was multiply imputed (200 imputations) from the observed distribution of change in the participant's own group, assuming that values were missing at random (MAR). Each completed dataset was analysed by a t test on change scores, and the results were pooled by Rubin's rules. Departures from that assumption were examined in two ways. A tipping-point analysis shifted the imputed changes of Yoga group withdrawals in the unfavourable direction until the 95% CI reached zero, and a conservative cross-group scenario assigned each Yoga group withdrawal the mean change of the Control group and each Control group withdrawal the mean change of the Yoga group. Details are given in Appendix 1.

Multiplicity

BMI and FBG were tested as two outcomes with Holm's procedure.

Clinical Categories and Responders

Within each group, shifts between categories were tested using the exact McNemar test (two categories) or the Stuart-Maxwell test (three or more). At Week 24, the proportion of women in each category, and the proportion meeting each responder definition, were compared between the Yoga and Control groups by Fisher's exact test and expressed as a risk difference (Yoga minus Control) with a Newcombe hybrid score 95% CI, a risk ratio (Yoga to Control) with its 95% CI, a risk ratio with its 95% CI and a number needed to treat (NNT), calculated as one divided by the risk difference. Three responder definitions were used: BMI lower by 5% or more; a return below 100 mg/dL in women whose baseline FBG was elevated (⩾100 mg/dL), repeated for the subgroup with impaired fasting glucose only (100-125 mg/dL); and a rise to ⩾100 mg/dL in women starting below 100 mg/dL. The last definition captures worsening, which can occur without treatment because dysglycaemia is more common in PCOS [3] and insulin sensitivity falls further as BMI rises [2].

Correlation Analyses

Associations were examined within each group with Pearson's ρ (95% CI by Fisher's z transformation). Spearman's ρ is shown alongside only as a sensitivity check, because age and the change in FBG in the Control group were not normally distributed; conclusions rest on Pearson's ρ. The pairs examined were baseline BMI with baseline FBG; Week-24 BMI with Week-24 FBG; the change in BMI with the change in FBG, both unadjusted and as a partial correlation controlling for the two baseline values; age with each change; and the baseline level of each outcome with its change. The last association was estimated by Oldham's method, which correlates the mean of the baseline and Week-24 values with the change and so avoids the spurious negative correlation that regression to the mean produces. Correlations were compared between groups with Fisher's z test. Across all participants, the association between changes was estimated only as a partial correlation controlling for group, because a pooled correlation without that adjustment would mainly reflect the difference between the groups rather than an association within individuals.

Exploratory Regression and Effect Modification

Within each group, the change in FBG was regressed on the change in BMI, adjusting for baseline FBG. The FBG ANCOVA was also refitted with the change in BMI as an additional covariate. Because BMI change occurred after randomisation, this model does not estimate a mediated effect; it shows only how far group and BMI change can be separated in these data. Effect modification was tested by adding interaction terms to the ANCOVA models: group × baseline glycaemic status (below 100 versus 100 mg/dL or above), group × obesity class (BMI below 30 versus 30 kg/m² or above) and group × age.

Bayesian Analysis

The ANCOVA model was refitted in a Bayesian framework under a reference prior and a sceptical prior; the specification and results are given in Appendix 2.

The exploratory analyses were not adjusted for multiplicity and are interpreted as hypothesis-generating.

Results

Participant flow and baseline characteristics

Screening covered 310 students. Of these, 179 failed at least one eligibility criterion, and the remaining 131 were randomised, 65 to yoga and 66 to the control condition (Figure *1*). The number excluded under each criterion was not recorded; every exclusion was for ineligibility, and all eligible students agreed to take part. Before the Week-24 visit, 11 women withdrew (Yoga: 5 of 65, 7.7%; Control: 6 of 66, 9.1%), and no withdrawal was attributed to the intervention. Two women from each group left during weeks 1-8 because lectures clashed with sessions or scheduling proved difficult; one Yoga and three Control participants left during weeks 9-16 because of mid-semester examinations, travel between semesters and project deadlines; and two Yoga participants and one Control participant left during weeks 17-24 because of illness unrelated to the study or relocation for internships.

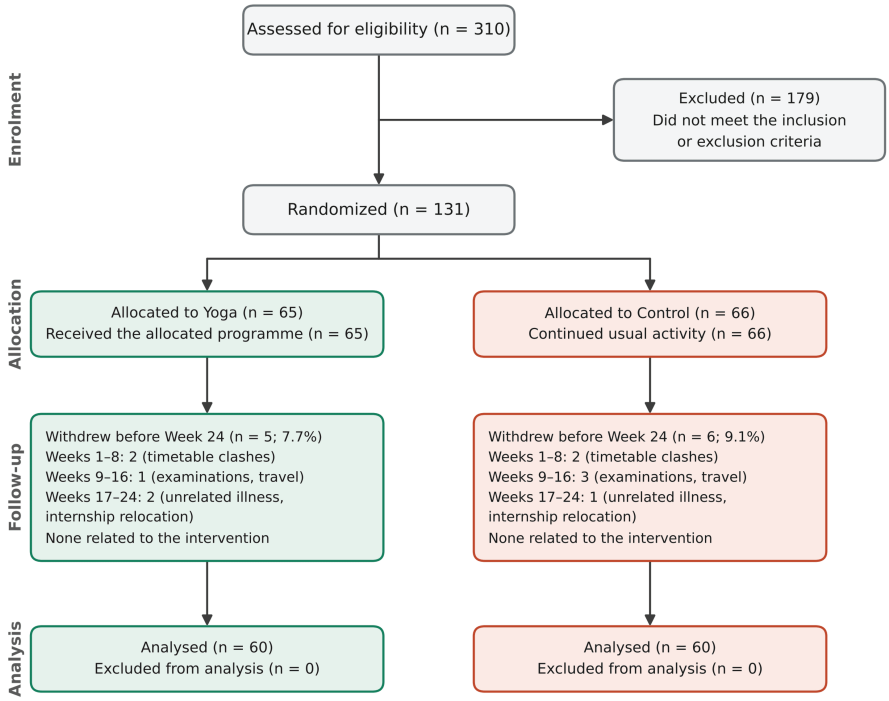


FIGURE 1: Flow of participants through each stage of the trial (CONSORT 2025)

The 120 women who completed follow-up (60 per group) had no missing values and make up the primary analysis set. Nobody reported an adverse event.

