Research Article

The effect of moderate-intensity intermittent training and coenzyme Q10 supplementation on serum VEGF, total antioxidant capacity, and hydrogen peroxide in sedentary obese men

Peyman Akraminia[1], Abdolali Banaiefar[1*], Vahid Imanipour[2], Hadi Khoran[3]

Abstract

Sedentary obesity is associated with chronic inflammation, oxidative stress, and impaired angiogenesis, leading to endothelial dysfunction. Interval training and CoQ10 each improve redox balance and vascular health, possibly via inter-organ crosstalk, but their combined effects in sedentary obese men are unclear. Sixty sedentary obese men (30–45 years) were randomized into four groups (n=15): control, CoQ10 (100 mg/day), moderate-intensity intermittent training (3×/week, 8 weeks), and training + CoQ10. Serum VEGF, TAC, and H_2O_2 were measured pre/post-n via ELISA and colorimetric assays. Data were analyzed with repeated-measures ANOVA (α=0.05). Effect sizes (partial η²) were reported. Training significantly raised VEGF ($p<0.001$, $\eta^2=0.363$) and TAC ($p<0.001$, $\eta^2=0.290$), while strongly reducing H_2O_2 ($p<0.001$, $\eta^2=0.520$). CoQ10 showed no significant main or interactive effects on TAC or H_2O_2; however, a significant three-way interaction (time×exercise×supplement) was observed for VEGF ($p=0.001$, $\eta^2=0.187$). Post-hoc analysis revealed that the exercise-only group showed a greater increase in VEGF compared to the combined group, though the direct comparison between TR and TR+SUP at post-intervention did not reach statistical significance ($p>0.05$). Eight weeks of interval training robustly improves VEGF and redox homeostasis in sedentary obese men, possibly through myokines/exerkines enhancing muscle–adipose–endothelial crosstalk and HIF-1α/VEGF signaling. CoQ10 provides no added benefit for most outcomes and may be associated with a modest attenuation of the exercise-induced VEGF response.

Key Words: Training, Coenzyme Q10, Vascular endothelial growth factor, Total antioxidant capacity, Hydrogen peroxide.

Introduction

Obesity has reached epidemic proportions, affecting more than 650 million adults worldwide (WHO, 2021), and is strongly exacerbated by sedentary lifestyles that promote metabolic syndrome and its complications (Saleh Fard et al., 2021). At the molecular level, the pathogenesis of obesity is closely linked to persistent low-grade inflammation and oxidative stress. Elevated levels of reactive oxygen species (ROS) (Asjodi et al., 2024), particularly hydrogen peroxide (H_2O_2), coupled with reduced total antioxidant capacity (TAC), provide biochemical evidence of this imbalance (Colak & Jain, 2021). These changes lead to endothelial dysfunction and disruptions in angiogenesis pathways, prominently manifested through altered vascular endothelial growth factor (VEGF) signaling (Radak et al., 2016).

Exercise-induced myokines and exerkines (exercise-induced signaling molecules) are believed to play a key role in these adaptations (Leal et al., 2018; Zhou et al., 2024). Interval training has emerged as one of the most effective exercise modalities for counteracting these obesity-related impairments (Sawyer et al., 2016). Compared with moderate continuous training, interval training produces superior improvements in endothelial function, capillary density, and physiological angiogenesis in obese individuals (Kolahdouzi et al., 2019; Khalafi et al., 2022). These benefits are largely mediated by inter-organ crosstalk, particularly the skeletal muscle–adipose–endothelial axis. It is hypothesized that these benefits are mediated by inter-organ crosstalk, particularly the skeletal muscle–adipose–endothelial axis. During interval training, contracting skeletal muscle releases a variety of myokines and exerkines (e.g., IL-6, irisin, FGF21) that act in an endocrine and paracrine manner to orchestrate communication between skeletal muscle, adipose tissue, and vascular endothelium (Leal et al., 2018; Zhou et al., 2024). This proposed signaling could, in turn, upregulate hypoxia-inducible factor 1-alpha (HIF-1α) &

1. Department of Exercise Physiology, ST.C., Islamic Azad University, Tehran, Iran. 2. Department of Physical Education, Pa.C., Islamic Azad University, Parand, Iran. 3. Department of Physical Education, ZARA.C., Islamic Azad University, Markazi, Iran.

