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Circuit training on oxidative stress and arterial health: a health promotion perspective for obese adult men

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Background: Obesity leads to increased oxidative stress, disruption of the antioxidant system, and decreased bioavailability of nitric oxide (NO). This, in turn, contributes to impaired endothelial function. The resulting increase in arterial stiffness (AS) has been associated with an increased risk of cardiovascular disease (CVD). Regular physical activity improves the antioxidant system and vascular function. Circuit training combines aerobic exercise and resistance training, encapsulating the benefits of both types of exercise, and helps improve vascular function. We aimed to investigate the effects of circuit training on total oxidant status (TOS), total antioxidant status (TAS), NO, and atherosclerosis in adult men with obesity.

Methods: A total of 25 obese men were randomly assigned to control (n = 12) or exercise groups (n = 13). The exercise group participated in circuit training three times per week for 12 weeks at an intensity corresponding to 60–80% of heart rate reserve (HRR). Anthropometrics, TOS, TAS, oxidative stress index (OSI), NO, and brachial-ankle pulse wave velocity (baPWV) were measured before and after the 12-week intervention.

Results: Body mass index (BMI) ($p < 0.001$), TAS ($p < 0.001$), OSI ($p < 0.05$), NO ($p < 0.05$), and baPWV (L, R) ($p < 0.05$) values improved significantly in the exercise group following the 12-week intervention, while TOS values did not demonstrate a significant change. Furthermore, no change was observed in the control group.

Conclusion: Our findings reveal that circuit training leads to improvements in BMI, TAS, OSI, NO, and baPWV in men with obesity, suggesting that it may contribute to an improvement in the antioxidant system and the prevention of CVD in obese men.

KEYWORDS

antioxidant, cardiovascular disease, circuit training, obesity, oxidative stress

Introduction

Obesity is defined as the accumulation of excessive fat due to physical inactivity, unhealthy eating patterns, and other factors, posing a serious public health problem worldwide (1, 2).

The increasing global prevalence of obesity among young adults is of particular concern (3), with rates among adults aged 20–39 years reported at 35.5% in the United States (4), 33.0%

for men and 22.3% for women in Japan (5), and, as of 2021, 49.2% for men and 27.8% for women aged 20 years and older in South Korea, where the highest prevalence was observed to be 55.4% among men in their 30s (6).

Obesity is recognized as an independent risk factor for increased oxidative stress, which is defined as an imbalance between oxidants and antioxidants (7, 8), as excessive fat accumulation induces a chronic inflammatory state through elevated secretion of pro-inflammatory cytokines such as TNF-α, IL-6, and MCP-1, along with enhanced activation of nicotinamide adenine dinucleotide phosphate (NADPH) oxidase, all of which contribute to the increased production of reactive oxygen species (ROS) (9, 10). Catoi et al. reported that obese individuals exhibited elevated levels of pro-oxidants (total oxidant status, TOS) compared with individuals with normal weight, noting that this increase in oxidative stress contributes to the development of various diseases, including diabetes, endothelial dysfunction, and cardiovascular disease (CVD) (11, 12).

Although the human body possesses an antioxidant system comprising both enzymatic and non-enzymatic antioxidants that counteract oxidative stress and maintain homeostasis (13), obesity has been shown to reduce antioxidant capacity, thereby increasing oxidative stress (14, 15), as evidenced by a lower antioxidants (total antioxidant status, TAS) in individuals with obesity compared with those with normal weight (8), as well as an inverse correlation between body fat percentage and antioxidant capacity (16).

In pathological conditions characterized by increased oxidative stress, dysfunction of endothelial nitric oxide synthase (eNOS), which is responsible for nitric oxide (NO) production (17) along with reduced NO bioavailability, results in endothelial dysfunction (18). This, in turn, leads to reduced arterial elasticity and increased arterial stiffness (AS), a primary contributor to elevated systolic blood pressure and pulse pressure (19, 20), thereby increasing the risk of cardiovascular disease (CVD) and mortality (21).

