

Research Article

The effect of aerobic interval-style continuous training combined with CoQ10 supplementation on MDA and TGF- β levels in inactive obese men

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Abstract

Obesity is associated with chronic low-grade inflammation and elevated oxidative stress. This study investigated the independent and combined effects of an 8-week aerobic interval-style continuous training program and CoQ10 supplementation on serum TGF- β and MDA concentrations in inactive obese men. Sixty inactive obese men (aged 30–45 years) were randomly assigned to four groups (n=15 each): control (CONT), CoQ10 supplementation (SUP; 100 mg/day), training (TR), and combined training + supplementation (TR+SUP). The supervised training protocol was performed three sessions per week for 8 weeks with progressive intensity. Fasting venous blood samples were collected before and after the intervention. Serum MDA was measured using colorimetric assay, and TGF- β was quantified via ELISA. Data was analyzed using two-way repeated-measures ANOVA. Significant time $\times$ exercise interactions were observed for both MDA ($F(1,56)=28.793$, $p<0.001$) and TGF- β ($F(1,56)=30.617$, $p<0.001$), with reductions in the exercise groups (MDA: exercise groups from 210 nmol/L to 156 nmol/L; TGF- β : from 35 nmol/L to 30 pg/mL). Time $\times$ supplementation interactions were also significant but smaller (MDA: $\eta^2_p = 0.082$; TGF- β : $\eta^2_p = 0.068$). No significant three-way interaction was detected for either marker. These findings highlight aerobic interval-style continuous training as a cornerstone intervention for mitigating obesity-related oxidative stress and fibrotic signaling. CoQ10 supplementation produced a small independent reduction in both markers, but no synergistic or additive interaction with exercise was observed.

Key Words: Interval training, Coenzyme Q10, Malondialdehyde, Transforming growth factor-beta

Introduction

Epidemiological data indicate that obesity affects over 650 million people globally, positioning it as a major public health crisis (Górczyńska-Kosiorz et al, 2024; Asjodi et al, 2024). Sedentary lifestyles significantly contribute to the onset of this condition and its related issues, such as metabolic syndrome (Saleh Fard et al, 2021). At the molecular level, obesity's development involves ongoing mild inflammation and heightened oxidative stress, evidenced by elevated reactive oxygen species and lipid peroxidation markers like malondialdehyde. These changes lead to impaired endothelial function and disruptions in fibrotic signaling, notably through alterations in transforming growth factor beta, culminating in fibrosis and structural changes within adipose tissue (Sharma, 2014; Saydi et al, 2024).

Obesity-driven oxidative stress primarily stems from mitochondrial impairments. Rising leptin concentrations enhance transforming growth factor beta signaling, which in turn fosters fibrosis by activating the Smad pathway (Morawietz et al, 2023). Research in models of obesity demonstrates that coenzyme Q10, a fat soluble antioxidant essential for the mitochondrial electron transport chain, offers protection against oxidative stress. It operates via mechanisms that include neutralizing reactive oxygen species and lowering lipid peroxidation indicators, including malondialdehyde. Clinical evidence further reveals that coenzyme Q10 supplementation in those with excess weight reduces inflammatory signals and enhances lipid profiles (Haghighi et al, 2023). Interval training, often referred to as aerobic interval training or intermittent aerobic exercise, involves alternating periods of moderate-to-high intensity effort with recovery phases, and has gained recognition as an effective, time-efficient strategy for addressing obesity-related metabolic disturbances in sedentary populations. In sedentary obese individuals, interval training pr-

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-otocols have been shown to significantly reduce serum malondialdehyde (MDA) levels (a reliable marker of lipid peroxidation and oxidative stress) while concurrently elevating total antioxidant capacity (saydi et al., 2024).

