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Moderate-intensity endurance training has higher effects suppression of oxidative stress secretion than strength training in obese students

El entrenamiento de resistencia de intensidad moderada tiene mayores efectos en la supresión de la secreción de estrés oxidativo que el entrenamiento de fuerza en estudiantes obesos

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Abstract. The objective of this research is to establish the impact of endurance training of moderate intensity and resistance exercises on the mitigation of oxidative stress in students with obesity. The research engaged a cohort of 30 female students, characterized by obesity, with an age average of 22.30±1.92 years and a Body Mass Index (BMI) averaging 31.77±2.72 kg/m^2. These participants were randomly allocated into three distinct groups: the control group (CN, n=10), the group undergoing endurance training (ET, n=10), and the group subjected to strength training (ST, n=10). The intervention involving endurance training was executed at an intensity level of 60-70% HR_{max}, with each training session lasting between 40 to 60 minutes. Whereas, the strength training regimen was implemented with a load intensity of 60-70% 1RM, 4-6 sets, and 12-15 repetitions for each set. The intervention was administered with a regularity of three times per week for four weeks. Data collection was carried out by taking blood samples before and 24 hours after the last intervention—analysis of MDA levels utilizing the TBARs technique served as an indicator of oxidative stress. The technique employed for data analysis was the ANOVA test, then continued with the LSD post hoc test with a significant level ($p \leq 0.05$). The results showed that the average pre-MDA levels in CN, ET, and ST were (31.07±5.97, 32.12±8.91, and 31.36±8.52 ng/mL) respectively p=0.953. The mean post-MDA levels in CN, ET, and ST were (32.14±11.46, 15.21±8.01, and 22.02±7.33 ng/mL) respectively p=0.001. The mean delta MDA levels in CN, ET, and ST were (1.08±14.83, -16.91±9.68, and -9.34±7.51 ng/mL) respectively p=0.005. The average changes in MDA levels in CN, ET, and ST were (9.09±48.42, -51.71±24.77, and - 27.85±25.31 %) respectively p=0.002. Our findings revealed that both the endurance and strength training interventions resulted in a reduction of oxidative stress, as evidenced by the observed decrease in MDA levels following the training sessions. However, endurance training intervention has a higher effect on suppressing the secretion of oxidative stress than strength training in obese students.
Keywords: Endurance training, MDA levels, obesity, oxidative stress, strength training

Resumen. El objetivo de esta investigación es establecer el impacto del entrenamiento de resistencia de intensidad moderada y ejercicios de resistencia en la mitigación del estrés oxidativo en estudiantes con obesidad. La investigación involucró a una cohorte de 30 estudiantes mujeres, caracterizadas por obesidad, con edad promedio de 22,30±1,92 años y índice de masa corporal (IMC) de 31,77±2,72 kg/m2 en promedio. Estos participantes fueron asignados aleatoriamente en tres grupos distintos: el grupo de control (CN, n=10), el grupo sometido a entrenamiento de resistencia (ET, n=10) y el grupo sometido a entrenamiento de fuerza (ST, n=10). . La intervención que implicó entrenamiento de resistencia se ejecutó a un nivel de intensidad del 60-70% FCmáx, y cada sesión de entrenamiento duró entre 40 y 60 minutos. Mientras que el régimen de entrenamiento de fuerza se implementó con una intensidad de carga de 60-70% 1RM, 4-6 series y 12-15 repeticiones para cada serie. La intervención se administró con una regularidad de tres veces por semana durante cuatro semanas. La recopilación de datos se llevó a cabo tomando muestras de sangre antes y 24 horas después de la última intervención; el análisis de los niveles de MDA utilizando la técnica TBAR sirvió como indicador de estrés oxidativo. La técnica empleada para el análisis de los datos fue la prueba ANOVA, luego se continuó con la prueba post hoc LSD con un nivel significativo ($p \leqslant 0.05$). Los resultados mostraron que los niveles promedio pre-MDA en CN, ET y ST fueron (31,07±5,97, 32,12±8,91 y 31,36±8,52 ng/mL) respectivamente p=0,953. Los niveles medios post-MDA en CN, ET y ST fueron (32,14±11,46, 15,21±8,01 y 22,02±7,33 ng/mL) respectivamente p=0,001. Los niveles medios de delta MDA en CN, ET y ST fueron (1,08±14,83, -16,91±9,68 y -9,34±7,51 ng/mL) respectivamente p=0,005. Los cambios promedio en los niveles de MDA en CN, ET y ST fueron (9,09±48,42, -51,71±24,77 y -27,85±25,31 %) respectivamente p=0,002. Nuestros hallazgos revelaron que tanto las intervenciones de entrenamiento de resistencia como de fuerza dieron como resultado una reducción del estrés oxidativo, como lo demuestra la disminución observada en los niveles de MDA después de las sesiones de entrenamiento. Sin embargo, la intervención de entrenamiento de resistencia tiene un mayor efecto en la supresión de la secreción de estrés oxidativo que el entrenamiento de fuerza en estudiantes obesos.
Palabras clave: Entrenamiento de resistencia, niveles de MDA, obesidad, estrés oxidativo, entrenamiento de fuerza.