*Author for correspondence: a.Banaeifar@iau.ir

P A: 0000-0002-1918-1846, A B: 0000-0002-3115-2467, V I: 0000-0002-1896-5439, H Kh: 0000-0003-1539-2513

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VEGF pathways, promoting healthy capillary sprouting and reducing adipose inflammation (Guo et al., 2024). In obese animal models, interval training has been shown to increase adipose VEGF-A expression, improve insulin sensitivity, and enhance vascular remodeling specifically through this muscle–adipose–endothelial communication (Kolahdouzi et al., 2019). Human studies similarly demonstrate that interval training elevates circulating VEGF and improves flow-mediated dilation more effectively than traditional endurance exercise, underscoring the role of exerkine-mediated crosstalk in obesity-related vascular-oxidative dysfunction (Habibi Maleki et al., 2020; Jost et al., 2025). Coenzyme Q10 (CoQ10), a lipophilic mitochondrial antioxidant, has also shown promise in mitigating oxidative stress (Haghighi et al., 2023). It participates in the electron transport chain, scavenges ROS, increases TAC, reduces H_2O_2, activates the Nrf2 pathway, suppresses NF-κB and TNF-α, and may support endothelial nitric oxide synthase (eNOS) while inhibiting inducible nitric oxide synthase (iNOS), potentially influencing VEGF signaling (Zhai et al., 2017; Mantle et al., 2025). Some evidence suggests synergistic effects when CoQ10 is combined with exercise, particularly in improving recovery from oxidative stress, modulating redox-sensitive pathways, and regulating vascular biomarkers, possibly by protecting mitochondrial function across muscle, adipose, and endothelial cells (Astani et al., 2022).

Despite these individual benefits, the exact mechanisms and outcomes of combining interval training and CoQ10 on oxidative stress markers (H_2O_2 and TAC) and VEGF in sedentary obese men—a high-risk group—remain poorly understood, particularly regarding how supplementation might modulate or interfere with exercise-induced exerkine-mediated inter-organ crosstalk. The present study was conducted to address this gap by investigating the effects of an 8-week combined intervention of interval training and coenzyme Q10 supplementation on VEGF, TAC, and H_2O_2 markers in sedentary obese men.

Materials and Methods

Subjects

Sixty sedentary obese men aged 30–45 years (BMI ≥ 30 kg/m²) were recruited from Tehran, Iran. Inclusion criteria included sedentary lifestyle (no regular exercise or dietary changes in the previous 6 months), non-smoking status, absence of chronic metabolic diseases or orthopedic limitations, and willingness to maintain habitual diet and avoid supplements/medications (except for short-term illness). Exclusion criteria were non-adherence (missing more than two consecutive exercise sessions), adverse reactions to CoQ10, or exercise-related injuries.

Sample size was calculated using G*Power software (version 3.1) for a two-way repeated-measures ANOVA (4 groups × 2 time points), with α=0.05, power=0.80, and effect size f=0.40 (Omidali et al, 2026)., yielding 56 participants. This was increased to 60 to account for potential dropout. Participants were selected via purposive sampling and randomly assigned to four groups (n=15 each): control (CONT), CoQ10 supplementation only (SUP), interval training only (TR), and interval training+CoQ10 (TR+SUP). All provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki.

Supplementation

Participants in the SUP and TR+SUP groups received a daily 100 mg capsule of CoQ10 (Dana Pharmaceutical Company, Iran) for 8 weeks. This dose was selected based on previous studies showing potential antioxidant effects in metabolic syndrome populations (Raygan et al, 2016). Compliance was monitored through weekly telephone calls and self-report logs.

Exercise training

The TR and TR+SUP groups performed supervised moderate-intensity intermittent 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 stretching) and ended with a 10-minute cool-down (slow walking and static stretching). The core training consisted of progressive interval running, with intensity prescribed as a percentage of maximum heart rate (220–age) and monitored continuously using Polar H10 heart rate monitors (Polar Electro Inc., USA). 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 (Sadeghifar et al, 2024).

