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Morning versus evening aerobic exercise and atherogenic and fat-distribution indices in older women with type 2 diabetes: a quasi-experimental controlled study

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Abstract

Purpose To compare the effects of 12 weeks of morning versus evening aerobic exercise on fat-distribution, insulin-resistance and atherogenic indices in older women with type 2 diabetes mellitus (T2DM).

Methods In this quasi-experimental controlled study, 45 sedentary postmenopausal women with T2DM aged 60–65 years were allocated by availability to supervised moderate-intensity aerobic training in the morning (09:00 h) or evening (21:00 h), three sessions per week for 12 weeks, or to a non-exercising control group. Thirteen anthropometric and biochemical indices were assessed before and after the intervention. Between-group differences were analysed by baseline-adjusted ANCOVA with false-discovery-rate control and confirmed by linear mixed-effects, change-score and non-parametric analyses.

Results Forty-two women (14 per group) completed the study. Significant group effects were found for insulin resistance (HOMA-IR), abdominal volume index, body adiposity index

(BAI), waist-to-height ratio, visceral adiposity index (VAI), triglyceride–glucose index (TyG), TyG–waist circumference (TyG–WC), TyG–body mass index, atherogenic index of plasma (AIP) and Castelli’s risk indices I and II (CRI-I, CRI-II) (all $p < 0.001$; $q < 0.001$). These indices improved in both exercise groups and mostly deteriorated in controls. Evening exercise was superior to morning exercise for BAI, VAI, AIP, CRI-I and CRI-II ($p = 0.044$ to $p < 0.001$), and change-score analysis additionally favoured evening exercise for TyG and TyG–WC.

Conclusion Aerobic exercise improved adiposity, insulin-resistance and atherogenic indices in older women with T2DM, with greater benefit from evening sessions for lipid-related and visceral-fat indices. Randomised trials are needed to confirm this timing effect.

Trial registration Iranian Registry of Clinical Trials, IRCT20181212041935N1; registered 18 February 2024.

Keywords Diabetes Mellitus, Type 2 · Exercise · Circadian Rhythm · Adiposity · Insulin Resistance · Postmenopause

Introduction

Diabetes mellitus is among the most prevalent chronic diseases worldwide. It is a chronic metabolic disorder characterised by persistent hyperglycaemia, insulin resistance and impaired lipid metabolism. Most affected individuals have type 2 diabetes mellitus (T2DM), which arises from defective insulin secretion by pancreatic beta cells together with the failure of insulin-sensitive tissues to respond appropriately to insulin [1]. T2DM leads to cardiovascular disorders, microvascular complications and increased mortality [1], and global prevalence in adults aged 20–79 years is projected to rise from 10.5% (536.6 million people) in 2021 to 12.2% (783.2 million) by 2045 [2].

Although T2DM is more frequent in men during early adulthood, its prevalence in women rises from menopause onwards [3]. The marked fall in oestrogen during menopause increases oxidative stress and inflammation in adipose tissue; under physiological conditions oestrogens attenuate both processes and thereby help prevent insulin resistance and cardiovascular disease. Oestrogen also participates in the regulation of insulin secretion and glucose homeostasis, so its decline may raise the risk of T2DM and related metabolic complications in postmenopausal women [3, 4].

Traditional anthropometric measures such as body mass index (BMI), waist circumference (WC) and waist-to-hip ratio are widely used to estimate obesity-related risk, but they cannot distinguish visceral from subcutaneous fat or from muscle mass. Newer composite indices — the abdominal volume index (AVI), body adiposity index (BAI), waist-to-height ratio (WHtR), visceral adiposity index (VAI), model of adipose distribution (MOAD), conicity index, triglyceride–glucose index (TyG) and its anthropometric combinations (TyG–WC, TyG–BMI) — are inexpensive, simple to calculate, and may characterise body-fat distribution and insulin resistance more accurately [4–6]. In parallel, plasma atherogenic indices such as the atherogenic index of plasma (AIP) and Castelli’s risk indices I and II (CRI-I, CRI-II) are among the strongest predictors of cardiovascular risk in diabetes [7, 8], and are sensitive to habitual physical activity [9].

The skeletal-muscle circadian clock responds to non-photic cues, including contractile activity, so circadian rhythmicity can be reset and reinforced by exercise even in metabolic disease [10]. Aerobic exercise enhances glucose and lipid metabolism and improves insulin sensitivity [11, 12], and modulates the expression of clock genes in peripheral tissues such as skeletal muscle and adipose tissue [13, 14]. Recent evidence suggests that afternoon or evening exercise yields greater improvements in glycaemic control and insulin sensitivity than morning exercise in people with T2DM [15–17]. Nevertheless, intervention studies

comparing exercise timing remain scarce, most have focused on glycaemic outcomes rather than on lipid-derived atherogenic and fat-distribution indices, and older women with T2DM are markedly under-represented [18]. The present study therefore compared the effects of 12 weeks of morning versus evening aerobic exercise on fat-distribution, insulin-resistance and atherogenic indices in older women with T2DM.

Materials and methods

Study design and participants

This was a quasi-experimental study with a non-equivalent control group and a pre-test–post-test design. Because participants could not attend arbitrary training times, allocation to the three groups — morning exercise, evening exercise and control — was made according to availability and scheduling preference rather than by randomisation. The study is reported in accordance with the TREND statement for non-randomised behavioural and public-health interventions.

The source population comprised women aged 60–65 years with T2DM residing in Chaharmahal and Bakhtiari Province, Iran, with medical records at the urban comprehensive health service centres of Kiar County. Inclusion criteria were: physician-confirmed T2DM; $\text{HbA1c} \geq 6.5\%$; fasting blood glucose ≥ 126 mg/dL (7 mmol/L); use of oral glucose-lowering medication; absence of hepatic steatosis on ultrasonography and medical history; and a sedentary lifestyle (no regular physical activity for at least six months). Exclusion criteria were: musculoskeletal injury or disability precluding exercise; neurological disorders affecting motor or cognitive function; inability to give informed consent; acute or chronic inflammatory disease (e.g. rheumatoid arthritis, systemic lupus erythematosus); advanced heart failure (NYHA class III–IV); and absence from more than three training sessions.

All participants were postmenopausal (amenorrhoea for ≥ 12 consecutive months and 17β -oestradiol < 30 pg/mL) and none were receiving hormone replacement therapy. Participants were on stable doses of glucose-lowering medication (e.g. metformin), unchanged for at least three months before enrolment, and none used drugs or corticosteroids known to affect lipid metabolism.

Of the 300 women screened for eligibility, 45 met the criteria and agreed to participate. Three withdrew during the intervention — one from the morning group (renal complications), one from the evening group (ocular surgery) and one from the control group (relocation) — leaving 42 participants (14 per group) for analysis (Fig. 1).

Sample size

The sample size was estimated for a one-way fixed-effects analysis of variance with three groups using G*Power 3.1. Assuming a large effect size ($f = 0.55$) based on previous exercise trials in T2DM [19, 20], $\alpha = 0.05$ and power $(1 - \beta) = 0.80$, a minimum of 36 participants (12 per group) was required. Forty-five women were enrolled to allow for approximately 20% attrition.

Ethical considerations

The study was approved by the Research Ethics Committee of the School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran (IR.SSU.SPH.REC.1403.093) and registered with the Iranian Registry of Clinical Trials (IRCT20181212041935N1) on 18 February 2024. Given the age and physical capacity of the participants, only low-risk exercise modalities (brisk walking and light jogging) were used. Participation was voluntary, written informed consent was obtained from every participant, and participants could withdraw at any time. Apart from the partial institutional support

described under Funding, the costs of laboratory testing, venue hire and instructor fees were met by the investigators.

Anthropometric and biochemical measurements

Body mass, BMI and body-fat percentage were measured with an InBody 230 bioelectrical impedance analyser (InBody Co. Ltd, Seoul, South Korea), and height and waist and hip circumferences were measured manually using standard procedures. All anthropometric measurements were taken before the first session and 48 h after the final session by the same trained assessor, who was unaware of group assignment.

Venous blood samples (10 mL) were drawn from the antecubital vein in the seated position 24 h before the first session and 48 h after the final session, after a 12-h overnight fast. Fasting glucose was determined by the glucose-oxidase method with Pars Azmoon kits (Pars Azmoon Co., Tehran, Iran) on a Hitachi clinical chemistry analyser (models 704 and 902; Hitachi Ltd, Tokyo, Japan). Serum insulin was measured by enzyme-linked immunosorbent assay on a microplate reader. Serum triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and fasting blood glucose (FBG) were measured with Delta Darman kits (Delta Darman Part Co., Tehran, Iran). All biochemical analyses were carried out in a single certified reference laboratory using identical equipment and procedures at both time points.