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Introduction

Physical activity triggers metabolic alterations within the organism, which in turn activates adaptive mechanisms to achieve a renewed dynamic equilibrium (Accattato et al., 2017). One of the most profound changes in this context takes place in muscle tissue, where the heightened energy requirement post-exercise leads to increased oxygen consumption by mitochondria (Benito et al., 2016). The primary source of free oxygen radical species is skeletal muscle, and during muscle contraction, there is an escalation in the transfer of unpaired electrons from complexes I and III in the electron transport chain, resulting in the generation of superoxide radical (O^{2-}), a principal member of reactive oxygen species (ROS) (Accattato et al., 2017). The harmful byproduct of this contractile activity is transformed into hydrogen peroxide (H_2O_2) by superoxide dismutase (SOD), which serves as the initial line of defense against radicals and

is detoxified by other enzymes such as catalase, glutathione peroxidase (GPX), and glutathione reductase (GR) (Bacanoiu et al., 2023). The equilibrium between ROS production and the expression of antioxidant enzyme activity is crucial to maintaining muscle redox homeostasis, keeping ROS below threshold levels, and preserving its function as a signaling molecule by mitigating the resultant toxic effects (Lu et al., 2021a). Conversely, the activity and capacity of ROS and antioxidants are correlated, hence they can be utilized as surrogate markers (Chatgilialoglu, 2024). The status of blood antioxidants may mirror the increased oxygen demand in muscle tissue during physical activity, as it is widely recognized that muscle contraction escalates ROS production in skeletal muscle (Powers et al., 2022). Thus, the circulating levels of these oxidative stress markers rise after acute and regular physical activity (Sies et al., 2017), potentially as a redox-mediated adaptation mechanism to safeguard against cellular oxidative damage (Thirupathi et al., 2021). However, the influence of diverse physical exercise protocols on antioxidant balance remains to be fully clarified. While obese individuals exhibit greater increases in oxidative biomarkers post-exercise than individuals of normal weight (Roh et al., 2017; Bouviere et al., 2021), other studies present conflicting findings (Vincent et al., 2006). For example, a systematic review suggests that lifestyle modifications can enhance oxidative stress biomarkers in non-communicable diseases, indicating a potential variability in oxidative stress responses among individuals (Husain et al., 2023). Moreover, there is evidence of elevated concentrations of total oxidative capacity and diminished concentrations of total antioxidative capacity in patients with recurrent hypertension, which may suggest different oxidative stress profiles based on body composition and metabolic conditions (Kościuszko, 2024). Therefore, the effect of exercise on reducing oxidative stress in obesity continues to be a subject of discussion.

Free radicals have the potential to instigate inflammation, lipid peroxidation, and chromosomal damage, which are linked with the onset of a variety of pathological conditions, such as cardiovascular disease, diabetes mellitus, chronic obstructive pulmonary disease, and cancer (Accattato et al., 2017). There is a close relationship and interconnection between oxidative stress and hypoxia, a condition that is frequently aggravated by the heightened production of oxygen radicals in various diseases (D'Aiuto et al., 2022). Recent research highlights the significant role of hypoxia in the pathogenesis of obesity and disorders related to obesity, leading to dysfunction of adipose tissue, aberrant gene expression, and ultimately resulting in systemic chronic, low-grade inflammatory states (Messineo et al., 2016; Kawai et al., 2021; Palma et al., 2022).