Measurements

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. Participants were instructed to maintain their habitual diet and physical activity levels outside of the intervention. No formal dietary monitoring was performed, which is a limitation. Serum was separated by centrifugation and analyzed for VEGF (ELISA kit, Arman Biotech), H_2O_2, and TAC (colorimetric assay kits, ZellBio, Germany).

Statistical analysis

Data were analyzed using SPSS version 26. Two-way repeated-measures ANOVA was employed with time (pre- vs. post-intervention) as the within-subjects factor and exercise (yes/no)

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

and supplementation (yes/no) as between-subjects factors, followed by Bonferroni post-hoc tests. The post-hoc comparison between TR and TR+SUP at post-intervention for VEGF was specifically extracted, including the 95% confidence interval for the difference. Effect sizes were reported as partial eta squared (η-p^2). Graphs were prepared with GraphPad Prism 8. Statistical significance was set at $p<0.05$.

Result

The normality of the data was confirmed using the Shapiro-Wilk test (all $p > 0.05$), indicating that the distributions of serum VEGF, TAC, and H_2O_2 levels were normal across all groups at both pre- and post-intervention time points. Baseline anthropometric measures, including age, height, body mass, BMI, and body fat percentage, showed no statistically significant differences across the four groups (all $p > 0.05$) (Table 2), confirming successful randomization and group equivalence at the start of the intervention. Following the 8-week period, modest reductions in body mass and BMI were observed in the exercise groups (TR and TR+SUP), while the non-exercise groups (CONT and SUP) exhibited minimal or no changes. Body fat percentage displayed slight declines in both exercise conditions, with no notable shifts in the control or supplementation-only groups (Table 2).

Pre- and post-intervention values for serum VEGF, TAC, and H_2O_2 are presented in Table 3. Descriptive statistics indicated baseline similarity across groups for all variables. Post-intervention, clear directional changes emerged primarily in the exercise-involved groups.

To evaluate the effects, repeated-measures ANOVA was applied, with time (pre- vs. post-intervention) as the within-subjects factor and exercise (performed vs. not performed) and supplementation (received vs. not received) as between-subjects factors.

VEGF

A significant main effect of time was detected ($F(1,56) = 37.987$, $p < 0.001$, $\eta^2 = 0.404$, large effect). A significant time × exercise interaction emerged ($F(1,56) = 31.921$, $p < 0.001$, $\eta^2 = 0.363$, large effect). The time × supplement interaction was not statistically significant ($F(1,56) = 0.120$, $p = 0.730$, $\eta^2 = 0.002$). However, a significant three-way interaction (time × exercise × supplement) was present ($F(1,56) = 12.890$, $p = 0.001$, $\eta^2 = 0.187$, medium-to-large effect).

Baseline VEGF levels were not significantly different across groups ($p>0.05$ for all pairwise comparisons). The exercise-only group (TR) exhibited the most substantial rise in VEGF (from 17.89±3.71 to 26.17±6.62 pg/mL). The combined group (TR+SUP) showed a smaller increase (from 17.25±4.72 to 21.96 ±4.83 pg/mL). The direct post-hoc comparison for the change in VEGF between TR and TR+SUP at post-intervention showed a difference of 4.21 pg/mL (95% CI: -1.52 to 9.94), which was not statistically significant ($p=0.148$). These patterns are illustrated in Figure 1. Collectively, these results indicate that interval training served as the principal stimulus for increasing serum VEGF, with the addition of CoQ10 yielding meaningful but attenuated responses compared to interval training performed in isolation.