Calculated indices

Indices were calculated as follows. Glucose and lipids are in mg/dL unless otherwise stated; for AIP and VAI, TG and HDL-C were converted to mmol/L (dividing by 88.57 and 38.67, respectively) in accordance with the original definitions of these indices.

$$AVI = [2 \times WC^2 + 0.7 \times (WC - \text{hip circumference})^2] / 1000, \text{ circumferences in cm} \quad [21]$$

$$BAI = [\text{hip circumference (cm)} / \text{height (m)}^{1.5}] - 18 \quad [22]$$

$$BMI = \text{body mass (kg)} / \text{height (m)}^2$$

$$\begin{aligned} \text{TyG} &= \ln[\text{TG} \times \text{FBG} / 2] \quad [23] \\ \text{TyG-BMI} &= \text{TyG} \times \text{BMI} ; \text{TyG-WC} = \text{TyG} \times \text{WC (cm)} \quad [24] \\ \text{CRI-I} &= \text{TC} / \text{HDL-C} ; \text{CRI-II} = \text{LDL-C} / \text{HDL-C} \quad [25] \\ \text{AIP} &= \log_{10} (\text{TG} / \text{HDL-C}), \text{ TG and HDL-C in mmol/L} \quad [26] \\ \text{VAI} &= [\text{WC} / (36.58 + 1.89 \times \text{BMI})] \times (\text{TG} / 0.81) \times (1.52 / \text{HDL-C}), \text{ WC in cm, TG and HDL-C in} \\ &\text{mmol/L} \quad [27] \\ \text{MOAD} &= \text{WC (cm)} / (36.58 + 1.89 \times \text{BMI}) \quad [28] \\ \text{WHtR} &= \text{WC (cm)} / \text{height (cm)} \quad [29] \\ \text{Conicity index} &= \text{WC (m)} / [0.109 \times \sqrt{(\text{body mass (kg)} / \text{height (m)})}] \quad [30] \\ \text{HOMA-IR} &= \text{fasting insulin } (\mu\text{U/mL}) \times \text{fasting glucose (mmol/L)} / 22.5 \quad [31] \end{aligned}$$

Training protocol

Exercise timing was based on the zeitgeber-time framework described by Sato et al. [32], with lights-on defined as 06:00 h (ZT0) and lights-off as 18:00 h (ZT12); sessions were therefore held three hours after lights-on (09:00 h, ZT3) or three hours after lights-off (21:00 h, ZT15). All sessions took place in the indoor sports hall of the Department of Sport and Youth at a controlled temperature of 25–26 °C.

Both exercise groups trained for 12 weeks, three non-consecutive sessions per week (36 sessions in total), each lasting 50 min [33]. Sessions comprised a warm-up of 11 dynamic stretching exercises (two sets of 15–20 s per position), 40 min of brisk walking or light jogging, and a 10-min cool-down (8 min of slow walking followed by 2 min of static stretching). Intensity was prescribed using the Karvonen method (target heart rate = resting heart rate + a percentage of heart-rate reserve, with maximal heart rate estimated as 220 – age); training began at 50% of heart-rate reserve and progressed to 60–70% after the fourth week in accordance with the principle of progressive overload. Heart rate was monitored continuously with Polar heart-rate monitors (Polar Electro Oy, Kempele, Finland). All sessions were supervised by a certified exercise instructor trained in the study protocol, who recorded attendance at every session. The control group received no exercise intervention and maintained habitual activity. All participants were asked to maintain their usual diet and to start no new nutritional supplements during the study.

Statistical analysis

Data are presented as mean $\pm$ standard deviation (SD) unless otherwise stated. Baseline comparability of the three groups was examined by one-way ANOVA. For every outcome, normality of the within-participant change scores was examined with the Shapiro–Wilk test, homogeneity of variance with Levene’s test, and homogeneity of regression slopes by testing the group $\times$ baseline interaction in the ANCOVA model (Online Resource 1).

The primary between-group analysis was an ANCOVA on post-intervention values with the corresponding baseline value as covariate, followed by Bonferroni-adjusted pairwise comparisons; adjusted mean differences are reported with unadjusted 95% confidence intervals. Because 13 outcomes were analysed, omnibus p values were additionally controlled for the false discovery rate using the Benjamini–Hochberg procedure, and the resulting q values are reported.

Because the homogeneity-of-slopes assumption was not met for every outcome and change scores were frequently non-normally distributed, three sensitivity analyses were performed. First, a linear mixed-effects model with group, time and the group $\times$ time interaction as fixed effects and a random intercept for participant was fitted, and the interaction was tested by likelihood-ratio test; this model does not require homogeneity of regression slopes. Second, between-group comparisons were repeated non-parametrically on change scores (Kruskal–Wallis test across the three groups; Mann–Whitney U test for evening versus morning exercise), and within-group changes were re-tested with the Wilcoxon signed-rank test. Third, to examine whether the evening–morning difference could reflect regression to the mean introduced by covariate adjustment, raw change scores (post – pre) were compared by one-way ANOVA and, for evening versus morning exercise, by independent-samples t-test.

Within-group changes were examined with paired-samples t-tests and are considered exploratory. Effect sizes are reported as partial eta-squared (η^2p) for the ANCOVA group effect, Cohen's d_z (mean change divided by the SD of the change) for within-group change, and Hedges' g for the evening–morning difference in change scores. Analyses were performed in SPSS version 25 (IBM Corp., Armonk, NY, USA) and Python 3.12 (statsmodels and SciPy); figures were produced with matplotlib. Two-sided $p < 0.05$ was considered statistically significant.

Results

Forty-two of the 45 enrolled women completed the intervention (Fig. 1); their mean age was 62.4 ± 1.8 years (range 60–65). Baseline characteristics are shown in Table 1. The groups were comparable on all demographic, anthropometric, glycaemic and lipid variables and on 11 of the 13 study indices (all $p \geq 0.080$); the exceptions were MOAD ($p = 0.008$) and the conicity index ($p = 0.005$), both of which were lower at baseline in the morning group. Pre- and post-intervention values are given in Table 2, ANCOVA results in Table 3, adjusted between-group differences in Table 4 and sensitivity analyses in Table 5.

Significant between-group effects were observed for 11 indices — AVI, BAI, WHtR, VAI, HOMA-IR, TyG, TyG–WC, TyG–BMI, AIP, CRI-I and CRI-II (all $p < 0.001$) — and all remained significant after false-discovery-rate correction (all $q < 0.001$). MOAD ($p = 0.198$) and the conicity index ($p = 0.159$) showed no group effect. For the 11 responsive indices, both exercise groups improved, whereas the control group either deteriorated or remained unchanged: controls showed significant increases in HOMA-IR (+21.8%), VAI (+13.6%), TyG (+1.6%), TyG–WC (+1.7%), TyG–BMI (+2.5%), AIP (+0.058), CRI-I (+10.5%) and CRI-II (+6.5%) (all $p < 0.001$) and no significant change in AVI, BAI or WHtR.

Fat-distribution indices. AVI decreased in both the morning (−3.1%) and evening (−5.9%) groups (both $p = 0.001$) but not in controls ($p = 0.161$); adjusted means were lower than control in both exercise groups (morning $p = 0.015$; evening $p < 0.001$), without a difference between exercise timings ($p = 0.364$) (Fig. 2). BAI decreased by 1.7% in the morning group ($p = 0.002$) and 6.2% in the evening group ($p < 0.001$); evening exercise produced a lower adjusted BAI than both control and morning exercise (both $p < 0.001$), whereas the morning–control difference was not significant ($p = 0.068$) (Fig. 3). WHtR decreased by 1.5% and 2.9% in the morning and evening groups (both $p < 0.001$) and was unchanged in controls ($p = 0.165$); both exercise groups differed from control (morning $p = 0.037$; evening $p < 0.001$; $F_{2,38} = 10.56$, $\eta^2p = 0.357$), with no difference between timings ($p = 0.188$) (Fig. 4). VAI decreased by 24.6% and 35.1% in the morning and evening groups (both $p < 0.001$) and increased by 13.6% in controls ($p < 0.001$); both exercise groups differed from control (both $p < 0.001$), and evening exercise was superior to morning exercise (−0.36, 95% CI −0.65 to −0.08; $p = 0.041$) (Fig. 5). MOAD and the conicity index changed little, with small within-group reductions only in the evening group (MOAD −1.6%, $p = 0.034$; conicity index −1.8%, $p = 0.017$) and no between-group effects (Figs. 6 and 7).

Insulin-resistance indices. HOMA-IR decreased by 38.7% in the morning group and 39.9% in the evening group and increased by 21.8% in controls (all $p < 0.001$; $dz = -1.32$, -2.00 and 1.26 , respectively). Adjusted HOMA-IR was lower than control in both the morning (−4.68, 95% CI −5.94 to −3.43) and evening (−4.84, 95% CI −6.10 to −3.57) groups (both $p < 0.001$; $F_{2,38} = 38.98$, $\eta^2p = 0.672$), with no difference between timings ($p = 1.000$) (Fig. 8). TyG, TyG–WC and TyG–BMI decreased in both exercise groups and increased in controls (all $p < 0.001$; Table 2), and adjusted means were lower than control in both exercise groups (all $p < 0.001$) (Figs. 9–11). The Bonferroni-adjusted evening–morning comparisons for these three

indices were not significant ($p = 0.106, 0.077$ and 0.082 , respectively), although the unadjusted 95% confidence intervals of the differences excluded zero (Table 4).