Moreover, interval training influences fibrotic pathways relevant to obesity-induced adipose tissue remodeling (Heidari et al, 2016). For instance, interval protocols have been associated with modulation of transforming growth factor beta (TGF- β) signaling (Czarkowska-Paczek et al., 2011). Emerging evidence points to the combined application of coenzyme Q10 and physical activity yielding strong synergistic benefits for health promotion (Talebi et al, 2024). Despite these insights, the precise workings of this combined approach remain unclear. Limited research has explored the joint impact of CoQ10 supplementation and interval training on oxidative stress indicators, including malondialdehyde, and inflammatory fibrotic elements like transforming growth factor beta in populations with obesity and sedentary lifestyles. The present research aims to address this shortfall by examining the influence of a combined interval training and coenzyme Q10 intervention on malondialdehyde levels and their association with transforming growth factor beta alterations.

Materials and Methods

Subjects

This pre-post quasi-experimental study examined the effect of simultaneous coenzyme Q10 supplementation and interval training on fibrotic and oxidative stress indices in inactive obese men aged 30 to 45. The study was conducted in Tehran, Iran, and all procedures were in accordance with the ethical principles set forth in the Declaration of Helsinki.

Criteria for participant's selection and exclusion

To be included in the study, males between 30 and 45 years old had to be inactive and obese, non-smokers, and not follow a particular diet or exercise regimen in the six months before the study, and no chronic metabolic disease (such as cancer, gastrointestinal problems, diabetes, or renal failure) or orthopedic issues. Inclusion criteria were presented as bullet points: Age 30–45 years, Body mass index (BMI) ≥ 30 kg/m², No participation in regular physical activity for at least 6 months, Non-smoker, No chronic metabolic or orthopedic diseases.

Participants were also instructed to stick to their regular eating habits without any modifications and to avoid any supplements or medications during the research period. no dietary logging was performed.

Non-adherence to the exercise or vitamin intake guidelines, such as missing more than two straight exercise sessions, metabolic or gastrointestinal issues brought on by Q10 capsules and incidence of sports-related injuries. Also, anyone who got long-term stomach or metabolic problems or physical injury during the intervention time was taken out of the study.

Sample size and statistical power

The sample size for this study was calculated using G*Power software (version 3.1) based on a two-way repeated measures analysis of variance (ANOVA), accounting for the between-subjects factor of group (4 levels) and the within-subjects factor of time (2 measurements: pre- and post-intervention). The priori power analysis assumed an alpha level (α) of 0.05, a minimum effect size (f) of 0.40 (corresponding to a large effect), and a desired statistical power of 0.80 (Omidali et al, 2026). This yielded an estimated total sample size of 56 participants. To account for potential attrition, the final recruitment target was set at 60 individuals. Participants were recruited from the population of sedentary, overweight adult men residing in Tehran using purposive sampling. Following screening, 60 eligible individuals (who were free of chronic diseases, non-smokers, without recent history of structured exercise or specific dietary regimens, and without diagnosed metabolic or musculoskeletal disorders) were enrolled. Participants provided written informed consent after a detailed briefing session in which the study purpose, procedures, potential risks, and benefits were fully explained.

Eligible participants were randomly allocated to one of four groups (n=15 per group): Control group (CONT), Coenzyme Q10 supplementation only (SUP), Aerobic interval-style continuous training only (TR), Combined interval training+Coenzyme Q10 supplementation (TR+SUP)

The entire study protocol was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Supplementation

Participants in the SUP and TR+SUP groups received a daily 100 mg capsule of CoQ10 (Dana Pharmaceutical Company, Tehran, Iran; product code: COQ-100) for 8 weeks. The dose of 100 mg/day was selected based on previous studies showing efficacy in reducing oxidative stress markers in overweight and obese populations (Raygan et al, 2016). although most evidence comes from metabolic syndrome patients.

Exercise training

The TR and TR+SUP groups performed supervised aerobic interval-style continuous training three times per week for 8 weeks in a controlled gym environment. Each session began with a 10-minute warm-up (low-intensity walking and dynamic stretch-

Table 1. The training program in this study.

