

Effects of Different Intensities of Aerobic Exercise on Cardiorespiratory Fitness and Levels of Plasma Malondialdehyde and Superoxide Dismutase

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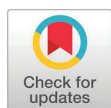
Received: January 22, 2024

Accepted: April 22, 2024

Published online: July 31, 2024

Keywords:

Cardiorespiratory Fitness
Malondialdehyde (MDA)
Superoxide Dismutase (SOD)



ABSTRACT

OBJECTIVES This study aimed to compare the effects of different intensities of aerobic exercise on cardiorespiratory fitness (CRF), plasma malondialdehyde (MDA), and superoxide dismutase (SOD) levels before and after an 8-week exercise.

METHODS Twenty-seven male university students were randomly divided into three intensity groups: light intensity (LI; 30-39% HRR), moderate intensity (MI; 40-59% HRR), and vigorous intensity (VI; 60-89% HRR). The study variables measured CRF factors such as maximal/peak oxygen uptake, ventilation, exercise time, and oxidative stress. MDA and SOD levels at rest, and following a graded exercise cessation before and after an 8-week exercise. Statistical analysis conducted two-way ANOVA with repeated measures after testing the normality of variables among groups using the Levene test.

Results The results showed significant increases in CRF factors, such as VO_2 peak/max, absolute VO_2 , and exercise time at both moderate and vigorous intensities after 8 weeks. Furthermore, there were significant increases including ventilation at all three intensities after the 8-week exercise. The SOD level showed a significant difference in the low intensity exercise group, but there was no significant difference at the three intensities after exercise. Plasma MDA differed significantly at low and moderate intensities after exercise.

Conclusions Based on these results, this study concluded significant improvements in cardiorespiratory fitness (CRF) factors such as $VO_{2peak/max}$, absolute VO_2 , and exercise time in both moderate and vigorous intensities exercise groups after exercise of 8 weeks. There were significant increases at three intensities after exercise of 8 weeks in ventilation. SOD levels also showed an increase in the low-intensity exercise group, while plasma MDA decreased in low and moderate-intensity exercise groups.

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Introduction

Physical Activity Guidelines recommend that adults engage in at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity aerobic physical activity per week to gain some health benefits [1]. There is a dose-response curve that the risk of coronary heart disease (CHD) or cardiovascular disease

(CVD) decreases linearly in association with increasing the amount of physical activity [2]. Cardiorespiratory fitness (CRF) can be evaluated as measured maximum or peak oxygen uptake for performing a graded exercise test. These are associated with cardiovascular health and longevity [3]. However, since the discovery of free radicals in living cells in 1954 [4], it has been known that oxidative stress can cause an increase in reactive oxygen species (ROS) as a result of aerobic metabolism involving oxygen consumption and oxidative phosphorylation [5]. Certain biological markers of oxidative stress indicate Malondialdehyde

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(MDA) and Superoxide dismutase (SOD) [6]. MDA, the final product of lipid peroxidation can be used as an indirect measure of cumulative lipid peroxidation [6]. SOD is the primary antioxidant that acts as the first line of defense and the first detoxification enzyme [7], and the most powerful antioxidant in the cell plays an indispensable role in the entire defense strategy of antioxidants [8]. Antioxidants play an important protective role against excessive production of free radicals, through scavenging or inhibiting their activities [9]. Oxidative stress (OS) occurs when there is an imbalance between reactive oxygen species (ROS) and enzymatic and nonenzymatic antioxidants [10]. This imbalance can cause damage to lipids, DNA, and proteins. Exercise, regardless of the intensity or duration, generates oxidative stress, leading to negative effects [11].

In many studies, OS, antioxidants, and exercise outcomes vary. Exercise leads to oxidative stress and lower levels of antioxidant enzymes in various tissues and organs. However, the intensity and type of exercise can result in varying degrees of oxidative stress [5]. Subjects with high VO₂ max showed significantly higher muscle catalase and superoxide dismutase activities than those with low-moderate fitness levels, according to the report [12]. Also, exercise training promotes an increase in primary antioxidant enzymes like SOD in cardiac and skeletal muscle [13]. This adaptation increases with exercise intensity and duration [14]. Antioxidant status can be improved through endurance training, which raises CRF levels [15]. However, in studies related to exercise intensity, exercise increases OS and/or antioxidant capacity. Previous researches conclude that OS is more activated with intense exercise [16]. The human body activates antioxidant enzymes to ensure hemostasis in response to the generation of OS after exercise [17]. Many studies reported that exercise at an intensity below 75% of VO₂ max can help minimize free radical damage [18] and prolonged treadmill running at 60% - 90% VO₂ max for 6 weeks does not prevent an exercise-induced increase in oxidative stress [19]. Lipid peroxides did not change at long-term or acute physical exercise on a cycle ergometer [20] or decreased during a graded exercise test [21]. These inconsistent outcomes were influenced by the intensity of exercise and the training level of the subjects [22]. In particular, information regarding the association between oxidative stress and cardio-pulmonary fitness could potentially be used to predict the risk