TAC

A significant main effect of time was found ($F(1,56)=7.915$, $p= 0.007$, $\eta^2=0.124$, medium effect). A significant time × exercise interaction was observed ($F(1,56)=22.833$, $p<0.001$, $\eta^2=0.290$, large effect). No significant three-way interaction (time × exercise ×supplement) was detected ($F(1,56)=0.980$, $p=0.326$, $\eta^2= 0.017$). These findings demonstrate that interval training was the main driver of improved serum TAC, with CoQ10 supplementation offering no additive advantage. Post-hoc evaluation of the time × exercise interaction showed that the exercise groups exhibited a substantial rise in TAC (from 988.13 µmol/L±22.25 µmol/L to 1075.01±20.34 µmol/L), while the non-exercise groups displayed a slight decline (from 941.35±22.25 µmol/L to 918.87±20.34 µmol/L). These changes are depicted in Figure 2, with significant group differences marked by asterisks (Figure 2).

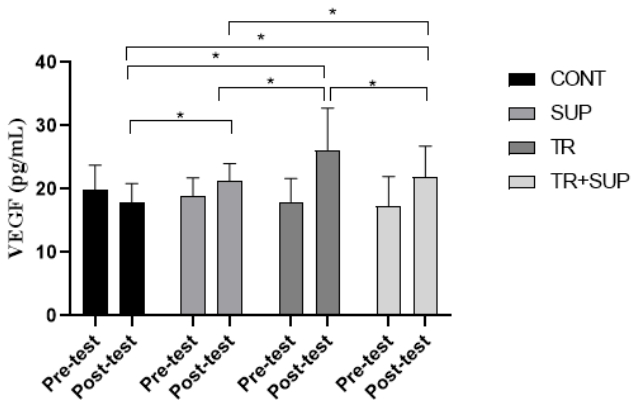

Figure1. VEGF changes levels in the four groups before and after intervention. *indicate a significant difference between groups ($p<0.05$).

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 VEGF, TAC and H_2O_2 across intervention groups.

Variable	Test	CONT	SUP	TR	TR+SUP
VEGF (pg/mL)	Pre-test	19.78 ± 3.88	18.83 ± 2.91	17.89 ± 3.71	17.25 ± 4.72
	Post-test	17.89 ± 2.98	21.28 ± 2.73	26.17 ± 6.62	21.96 ± 4.83
TAC (μmol/L)	Pre-test	911.25 ± 82.58	971.46 ± 88.29	1004.42 ± 175.24	971.85 ± 118.74
	Post-test	879.28 ± 118.55	958.45 ± 94.94	1104.48 ± 115.41	1045.54 ± 115.07
H_2O_2 (μmol/L)	Pre-test	43.72 ± 10.11	39.11 ± 5.64	46.39 ± 8.83	40.18 ± 21.50
	Post-test	40.67 ± 9.12	40.63 ± 4.40	19.49 ± 12.71	15.18 ± 5.60

Overall, these findings demonstrate that interval training was the main driver of improved serum TAC, with CoQ10 supplementation offering neither independent nor additive advantages when combined with exercise and showing limited standalone impact.

H_2O_2

A significant main effect of time was identified ($F(1,56) = 68.334$, $p<0.001$, $\eta^2=0.550$, large effect). A significant time × exercise interaction was present ($F(1,56)=60.718$, $p<0.001$, $\eta^2=0.520$, large effect). No significant three-way interaction was found ($F(1,56)=0.171$, $p=0.681$, $\eta^2 =0.003$). These results confirm that interval training was the primary factor driving the reduction in serum H_2O_2, with CoQ10 providing no further benefit. Post-hoc analysis of the time × exercise interaction revealed that the exercise groups showed a marked decline in H_2O_2 (from 43.28 ± 2.37 μmol/L to 17.33±1.57 μmol/L), whereas the non-exercise groups exhibited only minimal alteration (from 41.42±2.37 μmol/L to ≈ 40.65±1.57 μmol/L). These trends are shown in Figure 3, with significant differences indicated by asterisks (Figure 3).

In summary, these results confirm that interval training was the primary factor driving the reduction in serum H_2O_2, with CoQ10 supplementation providing no further benefit in combination with exercise and demonstrating limited independent influence.

