

Comparative evaluation of the impact of acute exercise performed in the morning and evening on serum fatty acid profiles in trained and untrained men

original paper

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ABSTRACT

Purpose. Exercise modulates lipid metabolism; however, the influence of the time of day on serum fatty acid responses remains incompletely understood. Although the effects of exercise intensity and duration are well documented, limited research has examined whether morning versus evening exercise produces different metabolic outcomes in individuals with varying training backgrounds. This study investigated the acute effects of morning compared with evening aerobic exercise on serum fatty acid profiles, including palmitic acid (C16:0), stearic acid (C18:0), oleic acid (C18:1), linoleic acid (C18:2), saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA), in trained and untrained men.

Methods. Twenty healthy men ($n = 10$ trained, $n = 10$ untrained) completed randomised morning (08:00–09:00) and evening (20:00 – 21:00) running sessions lasting approximately 45 minutes at 64–76% of their maximal heart rate (HR_{max}). Venous blood samples were collected before and after each session, and serum fatty acids were quantified using gas chromatography. A repeated-measures analysis of variance (ANOVA) was used to assess the effects of training status, exercise timing, and acute aerobic exercise.

Results. Levels of most serum fatty acids did not change significantly following exercise. However, in the untrained participants, evening exercise resulted in significant increases in C18:0 and C18:2 serum levels. Trained individuals demonstrated lower baseline C18:0 levels and lower evening SFA levels compared with the untrained participants. No significant differences were observed for C16:0, C18:1, MUFA, or PUFA levels.

Conclusions. The study findings suggest that exercise timing influences serum fatty acid responses, particularly in untrained individuals. Evening exercise increased levels of lipogenesis-related fatty acids, and trained individuals exhibited a more favourable basal lipid profile. Considering exercise timing may help optimise lipid regulation and reduce cardiometabolic risk.

Key words: exercise timing, fatty acids, serum lipids, circadian rhythm, trained *vs.* untrained

Introduction

According to the World Health Organization, health does not mean merely the absence of disease or infirmity but a state of complete physical, mental, and social well-being [1]. Evidence from clinical and sports sciences has consistently demonstrated that regular physical activity and exercise are essential determinants for maintaining this holistic concept of health [2, 3]. Indeed, exercise is a powerful tool for the prevention, treatment, and rehabilitation of cardiovascular, metabolic, and respiratory diseases [2, 4].

Discussions on exercise prescription often emphasise the parameters of intensity and duration. It is well

established that regular, moderate-to-vigorous exercise confers a broad spectrum of health benefits [5–10]. However, the “dose” of exercise is not defined solely by its intensity and duration, as the timing of exercise during the day has been increasingly recognised as an additional factor shaping physiological responses [11–14]. Yet, unlike intensity and duration, there is still no consensus in the literature regarding the “ideal time of day” to exercise.

The human body exhibits circadian rhythms across a wide range of physiological processes, including energy metabolism, cardiovascular regulation, endocrine responses, and cognitive-behavioural functions. The time of day can influence acute and chronic adaptations to

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exercise through changes in autonomic balance, hormonal secretion patterns, body temperature, glucose and lipid utilisation, and muscle performance [15, 16]. Notably, night exercise may uniquely interact with circadian regulation, thereby altering recovery, sleep architecture, and metabolic outcomes [16, 17].

In this context, an important but still unresolved question is whether performing the same exercise protocol at different times of the day elicits distinct effects on metabolic biomarkers. Among these, the serum fatty acid profile, including saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), and key fatty acids, such as C16:0, C18:0, C18:1, and C18:2, represents a sensitive indicator that may be influenced by both exercise timing and training status (trained *vs.* untrained individuals) [18, 19]. Chronic training induces fundamental adaptations in fatty acid mobilisation, oxidation, and re-esterification [20], while the superimposed effect of exercise timing may further modulate the magnitude and direction of acute responses [21, 22].