of developing cardiovascular and pulmonary diseases [23]. CRF factors can be measured by VO₂ consumption, which has been known to cause an increase in oxidative stress due to aerobic metabolism. However, there are still few studies regarding the association between CRF factors in chronic exercise of different intensities and oxidative stress.

The purpose of this study was to compare the effects of different intensities of aerobic exercise cardiorespiratory fitness factors, plasma malonaldehyde, and superoxide dismutase levels before and after the 8-week regular exercise.

Materials and Methods

Participants

Twenty-seven male university students without medical problems were divided randomly into three groups; light intensity, LI; n=9, moderate intensity, MI; n=9, and vigorous intensity, VI; n=9. Following the completion of a health screening questionnaire, written informed consent was obtained from all participants after explaining the study purpose, procedure, and possible risks. The physical characteristics of the participants are presented in <Table 1>

Study design

Physical characteristics were measured in height (cm), body weight (kg), percentage of body fat (%), and body mass index (BMI) at rest with an automatic height scale (TKK4343B, TAKEI Co., Ltd., Japan) and bio-impedance analyzer (Inbody 720, Biospace Co., Ltd., Korea) dressed in a short-sleeved cotton shirt and cotton shorts. The testing room was kept at a temperature between 68°F and 72°F, with humidity below 60% and adequate airflow. The participant's heart rate (HR), diastolic blood pressure (DBP), and systolic blood pressure (SBP) at rest were measured using electrocardiography (ECG) with the CH 2000 device from Cambridge Heart, Inc. (USA). The cardiorespiratory variables measured peak/maximum oxygen uptake (VO₂peak/max), ventilation (VE), and exercise time (seconds). Blood was collected to measure plasma malondialdehyde (MDA) and superoxide dismutase (SOD) at rest and following all-out of a graded exercise test before and after exercise of 8 weeks. The cardiorespiratory variables were measured with a breath-by-breath

Table 1. Physical characteristics of the participants.

(m ± SD)

Groups	Age (yrs)	Height (cm)	Weight (kg)	BMI (kg/m ²)	%BF	HR rest (bpm)	HR max (bpm)	SBP (mmHg)	DBP (mmHg)
LI (n=9)	23.11	175.57	71.12	23.45	21.56	85.11	196.44	114.44	68.66
	± 1.61	± 5.24	± 6.26	± 5.07	± 8.58	± 8.60	± 1.23	± 10.48	± 6.96
MI (n=9)	25.00	177.13	76.35	25.41	24.77	78.11	193.66	121.33	75.33
	± 2.06	± 6.82	± 9.11	± 3.09	± 4.48	± 11.21	± 4.15	± 7.14	± 9.98
VI (n=9)	24.56	178.66	73.40	22.73	18.82	77.22	195.66	115.00	70.22
	± 2.00	± 7.09	± 9.36	± 2.65	± 5.34	± 6.72	± 2.29	± 8.63	± 6.13
Levene test p*-values	.778	.659	.437	.485	.196	.196	.070	.161	.198

Values are presented as mean ± standard deviation. LI, light intensity 30~39% HRR; MI, moderate intensity 40~59% HRR; VI, vigorous intensity 60~89% HRR, bpm, beats per minute; BMI, body mass index, %BF, Percentage body fat; HR rest, rest heart rate; Age, years old; SBP, systolic blood pressure; DBP, diastolic blood pressure. HR max = 220 - age [24]. * No significant differences between groups; p-values using one-way analysis of variance.

gas analysis system (Quark b2, COSMED, Italy) with the As-trand exercise protocol using a cycle ergometer [25]. This proto-col began at 600 kgm · min⁻¹ (50rpm * 2kg; 100W) and increased by 300kgm · min⁻¹ every two minutes. The reasons for stopping exercise were exhaustion, RER >1.10, cardiovascular events, fa-tigue, or RPE >17 [26].