Physical activity is recognized as a significant regulator of cytokines and oxidative stress, and over the past decade, the potential of exercise to prevent and manage diseases with an inflammatory component by modulating cytokine production has been a focal point (Accattato et al., 2017). While the beneficial effects of exercise on inflammation are well established (Cordiano et al., 2023), the molecular mechanisms through which exercise imparts these effects remain elusive, and there is a lack of consistent information in the literature regarding the variation in markers related to physical exercise. Numerous studies have indicated that physical exercise can lead to an elevation in Malondialdehyde (MDA) levels, serving as a marker of oxidative stress (Diaba-Nuhoho et al., 2018; Park & Kwak, 2016; Bhutia et al., 2011). Conversely, other studies demonstrate a notable decrease in MDA levels following exercise (Pranoto et al., 2023; Wang et al., 2023; Lu et al., 2021b), thereby confirming the existing ambiguity surrounding this issue, potentially due to the complexity of adaptive mechanisms during physical exercise. Consequently, this study seeks to validate the impact of moderate-intensity endurance training and strength training on the reduction of oxidative stress in students with obesity.

Materials and Methods

This study was a true experiment employing a randomized pretest-posttest control group design using as subjects 30 obese female students aged 22.30±1.92 years, body mass index (BMI) 31.77±2.72 kg/m^2, normal of blood pressure, normal of resting heart rate (RHR), normal oxygen saturation (SpO_2), fasting blood glucose (FGB) ≤100 mg/dL, normal of hemoglobin (Hb) and randomly divided into three groups, namely CN (n=10, control group without training program), ET (n=10, endurance training group), and ST (n=10, strength training group). The endurance training intervention was implemented by performing a jog on a treadmill at an intensity of 60-70% HRmax for 40-60 minutes per training session. Strength training intervention was carried out using lat pull-down back, chest presses, leg presses, leg extensions, and leg curls with an intensity of 60-70% 1RM which was done for 4-6 sets @ 12-15 repetitions per training session. Control intensity during exercise using Polar Heart Rate Monitoring H-10. Routine intervention was carried out every 07.00-09.00 a.m with a frequency of 3x/week for 4 weeks at the Gym of the Faculty of Sports and Health Sciences, Universitas Negeri Surabaya (Indonesia). Meanwhile, the control group did not receive endurance training or strength training intervention and during the intervention period, the control group was not allowed to change their physical activity habits which were monitored every day with the International Physical Activity Questionnaire (IPAQ). All processes executed in this research adhered to the Declaration of Helsinki by the World Medical Association and received approval from the Ethics Commission for Health Research, Faculty of Medicine, Ciputra University, Surabaya, East Java, Indonesia, under the approval number 103/EC/KEPK-FKUC/II/2024. Data collection was carried out by taking 3 ml of blood samples from the cubital vein. Blood was taken 2 times, namely pre-training and 24 hours after the last post-training. Following a 15-minute centrifugation

process at 3000 revolutions per minute, the serum was isolated. Subsequently, an immediate assessment was conducted to determine the levels of MDA, a recognized indicator of oxidative stress, employing the TBARs technique. Body fat and skeletal muscle mass were measured using the TANITA DC-360 Body Composition Analyzer. Analytical procedures were performed using the SPSS version 20. The Shapiro-Wilk test was employed for normality testing, and Levene's test was used to assess homogeneity. Data that met the criteria of normal distribution and homogeneous variance underwent further testing with the paired sample t-test and one-way ANOVA, followed by the LSD post hoc test at a significance level of $p \leq 0.05$. The Pearson product-moment model was utilized to examine the correlation between variables. All data are expressed as the mean ± SD.