Although the influence of exercise on lipid metabolism is well established [18, 19], few studies have systematically compared how morning *versus* night exercise alters the serum fatty acid profile in individuals with different training backgrounds. The interaction between training status and exercise timing remains poorly understood, leaving a gap in the evidence base that this study seeks to address [23].

Understanding these mechanisms is not only of academic interest but also highly relevant to public health. Identifying optimal exercise timing to favourably modulate serum fatty acids could contribute to more effective strategies in the prevention and management of obesity, type 2 diabetes, and cardio-metabolic diseases that continue to impose a substantial global health burden [24].

The present study moves beyond the traditional focus on exercise intensity and duration by positioning timing as a central variable. To the best of our knowledge, it is among the first investigations to directly compare the acute effects of morning and night exercise on serum fatty acid profiles in both trained and untrained individuals under controlled conditions [21]. Specifically, we investigated the acute effects of morning *versus* night exercise on the serum fatty acid profile in trained and untrained individuals. In doing so, we aimed to strengthen the evidence base for the “time” component of the three central questions of exercise science – *how intense, how long, and at what time of day?* and to contribute to the development of time-sensitive recommendations for both clinical practice and performance optimisation.

Material and methods

Participants

The study included 20 healthy male volunteers: 10 trained subjects (20.6 ± 0.1 years, 62.6 ± 2.5 kg, 178 ± 3.6 cm) and 10 untrained individuals (20.8 ± 0.1 years, 64.3 ± 2.6 kg, 175 ± 2.4 cm). Participants were instructed to refrain from strenuous physical activity for 24 hours prior to each exercise session.

Inclusion and exclusion criteria

Participants were between 18–25 years of age and free from acute conditions such as respiratory infections or muscle inflammation, as well as chronic diseases including diabetes, obesity, cardiovascular disorders, and musculoskeletal diseases. Individuals who smoked, consumed alcohol, or used regular medication were excluded.

Trained group ($n = 10$) included licensed amateur or professional athletes who had been training regularly for at least three years, whereas untrained group ($n = 10$) included individuals with no history of regular exercise in the previous year, meeting the same health-related inclusion criteria.

Exercise protocol

Each participant performed treadmill running sessions in both the morning (08:00–09:00) and evening (20:00–21:00), in randomised order. At least three days of recovery were allowed between sessions. Participants consumed a light meal at least three hours before exercise and avoided caffeine, energy drinks, supplements, and other ergogenic aids.

Each session lasted approximately 45 minutes. Exercise intensity was determined using the Karvonen method and adjusted to 64–76% of maximal heart rate, as recommended by the American College of Sports Medicine (ACSM) [25, 26]. Heart rate was monitored continuously using a Polar Heart Rate Monitor (Polar T31-Coded, Hong Kong, China).

Blood sampling and fatty acid analysis

Blood samples were collected from the antecubital vein immediately before and within two minutes after exercise. Samples were centrifuged at 4,500 rpm for 10 min at 4°C, and serum was stored at –80°C until analysis.

Serum fatty acids were analysed by gas chromatography (GC) following lipid extraction and methylation. Total lipids were extracted using the Folch method (chloroform-methanol, 2:1 *v/v*). The extracted lipids were then converted to fatty acid methyl esters (FAMEs) with boron trifluoride in methanol.

Analyses were performed using an Agilent 6890N Gas Chromatograph equipped with a flame ionization detector (FID) and a DB-23 capillary column (60 m × 0.25 mm internal diameter × 0.25 µm film thickness; Agilent Technologies, California, USA). Helium was used as the carrier gas at a constant flow rate of 1.0 ml/min. The injector and detector temperatures were maintained at 250°C, and the oven temperature was programmed from 150°C to 230°C at a rate of 4°C/min.