It has been confirmed that regular physical activity offers many benefits, including improvements in the antioxidant system (22) and vascular health (23). It has been proposed as a non-pharmacological treatment option. Multiple studies have previously reported an alleviation of oxidative stress (24), reflected in changes in TAS (25), NO (26), and AS (27) levels subsequent to participation in regular exercise training.

Among the various forms of exercise training, circuit training combines the benefits of both aerobic and resistance training (28). It has been proposed as a time-efficient method to improve body composition and strength, obviating the need for specialized equipment or facilities (29). Further, previous studies have demonstrated improvements in body composition and cardiovascular risk factors with circuit training (30, 31).

Although circuit training has been well-documented for its benefits in improving body composition and reducing cardiovascular risk factors, there is a distinct lack of research exploring its direct impact on key vascular health markers, specifically oxidative stress, NO production, and AS in obese men. This study was designed to rigorously assess the effects of a structured circuit training regimen on TOS, TAS, NO, and AS levels in a high-risk patient cohort comprising obese men in Korea. By focusing on these critical biomarkers, the present study sought to elucidate the physiological pathways through which circuit training may mitigate CVD risk. Anchored in a health intervention framework, this study aimed to generate actionable evidence that can inform both clinical practice and public health policies geared toward the prevention and management of obesity-related vascular complications. We hypothesized that circuit training would significantly enhance antioxidant defense systems and vascular function, thereby offering a potent, scalable exercise intervention for obese men.

Methods

Participants

This study included 25 obese men (Body Mass Index [BMI] $\geq$ 25 kg/m^2) (6) aged between 30 and 35 years, who had not engaged in regular physical activity in the past 6 months and had no history of diabetes, dyslipidemia, or CVD. All participants were randomly assigned to the control (CON, n = 12) or circuit training groups (EX, n = 13) (Figure 1). All study protocols were reviewed and approved by the Research Ethics Committee of Korea Maritime and Ocean University (Institutional Review Board Approval Number: KMOU IRB 2024–05) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to the study.

Study design

After baseline measurements, participants were randomly assigned, using a parallel design, to either a non-exercise control group (CON, n = 12) or a circuit training group (EX, n = 13). Before and after the 12-week intervention period, all participants underwent blood sampling, AS assessment, and anthropometric evaluations. The EX group participated in a supervised circuit training program for 12 weeks, which included warm-up, main exercise (push-ups, squats, lunges, kickbacks, shoulder presses, pull-ups, hip-bridges, leg-raises, and crunches), and cool-down components. In contrast, the CON group did not engage in any structured physical activity and maintained their usual lifestyle.

Anthropometrics

Height was measured without shoes using a portable stadiometer (InLabS50, InBody, Korea), and weight was measured in light clothing using an electronic scale (DB-1, CAS, South Korea). Body mass index (BMI) was computed as body weight in kilograms divided by height in meters squared (kg/m^2).

Blood sampling and analysis

Blood samples were collected from the antecubital vein using ethylenediaminetetraacetic acid tubes both before and after the 12-week circuit training program. Samples were centrifuged at 3,500 rpm for 10 min at 4°C, and the resulting plasma was stored at −70°C for subsequent analysis of TOS, TAS and NO levels. Serum TOS levels were determined using a commercial assay kit (Total Oxidant Status, Rel Assay Diagnostics, Gaziantep, Turkey) and measured spectrophotometrically using an automated analyzer