Results

Derived from the analytical outcomes of the descriptive characteristics of the study participants for each variable, no significant differences were found between groups which can be seen in Table 1 below. The results of the analysis of MDA levels between pre and post in each group are shown in Figure 1, while the differences in MDA levels between groups with pre, post, delta, and change observations are presented in Figure 2. Figure 3 presents the relationship between MDA levels with body fat and skeletal muscle mass.

Table 1.
Descriptive characteristics of research subjects

Variable	CN (n=10)	ET (n=10)	ST (n=10)	p-value
Age, yrs	22.30±2.06	22.20±1.99	22.40±1.89	0.975
BW, kg	79.33±7.69	76.24±8.17	78.09±8.85	0.704
BH, m	1.59±0.05	1.55±0.06	1.54±0.06	0.264
BMI, kg/m^2	31.66±3.02	31.45±2.69	32.21±2.66	0.822
PBF, %	42.34±4.75	44.17±1.98	45.34±3.45	0.187
FM, kg	35.29±5.19	33.76±4.53	35.53±5.76	0.713
SM, kg	20.41±1.59	19.23±2.31	20.21±4.36	0.644
SBP, mmHg	115.30±9.18	110.00±10.88	116.10±9.36	0.336
DBP, mmHg	78.90±7.92	78.40±7.29	81.00±6.29	0.696
HR, bpm	78.60±9.80	77.30±7.88	82.20±8.16	0.434
SpO_2, %	98.20±0.92	98.00±1.05	97.90±0.99	0.790
FBG, mg/dL	92.60±5.15	89.50±6.66	90.80±6.94	0.550
Hb, g/dL	14.48±2.64	15.73±1.41	15.57±1.31	0.288

Description: CN: Control group; ET: Endurance training group; ST: Strength training group. The p-value was obtained using one-way ANOVA.

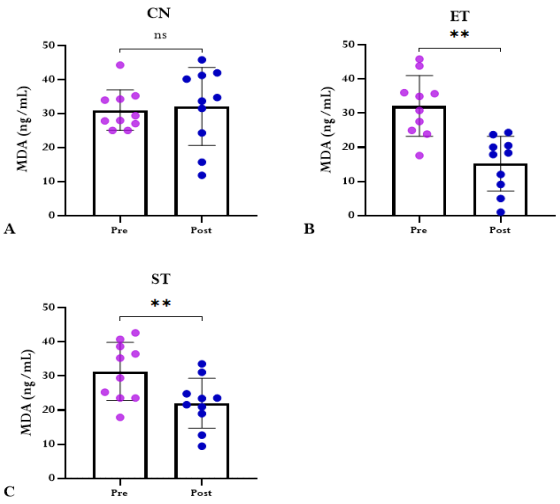

Figure 1. MDA levels (ng/mL) in each group

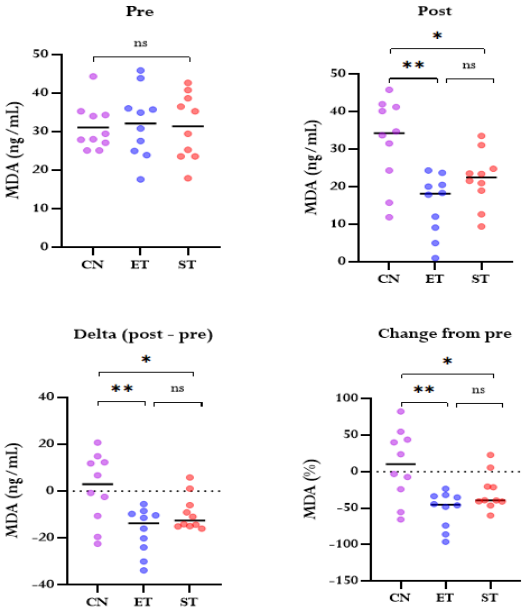

Figure 2. Comparison of MDA levels at pre, post, delta, and change from pre in three groups (CN vs ET vs ST)