Atherogenic indices. AIP decreased by 0.110 in the morning group and 0.185 in the evening group and increased by 0.058 in controls (all $p < 0.001$), with the largest within-group effect sizes observed in the study ($dz = -2.32$ and -2.65). Adjusted AIP was lower than control in both exercise groups (both $p < 0.001$) and lower after evening than after morning exercise (-0.067 , 95% CI -0.107 to -0.028 ; $p = 0.004$) (Fig. 12). CRI-I followed the same pattern (morning -17.4% , evening -29.9% , control $+10.5\%$; all $p < 0.001$), with both exercise groups lower than control (both $p < 0.001$) and evening lower than morning exercise (-0.50 , 95% CI -0.89 to -0.10 ; $p = 0.044$) (Fig. 13). CRI-II decreased by 19.8% and 33.8% in the morning and evening groups and increased by 6.5% in controls (all $p < 0.001$); both exercise groups differed from control (both $p < 0.001$), and the reduction was larger after evening exercise (-0.29 , 95% CI -0.50 to -0.07 ; $p = 0.032$) (Fig. 14).

Sensitivity analyses. The sensitivity analyses are summarised in Table 5. Mixed-effects models showed a significant group $\times$ time interaction for all 11 indices with an ANCOVA group effect (all $p < 0.001$) and no interaction for MOAD ($p = 0.090$); for the conicity index the interaction was nominally significant ($p = 0.045$) but was not supported by the ANCOVA, change-score or non-parametric analyses ($p = 0.056$ – 0.159). Change scores departed from normality for 11 of the 13 outcomes, but Kruskal–Wallis tests were significant for the same 11 indices (all $p < 0.001$), and Mann–Whitney U tests favoured evening over morning exercise for BAI ($p < 0.001$), AIP ($p = 0.005$), CRI-I ($p = 0.007$), CRI-II ($p = 0.016$), VAI ($p = 0.018$), TyG–WC ($p = 0.029$) and TyG ($p = 0.046$). Analysis of raw change scores, which involves no covariate adjustment, confirmed the between-group differences for the same 11 indices (all $p < 0.001$) and favoured evening over morning exercise for BAI ($p < 0.001$), AIP

($p = 0.003$), TyG–WC ($p = 0.011$), CRI-II ($p = 0.019$), VAI ($p = 0.021$), TyG ($p = 0.023$) and CRI-I ($p = 0.024$), with Hedges' g ranging from -0.88 to -1.87 .

Discussion

This study compared 12 weeks of morning and evening aerobic exercise in older women with T2DM. Irrespective of timing, aerobic training improved AVI, BAI, WHtR, VAI, HOMA-IR, TyG, TyG–WC, TyG–BMI, AIP, CRI-I and CRI-II, whereas most of these indices deteriorated in the non-exercising control group. Evening exercise produced greater improvements than morning exercise in BAI, VAI, AIP, CRI-I and CRI-II, and change-score analysis extended this advantage to TyG and TyG–WC. MOAD and the conicity index showed no consistent between-group effect.

Elevated atherogenic and fat-distribution indices are central to the development of cardiovascular complications in T2DM. AIP is a reliable marker of plasma atherogenicity and is responsive to physical activity [7, 26, 34], while the TyG index and VAI are robust predictors of cardiometabolic risk [23, 27, 35]. Combining TyG with anthropometric measures such as BMI and WC further improves risk prediction, because these composites capture both fat accumulation and insulin resistance [24, 36]. AVI and WHtR reflect central adiposity and BAI overall adiposity [5, 29], and the cholesterol ratios CRI-I and CRI-II, together with AIP, are established indicators of atherogenic dyslipidaemia [8, 25, 37].

The deterioration in the control group deserves emphasis. Over only 12 weeks, participants who did not exercise showed significant increases in HOMA-IR, VAI, TyG, TyG–WC, TyG–BMI, AIP, CRI-I and CRI-II. This is consistent with the progressive course of insulin resistance and dyslipidaemia in postmenopausal women with T2DM, and indicates that part of the benefit of exercise lies in preventing further deterioration; the between-group differences therefore reflect both improvement with exercise and worsening without it.

Aerobic exercise improves the lipid profile largely by increasing skeletal-muscle lipoprotein lipase activity, lowering circulating triglycerides and raising HDL-C [12]. The greater effect of evening exercise on atherogenic indices is consistent with circadian modulation of these adaptations. Exercise performed at different times of day can shift the phase of peripheral clocks, helping to correct circadian misalignment and improve metabolic efficiency [38]; mitochondrial function in human skeletal muscle peaks in the late afternoon [38], and time-of-day-specific metabolic responses to exercise have been demonstrated in animal models [32], which may favour lipid oxidation during later sessions.

Our findings are broadly concordant with the emerging literature. In the Look AHEAD cohort, bout-related moderate-to-vigorous physical activity performed in the afternoon was associated with the greatest reduction in HbA1c among adults with T2DM [16]. A recent systematic review and meta-analysis of intervention studies reported that fasting glucose was higher after morning than after afternoon or evening exercise in longer-term trials [17], and a recent review concluded that afternoon and evening exercise produce greater improvements in glycaemic control and insulin sensitivity than morning exercise in T2DM [15]. In a crossover trial in T2DM, Menek and Budak reported greater improvements in glycaemic and lipid parameters when exercise was timed to match participants' chronotype [39], and the ongoing Ex-Timing trial has been designed specifically to address this question [18]. Our data extend these observations from glycaemic endpoints to lipid-derived atherogenic and fat-distribution indices, and to a population — older postmenopausal women — that is rarely studied in this context.

A particular strength of the present analysis is that the superiority of evening exercise was established under several analytical frameworks. Because allocation was not randomised and the evening group began with numerically less favourable values on most indices, covariate-adjusted comparisons are vulnerable to regression to the mean. We therefore repeated the

between-group comparison on raw change scores, which involve no covariate adjustment; evening exercise remained superior for BAI, VAI, AIP, CRI-I and CRI-II, and additionally for TyG and TyG–WC, with Hedges’ g between -0.88 and -1.87 . Linear mixed-effects models, which do not require homogeneity of regression slopes, reproduced every ANCOVA group effect, and non-parametric tests reproduced the same pattern despite non-normal change scores. The only discrepancy — a nominal mixed-model interaction for the conicity index — was not supported by any other analysis. The convergence of approaches resting on different assumptions makes it unlikely that the timing effect is an analytical artefact, although it cannot overcome the non-randomised allocation itself.

Not all indices responded. MOAD and the conicity index, which depend only on waist circumference and body size, showed no between-group effect. For MOAD and the conicity index this is expected, since waist circumference and BMI changed only modestly over 12 weeks; notably, WHtR — also anthropometric, but scaling waist circumference to height rather than to a composite denominator — did show a clear group effect ($\eta^2p = 0.357$), suggesting that the choice of denominator determines how sensitive a shape index is to short-term training.

Strengths and limitations

Several limitations must be acknowledged. First, and most importantly, the design was quasi-experimental: allocation followed participant availability rather than randomisation, which limits causal inference. Although the groups were statistically comparable on most baseline variables (Table 1), the evening group had numerically less favourable values on most indices; the change-score analysis argues against regression to the mean as the explanation for the timing effect, but the between-timing differences should still be regarded as hypothesis-generating. Second, the sample was small (14 per group), female, and restricted to a narrow age range (60–65 years), limiting generalisability. Third, dietary intake was not

objectively recorded, although participants were instructed to maintain habitual eating patterns; because exercise timing and meal timing interact, unmeasured differences in chrononutrition may have contributed. Fourth, chronotype was not assessed, even though it differs systematically in people with T2DM and influences habitual activity timing [40]. Fifth, no circadian biomarkers (e.g. BMAL1, CLOCK, melatonin) were measured, so the proposed mechanism remains inferential. Sixth, the homogeneity-of-slopes assumption was not met for VAI, CRI-II, MOAD, TyG–WC and the conicity index and was borderline ($p = 0.050$ – 0.057) for AVI, HOMA-IR and CRI-I, and change scores departed from normality for most outcomes; the mixed-effects and non-parametric analyses reproduced the main conclusions, but the ANCOVA estimates for these indices should be interpreted together with Table 5. Seventh, baseline MOAD and conicity index were lower in the morning group ($p = 0.008$ and $p = 0.005$); neither index showed a between-group effect after the intervention, so these imbalances are unlikely to have influenced the main conclusions. Finally, follow-up ended 48 h after the last session, so the persistence of these adaptations is unknown.

Clinical implications and future directions

Future studies should use randomised designs with larger samples, include both sexes, control or record dietary intake and chronotype, incorporate molecular markers of the circadian clock, and compare exercise modalities and intensities across times of day. From a clinical standpoint, given the potential for post-exercise hypoglycaemia in this population, blood glucose should be monitored carefully after exercise, particularly following evening sessions.