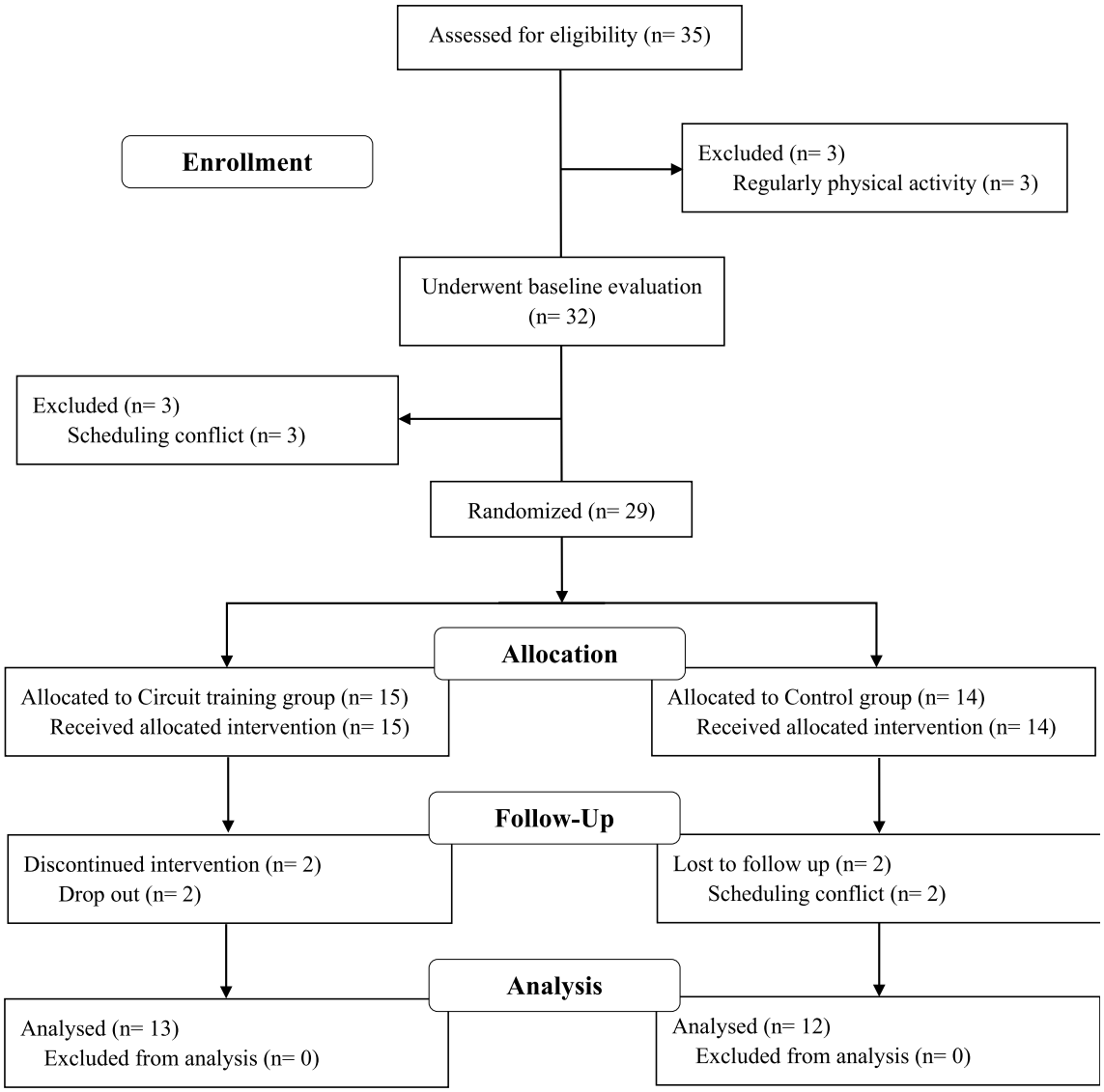

FIGURE 1
Diagram for the experimental study.

(Beckman Coulter AU680, Tokyo, Japan) (32). Serum TAS levels were assessed using a Total Antioxidant Status kit (Rel Assay Diagnostics) with the same analyzer (33). NO levels were analyzed using a Total NO/Nitrite/Nitrate Assay kit (R&D Systems, Minneapolis, MN, United States), and absorbance was measured spectrophotometrically at 540 nm using a VERSAmax microplate reader (Molecular Devices, Sunnyvale, CA, USA) (34). The oxidative stress index (OSI) was calculated as TOS divided by TAS multiplied by 100 (OSI = TOS/TAS × 100) (35).