Discussion

The present randomized controlled trial demonstrates that 8 weeks of supervised moderate-intensity intermittent training exerts robust positive effects on key circulating biomarkers of vascular health and redox balance in sedentary obese men aged 30–45 years. Specifically, interval training led to significant elevations in serum VEGF, enhancements in TAC, and substantial reductions in H_2O_2. The primary novel finding is that CoQ10 supplementation provided no additive benefit for TAC or H_2O_2; and while a significant three-way interaction suggested a

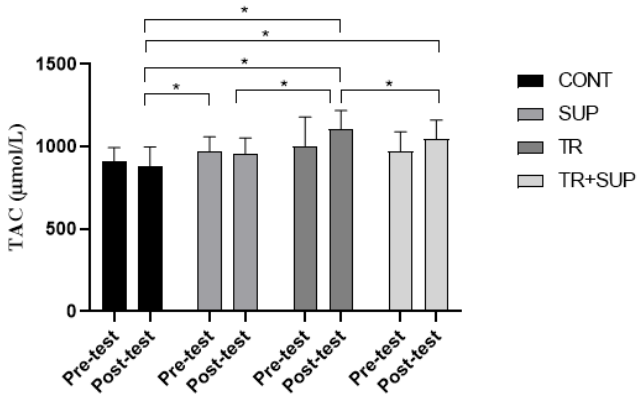

Figure2. TAC changes levels in the four groups before and after intervention. ∗indicate a significant difference between groups ($p<0.05$).

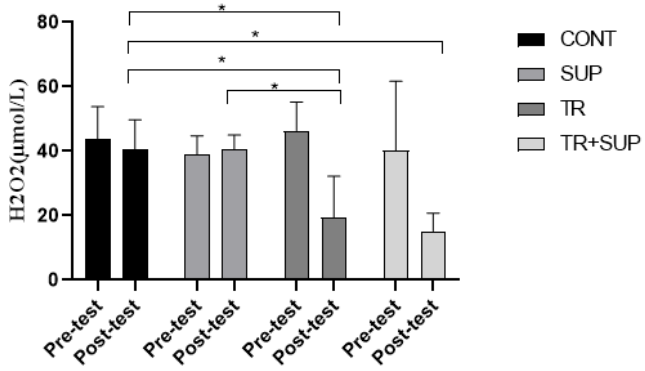

Figure3. H_2O_2 changes levels in the four groups before and after intervention. ∗indicate a significant difference between groups ($p<0.05$).

potential modifying effect on VEGF, direct post-hoc comparison did not confirm a statistically significant "blunting" effect. The marked increase in circulating VEGF following interval training aligns with its central role in promoting physiological angiogenesis and vascular remodeling, processes frequently compromised in obesity due to chronic adipose tissue hypoxia, inflammation, and endothelial dysfunction (Shafiee et al., 2023). It is hypothesized, based on existing literature, that this effect is mediated by muscle-derived myokines and exerkines (e.g., IL-6, irisin) that exert endocrine effects on adipose tissue and vascular endothelium (Leal et al., 2018; Zhou et al., 2024). These proposed signals may upregulate HIF-1α expression, thereby enhancing VEGF production (Kolahdouzi et al., 2019). This mechanism is supported by animal studies showing that interval training elevates adipose VEGF-A expression (Kolahdouzi et al., 2019) and by human meta-analyses confirming interval training's superiority for improving endothelial function (Khalafi et al., 2022).

A key finding of this study is that CoQ10 supplementation (100 mg/day) did not augment the training-induced improvements in TAC or H_2O_2 and was associated with a non-statistically significant trend toward lower VEGF levels when combined with exercise (p=0.148). Although CoQ10 functions as a mitochondrial electron carrier and lipophilic antioxidant capable of scavenging ROS, activating Nrf2 pathways, suppressing NF-κB/TNF-α, and supporting eNOS while inhibiting iNOS (Zhai et al., 2017), its effects here were neutral or mildly inhibitory. This pattern may reflect interference with physiological ROS-dependent signaling: exercise generates low-level, transient ROS that serve as hormetic signals to trigger adaptive cascades, including HIF-1α stabilization and VEGF expression. Excessive antioxidant buffering by CoQ10 could dampen these mild ROS cues. This pattern is consistent with other reports of antioxidant supplementation blunting exercise adaptations (Astani et al., 2022), but our data do not support a definitive "blunting" claim, as the direct comparison was not statistically significant.