At entry, the groups were well matched (Table *2*), with mean ages of 20.95 ± 2.10 years (Yoga) and 21.20 ± 2.18 years (Control). Nearly all women exceeded the Asian Indian obesity cut-off of 25 kg/m² (Yoga: 59, Control: 54), most had impaired fasting glucose (Yoga: 47, Control: 40) and mean FBG in each group was about 6.0 mmol/L. Baseline differences were small and non-significant for age ($p = 0.524$), BMI ($p = 0.246$) and FBG ($p = 0.596$), and for the distributions across BMI categories ($p = 0.269$) and glycaemic categories ($p = 0.359$).

Characteristic	Statistic	Yoga group (n = 60)	Control group (n = 60)
Age (years)	Mean	20.95	21.20
Age (years)	SD	2.10	2.18
BMI (kg/m²)	Mean	28.21	27.73
BMI (kg/m²)	SD	2.00	2.42
Normal BMI, <23 kg/m²	n	0	1
Normal BMI, <23 kg/m²	%	0.0	1.7
Overweight, 23.0-24.9 kg/m²	n	1	5
Overweight, 23.0-24.9 kg/m²	%	1.7	8.3
Obese, ≥25 kg/m²	n	59	54
Obese, ≥25 kg/m²	%	98.3	90.0
BMI ≥ 30 kg/m² (subset of obese)	n	9	9
BMI ≥ 30 kg/m² (subset of obese)	%	15	15
FBG (mg/dL)	Mean	107.85	106.83
FBG (mg/dL)	SD	9.43	11.56
FBG (mmol/L)	Mean	5.99	5.93
FBG (mmol/L)	SD	0.52	0.64
Normal FBG, <100 mg/dL	n	11	17
Normal FBG, <100 mg/dL	%	18.3	28.3
Impaired fasting glucose, 100-125 mg/dL	n	47	40
Impaired fasting glucose, 100-125 mg/dL	%	78.3	66.7
Diabetes range, ≥126 mg/dL	n	2	3
Diabetes range, ≥126 mg/dL	%	3.3	5.0

TABLE 2: Baseline characteristics of the analysed participants

Data are mean ± SD or number (%). Categories use the Asian Indian BMI cut-offs [4] and the American Diabetes Association FBG cut-offs [18].

BMI: body mass index, FBG: fasting blood glucose, SD: standard deviation

Effect of the programme on BMI and FBG

BMI in the Yoga group declined from a mean of 28.21 to 25.92 kg/m² (change: -2.29 kg/m², 95% CI: -2.44 to -2.14; -8.1%), whereas in the Control group, it increased from 27.73 to 28.15 kg/m² (change: +0.41, 95% CI: 0.23 to 0.60; +1.5%). With baseline BMI taken into account, the Yoga group's Week-24 mean was lower by 2.74 kg/m² (95% CI: -2.97 to -2.51; $F(1, 117) = 570.9$; $p < 0.001$; partial η^2 : 0.83, 95% CI: 0.78 to 0.86), a standardised difference of -1.23 (95% CI: -1.62 to -0.84; Table *3*, Figure *2*).

Quantity	BMI (kg/m²), Yoga	BMI (kg/m²), Control	FBG (mg/dL), Yoga	FBG (mg/dL), Control
Baseline, mean	28.21	27.73	107.85	106.83
Baseline, SD	2.00	2.42	9.43	11.56
Week 24, mean	25.92	28.15	98.58	107.34
Week 24, SD	2.11	2.77	9.62	11.15
Change, mean	-2.29	+0.41	-9.28	+0.51
Change, 95% CI lower limit	-2.44	0.23	-11.31	-1.99
Change, 95% CI upper limit	-2.14	0.60	-7.24	3.01
Change, % of baseline mean	-8.1	+1.5	-8.6	+0.5
Paired t	-31.19	4.53	-9.12	0.41
Paired t test, p	<0.001	<0.001	<0.001	0.685
Effect size, dz	-4.03	0.58	-1.18	0.05
Adjusted Week-24 mean	25.66	28.40	98.25	107.67
Adjusted mean, 95% CI lower limit	25.50	28.24	96.21	105.62
Adjusted mean, 95% CI upper limit	25.82	28.56	100.29	109.71

TABLE 3: BMI and FBG at baseline and Week 24, within-group change and between-group differences

Note: Group rows show mean ± SD or mean (95% CI). Adjusted means come from ANCOVA with the baseline value as covariate, evaluated at the overall baseline mean. In the difference rows, the baseline and Week-24 columns come from independent-samples t tests, the change column from a t test on change scores and the last column from ANCOVA, the primary analysis (bold).

ANCOVA: analysis of covariance, BMI: body mass index, CI: confidence interval, FBG: fasting blood glucose, SD: standard deviation

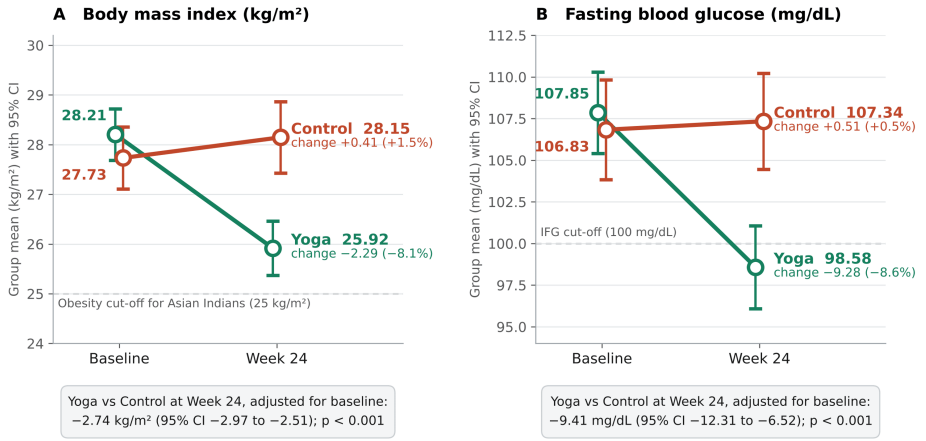


FIGURE 2: Group means for BMI (A) and FBG (B) at baseline and Week 24, with 95% CIs

Labels on the right show each group's Week-24 mean and its change from baseline, and the box beneath each panel gives the baseline-adjusted (ANCOVA) difference between groups. Dashed lines mark 25 kg/m², the obesity cut-off for Asian Indian adults, and 100 mg/dL, the lower limit of IFG.