Weeks	Intervals	Intensity (% of Maximum Heart Rate)	Rest Between Intervals	Total Activity Time
Weeks 1–2	3 × 5 min	55%	3 min inactive rest	15 min
Weeks 3–4	2 × 10 min	55–60%	3 min rest	20 min
Weeks 5–6	2 × 15 min	65–70%	5 min rest	30 min
Weeks 7–8	2 × 20 min	70–75%	5 min rest	40 min

-ing) and ended with a 10-minute cool-down (slow walking and static stretching). The core training consisted of progressive interval-style running, with intensity prescribed as a percentage maximum heart rate ($HR_{max}=220-\text{age}$) and monitored continuously using Polar heart rate monitors (Polar H10, Polar Electro Inc., Kempele, Finland). The protocol started at moderate intensity (55% HR_{max}) to ensure safety and adherence in sedentary obese individuals, progressing to 70-75%. We acknowledge that this intensity range (55-75% HR_{max}) is lower than traditional interval training (85-95% HR_{max}); therefore, we have renamed the intervention to "aerobic interval-style continuous training" throughout the manuscript (Sadeghifar et al, 2024). The detailed protocol is presented in Table 1. All sessions were supervised by a qualified trainer, and participants who missed more than two consecutive sessions were excluded.

Biological and physical evaluations

Anthropometric assessments (body mass, waist circumference, BMI, and body fat percentage) were performed at baseline and post-intervention. Fasting venous blood samples (5 mL) were collected pre- and post-intervention. Colorimetric assay kits (made by ZellBio Germany, distributed by Farateb) were used to measure MDA. TGF- β was quantified using ELISA kits from Arman Biotech Company.

Statistical analysis

In SPSS version 26, data were examined using descriptive and inferential statistics including two-way analysis of variance (ANOVA) and Bonferroni post hoc test. Graphs were created with GraphPad Prism 8. The statistical significance level was established as $\alpha = 0.05$.

Result

The normality of the data was confirmed using the Shapiro-Wilk test (all $p>0.05$), indicating that the distributions of serum MDA and TGF- β levels were normal across all groups at both pre- and post-intervention time points. Baseline characteristics (age, height, body mass, BMI) showed no significant differences across groups (all $p>0.05$) (Table 2).

MDA

There was a significant main effect of time ($F(1,56) = 41.236, p < 0.001, \eta^2_{sub>p</sub>= 0.424$). A significant time $\times$ exercise

interaction was observed ($F(1,56)=28.793, p<0.001, \eta^2_{sub>p</sub>=0.340$). A significant time $\times$ supplement interaction was also found ($F(1,56)=5.031, p=0.029, \eta^2_{sub>p</sub>=0.082$). No significant three-way interaction was detected ($F(1,56)=0.022, p=0.882, \eta^2_{sub>p</sub>< 0.001$) (Figure 1).

MDA mean difference: -54.07 nmol/L (95% CI: -72.14 to -36.00), $p < 0.001$. Post-hoc examination of the time $\times$ exercise interaction revealed that the exercise groups exhibited a marked decrease in MDA levels (from 210.23 $\pm$ 6.15 nmol/L to 156.16 $\pm$ 6.90 nmol/L), whereas the no-exercise groups displayed only a minimal decrease (from 196.31 $\pm$ 6.15 nmol/L to 191.47 $\pm$ 6.90 nmol/L).

Post-hoc examination of the time $\times$ supplement interaction showed that the supplement groups demonstrated a greater decrease in MDA levels (from 210.71 $\pm$ 6.15 nmol/L to 170.97 $\pm$ 6.90 nmol/L) compared to the no-supplement groups (from 195.83 $\pm$ 6.15 nmol/L to 176.66 $\pm$ 6.90 nmol/L). These results suggest that both exercise and supplementation independently contributed to the reduction in serum MDA levels, with exercise having a stronger effect (as evidenced by the larger eta squared value), and no synergistic or antagonistic interaction between them (Figure 1).