Conclusion

Twelve weeks of moderate-intensity aerobic exercise improved adiposity, insulin-resistance and atherogenic indices in older women with type 2 diabetes irrespective of the

time of day, while most of these indices deteriorated in non-exercising controls. Evening exercise produced greater improvements in lipid-related and visceral-fat indices than morning exercise, suggesting a possible circadian influence on exercise-induced metabolic adaptation. Given the quasi-experimental design and small sample, this timing effect should be confirmed in adequately powered randomised trials.

Statements and Declarations

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Competing interests. The authors have no relevant financial or non-financial interests to disclose.

Ethics approval. All procedures were in accordance with the ethical standards of the institutional research committee and with the 1964 Declaration of Helsinki and its later amendments. The study was approved by the Research Ethics Committee of the School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran (IR.SSU.SPH.REC.1403.093), and registered with the Iranian Registry of Clinical Trials (IRCT20181212041935N1) on 18 February 2024.

Consent to participate. Written informed consent was obtained from all individual participants included in the study.

Consent to publish. Not applicable; the manuscript contains no individually identifiable data.

Data availability. The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Author contributions. All authors contributed to the study conception and design, material preparation, data collection and analysis, and the drafting and critical revision of the manuscript. zahra hemati and Shahrzad Habibi Ghahfarrokhi coordinated the study and were involved in all of its stages. All authors read and approved the final manuscript.

Table 1 Baseline characteristics of the three groups (n = 14 per group)

Variable	Control	Morning exercise	Evening exercise	F (2, 39)	p
Age (years)	62.57 ± 1.55	62.50 ± 1.91	62.21 ± 1.93	0.15	0.858
Height (cm)	158.36 ± 4.41	156.71 ± 4.75	156.64 ± 8.19	0.36	0.698
Body mass (kg)	71.86 ± 7.54	73.49 ± 9.78	72.57 ± 10.28	0.11	0.898
BMI (kg/m ²)	28.67 ± 2.82	29.85 ± 2.90	29.71 ± 4.47	0.48	0.623
Waist circumference (cm)	105.21 ± 8.93	101.77 ± 9.41	108.43 ± 8.72	1.91	0.162
Hip circumference (cm)	106.07 ± 8.40	101.14 ± 8.43	108.14 ± 8.47	2.55	0.091
HbA1c (%)	7.01 ± 0.78	7.40 ± 1.61	7.94 ± 1.57	1.61	0.212
Fasting blood glucose (mg/dL)	153.29 ± 28.50	154.07 ± 47.43	180.57 ± 44.95	1.99	0.150
Systolic blood pressure (mmHg)	125.57 ± 7.74	126.00 ± 10.24	129.50 ± 13.20	0.58	0.567
Diastolic blood pressure (mmHg)	76.07 ± 5.94	76.86 ± 6.29	80.50 ± 6.48	2.01	0.148
Triglycerides (mg/dL)	175.29 ± 40.51	164.43 ± 43.99	180.86 ± 49.70	0.48	0.619
Total cholesterol (mg/dL)	183.86 ± 31.25	189.21 ± 30.10	200.86 ± 45.84	0.80	0.458
HDL-C (mg/dL)	50.64 ± 7.90	56.79 ± 14.34	51.86 ± 8.54	1.30	0.283
LDL-C (mg/dL)	106.14 ± 34.16	93.64 ± 14.84	103.00 ± 28.93	0.80	0.457
AVI	22.30 ± 3.85	20.91 ± 3.92	23.68 ± 3.73	1.82	0.175
BAI	35.28 ± 4.51	33.56 ± 3.91	37.46 ± 6.41	2.09	0.137
WHtR	0.665 ± 0.063	0.649 ± 0.053	0.694 ± 0.065	1.97	0.154
VAI	3.38 ± 1.00	2.92 ± 1.46	3.51 ± 1.38	0.79	0.462
MOAD	1.159 ± 0.059	1.094 ± 0.074	1.172 ± 0.066	5.46	0.008
Conicity index	1.434 ± 0.083	1.366 ± 0.088	1.464 ± 0.053	6.03	0.005
HOMA-IR	7.65 ± 2.91	8.49 ± 3.66	9.06 ± 3.67	0.60	0.553
TyG	9.46 ± 0.38	9.38 ± 0.39	9.63 ± 0.43	1.46	0.245
TyG–WC	995.96 ± 94.81	955.91 ± 107.13	1045.54 ± 104.72	2.69	0.080
TyG–BMI	271.52 ± 29.83	280.04 ± 29.72	286.79 ± 48.49	0.60	0.556
AIP	0.172 ± 0.144	0.100 ± 0.215	0.171 ± 0.171	0.74	0.482
CRI-I	3.73 ± 0.97	3.52 ± 1.02	3.99 ± 1.19	0.68	0.511
CRI-II	2.18 ± 0.85	1.75 ± 0.50	2.09 ± 0.85	1.27	0.292

Values are mean ± SD. Groups were compared by one-way ANOVA. The groups were comparable on every variable except MOAD (p = 0.008) and the conicity index (p = 0.005), both lower at baseline in the morning group. AIP and VAI were calculated with triglycerides and HDL-C in mmol/L. BMI body mass index, HbA1c glycated haemoglobin, HDL-C high-density lipoprotein cholesterol, LDL-C low-density lipoprotein cholesterol, AVI abdominal volume index, BAI body adiposity index, WHtR waist-to-height ratio, VAI visceral adiposity index, MOAD model of adipose distribution, HOMA-IR homeostatic model assessment of insulin resistance, TyG triglyceride–glucose index, WC waist circumference, AIP atherogenic index of plasma, CRI Castelli’s risk index

Table 2 Pre- and post-intervention values, within-group change and within-group effect size (n = 14 per group)

Variable	Group	Pre-test	Post-test	Change	Δ (%)	p (paired t)	dz
AVI	Control	22.30 ± 3.85	22.36 ± 3.87	+0.06	+0.3	0.161	0.40
	Morning	20.91 ± 3.92	20.26 ± 3.48	-0.66	-3.1	0.001	-1.08

Variable	Group	Pre-test	Post-test	Change	Δ (%)	p (paired t)	dz
BAI	Evening	23.68 ± 3.73	22.29 ± 3.26	-1.39	-5.9	0.001	-1.09
	Control	35.28 ± 4.51	35.35 ± 4.54	+0.07	+0.2	0.165	0.39
	Morning	33.56 ± 3.91	33.00 ± 3.64	-0.55	-1.7	0.002	-1.04
WHtR	Evening	37.46 ± 6.41	35.15 ± 6.34	-2.30	-6.2	< 0.001	-1.98
	Control	0.665 ± 0.063	0.666 ± 0.063	+0.001	+0.1	0.165	0.39
	Morning	0.649 ± 0.053	0.639 ± 0.049	-0.010	-1.5	< 0.001	-1.16
VAI	Evening	0.694 ± 0.065	0.674 ± 0.064	-0.020	-2.9	< 0.001	-1.13
	Control	3.38 ± 1.00	3.84 ± 1.10	+0.46	+13.6	< 0.001	1.37
	Morning	2.92 ± 1.46	2.20 ± 1.03	-0.72	-24.6	< 0.001	-1.48
MOAD	Evening	3.51 ± 1.38	2.27 ± 0.94	-1.23	-35.1	< 0.001	-2.03
	Control	1.159 ± 0.059	1.154 ± 0.054	-0.005	-0.4	0.084	-0.50
	Morning	1.094 ± 0.074	1.091 ± 0.071	-0.004	-0.3	0.468	-0.20
Conicity index	Evening	1.172 ± 0.066	1.153 ± 0.050	-0.019	-1.6	0.034	-0.63
	Control	1.434 ± 0.083	1.429 ± 0.078	-0.005	-0.4	0.067	-0.53
	Morning	1.366 ± 0.088	1.359 ± 0.084	-0.007	-0.5	0.239	-0.33
HOMA-IR	Evening	1.464 ± 0.053	1.438 ± 0.044	-0.026	-1.8	0.017	-0.73
	Control	7.65 ± 2.91	9.31 ± 3.21	+1.66	+21.8	< 0.001	1.26
	Morning	8.49 ± 3.66	5.20 ± 2.94	-3.29	-38.7	< 0.001	-1.32
TyG	Evening	9.06 ± 3.67	5.44 ± 2.32	-3.62	-39.9	< 0.001	-2.00
	Control	9.46 ± 0.38	9.61 ± 0.41	+0.15	+1.6	< 0.001	1.19
	Morning	9.38 ± 0.39	9.05 ± 0.29	-0.33	-3.5	< 0.001	-1.80
TyG–WC	Evening	9.63 ± 0.43	9.11 ± 0.42	-0.52	-5.4	< 0.001	-2.16
	Control	995.96 ± 94.81	1013.29 ± 100.55	+17.33	+1.7	< 0.001	1.15
	Morning	955.91 ± 107.13	908.14 ± 91.48	-47.77	-5.0	< 0.001	-1.79
TyG–BMI	Evening	1045.54 ± 104.72	959.33 ± 89.61	-86.20	-8.2	< 0.001	-1.91
	Control	271.52 ± 29.83	278.25 ± 30.92	+6.72	+2.5	< 0.001	1.32
	Morning	280.04 ± 29.72	265.11 ± 28.58	-14.93	-5.3	< 0.001	-1.95
AIP	Evening	286.79 ± 48.49	264.65 ± 42.09	-22.14	-7.7	< 0.001	-2.03
	Control	0.172 ± 0.144	0.230 ± 0.141	+0.058	—	< 0.001	1.45
	Morning	0.100 ± 0.215	-0.010 ± 0.183	-0.110	—	< 0.001	-2.32
CRI-I	Evening	0.171 ± 0.171	-0.014 ± 0.177	-0.185	—	< 0.001	-2.65
	Control	3.73 ± 0.97	4.13 ± 1.05	+0.39	+10.5	< 0.001	2.18
	Morning	3.52 ± 1.02	2.91 ± 0.91	-0.61	-17.4	< 0.001	-1.72
CRI-II	Evening	3.99 ± 1.19	2.80 ± 1.04	-1.19	-29.9	< 0.001	-1.43
	Control	2.18 ± 0.85	2.32 ± 0.85	+0.14	+6.5	< 0.001	1.23
	Morning	1.75 ± 0.50	1.40 ± 0.43	-0.35	-19.8	< 0.001	-1.19
	Evening	2.09 ± 0.85	1.38 ± 0.60	-0.71	-33.8	< 0.001	-1.56