Fatty acids were identified by comparing their retention times with those of authentic standards (Supelco 37 Component FAME Mix, Sigma-Aldrich, Pennsylvania, USA) and expressed as the percentage of total identified fatty acids. The following fatty acids were quantified: palmitic acid (C16:0), stearic acid (C18:0), oleic acid (C18:1), linoleic acid (C18:2), and total saturated (SFA), monounsaturated (MUFA), and polyunsaturated (PUFA) fatty acids.

Statistical analysis

A priori power analysis was conducted using G*Power software (version 3.1; Heinrich Heine University, Düsseldorf, Germany). Assuming a medium effect size ($f = 0.25$), an alpha level of 0.05, and a statistical power of 0.80, a minimum of 18 participants was required. To compensate for possible dropouts, 20 participants (10 trained and 10 untrained) were included in the study.

All statistical analyses were performed using SPSS (version 26.0; IBM Corp., Armonk, NY, USA). Data are expressed as mean ± standard deviation (SD). The Shapiro-Wilk test was used to assess normality of data distribution.

A three-way repeated-measures analysis of variance (ANOVA) was used to evaluate the effects of Group (trained *vs.* untrained; between-subjects), Time of day (morning *vs.* evening; within-subjects), and Exercise (pre- *vs.* post-exercise; within-subjects) on serum fatty acid concentrations. When significant main or interaction effects were detected, Bonferroni-adjusted post hoc tests were applied. Effect sizes were reported as partial eta squared (η^2), and statistical significance was set at $p < 0.05$.

Results

C16:0 (palmitic acid)

In both groups, there was no significant effect of morning or evening exercise on C16:0 serum level. In trained subjects, basal and post-exercise C16:0 levels were 26.21 ± 2.15 *vs.* 26.00 ± 2.32 in the morning ($p = 0.8$) and 26.39 ± 2.87 *vs.* 25.17 ± 2.56 in the evening ($p = 0.2$). Baseline C16:0 values were higher in the untrained group than in the trained group, but the difference was not statistically significant ($p = 0.37$). In the untrained group, values were 27.27 ± 3.00 *vs.* 27.79 ± 2.07 in the morning ($p = 0.6$) and 29.08 ± 3.12 *vs.* 29.47 ± 2.19 in the evening ($p = 0.6$).

C18:0 (stearic acid)

The impact of morning exercise on C18:0 levels differed between the groups. A non-significant decrease was observed in the trained group following morning exercise (8.29 ± 1.26 *vs.* 7.33 ± 0.94 , $p = 0.5$) and evening exercise (7.89 ± 1.06 *vs.* 7.63 ± 0.86 , $p = 0.5$).

However, in untrained individuals, a non-significant increase was observed following morning exercise (9.87 ± 1.09 *vs.* 10.08 ± 1.71 , $p = 0.5$), but a significant increase was observed following evening exercise (10.54 ± 1.17 *vs.* 11.32 ± 1.25 , $p = 0.03$). Statistically significant differences in baseline C18:0 levels were observed between untrained and trained individuals during morning exercise ($p = 0.007$) and evening exercise ($p = 0.007$).

C18:1 (oleic acid)

The impact of morning and evening exercises on C18:1 levels did not differ between the groups. In the trained group, they were at 20.32 ± 2.08 *vs.* 20.36 ± 2.29 ($p = 0.93$) in the morning and 21.34 ± 1.92 *vs.* 20.61 ± 2.21 ($p = 0.33$) in the evening. In the untrained group, the respective values were at: 21.61 ± 2.49 *vs.* 21.76 ± 2.93 ($p = 0.79$) for morning; 20.45 ± 2.72 *vs.* 19.58 ± 3.97 ($p = 0.33$) for evening.

There were no statistically significant differences in baseline C18:1 levels between untrained and trained individuals during morning exercise ($p = 0.22$) or evening exercise ($p = 0.40$).