TGF- β

There was a significant main effect of time ($F(1,56)=22.500, p< 0.001, \eta^2_{sub>p</sub>=0.287$). A significant time $\times$ exercise interaction was observed ($F(1,56)=30.617, p<0.001, \eta^2_{sub>$

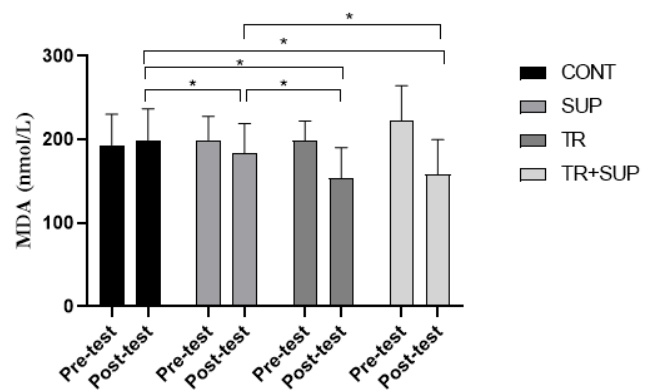


Figure1. MDA changes levels in the four groups before and after intervention. *indicate a significant difference between groups ($p<0.05$).

Table 2. Anthropometric characteristics of participants across groups.

Group	Test	Age (years)	Height (cm)	Body Mass (kg)	BMI (kg/m²)	Body Fat (%)
CONT	Pre	43.44 ± 3.20	174.75	93.42 ± 2.91	30.59 ± 0.76	29.83 ± 0.36
	Post		174.75	93.71 ± 2.86	30.69 ± 0.79	29.52 ± 0.46
SUP	Pre	44.01 ± 6.90	175.01	94.54 ± 1.86	30.93 ± 0.39	29.93 ± 0.47
	Post		175.01	94.45 ± 2.42	30.90 ± 0.49	29.67 ± 0.87
TR	Pre	44.04 ± 5.27	174.91	94.46 ± 2.07	30.87 ± 0.45	29.71 ± 0.67
	Post		174.91	91.75 ± 1.85	29.99 ± 0.38	29.99 ± 0.47
TR+SUP	Pre	44.22 ± 3.29	175.50	94.54 ± 1.80	30.74 ± 0.86	28.38 ± 0.54
	Post		175.50	92.21 ± 1.51	29.98 ± 0.85	26.57 ± 0.75

Table 3. Mean ± SD of Serum MDA and TGF-β across intervention groups.

Variable	Test	CONT	SUP	TR	TR+SUP
MDA (nmol/L)	Pre-test	193.33 ± 37.10	199.28 ± 28.34	198.32 ± 23.69	222.15 ± 42.48
	Post-test	199.47 ± 37.36	183.47 ± 35.68	153.85 ± 36.52	158.47 ± 41.29
TGF-β (pg/mL)	Pre-test	33.69 ± 4.12	33.52 ± 4.90	35.29 ± 4.79	34.17 ± 4.54
	Post-test	35.06 ± 3.57	32.82 ± 4.45	31.60 ± 4.80	29.12 ± 5.72

$p</sub>^2=0.353$). A significant time×supplement interaction was also found ($F(1,56)=4.066$, $p=0.049$, $\eta</sub>p</sub>^2 = 0.068). No significant three-way interaction was detected ($F(1,56) = 0.175$, $p=0.677$, $\eta</sub>p</sub>^2=0.003) (Figure 2). TGF-β mean difference: -4.37 pg/mL (95% CI: -6.02 to -2.72), $p < 0.001$. Examination of the estimated marginal means for the exercise×supplement combinations (averaged across time) revealed the following overall levels: control: 34.37; supplement-only: 33.17; exercise-only: 33.44; combined: 31.65. This pattern suggests modestly lower overall TGF-β levels in the combined group, though differences were not statistically significant. Post-hoc examination of the time×exercise interaction revealed that the exercise groups exhibited a marked decrease in TGF-β levels (from 34.73±0.84 to 30.36±0.86), whereas the no-exercise groups showed a slight increase (from 33.61±0.84 to 33.94±0.86). Post-hoc examination of the time×supplement interaction showed that the supplement groups demonstrated a greater decrease in TGF-β levels (from 33.85±0.84 to 30.97±0.86) compared to the no-supplement groups (from 34.49±0.84 to 33.33±0.86) (Figure 2).$$