Values are mean ± SD. Change = mean post-test minus pre-test value; Δ (%) = change as a percentage of the pre-test mean (not shown for AIP, which is a logarithm of a ratio and may take negative values). dz = Cohen's d for paired data. Within-group p values are from paired-samples t-tests and are exploratory; Wilcoxon signed-rank results are given in Online Resource 1

413 Table 3 ANCOVA on post-intervention values adjusted for baseline

Variable	F (2, 38)	p	q (FDR)	η^2p	Control (adj.)	Morning (adj.)	Evening (adj.)
AVI	11.15	< 0.001	< 0.001	0.370	22.36 ± 0.20	21.51 ± 0.20	21.04 ± 0.20
BAI	34.00	< 0.001	< 0.001	0.641	35.50 ± 0.20	34.82 ± 0.20	33.18 ± 0.20
WHtR	10.56	< 0.001	< 0.001	0.357	0.670 ± 0.003	0.659 ± 0.003	0.650 ± 0.003
VAI	80.12	< 0.001	< 0.001	0.808	3.75 ± 0.10	2.46 ± 0.10	2.10 ± 0.10
MOAD	1.69	0.198	0.198	0.082	1.140 ± 0.005	1.130 ± 0.005	1.128 ± 0.005
Conicity index	1.93	0.159	0.172	0.092	1.417 ± 0.006	1.408 ± 0.007	1.400 ± 0.007
HOMA-IR	38.98	< 0.001	< 0.001	0.672	9.82 ± 0.44	5.14 ± 0.43	4.99 ± 0.44
TyG	48.49	< 0.001	< 0.001	0.718	9.64 ± 0.05	9.15 ± 0.05	8.99 ± 0.05
TyG–WC	41.21	< 0.001	< 0.001	0.684	1016.07 ± 7.77	945.94 ± 8.01	918.75 ± 8.05
TyG–BMI	48.93	< 0.001	< 0.001	0.720	285.42 ± 2.03	264.58 ± 2.02	258.00 ± 2.03
AIP	83.67	< 0.001	< 0.001	0.815	0.208 ± 0.014	0.033 ± 0.014	-0.034 ± 0.014
CRI-I	33.93	< 0.001	< 0.001	0.641	4.14 ± 0.13	3.10 ± 0.14	2.60 ± 0.14
CRI-II	35.36	< 0.001	< 0.001	0.650	2.18 ± 0.07	1.60 ± 0.08	1.32 ± 0.07

414 Adjusted values are estimated marginal means ± SE from ANCOVA with the corresponding baseline value as covariate. q
415 (FDR) = Benjamini–Hochberg-adjusted p value across the 13 outcomes; η^2p = partial eta-squared. Assumption checks are
416 reported in Online Resource 1, and sensitivity analyses in Table 5

417 Table 4 Baseline-adjusted mean differences between groups (95% confidence intervals) and Bonferroni-
418 adjusted pairwise p values

Variable	Morning – Control (95% CI)	p	Evening – Control (95% CI)	p	Evening – Morning (95% CI)	p
AVI	-0.85 (-1.43, -0.27)	0.015	-1.32 (-1.90, -0.74)	< 0.001	-0.47 (-1.06, +0.13)	0.364
BAI	-0.67 (-1.25, -0.10)	0.068	-2.31 (-2.89, -1.74)	< 0.001	-1.64 (-2.24, -1.04)	< 0.001
WHtR	-0.011 (-0.020, -0.003)	0.037	-0.020 (-0.029, -0.011)	< 0.001	-0.0085 (-0.0175, +0.0005)	0.188
VAI	-1.29 (-1.58, -1.01)	< 0.001	-1.66 (-1.94, -1.38)	< 0.001	-0.36 (-0.65, -0.08)	0.041
MOAD	-0.009 (-0.024, +0.006)	0.673	-0.012 (-0.026, +0.002)	0.258	-0.003 (-0.018, +0.012)	1.000
Conicity index	-0.010 (-0.029, +0.009)	0.915	-0.0173 (-0.0352, +0.0007)	0.177	-0.008 (-0.028, +0.013)	1.000
HOMA-IR	-4.68 (-5.94, -3.43)	< 0.001	-4.84 (-6.10, -3.57)	< 0.001	-0.15 (-1.40, +1.10)	1.000
TyG	-0.49 (-0.63, -0.35)	< 0.001	-0.65 (-0.79, -0.51)	< 0.001	-0.15 (-0.30, -0.01)	0.106
TyG–WC	-70.12 (-92.67, -47.57)	< 0.001	-97.31 (-120.02, -74.60)	< 0.001	-27.19 (-50.91, -3.47)	0.077
TyG–BMI	-20.84 (-26.65, -15.04)	< 0.001	-27.42 (-33.28, -21.55)	< 0.001	-6.57 (-12.36, -0.78)	0.082
AIP	-0.176 (-0.215, -0.136)	< 0.001	-0.243 (-0.282, -0.204)	< 0.001	-0.067 (-0.107, -0.028)	0.004
CRI-I	-1.04 (-1.43, -0.66)	< 0.001	-1.54 (-1.93, -1.15)	< 0.001	-0.50 (-0.89, -0.10)	0.044
CRI-II	-0.58 (-0.80, -0.36)	< 0.001	-0.87 (-1.08, -0.65)	< 0.001	-0.29 (-0.50, -0.07)	0.032

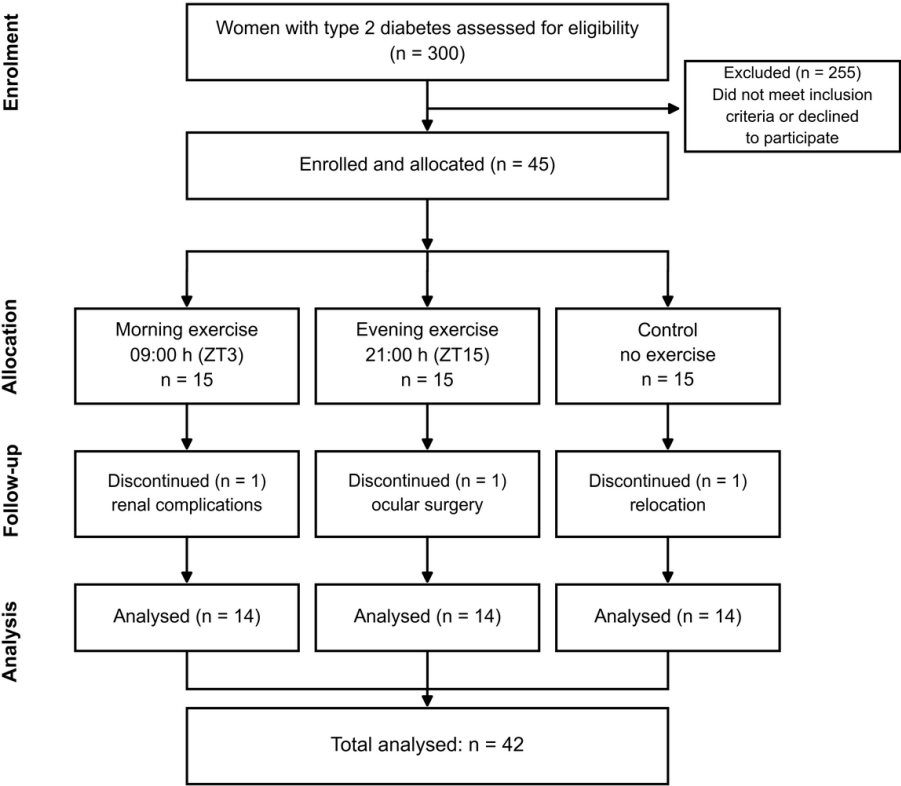
Variable	Morning – Control (95% CI)	p	Evening – Control (95% CI)	p	Evening – Morning (95% CI)	p
		0.001		0.001		