ANCOVA: analysis of covariance, BMI: body mass index, CI: confidence interval, FBG: fasting blood glucose, IFG: impaired fasting glucose

FBG in the Yoga group dropped from a mean of 107.85 to 98.58 mg/dL (change: -9.28 mg/dL, 95% CI: -11.31

to -7.24; -8.6%) but barely moved in the Control group (106.83 to 107.34 mg/dL; change: +0.51, 95% CI: -1.99 to 3.01). Adjusted for baseline, Week-24 FBG differed by -9.41 mg/dL between groups (95% CI: -12.31 to -6.52; F(1, 117) = 41.6; p < 0.001; partial η^2 : 0.26, 95% CI: 0.14 to 0.38), or -0.52 mmol/L (-0.68 to -0.36), with a standardised difference of -0.89 (-1.27 to -0.52). Both results remained significant after Holm correction for the two outcomes (p < 0.001 for both).

Within the Yoga group, BMI decreased in all 60 participants (paired t(59) = -31.19, p < 0.001, d_z = -4.03) and FBG decreased in 55 (t(59) = -9.12, p < 0.001, d_z = -1.18). Within the Control group, BMI increased in 44 of 60 participants (t(59) = 4.53, p < 0.001, d_z = 0.58), and FBG did not change (t(59) = 0.41, p = 0.685, d_z = 0.05). Wilcoxon signed-rank tests, run as a sensitivity check, gave the same conclusions.

Consistency, robustness and sensitivity analyses

The conclusion did not depend on the analytic frame (Table 3 and Table 4).

Outcome	Analysis	Difference, Yoga - Control	95% CI lower limit	95% CI upper limit	t	p	Cohen's d	d, 95% CI lower limit	d, 95% CI upper limit
BMI (kg/m²)	Baseline, t test	+0.47	-0.33	1.28	1.17	0.246	0.21	-0.15	0.57
BMI (kg/m²)	Week 24, unadjusted t test	-2.23	-3.12	-1.34	-4.96	<0.001	-0.91	-1.28	-0.53
BMI (kg/m²)	Change score, t test	-2.70	-2.94	-2.47	-23.05	<0.001	-4.21	-4.85	-3.57
BMI (kg/m²)	Week 24 adjusted for baseline, ANCOVA (primary)	-2.74	-2.97	-2.51	-23.89	<0.001	-1.23	-1.62	-0.84
FBG (mg/dL)	Baseline, t test	+1.02	-2.79	4.84	0.53	0.596	0.10	-0.26	0.46
FBG (mg/dL)	Week 24, unadjusted t test	-8.76	-12.53	-5.00	-4.61	<0.001	-0.84	-1.21	-0.47
FBG (mg/dL)	Change score, t test	-9.79	-12.98	-6.59	-6.07	<0.001	-1.11	-1.49	-0.72
FBG (mg/dL)	Week 24 adjusted for baseline, ANCOVA (primary)	-9.41	-12.31	-6.52	-6.45	<0.001	-0.89	-1.27	-0.52

TABLE 4: Between-group comparison under alternative analytic frames

Note: Values in brackets are 95% CIs; d, Cohen's d; g, Hedges' g. t(118) and F(1, 118) give each test statistic with its degrees of freedom. All p-values were below 0.001, and Welch t tests gave the same t values. For two groups and two time points, the interaction F equals the square of the change-score t.

ANCOVA: analysis of covariance, BMI: body mass index, CI: confidence interval

The ANCOVA assumptions were met. Residuals were normally distributed (Shapiro-Wilk p = 0.795 for BMI and 0.763 for FBG), variances were homogeneous (Levene's test p = 0.378 and 0.316) and no observation was influential (maximum Cook's distance: 0.065 and 0.167).

The group × baseline interaction was absent for FBG (p = 0.694) and borderline for BMI (p = 0.051). The BMI benefit tended to be larger at higher baseline BMI, by -0.10 kg/m² per 1 kg/m² of baseline BMI (95% CI: -0.21 to 0.00), and the effect at the mean baseline value was unchanged (-2.73 kg/m²).

Every robustness and sensitivity analysis supported the primary result (Figure 3; details in Appendix 1). Estimates with robust and bootstrap CIs, after adjustment for age, after exclusion of the one outlying FBG observation and after exclusion of the five participants with baseline FBG of 126 mg/dL or above differed from the primary estimate by no more than 0.01 kg/m² for BMI and 0.41 mg/dL for FBG. The Mann-Whitney test on change scores gave the same conclusion (Hodges-Lehmann shift: -2.65 kg/m² and -8.70 mg/dL; both p < 0.001).

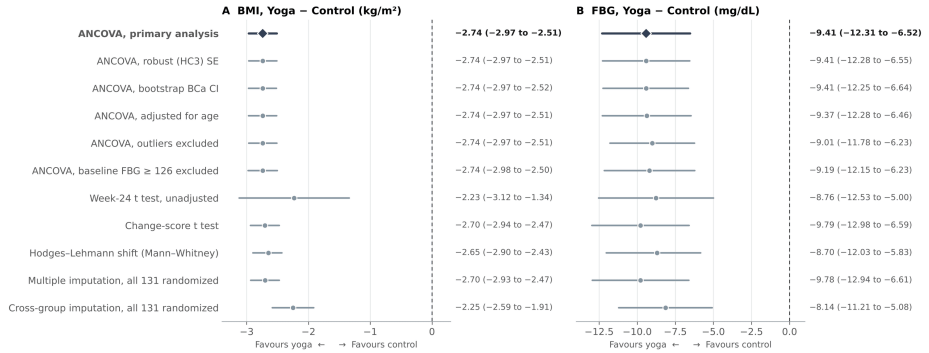


FIGURE 3: Between-group difference (Yoga - Control) in BMI (A) and FBG (B), with 95% CIs, for the primary analysis (diamond) and the consistency, sensitivity and missing-data analyses (circles)

BMI had no outlying observations, so its outlier-excluded row repeats the primary estimate. The Hodges-Lehmann shift is the median of all pairwise differences in change scores. The two imputation rows include all 131 randomised participants and compare change scores.