Arterial stiffness

Participants arrived at the laboratory 30 min prior to the measurement. After lying in a supine position and resting for approximately 10 min to achieve maximal relaxation, AS was assessed using an automated vascular screening device (VP-1000 Plus, Omron Healthcare Co., Ltd., Kyoto, Japan) to determine baPWV (36). The measurement was performed by wrapping cuffs around both arms and ankles and attaching electrocardiogram electrodes to both wrists.

Circuit training program

The design of the circuit training program was based on the exercise regimen proposed by Bocalini et al. (37) that has been modified for the present study. It consisted of 9 exercises: push-ups, squats, lunges, kickbacks, shoulder presses, pull-ups, hip-bridges, leg-raises, and crunches that were performed three times per week for 12 weeks. All participants were encouraged to complete each exercise within 60 s and to rest for 50 s between sets, performing three sets of each exercise during weeks 1–6 and increasing to four sets during weeks 7–12. Exercise intensity was monitored using a wrist-worn heart rate monitor (Polar RS400sd, APAC, USA) to ensure that heart rate reached 60–80% of heart rate reserve (Table 1).

Data analysis

SPSS Statistics software version 25.0 (IBM Corp, Armonk, New York, USA) was used to conduct all statistical analyses. The Shapiro–Wilk test was used to assess data normality, and the effects of the circuit training program on BMI, TOS, TAS, NO, and baPWV were identified using a two-way repeated measures analysis of variance with group (EX and CON) and time (pre- and post-12 week) as independent variables. When significant interactions were noted, paired t-tests were used for *post hoc* comparisons. Data are presented as mean ± standard deviation. Statistical significance was set as $p < 0.05$.

Results

No participants reported any adverse events or unfavorable symptom effects resulting from a circuit training program. BMI, TOS, TAC, NO and cfPWV pre and post 12 weeks for CON and EX groups are presented (Tables 2–4). Participants in the EX group completed 95% of the circuit training program under the supervision of a professional. No statistically significant differences were observed between the groups in the pre-test measurements. We observed significant group versus time interactions for BMI ($F = 48.714$, $p < 0.001$), TAS ($F = 23.000$, $p < 0.001$), OSI ($F = 5.133$, $p < 0.05$,), NO ($F = 6.723$, $p < 0.05$), baPWV (L) ($F = 6.000$, $p < 0.05$), and baPWV (R) ($F = 4.715$, $p < 0.05$). BMI (from 26.84 ± 1.57 to 25.77 ± 1.64 kg/m^2, $t = 9.298$, $p < 0.001$) was significantly reduced in the EX group (Table 2). OSI (from 0.23 ± 0.04 to 0.20 ± 0.04, $t = 3.049$, $p < 0.05$), baPWV(L) (from 1263.38 ± 119.25 to 1222.46 ± 99.47 m/s, $t = 2.210$, $p < 0.05$), and baPWV(R) (from 1301.15 ± 99.47 to 1254.38 ± 87.71 m/s, $t = 3.034$, $p < 0.05$) were significantly reduced in the EX group (Tables 3, 4). TAS (from 1707.69 ± 122.69 to 1850.77 ± 130.03 μmol/L, $t = -4.873$, $p < 0.001$) was significantly elevated in the EX group (Table 3). NO (from 40.95 ± 7.05 to 44.71 ± 6.16 μmol/L, $t = -2.702$, $p < 0.05$) was significantly elevated in the EX group (Table 3).

Discussion

This study was conducted to verify the hypothesis that circuit training would have a positive effect on post-exercise TOS, TAS, NO, and AS levels in obese men. Several notable findings were observed in this study. First, there was an increase in TAS and NO levels. Second, significant reductions were observed in OSI and baPWV values. These findings suggest that circuit training is a viable intervention for enhancing vascular function, as evidenced by improvements in TAS, OSI, NO, and AS in obese men.