Differences are adjusted mean differences from the ANCOVA models in Table 3; negative values indicate a lower post-intervention value in the first-named group. Confidence intervals are unadjusted; p values are Bonferroni-adjusted

Table 5 Sensitivity analyses

Variable	Shapiro–Wilk (min p)	LMM group × time p	Δ ANOVA p	Δ E vs M p (t-test)	Kruskal–Wallis p	Mann–Whitney E vs M p	Hedges' g
AVI	< 0.001	< 0.001	< 0.001	0.064	< 0.001	0.124	-0.71
BAI	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001	-1.87
WHtR	< 0.001	< 0.001	< 0.001	0.061	< 0.001	0.123	-0.72
VAI	0.022	< 0.001	< 0.001	0.021	< 0.001	0.018	-0.90
MOAD	0.001	0.090	0.106	0.110	0.279	0.206	-0.61
Conicity index	0.006	0.045	0.056	0.095	0.116	0.148	-0.64
HOMA-IR	0.002	< 0.001	< 0.001	0.690	< 0.001	0.395	-0.15
TyG	0.012	< 0.001	< 0.001	0.023	< 0.001	0.046	-0.89
TyG–WC	0.043	< 0.001	< 0.001	0.011	< 0.001	0.029	-1.01
TyG–BMI	0.082	< 0.001	< 0.001	0.053	< 0.001	0.148	-0.74
AIP	0.357	< 0.001	< 0.001	0.003	< 0.001	0.005	-1.22
CRI-I	< 0.001	< 0.001	< 0.001	0.024	< 0.001	0.007	-0.88
CRI-II	< 0.001	< 0.001	< 0.001	0.019	< 0.001	0.016	-0.92

Shapiro–Wilk (min p) is the smallest p value across the three groups for within-participant change scores; values below 0.05 indicate departure from normality. LMM = linear mixed-effects model with a random intercept for participant; p is the likelihood-ratio test of the group × time interaction. Δ = raw change score (post minus pre) analysed without covariate adjustment. Hedges' g is the evening-versus-morning difference in change scores; negative values favour evening exercise. E evening exercise, M morning exercise



429
430 **Fig. 1** Flow of participants through the study

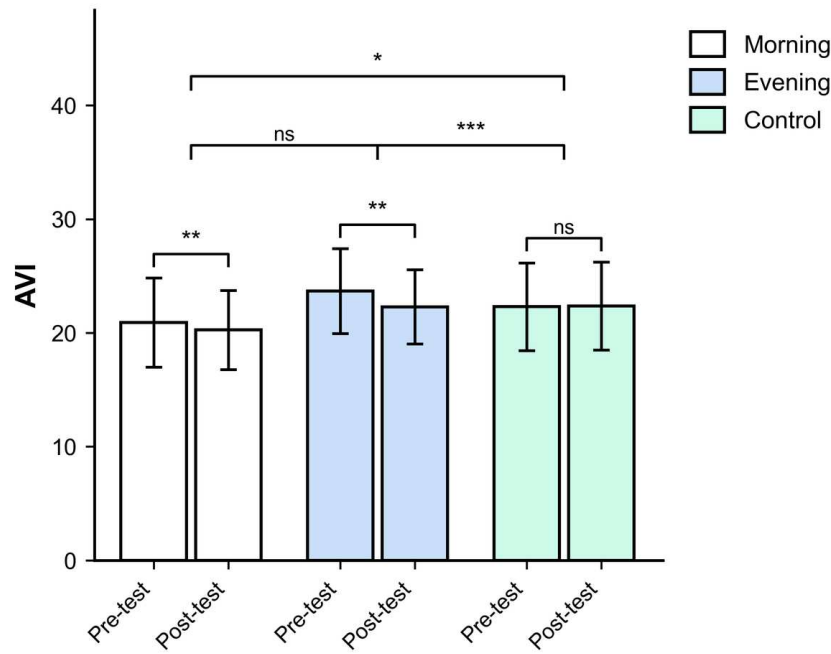


Fig. 2 Effect of morning and evening aerobic exercise on the abdominal volume index (AVI). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. *p < 0.05; **p < 0.01; ***p < 0.001; ns not significant

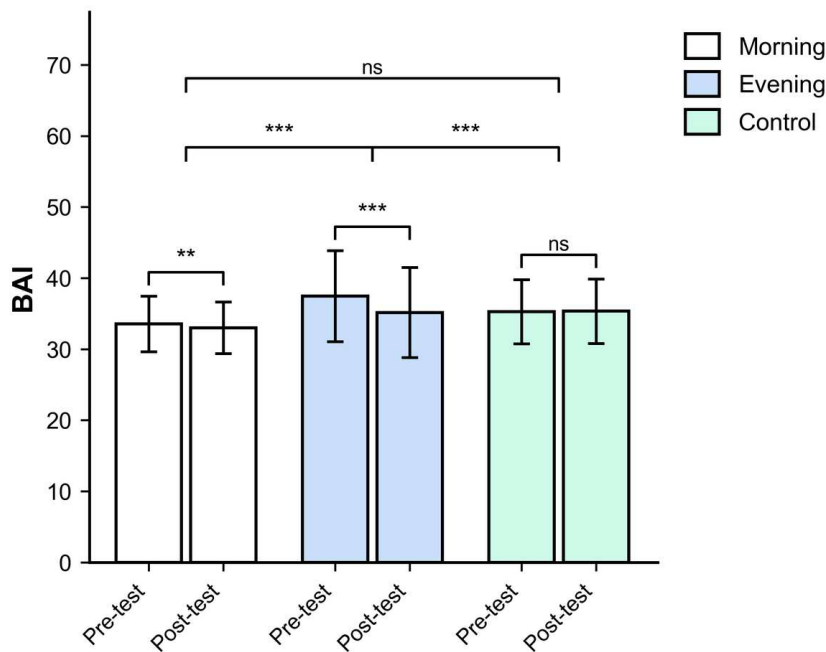


Fig. 3 Effect of morning and evening aerobic exercise on the body adiposity index (BAI). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of

baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

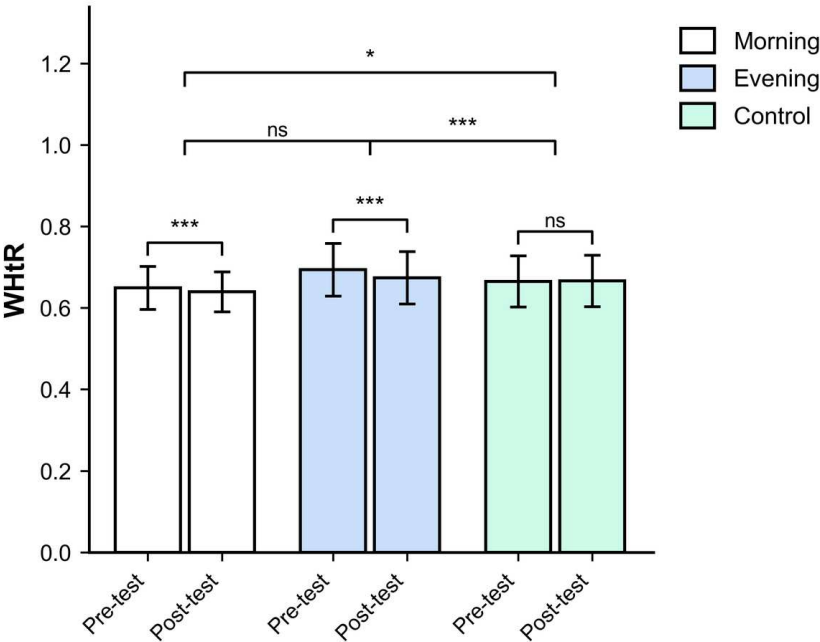


Fig. 4 Effect of morning and evening aerobic exercise on the waist-to-height ratio (WHtR). Bars are mean $\pm$ SD ($n = 14$ per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

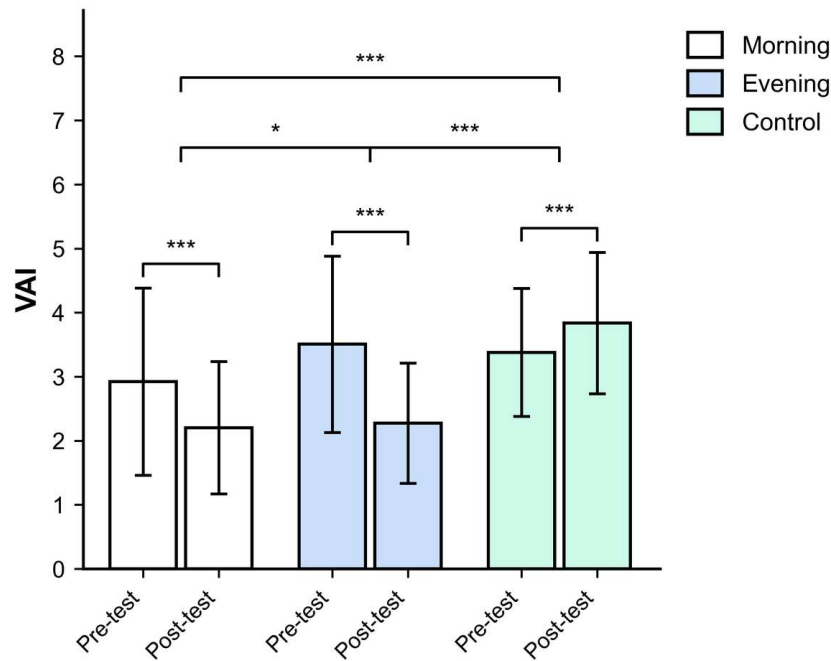


Fig. 5 Effect of morning and evening aerobic exercise on the visceral adiposity index (VAI). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. *p < 0.05; **p < 0.01; ***p < 0.001; ns not significant