ANCOVA: analysis of covariance, BCa: bias-corrected and accelerated, BMI: body mass index, CI: confidence interval, HC3: heteroscedasticity-consistent standard error, FBG: fasting blood glucose

In the intention-to-treat analysis of all 131 randomised participants, with multiple imputation for the 11 withdrawals, the difference in mean change was -2.70 kg/m² for BMI (95% CI: -2.93 to -2.47) and -9.78 mg/dL for FBG (95% CI: -12.94 to -6.61), close to the complete-case change-score estimates (-2.70 kg/m² and -9.79 mg/dL). In the tipping-point analysis, the BMI difference lost significance only if the five Yoga-group withdrawals had gained an average of about 16.7 kg/m² over 24 weeks, and the FBG difference only if their FBG had risen by an average of about 53 mg/dL; Yoga participants who completed the trial lost 2.29 kg/m² and 9.28 mg/dL on average. The conservative cross-group scenario also retained both effects (BMI: -2.25 kg/m², 95% CI: -2.59 to -1.91; FBG: -8.14 mg/dL, 95% CI: -11.21 to -5.08; both $p < 0.001$). A Bayesian re-analysis under reference and sceptical priors gave the same estimates and conclusions (Appendix 2).

Clinical categories and responders

Participants in the Yoga group moved out of the high-risk categories, whereas the Control group stayed where it started or moved upward (Table *5*, Figure *4*).

Outcome	Yoga, n	Yoga, N	Control, n	Control, N	Risk difference (percentage points)	95% CI lower limit	95% CI upper limit	NNT	p
BMI ≥ 25 kg/m² at Week 24	37	60	53	60	-26.7	-40.6	-11.3	3.8	0.001
BMI ≥ 30 kg/m² at Week 24	4	60	14	60	-16.7	-29.4	-3.8	6.0	0.019
BMI reduction ≥ 5% from baseline	58	60	0	60	96.7	86.6	99.1	1.0	<0.001
FBG ≥ 100 mg/dL at Week 24	28	60	44	60	-26.7	-42.0	-9.1	3.8	0.005
FBG ≥ 126 mg/dL at Week 24	0	60	3	60	-5.0	-13.7	1.9	Not estimable	0.244
FBG < 100 mg/dL at Week 24, among those with baseline FBG ≥ 100 mg/dL	21	49	6	43	28.9	10.4	44.6	3.5	0.003
FBG < 100 mg/dL at Week 24, among those with baseline FBG 100-125 mg/dL	20	47	6	40	27.6	8.4	43.8	3.6	0.009
FBG ≥ 100 mg/dL at Week 24, among those with baseline FBG < 100 mg/dL	0	11	7	17	-41.2	-64.0	-8.7	2.4	0.023

TABLE 5: Clinical categories and responders

Note: Counts are participants out of 60 per group unless stated. Risk differences carry Newcombe hybrid-score CIs; p-values are from Fisher's exact test at Week 24. Within-group change (exact McNemar test): for BMI ≥ 25 kg/m² and FBG ≥ 100 mg/dL, Yoga p < 0.001 and Control p = 1.000 for both; for BMI ≥ 30 kg/m², p = 0.063 in each group. Risk ratios are omitted where a zero cell makes them unstable. Impaired fasting glucose is 100-125 mg/dL; the row for baseline FBG ≥ 100 mg/dL also includes the five participants with diabetes-range values.

BMI: body mass index, FBG: fasting blood glucose, CI: confidence interval, NNT: number needed to treat

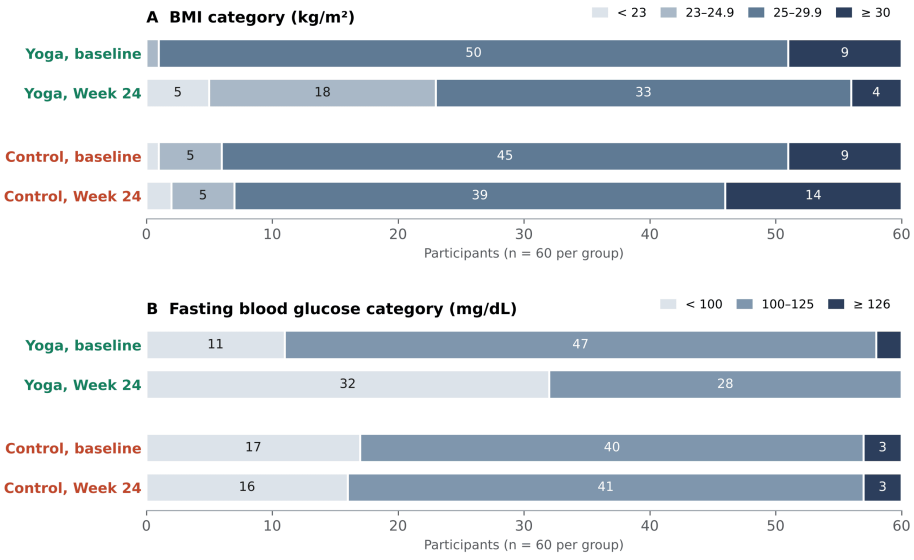


FIGURE 4: Distribution of participants across BMI categories (A; Asian Indian thresholds) and fasting blood glucose categories (B; American Diabetes Association thresholds) at baseline and Week 24

Counts below 3 are not printed.

BMI: body mass index

BMI Category

The distribution across BMI bands (<23, 23-24.9, 25-29.9 and ≥30 kg/m²) shifted in the Yoga group (Stuart-Maxwell $\chi^2(3) = 27.23$, p < 0.001), but not in the Control group ($\chi^2(3) = 7.00$, p = 0.072). At Week 24, 37 Yoga and 53 Control participants remained in the obese range, a risk difference of -26.7 percentage points (NNT: 3.8). Fifty-eight Yoga participants (96.7%) and no Control participants achieved a BMI reduction of at least 5%.

Glycaemic Category

The glycaemic distribution shifted in the Yoga group ($\chi^2(2) = 21.51$, p < 0.001), but not in the Control group ($\chi^2(2) = 0.08$, p = 0.962). At Week 24, 28 Yoga and 44 Control participants had FBG of 100 mg/dL or above (risk ratio: 0.64; NNT: 3.8). Among participants with elevated FBG (100 mg/dL or above) at baseline, 21 of 49 in the Yoga group (42.9%) and 6 of 43 in the Control group (14.0%) had FBG below 100 mg/dL at Week 24 (risk ratio: 3.07; NNT: 3.5). Restricted to impaired fasting glucose (100-125 mg/dL), the proportions were 20 of 47 (42.6%) and 6 of 40 (15.0%) (risk ratio: 2.84; NNT: 3.6). Among those with FBG below 100 mg/dL at baseline, none of 11 in the Yoga group and 7 of 17 in the Control group (41.2%) had moved into the impaired range by Week 24 (p = 0.023), and none reached 126 mg/dL.