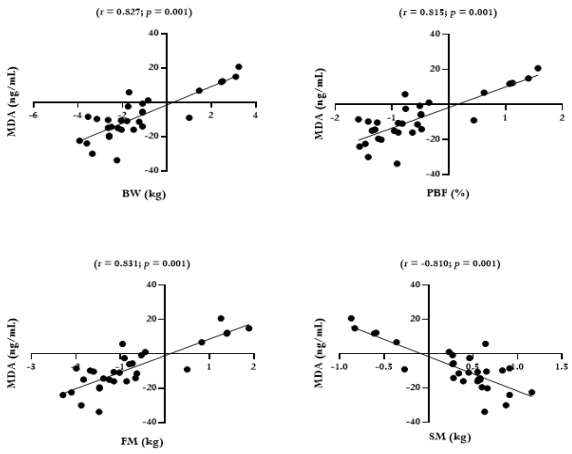

Figure 3. The association between oxidative stress with body fat and skeletal muscle mass

Description: (**A**) Control group (CN). (**B**) Endurance training group (ET). (**C**) Strength training group (ST). The p-value was obtained using paired sample t-test analysis. (ns) Not significant at pre from CN ($p \geq 0.05$). (**) Significant at pre-from ET and ST ($p \leq 0.001$).

Description: The p-value was obtained using one-way ANOVA and followed by an LSD post-hoc test. (ns) Not significant ($p \geq 0.05$). (**) Significant at ET ($p \leq 0.001$). (*) Significant at ST ($p \leq 0.05$).

Description: Pearson correlation coefficients (r) and p-values are shown in each graph. Malondialdehyde (MDA), Body Weight (BW), Percentage of Body Fat (PBF), Fat Mass (FM), Skeletal Muscle Mass (SM).

Discussion

According to the findings of the study, it is evident that both forms of exercise considerably diminish oxidative stress in obesity, as indicated by a reduction in MDA levels (Figure 1). This is consistent with the study conducted by Pranoto et al. (2023), who observed that moderate-intensity endurance training reduces oxidative stress, characterized by a decrease in MDA levels in obese women. Likewise, a study conducted by Yosika et al. (2020) found that resistance training led to a decrease in MDA levels in women with obesity. This suggests that exercise of moderate intensity has been demonstrated to be beneficial in mitigating oxidative stress in individuals with obesity. A study by Jiang et al. (2023) further elucidates that exercise of moderate intensity is a viable strategy for preserving redox homeostasis and increasing anti-oxidative capacity. The reduction in oxidative stress is likely because exercise can increase the regulation of endogenous antioxidants as a balance to protect against damage and inflammation caused by increased reactive oxygen species (ROS) (Li et al., 2017; Gutiérrez-López et al., 2021; Thomas et al., 2022).

It is widely recognized that obesity correlates with elevated oxidative stress levels (Jiang et al., 2021). Obesity augments the likelihood of oxidative stress via several biochemical routes, encompassing auto-oxidation of glyceraldehyde, superoxide generation from NADPH oxidase, oxidative phosphorylation, and the polyol and hexosamine pathways (Manna & Jain, 2015). Furthermore, other elements such as diminished antioxidant defenses, persistent inflammation, hyperleptinemia, and ROS production post-meal contribute to obesity progression via oxidative stress (Savini et al., 2013). Therefore, moderate-intensity exercise performed regularly can be used as an effective therapy in reducing oxidative stress in obesity (Li et al., 2017; Roh et al., 2020; Gutiérrez-López et al., 2021).