Parallel improvements in redox homeostasis were evident: interval training robustly increased TAC (large effect) and decreased H_2O_2 (strong effect), driven by enhanced mitochondrial efficiency, upregulation of endogenous antioxidant enzymes. This is consistent with the hypothesis that physiological exercise-induced ROS acts hormetically to boost antioxidant defenses (Flensted-Jensen et al., 2021) and interval training-mediated decreases in muscle H_2O_2 emission alongside elevated MnSOD in obese contexts (Flensted-Jensen et al., 2021). Divergent acute studies highlight the distinction between acute stress and chronic adaptive responses, where repeated interval training induces net redox improvements (Melo et al., 2019).

Again, CoQ10 provided no additive redox benefit, consistent with evidence that exogenous antioxidants can blunt exercise hormesis in certain contexts (e.g., interfering with ROS-triggered Nrf2 activation or mitochondrial adaptations). In sedentary obese men, interval training alone appears sufficient to restore redox balance through integrated organ communication. In summary, our data supports the role of interval training (performed as moderate-intensity intermittent training) as an effective modulator of inter-organ communication. By stimulating myokine/exerkine release, interval training potentially orchestrates beneficial signals to adipose tissue and endothelium, while simultaneously restoring systemic antioxidant capacity. The lack of synergy from CoQ10, and the non-significant trend toward attenuated VEGF adaptations, suggests a potential risk of disrupting ROS-dependent signaling. Future research should explore tissue-specific exerkine profiles and optimized CoQ10 dosing.

Conclusion

Eight weeks of moderate-intensity intermittent training improves vascular (VEGF) and redox (TAC, H_2O_2) homeostasis in sedentary obese men. CoQ10 supplementation (100 mg/day) provided no additional benefit and was associated with a non-significant trend toward a lower VEGF response compared to training alone. These findings position interval training as a superior non-pharmacological strategy for improving vascular and oxidative health in this population. Several limitations should be acknowledged. First, the absence of a formal dietary control represents a potential confounder, particularly for TAC and H_2O_2, as variations in habitual antioxidant intake could have influenced the results. Second, the lack of prospective trial registration (IRCT) limits the transparency of the study; therefore, these findings should be considered exploratory and interpreted with caution. Third, the modest intensity of the intermittent training (≤75% HRmax) may explain the lack of significant body fat reduction in the training-only group, suggesting that higher intensities might be required for adipose tissue changes despite improvements in vascular and redox biomarkers. Fourth, the relatively short duration (8 weeks) precludes conclusions about long-term adaptations. Fifth, the generalizability of these findings is limited to sedentary obese men aged 30–45 years and cannot be extended to women, other age groups, or clinically obese patients with comorbidities.

What is already known on this subject?

Moderate-intensity intermittent training enhances cardiometabolic health in obesity by promoting angiogenesis (↑VEGF), improving antioxidant capacity (↑TAC), and reducing oxidative stress (↓H_2O_2), largely through muscle-derived myokines/exerkines that activate HIF-1α/VEGF signaling.

Although CoQ10 lowers oxidative stress in various conditions, its combined effect with interval training in sedentary obesity is unclear.

What this study adds?

Eight weeks of moderate-intensity intermittent training significantly increased VEGF, markedly elevated TAC, and strongly decreased H_2O_2 in sedentary obese men. CoQ10 supplementation provided no additional benefit and slightly blunted some training-related vascular responses. Thus, interval training alone is the primary driver of vascular and redox adaptations, with no synergistic effect from CoQ10.

Organ Cross-Talk Tips:

- Moderate-intensity intermittent training activates skeletal muscle signaling to adipose tissue and endothelium, enhancing angiogenesis and antioxidant defense. Exercise-induced physiological ROS acts as a hormetic trigger improving TAC and reducing H_2O_2. This restores muscle–adipose–endothelial communication and systemic redox balance more effectively than CoQ10 alone.

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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. Written informed consent was obtained from all participants. Trial registration was not obtained prospectively (IRCT); this is acknowledged as a limitation, and the study was conducted as an exploration trial.

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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