Diabetes-Range Values

Both Yoga participants with a baseline value of 126 mg/dL or above fell below that level by Week 24 (one to below 100 mg/dL), as did two of the three such Control participants. Two other Control participants rose into this range, so the Control count remained at three (p = 0.244 at Week 24). These are single fasting values and do not establish a diagnosis of diabetes or its reversal [18].

Correlations and effect modification

Changes in FBG were not significantly correlated with changes in BMI (Table *6*, Figure *5*). Within neither group were the two changes associated (Yoga: r = -0.06, 95% CI: -0.31 to 0.20; Control: r = 0.11, 95% CI: -0.14 to 0.36), and adjusting for the baseline values made little difference (partial r = -0.09 and 0.13). The CIs include weak to moderate associations in either direction. Across all participants, the partial correlation controlling for group was 0.05 (95% CI: -0.14 to 0.22; p = 0.623); the unadjusted pooled correlation of 0.46 reflected the difference between the groups, not an association within individuals. In the Yoga group, the change in FBG per 1 kg/m² change in BMI, adjusted for baseline FBG, was -1.06 mg/dL (95% CI: -4.43 to 2.31; p = 0.530).

Variables	Group	Pearson r	r, 95% CI lower limit	r, 95% CI upper limit	p for r	Spearman ρ
Baseline BMI and baseline FBG	Yoga	-0.10	-0.35	0.16	0.443	-0.12
Baseline BMI and baseline FBG	Control	0.16	-0.10	0.39	0.234	0.13
Week-24 BMI and Week-24 FBG	Yoga	-0.05	-0.30	0.20	0.694	-0.04
Week-24 BMI and Week-24 FBG	Control	0.06	-0.20	0.31	0.641	-0.05
Change in BMI and change in FBG	Yoga	-0.06	-0.31	0.20	0.653	-0.06
Change in BMI and change in FBG	Control	0.11	-0.14	0.36	0.386	0.09
Change in BMI and change in FBG, partial	Yoga	-0.09	-0.34	0.18	0.523	-0.11
Change in BMI and change in FBG, partial	Control	0.13	-0.13	0.38	0.334	0.17
Age and change in BMI	Yoga	-0.03	-0.28	0.22	0.801	-0.03
Age and change in BMI	Control	-0.01	-0.26	0.24	0.934	-0.07
Age and change in FBG	Yoga	0.04	-0.21	0.29	0.740	0.05
Age and change in FBG	Control	-0.08	-0.33	0.18	0.544	0.00
Baseline BMI and change in BMI (Oldham)	Yoga	0.18	-0.07	0.42	0.161	0.20
Baseline BMI and change in BMI (Oldham)	Control	0.51	0.29	0.67	<0.001	0.48
Baseline FBG and change in FBG (Oldham)	Yoga	0.03	-0.23	0.28	0.847	0.07
Baseline FBG and change in FBG (Oldham)	Control	-0.05	-0.30	0.21	0.723	0.01

TABLE 6: Correlations within each group

Note: Pearson's r is given with a Fisher-z 95% CI; ρ is Spearman's rank correlation. Partial correlations are adjusted for baseline BMI and baseline FBG (the partial ρ is computed on ranks). Oldham's method correlates the mean of the baseline and Week-24 values with the change. Fisher's z test comparing the two groups gave p = 0.168 for baseline BMI and baseline FBG, 0.545 for Week-24 BMI and Week-24 FBG and 0.354 for change in BMI and change in FBG.

BMI: body mass index, CI: confidence interval, FBG: fasting blood glucose

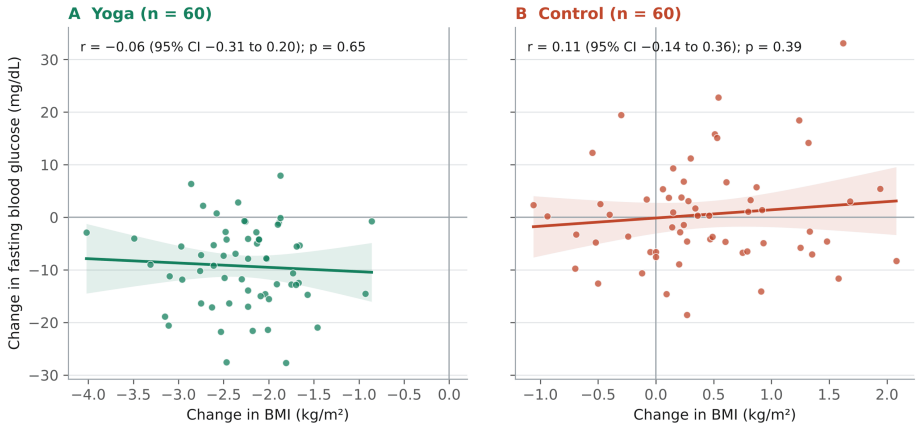


FIGURE 5: Change in fasting blood glucose against change in BMI within the Yoga group (A) and the Control group (B), with the least-squares line and its 95% confidence band

The correlations are within-group Pearson coefficients.

BMI: body mass index

Group and BMI change could not be cleanly separated. BMI change was almost collinear with group (r = -0.90): every Yoga participant lost BMI, whereas 44 of 60 Control participants gained. When change in BMI was added to the FBG ANCOVA, the group difference was similar (-8.60 mg/dL), but its CI widened (95% CI: -15.40 to -1.79; variance inflation factor: 5.5), and the coefficient for change in BMI was 0.30 mg/dL per kg/m² (95% CI: -1.98 to 2.58).

BMI and FBG levels were not correlated at baseline or at Week 24 in either group (all $|r| \leq 0.16$), and age was unrelated to either change. The simple correlation between baseline FBG and its change was negative in both groups (r = -0.39 and -0.46), but by Oldham's method, there was no association (r = 0.03 and -0.05), indicating regression to the mean. For BMI, Oldham's method showed that Control participants with higher BMI gained more (r = 0.51, $p < 0.001$); there was no such association in the Yoga group (r = 0.18, p = 0.161).