C18:2 (linoleic acid)

Morning and evening exercises did not significantly affect C18:2 levels in the trained group: 42.38 ± 4.47

vs. 43.25 ± 3.67 ($p = 0.40$) for the morning; 39.56 ± 6.72 vs. 41.36 ± 3.06 ($p = 0.45$) for the evening. However, a varied response in C18:2 levels was observed in the untrained group depending on exercise time: a non-significant decrease was noted in the morning (40.23 ± 7.13 vs. 37.79 ± 4.71 , $p = 0.27$) but a significant increase was found in the evening (36.95 ± 5.44 vs. 40.25 ± 6.62 , $p = 0.01$).

In the trained participants, baseline C18:2 levels were not significantly affected by exercise time, and evening exercise also produced no change. In the untrained individuals, however, evening exercise led to a significant increase in C18:2 levels.

PUFA

Morning (44.52 ± 4.30 vs. 44.96 ± 4.32 , $p = 0.72$) and evening (42.79 ± 5.53 vs. 45.21 ± 4.13 , $p = 0.2$) exercise had no significant impact on PUFA levels in the trained group. Similarly, morning (41.49 ± 6.07 vs. 39.23 ± 4.60 , $p = 0.20$) and evening (38.94 ± 5.17 vs. 40.30 ± 6.56 , $p = 0.21$) exercise did not significantly affect PUFA levels in the untrained group. The comparisons of baseline values showed no significant differences between the trained and untrained individuals.

MUFA

There were no statistically significant differences in MUFA levels in the trained and untrained individuals during morning exercise (20.84 ± 2.33 vs. 21.54 ± 2.75 ; 22.13 ± 2.93 vs. 22.59 ± 3.48 , respectively) or evening exercise (22.15 ± 2.37 vs. 21.28 ± 2.57 ; 21.11 ± 2.73 vs. 20.14 ± 4.03 , respectively). Baseline MUFA values were also not significantly different between the trained and untrained participants.

SFA

There were no statistically significant differences in SFA levels during morning exercise (trained: 34.64 ± 2.42 vs. 33.49 ± 2.40 ; untrained: 37.37 ± 2.55 vs. 38.06 ± 2.93) or evening exercise (trained: 35.05 ± 4.38 vs. 33.51 ± 2.96 ; untrained: 40.08 ± 3.57 vs. 39.67 ± 3.61) in the trained and untrained individuals. However, baseline SFA levels in the morning ($p = 0.01$) and evening ($p = 0.01$) were significantly higher in the untrained group than in the trained group (Figure 1).

Discussion

The present study investigated the acute effects of morning *versus* evening exercise on serum fatty acid

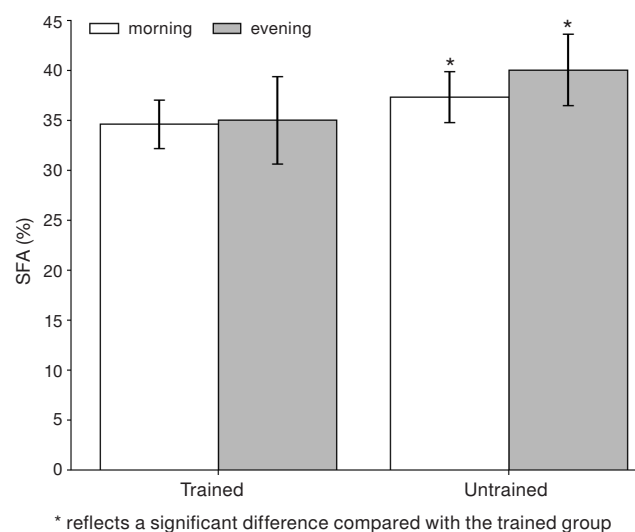


Figure 1. Baseline saturated fatty acid (SFA) values in the trained and untrained groups

profiles in trained and untrained individuals. Circadian regulation of autonomic activity, hormone secretion, and substrate utilisation influences metabolic responses to exercise throughout the day [21, 22]. The primary finding is that exercise timing *per se* does not uniformly alter circulating fatty acid composition following a single bout of moderate-intensity exercise. Instead, the metabolic response appears to depend on both training status of an individual and fatty acid specificity. While most fatty acid parameters (C16:0, C18:1, total SFA, MUFA, and PUFA) remained unchanged across time-of-day conditions, selective and physiologically meaningful alterations were observed in C18:0 and C18:2, particularly in untrained individuals following evening exercise.