TOS

Obesity has been associated with increased oxidative stress (38), primarily due to obesity-induced mitochondrial dysfunction, which promotes the overproduction of reactive oxygen species (ROS), ultimately resulting in elevated oxidative stress (39).

Furukawa et al. reported that fat accumulation increases oxidative stress (9), and Cagnacci et al. (40) reported a positive correlation between abdominal obesity and oxidative stress. Increased oxidative stress has been demonstrated to contribute to various pathological events, including insulin resistance, diabetes, and cardiovascular complications (41, 42). It is recognized as a promoter of the development of endothelial dysfunction and CVDs (43). A reduction in oxidative stress has been identified as a means to enhance vascular function (44). Physical activity or exercise has been proposed as a potential intervention strategy to improve vascular health due to its ability to reduce oxidative stress (45). This was demonstrated in a study by Roh et al. (46) which reported that aerobic exercise effectively reduces oxidative stress in obese adults.

Rosety-Rodriguez et al. (25) reported a reduction in oxidative damage after 12 weeks of circuit training in adults with Down syndrome, attributing this outcome to an increase in antioxidant enzymes such as superoxide dismutase and catalase, as well as an improvement in redox balance, which led to a reduction in oxidative stress (45). However, Deminice et al. (47) reported no change in oxidative stress after an acute session of circuit training in healthy young men. These results were attributed to factors such as duration of exercise, exercise mode, and the exercise protocol (48).

Our findings revealed that oxidative stress levels did not demonstrate a statistically significant difference between the EX and CON groups; however, a statistical trend toward slightly lowered oxidative stress was observed in the EX group, suggesting that regular participation in circuit training may contribute to a statistically significant reduction in oxidative stress. However, the impact of exercise on oxidative stress remains controversial (49, 50),

TABLE 1 Circuit training program.

Order	Exercise	Duration, min	Week	Intensity	Sets	Frequency
Warm-up	Static stretching	10				3 times/wk
Main exercise	Push-ups squats, lunges, kick backs	35	1–6	60-70%HRR	3 sets	
	shoulder presses pull-ups hip-bridges leg-raises crunches	45	7–12	70-80%HRR	4 sets	
Cool-down	Static stretching	10				

TABLE 2 Characteristics of participants.

	CON (n = 12)			EX (n = 13)		
	Pre	Post	Δ	Pre	Post	Δ
Age, y	32.58 ± 1.73	-	-	32.15 ± 1.21	-	-
Height, cm	176.50 ± 3.55	-	-	177.23 ± 6.29	-	-
Weight, kg	83.97 ± 6.82	83.97 ± 7.72	0.0 ± 0.90	84.45 ± 8.45	81.06 ± 8.28	−3.39 ± 0.17
BMI, kg/m^2	26.94 ± 1.90	26.94 ± 2.15	0.0 ± 0.52	26.84 ± 1.57	25.77 ± 1.64***	−1.07 ± 0.07$^{\#\#\#}$

Values are presented as mean (M) ± standard deviation (SD). BMI: body mass index; CON: control group; EX: exercise group. ***$p < 0.001$ different than Pre. $^{\#\#\#}$ $p < 0.001$ different than CON.

TABLE 3 Change in TOS, TAS, OSI and NO pre and post in CON and EX group.

	CON (n = 12)			EX (n = 13)		
	Pre	Post	Δ	Pre	Post	Δ
TOS, μmol/L	3.90 ± 0.40	3.94 ± 0.53	0.40 ± 0.13	3.85 ± 0.54	3.59 ± 0.57	−0.26 ±0.03
TAS, μmol/L	1717.50 ± 152.62	1707.50 ± 143.37	10.00 ± 9.25	1707.69 ± 122.69	1850.77 ± 130.03***	143.08 ± 7.34$^{\#\#\#}$
OSI, arbitrary unit	0.23 ± 0.03	0.23 ± 0.04	0.00 ± 0.01	0.23 ± 0.04	0.20 ± 0.04*	−0.03 ± 0.00$^{\#}$
NO, μmol/L	42.70 ± 8.61	41.22 ± 7.51	−1.48 ± 1.10	40.95 ± 7.05	44.71 ± 6.16*	3.76 ± 0.89$^{\#}$

Values are presented as mean (M) ± standard deviation (SD). CON: control group; EX: exercise group; TOS: Total Oxidant Status; TAS; Total Antioxidant Status; NO: nitric oxide. *$p < 0.05$, ***$p < 0.001$ different than Pre. $^{\#}p < 0.05$, ###$p < 0.001$ different than CON.

necessitating further research that encompasses varying parameters such as exercise intensity and duration.