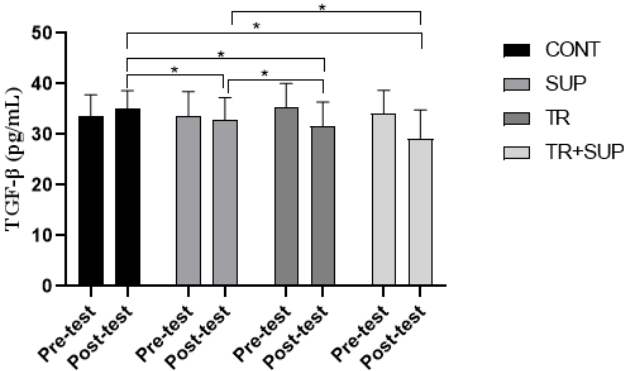


Figure2. TGF-β changes levels in the four groups before and after intervention. *indicate a significant difference between groups ($p<0.05$).

Discussion

The findings from the present study indicate that eight weeks of aerobic interval-style continuous training was associated with lower serum TGF-β levels in inactive obese men. However, supplementation with CoQ10, either alone or in combination with training, did not yield a synergistic effect on TGF-β levels, suggesting that the intervention's impact was primarily driven by the exercise component. These results are consistent with previous research demonstrating that aerobic exercise protocols can modulate TGF-β family members in obesity contexts. For instance, interval resistance training at varying intensities significantly decreased TGF-β1 levels in males with obesity, alongside improvements in cardiometabolic risk factors, potentially through reduced adipose tissue inflammation and enhanced metabolic adaptations (Czarkowska-Paczek et al., 2011). Although some animal studies have reported similar findings (Xu et al., 2022; Dropmann et al., 2016), the present study focuses on human data, and caution is warranted when extrapolating from animal models to human obesity.

Similarly, aerobic interval training has been shown to reduce TGF-β1 gene expression in subcutaneous adipose tissue of rats fed a high-fat diet, preventing increases in fat cell size and highlighting its role in mitigating obesity-induced fibrosis (Czarkowska-Paczek et al., 2011). Although our study focused on aerobic interval training, the observed reduction in TGF-β may similarly stem from training-induced decreases in fat mass, improved insulin sensitivity, and modulation of glycemic indices, as hyperglycemia is a known inducer of TGF-β1 via pathways such as diacylglycerol-protein kinase C and glucosamine (Zhang et al., 2020). In overweight men, interval training protocols have also been shown to prevent significant declines in serum TGF-β2 levels, supporting maintenance of anti-fibrotic or adaptive responses in adipose tissue (Dropmann et al., 2016). Furthermore, high-intensity interval training ameliorates renal &

liver fibrosis in aging and diabetic models by suppressing the TGF- β 1/Smad pathway, reducing collagen deposition, and alleviating pro-fibrotic signaling (Zhang et al., 2020). Conversely, our results contrast with some earlier studies where certain exercise modalities, such as resistance or prolonged continuous exercise, increased TGF- β expression, possibly due to acute hypoxic responses or temporary activation of hypoxia-inducible factor-1 (HIF-1), which upregulates TGF- β mRNA without necessarily altering protein content (Czarkowska-Paczek et al., 2011). The discrepancy may arise from differences in exercise protocols (e.g., interval vs. continuous), duration, intensity, or participant characteristics (e.g., sedentary obese men vs. young active individuals), underscoring the context-dependent nature of TGF- β responses to physical activity (Han et al., 2021).

For MDA, a marker of lipid peroxidation and oxidative stress, interval training significantly lowered levels (mean post-intervention: 158.48 nmol/L in trained groups vs. 188.98 nmol/L in non-trained; $p=0.001$), with a moderate effect size, alongside increased total antioxidant capacity (TAC) and reduced hydrogen peroxide (H_2O_2). This supports the role of regular interval training in enhancing antioxidant defenses and mitigating oxidative damage in obesity and sedentary states. These findings are consistent with studies showing that aerobic interval or intermittent training protocols reduce serum MDA and improve oxidative stress profiles in obese or sedentary populations, often by bolstering endogenous antioxidant systems such as superoxide dismutase (SOD) and catalase (CAT) (Delwing-de Lima et al., 2017). For example, high-intensity interval training has been demonstrated to decrease MDA content in tissues like the testes and liver of obese mice, with greater reductions compared to moderate-intensity continuous training, alongside enhancements in antioxidant enzyme activities (Saydi et al., 2024). In human studies, interval training protocols reduced plasma MDA in overweight and obese individuals, indicating a decrease in oxidative stress. Chronic interval training adaptations appear to outweigh transient acute increases in oxidative stress observed in some short-term or high-intensity protocols (Attarzadeh et al., 2020). Contrary to our hypothesis, CoQ10 supplementation, alone or combined with training, did not produce statistically significant additive benefits beyond exercise alone. This contrasts with some studies where CoQ10 reduced MDA in specific clinical populations but aligns with divergent studies showing no change, potentially due to variations in dosage (e.g., 100 mg/day), participant health status, or body weight/obesity degree (Gholami et al., 2018).