Effect Modification

The FBG effect was similar in women with FBG below 100 mg/dL at baseline (-11.82 mg/dL, 95% CI: -19.60 to -4.04; n = 28) and in those with FBG of 100 mg/dL or above (-8.39 mg/dL, 95% CI: -11.50 to -5.27; n = 92; interaction p = 0.271); restricted to the 87 with impaired fasting glucose, it was -7.97 mg/dL (95% CI: -11.13 to -4.81). The FBG effect was also similar by obesity class (p = 0.856) and age (p = 0.611). The BMI effect did not vary by baseline glycaemic status (p = 0.478), obesity class (p = 0.436) or age (p = 0.743).

Discussion

Principal findings

College women with PCOS who were randomised to 24 weeks of supervised yoga ended the trial with lower BMI and FBG than those who kept to their usual activity. The baseline-adjusted Week-24 means were 2.74 kg/m² and 9.41 mg/dL (0.52 mmol/L) lower with yoga, and these differences persisted in every alternative and sensitivity analysis.

Three further findings shape the interpretation. First, the response was clinically meaningful: almost every Yoga participant lost at least 5% of her BMI, and among women with elevated FBG at baseline, those in the Yoga group were about three times as likely as controls to have FBG below 100 mg/dL at Week 24; none of those with normal FBG at baseline moved into the impaired range. Second, usual activity was not neutral, because the Control group gained weight over the 24 weeks. Third, within each group, the change in FBG was not significantly correlated with the change in BMI. This exploratory finding cannot show whether weight change mediated the glycaemic difference, because nearly every Yoga participant lost weight and most controls gained it.

The earlier reports from this trial, on other outcomes, used a Week-24 comparison [6] and a change-score comparison [15] as their primary analyses. Both are reported here for BMI and FBG alongside ANCOVA, and all three agreed in direction, magnitude and significance.

Comparison with previous studies

The glycaemic effect is larger than pooled estimates. A meta-analysis of yoga in PCOS found a reduction in FBG of 0.22 mmol/L, about 4 mg/dL, from two small trials of low certainty [10], against 0.52 mmol/L here. In type 2 diabetes, yoga improves FBG with a standardised effect of 0.57 [9], compared with 0.89 here. The principal PCOS trial reported improved fasting glucose and insulin resistance after 12 weeks in adolescents, against an active exercise comparator [11]. Three features of the present trial may explain a larger effect: the programme lasted twice as long and delivered 120 supervised sessions, the comparator was usual activity rather than exercise and most participants started with impaired fasting glucose and therefore had more room to improve.

The BMI effect is larger still against the literature. Lifestyle interventions in PCOS reduce BMI by 0.34 kg/m² on average [12], and yoga trials show no overall effect on BMI, with a non-robust reduction in overweight and obese adults [13]. A fall of 8% in mean BMI over 24 weeks, without dietary prescription, therefore sits well above what is usually reported. This calls for the caution discussed below.

The control group gained weight

BMI did not stay static without the intervention. Controls gained a mean of 0.41 kg/m², 44 of the 60 put on weight and those with a BMI of 30 kg/m² or more increased from 9 to 14. Such gain is typical of student life: pooled data on first-year university students show that 61% gain weight, about 1.36 kg on average over roughly five months [20]. By Oldham's method, the controls who were heaviest at baseline gained the most. The between-group difference therefore combines weight that yoga removed with weight gain that it prevented, measured against a trajectory that, under ordinary student conditions, drifts upward.

Clinical significance

The 2018 international PCOS guideline describes weight loss of 5%-10% in women with excess weight as an achievable goal that yields significant clinical improvement [21]. Fifty-eight of 60 Yoga participants reached at least the lower end of that range, and no control participant did. In Asian Indian terms, the obese category shrank from 59 to 37 in the Yoga group and was unchanged in the Control group, a number needed to treat of about four.

Glycaemia points the same way. Offering the programme to roughly four women would leave one fewer with FBG of 100 mg/dL or more at Week 24. In women whose baseline FBG lay in the impaired range (100-125 mg/dL), 43% had normal values at Week 24 after yoga, compared with 15% after usual activity. The Yoga group's mean FBG crossed the 100 mg/dL threshold, falling from 107.85 to 98.58 mg/dL, although this is a group average and not true of every woman. Because each value comes from a single fasting sample, the results show a difference in fasting glycaemia rather than a confirmed change in glucose tolerance or diabetes status, for which repeat testing or a glucose tolerance test would be needed [1,18]. Even so, young women whose odds of type 2 diabetes are roughly four times higher [3] stand to gain most from prevention begun early in adulthood.

The mechanisms below were not measured in this trial, and the programme was evaluated as a whole, so none of its components can be credited individually. Each subsection sets the traditional rationale beside the physiology that could underlie it and the evidence for that physiology.

Āsana as Muscular Work

The Hatha Yoga Pradipika presents āsana as the source of steadiness, health and lightness of body [8]. In physiological terms, the programme placed sustained demands on large muscle groups. Most yoga is light-intensity activity, averaging about 3.3 metabolic equivalents, but Surya Namaskar is among the few sequences that reach moderate-to-vigorous intensity [22]. The standing and squatting holds of Utkata Konasana, Malasana and Prasarita Padottanasana add prolonged isometric work in the lower limbs.

Contracting muscle takes up glucose through translocation of glucose transporter type 4 (GLUT4), a route that does not require insulin. After exercise, the muscle's sensitivity to insulin remains raised for up to 48 hours [23]. In animal studies, this post-exercise sensitisation depends on signalling through AMP-activated protein kinase (AMPK) [24]. Because FBG reflects mainly hepatic glucose output [5], the effect of muscular work on fasting glucose is likely to be indirect and cumulative. Exercise training reduces liver fat by about 3.3 percentage points and improves hepatic insulin sensitivity [25], and a less fatty, more insulin-sensitive liver releases less glucose overnight.

Abdominal Postures

The Gheranda Samhita states that Bhujangasana increases the bodily fire and destroys disease [17]. The Hatha Yoga Pradipika attributes a similar kindling of the digestive fire to Matsyendrasana, of which Vakrasana is a simplified form [8]. A physiological counterpart can be proposed but has not been tested. In the prone postures (Bhujangasana and Dhanurasana), the abdomen bears on the floor, and the seated twist (Vakrasana) compresses and releases the trunk. Both change intra-abdominal pressure. Studies in healthy adults show that a sudden increase in abdominal pressure quickly drives blood out of the splanchnic vascular bed, probably largely from the liver, allowing the abdomen to work as an additional pump [26]. Whether repeated postural changes of this kind alter hepatic blood flow or glucose metabolism has not been studied, and the idea should be treated as a hypothesis.