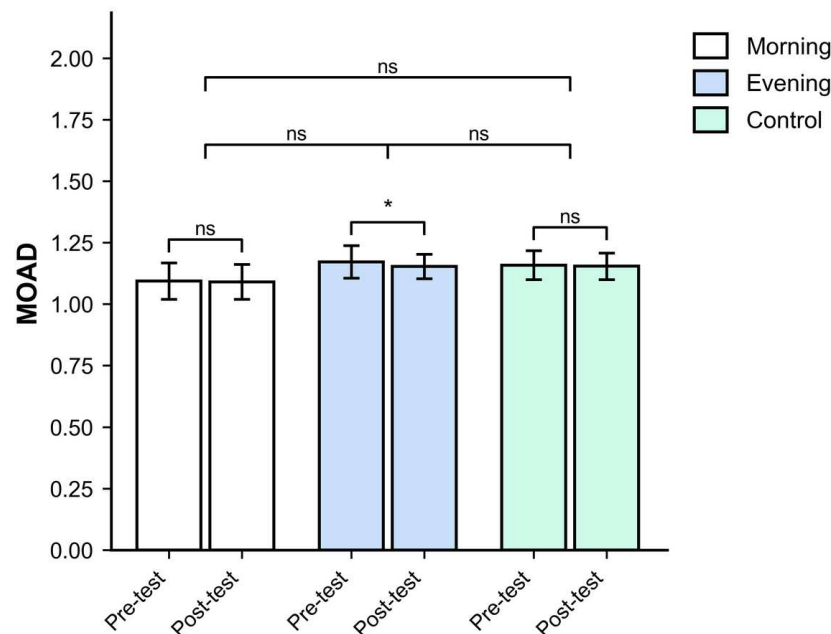


Fig. 6 Effect of morning and evening aerobic exercise on the model of adipose distribution (MOAD). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons

of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

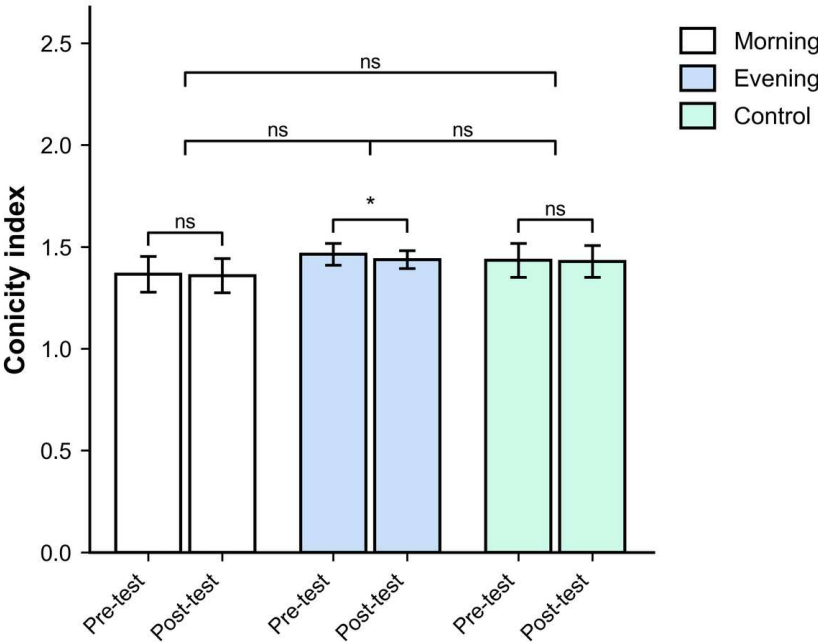


Fig. 7 Effect of morning and evening aerobic exercise on the conicity index. Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

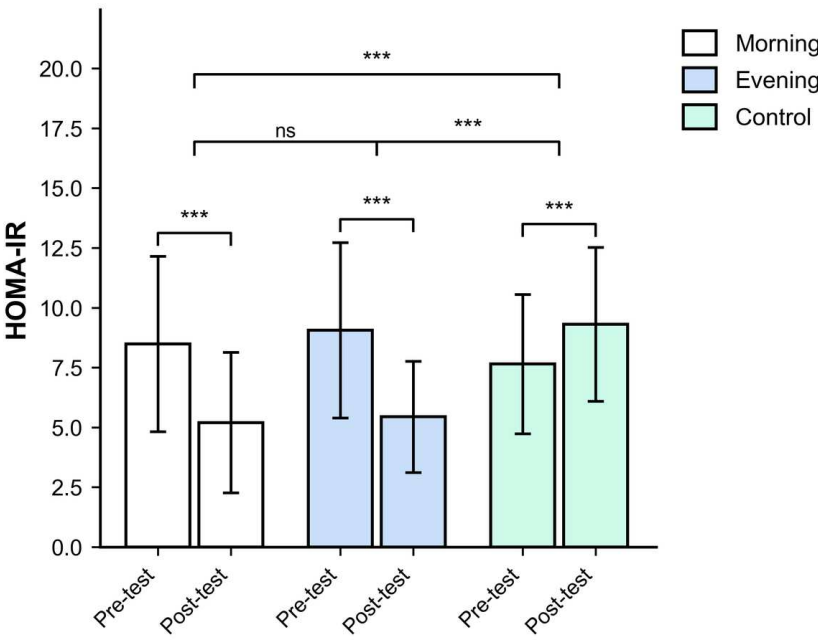


Fig. 8 Effect of morning and evening aerobic exercise on the homeostatic model assessment of insulin resistance (HOMA-IR). Bars are mean $\pm$ SD (n = 14 per group). Brackets within

each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

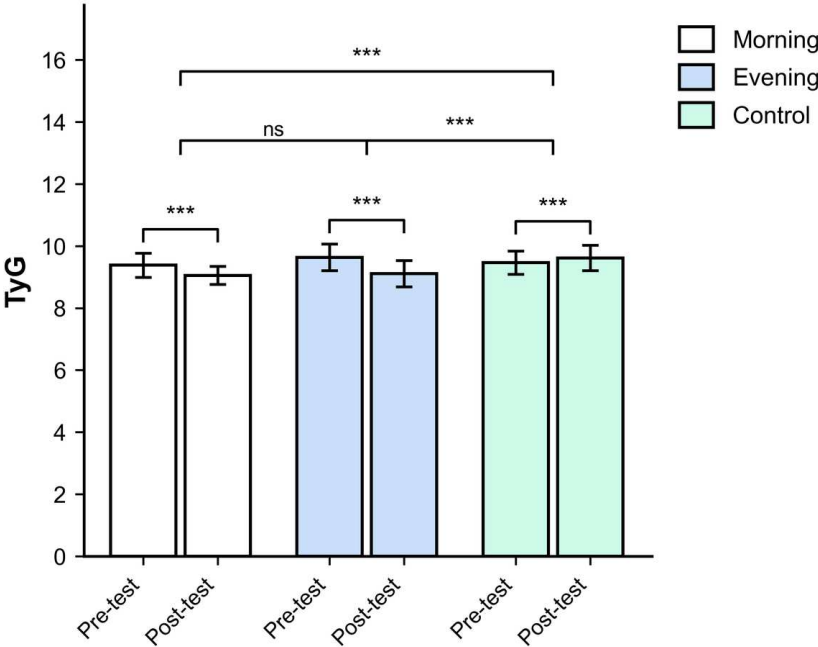


Fig. 9 Effect of morning and evening aerobic exercise on the triglyceride–glucose index (TyG). Bars are mean ± SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not significant