TAS

Obese individuals have higher levels of oxidative stress compared with those having a normal body weight (51), and this increase in oxidative stress disrupts the homeostasis of the antioxidant system (52). It has been established that an increase in body weight is associated with a reduction in the antioxidant capacity of plasma (53). Chrysohoou et al. (7) reported that markers of antioxidant defense exhibited an inverse correlation with body fat percentage and abdominal obesity. Furthermore, Catoi et al. (11) revealed that serum levels of TAS, a marker of overall antioxidant status encompassing both enzymatic and non-enzymatic antioxidants, were diminished in obese individuals compared with normal-weight individuals.

Willcox et al. (54) reported that lower levels of antioxidants were associated with increased oxidative stress, which contributed to lipid peroxidation and tissue damage, thereby adversely affecting vascular function and structure (55, 56), while elevated antioxidant levels helped prevent the accumulation of ROS and reduced the risk of CVD (57).

Regular physical activity has been reported to stimulate antioxidant enzyme activity (58). Attarzadeh Hosseini et al. (59) further demonstrated that both high-intensity interval training and moderate-intensity continuous exercise significantly enhanced total antioxidant capacity in overweight and obese women. Zhang et al. (60) reported an increase in antioxidant enzyme levels after 8 weeks of circuit training in high-school wrestlers. It has been suggested that oxidative stress is mitigated by a reduction in body fat mass and the activation of erythroid-related nuclear factor 2 and antioxidant-responsive elements (61).

Our current findings revealed a significant TAS reduction in the exercise group, corroborating previous studies. Additionally, the OSI value, which provides information about the interactions between the TOS and TAS ratio (62), displayed a significant reduction in the exercise group, suggesting that antioxidant activity predominates over oxidative processes (35).

Our data suggest that a reduction in BMI through circuit training may help improve antioxidant capacity and redox equilibrium in individuals with obesity.

Nitric oxide

Increased oxidative stress has been demonstrated to increase endothelium-derived contractile factors and impair the activation of eNOS, which reduces NO production, a key regulator of endothelial function that involves the prevention of platelet aggregation and adhesion and vasorelaxation (63, 64). An animal study by DeMarco et al. reported that increased oxidative stress decreased NO bioavailability in mice that had gained weight from a Western diet (65). This decline in the production and bioavailability of NO has been linked to various health complications, including atherosclerosis, hypertension, and endothelial dysfunction (66, 67).

Conversely, regular physical activity has been suggested to increase the bioavailability of NO (68), as demonstrated by elevated NO levels following consistent exercise in our previous study (69), by Ghadery et al. (70) who reported an upregulation of eNOS after 6 weeks of high-intensity interval training in obesity-induced rats, and by Guzel et al. (71) who observed a significant increase in NO after a single bout of high-intensity circuit training in sedentary men, where this effect was speculated to result from the rise in shear force during physical exertion that activates the Akt/eNOS signaling pathway, thereby enhancing NO production (72).

TABLE 4 Change in baPWV pre and post in CON and EX group.

	CON (n = 12)			EX (n = 13)		
	Pre	Post	Δ	Pre	Post	Δ
baPWV (L), m/s	1263.42 ± 124.91	1295.00 ± 129.44	31.58 ± 4.53	1263.38 ± 119.25	1222.46 ± 99.47*	−40.92 ± 19.78#
baPWV(R), m/s	1295.25 ± 90.16	1320.17 ± 85.03	24.92 ± 5.13	1301.15 ± 99.47	1254.38 ± 87.71*	−46.77 ± 11.76#

Values are presented as mean (M) ± standard deviation (SD). CON: control group; EX: exercise group; baPWV: Brachial-Ankle Pulse Wave Velocity. *$p < 0.05$ different than Pre. #$p < 0.05$ different than CON.