Prāṇāyāma and Dhyāna

Patanjali places breath regulation and meditation among the limbs of yoga [7], and the Hatha Yoga Pradipika holds that correctly practised prāṇāyāma wards off disease [8]. Nadi Shodhana and Bhramari slow the breath and lengthen exhalation. Slow breathing is consistently associated with greater vagal activity and lower arousal and anxiety [27]. Compared with active control conditions, yoga practice has been linked to reduced waking and evening cortisol, a slower resting heart rate and lower fasting glucose [28]. Cortisol raises fasting glycaemia by stimulating hepatic gluconeogenesis and opposing insulin action [29]. A sustained reduction in stress-axis and sympathetic output is therefore a plausible route to lower fasting glucose that need not depend on weight loss. Psychological outcomes from this trial are reported separately, and cortisol was not measured.

Abhyāsa as Dose

Patanjali holds that practice becomes firm only when it is pursued over a long period, without interruption and with dedication [7]. This principle has a direct physiological counterpart. Because exercise-induced insulin sensitisation fades within about two days [23], a programme of five sessions a week keeps successive windows of heightened sensitivity overlapping rather than intermittent. Over 24 weeks, it also provides the sustained stimulus that structural changes such as reduced liver fat require [25]. Whether this mechanism operated here cannot be determined, because insulin sensitivity was not measured.

What the Present Data Add

Within the Yoga group, the size of the fall in FBG was not significantly correlated with the size of the fall in BMI (r = -0.06, 95% CI: -0.31 to 0.20), and the FBG effect was similar in women with and without obesity and with and without elevated FBG at baseline. If glycaemic improvement were simply a by-product of weight loss, a positive association would be expected. The pattern is therefore compatible with routes that act partly independently of body weight, such as hepatic insulin sensitivity and stress-hormone output, but it does not establish them.

Three limits apply. The CI for the correlation includes modest associations, the trial could reliably detect only correlations of about 0.35 or larger, and FBG varies from day to day. Most importantly, group and BMI change were almost collinear, so the trial cannot separate a weight-mediated effect from other effects of the programme. The finding is nonetheless consistent with clamp evidence that insulin resistance in PCOS is not explained by adiposity alone [2] and with the observation from this cohort that anthropometric and biochemical markers behave as distinct dimensions [6].

Body Composition

Sustained postural holds may preserve or build lean mass while fat is lost, which would lower glycaemia beyond what BMI reflects. BMI cannot separate fat from lean tissue, and body composition was not measured, so this remains a hypothesis.

Endocrine Relevance

In PCOS, excess insulin stimulates androgen synthesis in the ovary and suppresses hepatic output of sex hormone-binding globulin, and the two effects together intensify hyperandrogenism [30]. An intervention that improves glycaemic control could therefore act on the reproductive axis as well. Hormonal outcomes from this trial are reported separately, and no inference about them is drawn here.

Magnitude and plausibility

The size and consistency of the BMI response need comment. Every Yoga participant lost BMI, and the change-score distributions of the two groups barely overlapped. Because change varied little within each group (SD: 0.57 and 0.71 kg/m²), the standardised difference for change scores was -4.21; the difference standardised by the baseline SD, −1.23, is the more interpretable figure. Several features may contribute to the size and consistency of the response: an intensive and fully supervised programme delivered by one experienced instructor, a young cohort that started almost entirely in the obese range and a comparator group that gained weight.

Measurement by blinded assessors, the agreement of every analytic frame and the intention-to-treat (multiple-imputation) and tipping-point analyses argue against a statistical artefact or an effect of attrition. They cannot, however, exclude unmeasured changes in diet or activity outside the sessions, which the pragmatic design did not record. An effect of this size and uniformity is uncommon in behavioural research and should be regarded as provisional until it is replicated by independent investigators.

Strengths and limitations

The trial's strengths are prospective registration, concealed randomisation, blinded outcome assessment with a single accredited laboratory, a written and standardised 24-week protocol and complete data for all retained participants. The analysis triangulated ANCOVA, Week-24, change-score, non-parametric, robust, intention-to-treat (multiple-imputation) and Bayesian estimates and Bayesian estimates, reported clinically interpretable categorical and responder outcomes and used correlation methods designed to avoid the artefacts of pooling and regression to the mean.

Several limitations apply. A single fasting sample measured glycaemia; neither glycated haemoglobin (HbA1c) nor an oral glucose tolerance test was performed, although the international PCOS guideline regards the latter as the best test of glucose status [1], and insulin resistance could not be estimated because fasting insulin was not measured. BMI cannot separate fat from lean tissue. Only women retained to Week 24 entered the primary analysis; the intention-to-treat analysis with multiple imputation and the tipping-point analysis indicate that the 11 withdrawals were very unlikely to overturn the result, but both depend on assumptions about data that were never observed and cannot rule out every bias. One instructor at one university delivered the programme, which restricts generalisability, and diet and activity outside the sessions went unrecorded. Neither the participants nor the instructor were blind to allocation, leaving room for expectancy effects, and comparison with usual activity cannot distinguish what is specific to yoga from the effects of supervised activity and attention.

Although attendance was taken at every session, the registers were not available for analysis, so adherence is not reported and dose-response could not be explored. Exclusions at screening were not counted by

criterion. The trial had no stand-alone statistical analysis plan, and ANCOVA was adopted as the primary analysis for this report, although all other analyses agreed with it. Lastly, none of the proposed mechanisms was measured, and how long the changes last once supervised sessions end is not known.

Future research

Replication now requires independent trials at several centres that compare yoga with an active control matched for time and supervision. These trials should assess glycaemia more completely (fasting insulin, glycated haemoglobin, glucose tolerance testing and, if feasible, clamp-measured insulin sensitivity), image body composition and liver fat, and record possible mediators such as cortisol and heart rate variability so that the proposed pathways can be tested directly. Analysing attendance and continuing follow-up after supervised sessions end would show whether benefit depends on dose and how long it lasts.

Conclusions

In college women with PCOS who completed this 24-week trial, a supervised integrated yoga programme, practised five days a week, was associated with lower BMI and lower fasting blood glucose than usual activity. The baseline-adjusted differences were -2.74 kg/m² and -9.41 mg/dL, and analyses that included all randomized participants gave the same conclusion. Almost all women in the Yoga group achieved a weight-loss response of at least 5%, and among those with elevated fasting glucose at baseline, women in the Yoga group were about three times as likely to have fasting glucose below 100 mg/dL at Week 24. Women continuing their usual routines gained weight. Within each group, changes in glucose and BMI were not significantly correlated; this exploratory finding does not establish whether the glycaemic difference was independent of weight loss.