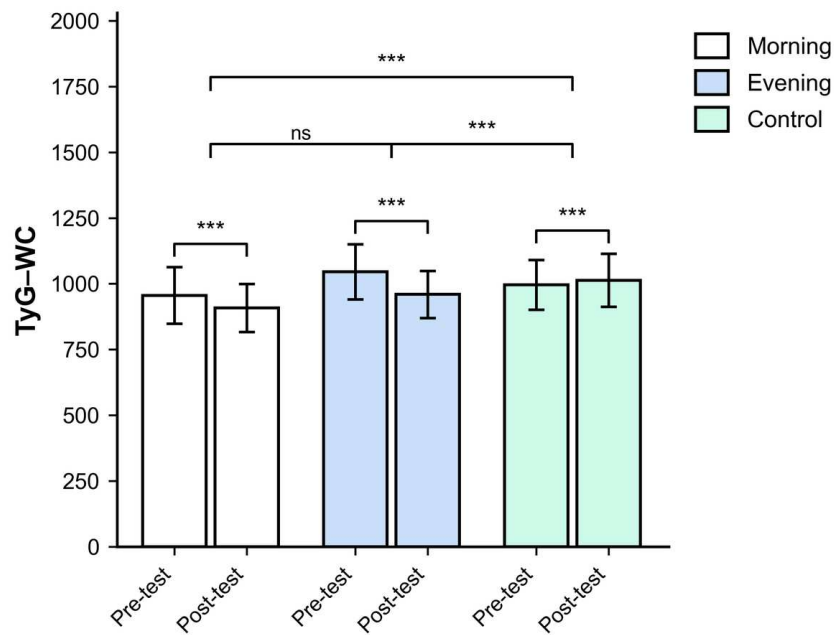


Fig. 10 Effect of morning and evening aerobic exercise on the triglyceride–glucose–waist circumference index (TyG–WC). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. *p < 0.05; **p < 0.01; ***p < 0.001; ns not significant

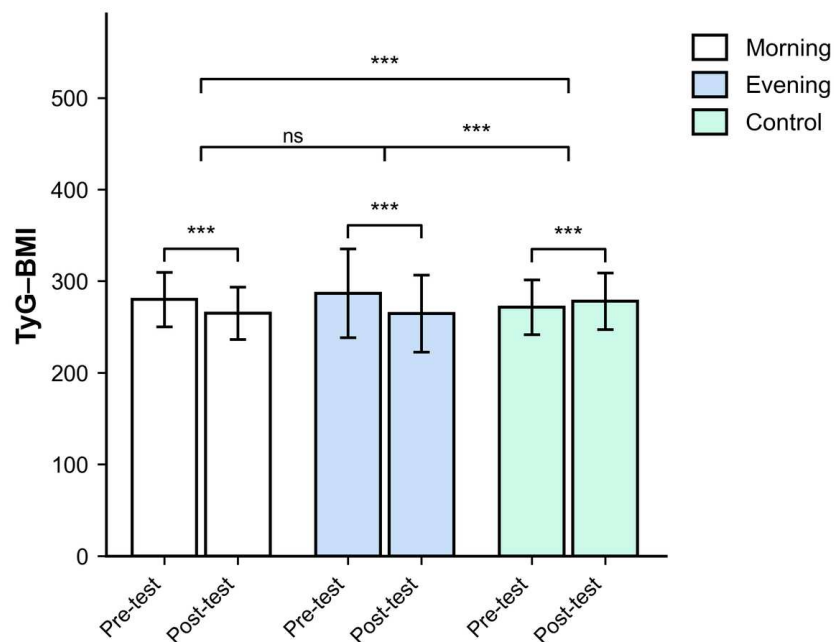
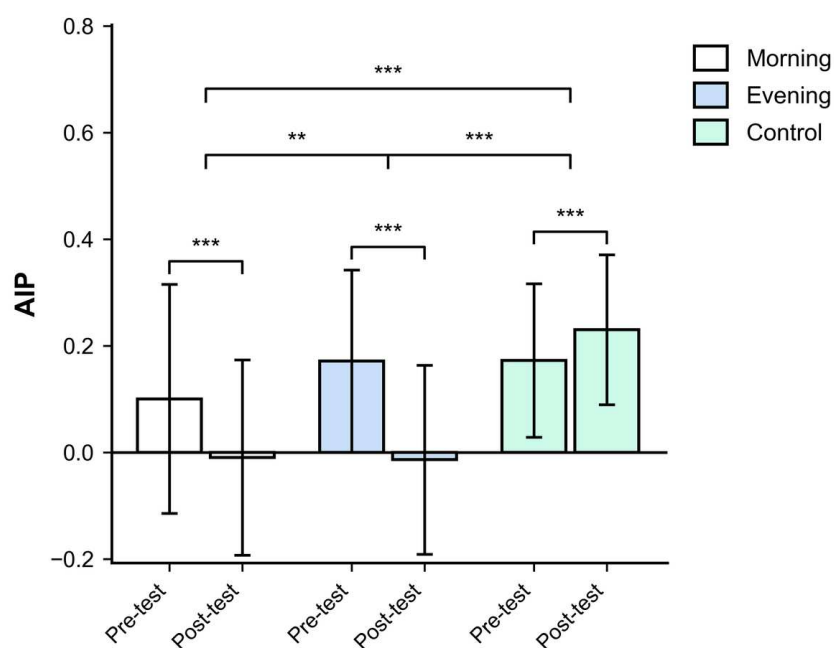


Fig. 11 Effect of morning and evening aerobic exercise on the triglyceride–glucose–body mass index (TyG–BMI). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise

488 comparisons of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p <$
 489 0.001 ; ns not significant



490
 491 **Fig. 12** Effect of morning and evening aerobic exercise on the atherogenic index of plasma
 492 (AIP). Bars are mean $\pm$ SD ($n = 14$ per group). Brackets within each group denote paired-
 493 samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons
 494 of baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not
 495 significant

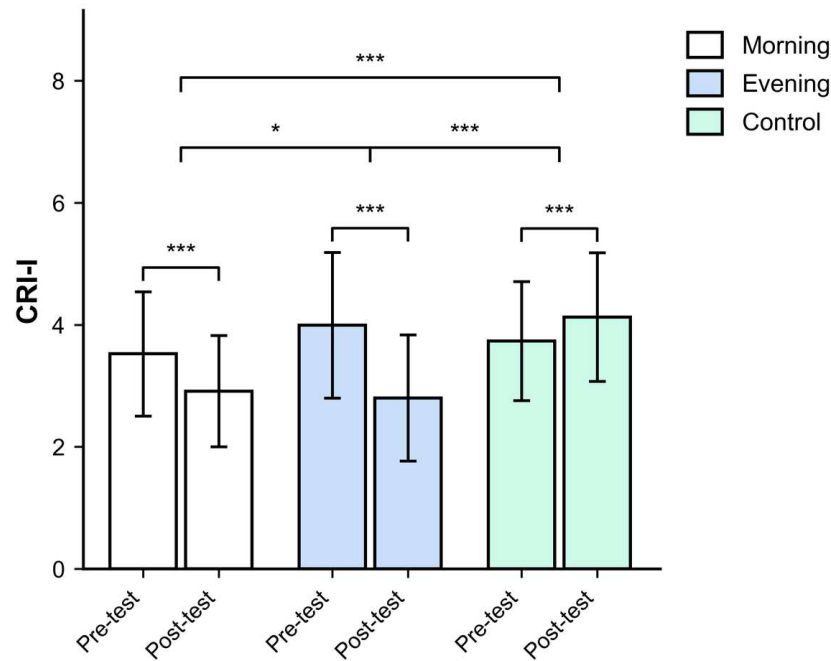


Fig. 13 Effect of morning and evening aerobic exercise on Castelli's risk index I (CRI-I). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of baseline-adjusted post-intervention means. *p < 0.05; **p < 0.01; ***p < 0.001; ns not significant

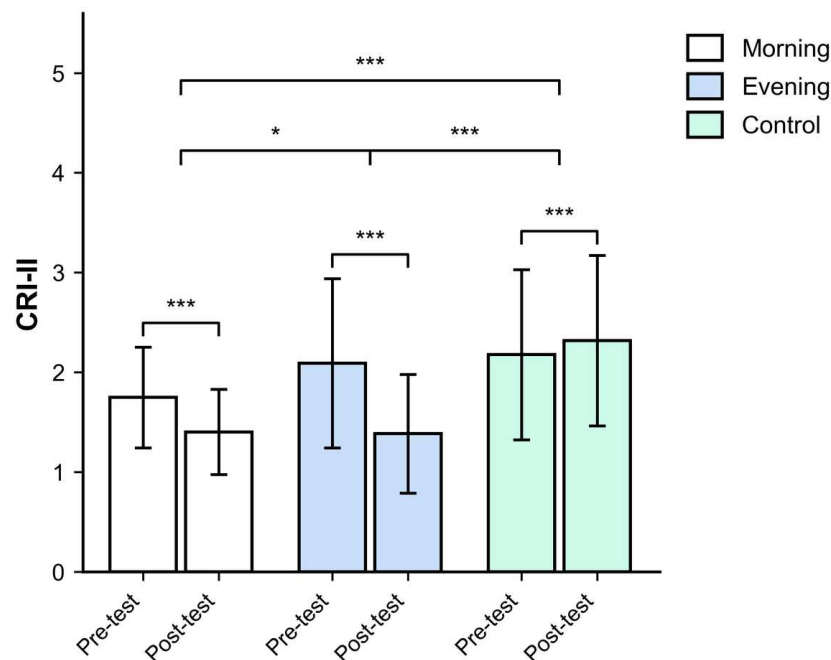


Fig. 14 Effect of morning and evening aerobic exercise on Castelli's risk index II (CRI-II). Bars are mean $\pm$ SD (n = 14 per group). Brackets within each group denote paired-samples t-tests; brackets above the groups denote Bonferroni-adjusted pairwise comparisons of

506 baseline-adjusted post-intervention means. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; ns not
507 significant

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