Notably, cross-talk between oxidative stress pathways and TGF- β signaling appears relevant here. Although we did not directly measure NF- κ B or other mediators, it is plausible that interval training may disrupt this interplay by enhancing antioxidant capa-

-city and reducing lipid peroxidation, thereby indirectly suppressing TGF- β expression (Han et al., 2021)

Limitations and future directions

The study lacks direct measurements of glycemic control or key inflammatory mediators (e.g., NF- κ B, TNF- α), which could clarify underlying mechanisms. No dietary logging was performed; therefore, changes in antioxidant intake from food sources cannot be ruled out as a confounding variable. The fixed CoQ10 dose (100 mg/day) may have been suboptimal for synergy. The exercise protocol (55-75% HRmax) is lower in intensity than traditional interval training; therefore, the intervention was renamed "aerobic interval-style continuous training." Future work should examine dose-response relationships, longer interventions, and diverse populations using multi-omics to dissect organ cross-talk more precisely. Trial registration was not obtained prospectively (IRCT); this is acknowledged as a limitation, and the study was conducted as an exploration trial.

Conclusion

This study shows that 8 weeks of aerobic interval-style continuous training was associated with lower serum levels of TGF- β and MDA in inactive obese men. These findings highlight the potential anti-fibrotic and antioxidant effects of regular moderate-to-high intensity exercise in this population. CoQ10 supplementation at 100 mg/day produced a small independent reduction in both markers, but no synergistic or additive interaction was observed when combined with training, as indicated by non-significant three-way interaction effects. This indicates that the exercise stimulus itself appears to be the primary driver of the observed improvements. Overall, these findings reinforce aerobic interval-style continuous training as a potential non-pharmacological strategy for counteracting obesity-related oxidative stress and fibrotic signaling, with CoQ10 offering small independent effects at the tested dose.

What is already known on this subject?

Obesity is associated with elevated MDA (oxidative stress marker) and upregulated TGF- β signaling (fibrotic pathway). Interval training improves antioxidant defenses while CoQ10 neutralizes ROS. However, evidence on their combined effects in sedentary obese men is limited.

What this study adds?

This is among the first studies to show that 8 weeks of aerobic interval-style continuous training significantly reduces serum MDA and TGF- β in inactive obese men, while CoQ10 (100 mg/day) provides small independent effects without synergistic interaction with exercise.

Organ Cross-Talk Tips:

- Moderate-intensity intermittent training activates skeletal muscle signaling to adipose tissue and endothelium, enhancing antioxidant defense.

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None.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Compliance with ethical standards

Conflict of interest The authors declare that there is no conflict of interest in the present research.

Ethical approval The study protocol was approved by the Ethics Committee of Islamic Azad University, Varamin-Pishva Branch (ethics code: IR.IAU.VARAMIN.REC.1403.026). All procedures were performed in accordance with the ethical standards of the Declaration of Helsinki and its later amendments.

Informed consent Performed.

Author contributions

Conceptualization: P.A., Methodology: A.B., Software: V.I., Validation: H.Kh., Formal analysis: P.A., Investigation: V.I., Resources: H.Kh., Data curation: H.Kh., Writing - original draft: p.A., Writing-review & editing V.I., Visualization: P.A., Supervision: A.B., Project administration: V.I., Funding acquisition: P.A.

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