These findings support sustained, supervised practice, the principle the yoga tradition calls abhyāsa, as a promising lifestyle option for young women with PCOS. Because the comparison was with usual activity, the trial cannot separate effects specific to yoga from those of regular supervised physical activity and attention. Because glycaemia was assessed from a single fasting sample, the effects are larger than pooled estimates and the BMI response was unusually uniform, the findings should be regarded as provisional. Independent replication with an active comparator, oral glucose tolerance testing, insulin measurement and body-composition imaging is needed.

Appendices

Appendix 1: Robustness, sensitivity and missing data analyses

Robustness and Sensitivity

The estimates and 95% CIs of the robustness and sensitivity analyses are shown in Figure *3*. The Mann-Whitney test on change scores gave U = 4 (z = -9.43) for BMI and U = 772 (z = -5.40) for FBG, both $p < 0.001$. No BMI observation had a studentised deleted residual beyond ±3; one FBG observation did, and excluding it changed the FBG estimate from -9.41 to -9.01 mg/dL.

Multiple Imputation

Each withdrawal's change from baseline was imputed from a normal model fitted to the observed changes in the participant's own group, with the group mean and variance drawn from their posterior distributions under a non-informative prior in each of 200 imputations. Each completed dataset of 131 participants was analysed with a t test on change scores, and the results were pooled by Rubin's rules. The fraction of missing information was 0.08 for both outcomes. The pooled differences were -2.70 kg/m² (95% CI: -2.93 to -2.47) for BMI and -9.78 mg/dL (95% CI: -12.94 to -6.61) for FBG.

Tipping-Point Analysis

The imputed changes of the five Yoga-group withdrawals were shifted in the unfavourable direction, in steps of 0.25 kg/m² and 1 mg/dL, until the upper 95% confidence limit of the difference reached zero. This required a shift of 19.0 kg/m² for BMI, so that these women would have gained 16.7 kg/m² on average (a shift of 33 SDs of the observed change in the Yoga group), and a shift of 62 mg/dL for FBG, so that their FBG would have risen by 52.7 mg/dL on average (7.9 SDs). When the imputed changes of the six Control-group withdrawals were also shifted by the same amount in the favourable direction, the tipping points were 10.25 kg/m² and 33 mg/dL. In every scenario, the average change required of the withdrawals lies beyond the largest individual change observed among completers in the same group.

Cross-Group Scenario

Assigning each Yoga-group withdrawal the mean change of the Control group, and each Control-group withdrawal the mean change of the Yoga group, gave differences of -2.25 kg/m² (95% CI: -2.59 to -1.91) and -8.14 mg/dL (95% CI: -11.21 to -5.08), both $p < 0.001$.

Appendix 2: Bayesian analysis

Specification

The ANCOVA model (Week-24 value on group and baseline value) was refitted as a Bayesian linear regression. Under a reference (non-informative) prior, the posterior distribution of the group effect is a t distribution with 117 degrees of freedom centred on the least-squares estimate. To test sensitivity to the prior, the group effect was also given a sceptical normal prior centred on no effect (SD: 1 kg/m² for BMI and 5 mg/dL for FBG), combined with the likelihood by a normal approximation. From each posterior, we computed the probability that the effect exceeded clinically relevant reductions, defined as 1 kg/m² for BMI and 5 mg/dL for FBG. Evidence for a group effect was summarised as a Bayes factor (BF_{10}) comparing the ANCOVA models with and without group under the Jeffreys-Zellner-Siow prior (scale $r = \sqrt{2}/4$, with r = 1 as a sensitivity check), computed by numerical integration. All posterior quantities were computed analytically or by numerical integration in Python, without sampling.

Results

The posterior estimates matched the frequentist results and changed little under the sceptical priors (Table 7). The Bayes factors favouring a group effect were about 2×10^{43} for BMI and 4×10^{6} for FBG, and were essentially unchanged with r = 1.

Variables	Group	Pearson r	r, 95% CI lower limit	r, 95% CI upper limit	p for r	Spearman ρ
Baseline BMI and baseline FBG	Yoga	-0.10	-0.35	0.16	0.443	-0.12
Baseline BMI and baseline FBG	Control	0.16	-0.10	0.39	0.234	0.13
Week-24 BMI and Week-24 FBG	Yoga	-0.05	-0.30	0.20	0.694	-0.04
Week-24 BMI and Week-24 FBG	Control	0.06	-0.20	0.31	0.641	-0.05
Change in BMI and change in FBG	Yoga	-0.06	-0.31	0.20	0.653	-0.06
Change in BMI and change in FBG	Control	0.11	-0.14	0.36	0.386	0.09
Change in BMI and change in FBG, partial	Yoga	-0.09	-0.34	0.18	0.523	-0.11
Change in BMI and change in FBG, partial	Control	0.13	-0.13	0.38	0.334	0.17
Age and change in BMI	Yoga	-0.03	-0.28	0.22	0.801	-0.03
Age and change in BMI	Control	-0.01	-0.26	0.24	0.934	-0.07
Age and change in FBG	Yoga	0.04	-0.21	0.29	0.740	0.05
Age and change in FBG	Control	-0.08	-0.33	0.18	0.544	0.00
Baseline BMI and change in BMI (Oldham)	Yoga	0.18	-0.07	0.42	0.161	0.20
Baseline BMI and change in BMI (Oldham)	Control	0.51	0.29	0.67	<0.001	0.48
Baseline FBG and change in FBG (Oldham)	Yoga	0.03	-0.23	0.28	0.847	0.07
Baseline FBG and change in FBG (Oldham)	Control	-0.05	-0.30	0.21	0.723	0.01

TABLE 7: Bayesian re-analysis of the primary ANCOVA

Note: The sceptical priors were normal with SD 1 kg/m² for BMI and 5 mg/dL for FBG. $\log_{10} \mathrm{BF}_{10}$ was 43.3 for BMI and 6.6 for FBG ($r = \sqrt{2}/4$). CrI, credible interval; BF_{10} , Bayes factor for a group effect against no effect.

ANCOVA: analysis of covariance, BMI: body mass index, CI: confidence interval, FBG: fasting blood glucose, SD: standard deviation

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the

work.

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