Obesity is defined as an abnormal buildup of fat or adipose tissue in the body (Wen et al., 2022). This excessive fat accumulation in obese individuals results in pathologically elevated concentrations of serum free fatty acids (FFAs), which can interfere with glucose metabolism, promote the accumulation of energy substrates (glucose and fat) in the liver, muscle, and adipose tissue, and initiate mitochondrial and peroxisomal oxidation (Čolak & Pap, 2021). The increase in oxidative stress due to fat accumulation is an early indicator of metabolic syndrome, and the redox state in adipose tissue is a potential therapeutic target for metabolic syndrome related to obesity (Martínez-Martínez & Cachofeiro, 2022). Moreover, a diet high in fat (HFD) elevates the levels of chylomicrons in the intestine (Tan & Norhaizan, 2019). These chylomicrons enter the bloodstream, leading to the formation of free fatty acids that are absorbed by the liver. These fatty acids from the liver can undergo β-oxidation in the mitochondria or be esterified into triglycerides. Triglycerides can accumulate in hepatocytes as small granules or produce VLDL, which is subsequently converted into low-density lipoprotein (LDL). An excessive LDL burden in the blood can lead to the formation of oxidized LDL (Ox-LDL) due to overaccumulation or a deficiency of LDL receptors in hepatocytes, which are then ingested by macrophages and transformed into foam cells. Subsequently, these foam cells accumulate on the arterial endothelium, forming plaque. In the end, this plaque causes cardiovascular and circulatory disorders. Carrying out regular physical activity is well known to improve muscle strength, aerobic capacity, endothelial function, and body composition (Almuraikhy et al., 2024). Furthermore, obesity is associated with adipocyte atrophy, a condition where fat cells shrink or lose their functionality (Zatterale et al., 2020). This atrophy contributes to a state of chronic low-grade inflammation within the adipose tissue (Hagberg & Spalding, 2024). The dysfunction of adipocytes leads to an altered secretion of adipokines and cytokines, which serve as crucial signaling molecules in the regulation of metabolic processes and immune responses (Kawai et al., 2021). As adipocytes atrophy, the balance of these secretions shifts towards a profile that promotes inflammation, marked by elevated concentrations of cytokines such as TNF-α, IL-6, and MCP-1 (Calder et al., 2011). These pro-inflammatory cytokines intensify the condition of inflammation, attracting immune cells like macrophages to the adipose tissue and fostering the shift of these macrophages towards a phenotype that promotes inflammation (M1) (Zatterale et al., 2020). The persistent presence of these activated immune cells further sustains the inflammatory environment, contributing to insulin resistance and the advancement of metabolic syndrome in individuals with obesity (Zatterale et al., 2020; Hagberg & Spalding, 2024; Kawai et al., 2021; Calder et al., 2011).

Increased oxidative stress in obesity can be caused by excessive fat accumulation (Marseglia et al., 2014). According to the findings of this research, it is demonstrated that both endurance and strength training effectively lower MDA levels, serving as a biomarker of oxidative stress (Figure 1-2). It was observed that there was a reduction in fat accumulation and an enhancement in muscle mass following the training. Further correlation analysis in this study found a positive relationship between a decrease in MDA levels and a decrease in body fat parameters post-training and a negative relationship with an increase in skeletal muscle mass post-training (Figure 3). Therefore, strategies to reduce oxidative stress in obesity are through reducing body weight, fat accumulation, and increasing skeletal muscle mass (Savini et al., 2013).

The results of this study contribute positively to the prevention of cardiovascular disease risks associated with obesity. However, the study acknowledges certain limitations, including the exclusive measurement of body fat and select oxidative stress markers. Notably, oxidative stress markers such as protein carbonyls have been recognized as significant indicators of oxidative damage and should be considered in future research (Colombo et al., 2020). Additionally, the dietary intake of antioxidants plays a crucial role in modulating oxidative status, which is an important endogenous

variable that can influence the results (Zare et al., 2024). Therefore, it is recommended that subsequent studies expand their scope to include a broader range of parameters, such as insulin, fasting blood glucose, HOMA-IR, and other markers of oxidative stress, to comprehensively evaluate their association with obesity complications and insulin resistance (IR). This holistic approach will provide a clearer picture of IR's impact on the risk of developing metabolic syndrome in obesity.

Conclusion

Overall it can be concluded that both interventions diminish oxidative stress, as evidenced by a reduction in MDA levels following training. However, moderate-intensity resistance training has a higher suppressive effect on oxidative stress secretion than moderate-intensity strength training in obese students. Interestingly, this research also discovered a positive correlation between the reduction in MDA levels post-training and body fat indicators (weight, body fat percentage, and fat mass). In addition, a decrease in post-training MDA levels was also negatively related to an increase in skeletal muscle mass. Therefore, endurance training with moderate intensity carried out at least 3x/week for more than 4 weeks is recommended for individuals with obesity as a non-drug treatment for obesity to mitigate the risk of diseases associated with oxidative stress, such as cardiovascular disease.

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