Our present data showed a significant increase in NO in the exercise group, indicating that circuit training may contribute to the reduction of BMI and the increase of antioxidant capacity, thereby improving NO levels and endothelial dysfunction.

Arterial stiffness

Obesity-induced oxidative stress reduces the activation of eNOS, leading to decreased NO bioavailability, endothelial dysfunction, and vascular hypertrophy, which ultimately contribute to increased AS (73, 74), resulting in structural vascular changes, including alterations in elasticity, capacitance (75), and resistance that are associated with a higher incidence and mortality of CVD (76).

Safar et al. reported that the risk of AS increases in individuals with obesity, regardless of ethnicity, age, or blood pressure (77), and a cohort study by Ohkuma et al. (78) demonstrated that each 1 m/s increase in baPWV is associated with an approximately 12% higher risk of CVD. However, a reduction in BMI through increased and regular physical activity has been shown to improve vascular function (79). A study by Vlachopoulos et al. (80) reported that a 1 m/s reduction in PWV was associated with an approximately 7% reduction in CVD risk. Previous studies have reported a decrease in baPWV following twice-weekly circuit training in older adult women (81) and following 12 weeks of circuit training in postmenopausal women with hypertension (82). This indicates that the rise in NO, a vasodilator, resulting from exercise leads to a reduction in AS (83, 84).

Our present findings revealed a substantial reduction in AS in the exercise group, which was consistent with previous reports. Our data suggest that a reduction in BMI achieved through circuit training may lead to an increase in total antioxidant capacity and a subsequent rise in NO levels, which together may improve AS and help prevent CVD.

However, this study has several methodological limitations. First, dietary habits, particularly sodium intake, were not controlled, which may be associated with AS (85). Second, the daily routines of the study participants, such as physical activity and smoking, were not monitored, which may have influenced the potential effectiveness of the circuit training. Third, since the participants were obese men in their 30s, the findings may not be generalizable to broader populations, including women and individuals of different age groups. Fourth, genetic and epigenetic factors, which can affect individual responses to circuit training in relation to oxidative stress and arterial health, were not considered. Fifth, psychosocial factors such as motivation and mental well-being, which could influence adherence to circuit training programs, were also not assessed. Finally, the sample size was limited to 25 participants. Further studies with larger sample sizes that incorporate dietary, lifestyle, genetic, and psychosocial factors are warranted to substantiate and expand upon the findings of this study.

Conclusion

This study investigated the effects of circuit training on oxidative stress, NO levels, and AS in obese men. Our results showed significant increases in TAS and NO levels, alongside improvements in OSI and baPWV, indicating that circuit training is an effective strategy for enhancing vascular health and reducing CVD risk in this population. These findings contribute to the existing literature by providing new evidence on the benefits of circuit training, with relevance for both developed and developing countries where obesity remains a growing health concern. The study underscores the importance of incorporating structured exercise programs into public health initiatives and clinical practice to manage oxidative stress and AS. We recommend further research with diverse populations and varied training protocols to confirm and extend these findings and encourage policymakers to adopt circuit training as part of more comprehensive obesity and cardiovascular health strategies.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

Ethics statement

The studies involving humans were approved by KOREA MARITIME & OCEAN UNIVERSITY Institutional Review Board. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

W-HS: Writing – original draft, Writing – review & editing, Data curation, Investigation. Y-SK: Writing – review & editing, Formal analysis. M-SH: Writing – review & editing, Data curation, Methodology.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

The author(s) declared that they were an editorial board member of Frontiers, at the time of submission. This had no impact on the peer review process and the final decision.

Generative AI statement

The authors declare that no Gen AI was used in the creation of this manuscript.

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