The overall stability of major fatty acid classes is consistent with the concept that acute moderate-intensity exercise elicits tightly regulated lipid responses aimed at preserving systemic homeostasis. Previous works have demonstrated that significant perturbations in circulating fatty acids typically occur under conditions of prolonged exercise, high-intensity workloads, or increased metabolic demand such as fasting [18, 27]. In this context, the absence of significant changes in total SFA, MUFA, and PUFA in both trained and untrained groups suggests that the applied exercise stimulus (64–76% HR_{max}, ~45 min) may not have exceeded the threshold required to disrupt steady-state lipid turnover. This interpretation is further supported by evidence indicating that circulating fatty acid levels reflect a balance between adipose tissue lipolysis, skeletal muscle uptake, and hepatic re-esterification processes, all of which are tightly coordinated during moderate exercise [18].

The fatty acid-specific responses observed in the present study provide important mechanistic insights. The significant increase in C18:0 following evening exercise in the untrained individuals may indicate an imbalance between fatty acid mobilisation and oxidative capacity. Untrained individuals typically exhibit reduced mitochondrial density, lower oxidative enzyme activity, and diminished capacity for β -oxidation, which can result in transient accumulation of circulating saturated fatty acids under metabolic stress [28]. Importantly, this response was observed only during evening exercise, suggesting a potential interaction with circadian regulation. Circadian variations in hormonal milieu – particularly reduced insulin sensitivity and altered cortisol rhythms in the evening – may impair substrate utilisation efficiency and favour the accumulation of certain lipid species [29–31].

A similar pattern was evident for C18:2 (linoleic acid), which increased significantly following evening exercise exclusively in the untrained group. This essential polyunsaturated fatty acid plays a critical role in membrane structure and inflammatory signalling pathways. Its increase may reflect enhanced lipolytic release from adipose tissue or altered incorporation into circulating lipid pools. Notably, the trained individuals did not exhibit these changes, supporting the concept of “metabolic flexibility,” defined as the ability to efficiently switch between lipid and carbohydrate oxidation in response to physiological demands [31]. Chronic exercise training is known to enhance mitochondrial function, upregulate fatty acid transport proteins (e.g., CD36), and improve enzymatic control of lipid oxidation pathways, thereby stabilising circulating lipid levels across varying conditions [31, 32].

Another key observation is the significantly higher baseline SFA levels in sedentary individuals compared to trained participants. This finding aligns with a large body of evidence linking physical inactivity to impaired lipid metabolism and increased cardio-metabolic risk. Elevated circulating saturated fatty acids have been associated with insulin resistance, low-grade inflammation, and endothelial dysfunction. Regular endurance training, on the other hand, promotes enhanced fatty acid oxidation and reduces reliance on circulating lipid pools, thereby contributing to a more favourable metabolic profile [27, 28]. The absence of differences in MUFA and PUFA levels between the studied groups may suggest that these fractions are either less sensitive to training status in acute settings or more strongly influenced by dietary intake, which was not fully controlled in the present study.

Contrary to some previous reports indicating diurnal variation in substrate utilisation, the present findings demonstrate limited time-of-day effects on circulating fatty acids. This discrepancy may be explained by methodological differences, including exercise intensity, nutritional standardisation, and participant characteristics. Importantly, pre-exercise nutritional intake was standardised in this study, which may have attenuated circadian fluctuations in substrate availability and utilisation. Additionally, moderate exercise intensity may not be sufficient to amplify underlying circadian differences in metabolic pathways [29–31].

From a mechanistic standpoint, the findings suggest that acute fatty acid responses are predominantly governed by intrinsic metabolic capacity rather than circadian timing alone. Nevertheless, the selective sensitivity of C18:0 and C18:2 to evening exercise in the untrained individuals highlights a potential interaction between circadian biology and lipid metabolism. This interaction may involve clock gene expression, time-of-day-dependent variation in key metabolic regulators (e.g., AMPK, CPT-1), and hormonal oscillations, although these pathways were not directly assessed in the present study [31, 32]. A previous study has shown that night exercise can enhance lipid utilisation and improve systemic metabolic flexibility [33].

Several limitations should be acknowledged. First, the relatively small sample size may limit statistical power, particularly for detecting subtle interaction effects. Second, the inclusion of only young male participants restricts the generalisability of the findings to other populations, including females, older adults, and clinical cohorts. Third, although pre-exercise conditions were standardised, habitual dietary intake was not strictly controlled, which may have influenced baseline fatty acid composition. Finally, the study focused on acute responses; therefore, the long-term effects of exercise timing on fatty acid metabolism remain to be determined.

Despite these limitations, this study provides novel and clinically relevant insights into the interaction between exercise timing and training status. By demonstrating that fatty acid responses are largely stable but selectively modulated in untrained individuals during evening exercise, the findings contribute to a more nuanced understanding of time-dependent metabolic regulation.

Conclusions

In conclusion, acute moderate-intensity exercise does not induce substantial changes in the overall se-

rum fatty acid profile, regardless of whether it is performed in the morning or evening. However, specific fatty acids, particularly C18:0 and C18:2, exhibit time-dependent alterations in untrained individuals, with significant increases observed following evening exercise. These findings suggest that training status is a primary determinant of fatty acid metabolism, while the influence of exercise timing is modest and fatty acid-specific.

Practical implications

From a practical perspective, these findings indicate that exercise timing is not a critical determinant of overall lipid metabolism in healthy young men performing moderate-intensity exercise. Therefore, adherence, consistency, and total exercise volume should remain the primary considerations in exercise prescription.

However, in untrained individuals or those with reduced metabolic flexibility, evening exercise may induce less favourable acute lipid responses, particularly in saturated and polyunsaturated fatty acids. In such populations, morning exercise may represent a more metabolically stable alternative, although this hypothesis requires confirmation in longitudinal studies.

For trained individuals, the absence of timing-dependent differences suggests a high degree of metabolic adaptability, allowing flexibility in training schedules without compromising lipid metabolism. This has important implications for both athletic performance and lifestyle-based exercise interventions.

Future perspectives

Future studies should explore chronic adaptations to exercise timing, incorporate diverse populations, and investigate underlying molecular mechanisms, including circadian gene expression and enzyme activity, to further elucidate the role of timing in metabolic regulation. Expanding research to include women, older adults, and clinical populations will also be essential for broader applicability. Combining lipidomic profiling with molecular markers of lipid metabolism (e.g., desaturase and elongase activity, mitochondrial function) could further elucidate the mechanisms behind time-of-day differences. Ultimately, integrating chronobiology with exercise science holds promise for developing time-sensitive, personalised exercise recommendations to improve metabolic health.

Data availability statement

The datasets generated and analysed during the current study are not publicly available due to privacy and ethical restrictions, but are available from the corresponding author upon reasonable request.

Ethical approval

The research related to human use complied with all the relevant national regulations and institutional policies, followed the tenets of the Declaration of Helsinki, and was approved by the Van Yuzuncu Yil University ethical committee (approval No.: 11.03.2014-05/20).

Informed consent

All participants provided written informed consent after receiving a detailed explanation of the study procedures, potential risks, and benefits.

Conflict of interest

The authors declare no conflicts of interest.

Disclosure statement

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