Exercise intervention

The exercise program consisted of a 5-minute warm-up, 30 minutes of aerobic exercise, and a 5-minute cool-down on a treadmill machine three times a week for 8 weeks. The exercise intensity was classified based on the percentage of Heart Rate Reserve (HRR) using the guidelines outlined in the ACSM [25]. The target heart rate (THR) of the subject was calculated using the Karvonen formula [27], which is [(HR max - HR rest) × % intensity desired] + HR rest. HR max was estimated using the formula 220 - age [24]. The exercise groups of three intensities were randomly divided into light intensity (LI; 30-39% HRR), moderate intensity (MI; 40-59% HRR), and vigorous intensity (VI; 60-89% HRR) [25]. The estimated lower and upper limits of the target heart rate (THR) range were LI (114.31±7.76 ~ 124.79±6.89 bpm), MI (125.95±6.80 ~ 148.06±5.01 bpm), and VI (149.22±4.92 ~ 182.97±2.53 bpm) monitored by a wireless heart rate system (Bodypro M100, Du-sung tech. co., Korea) during the aerobic exercise on treadmill machine.

Blood Analysis

Participants were instructed to fast for 8 hours before the

assessment, refrain from smoking or drinking alcohol, and avoid strenuous physical activity on the day of the measurement for blood analysis. Blood samples were collected from finger-tip capillaries to measure plasma malondialdehyde (MDA) and superoxide dismutase (SOD). Blood collection was used a 22-gauge needle from the antecubital vein into SST tubes. The collected blood was centrifuged, and the plasma supernatant was separated from the white buffy layer. The plasma supernatant was then refrigerated at -80°C for further analysis. Plasma MDA concentration was analyzed using a colorimetric assay in a Hi-Tech Scientific (USA) spectrophotometer, with a BIOXYTECH kit (Oxis Biotech, Tampa, FL, USA). SOD activity was measured using a colorimetric method with a Superoxide Dismutase Assay Kit (#CM706002, IBL International, Germany) and a microplate reader (GENios, TECAN, Austria).

Statistical analysis

Statistical analysis was analyzed using the Statistical Pack-age for Social Sciences, ver. 25, KOREA. Data were presented as mean and standard deviation (SD). After the normality of variables among groups was conducted using the Levene test, the analysis of variance (ANOVA) with the two-way repeated measures was used to determine the main effects (times and in-tensities) among the three groups after exercise. If there is a sig-nificant difference between pre-and post-exercise results within each group, post hoc analysis used the paired t-test and tested the difference with Tukey's HSD test by one-way ANOVA within the groups after exercise. Also, the effect size (ES) was classified

[28] as follows: small for values <0.5, moderate for values 0.50-0.79, and large for values >0.8. The significance level for statistical analysis will be established at a p-value of less than 0.05.

Results

The results of the cardiorespiratory fitness (CRF) factors before and after an 8-week exercise program are presented in <Table 2>. There were significant differences between MI (B) and (E), VI (C) and (F) before and after an 8-week exercise ($F=83.402$, $p<0.01$) in VO_{2peak}/max ($ml \cdot kg^{-1} \cdot min^{-1}$). After the 8-week exercise program, there were significant differences among the three groups (D < E < F) in VO_{2peak}/max and absolute VO_2 ($L \cdot min^{-1}$), respectively ($p<0.01$). VO_{2peak}/max and absolute VO_2 showed an interaction effect between intensity and time ($F=78.253$, $P<0.01$), and ($F=4.38$, $P<0.01$), respectively.

There was found a significant difference between LI (D) and VI (F) ($p<0.05$) in ventilation ($L \cdot min^{-1}$). Furthermore, there were significant differences noted between MI (B) and (E), as well as VI (C) and (F) ($p<0.05$) in absolute VO_2 . Additionally, there was a significant difference observed between the three groups in post-exercise ($p<0.05$) in exercise time (seconds). The plasma SOD & MAD levels are presented in <Table 3>. SOD levels showed a significant difference between LI (A) (1.97 ± 0.43 U/L) and LI (D) (2.30 ± 0.43 U/L) ($p<0.01$), but there were no significant differences between the three groups in post-exercise. There were significant differences in plasma MDA concentration between LI (A) with 829.66 ± 25.34 Umol/L and LI (D) with 733.55 ± 48.29 Umol/L, as well as between LI (B) with 818.00 ± 46.79 Umol/L and LI (E) with 740.33 ± 56.67 Umol/L. The plasma MAD levels showed an interaction effect between intensity and time ($F=4.360$, $P<0.05$).

Table 2. The variables of the cardiorespiratory fitness.

Variables	Time	Intensity	m	SD	F	p-value	post hoc	ES	
VO ₂ max/peak kg·mL·min ⁻¹	pre	LI (A)	27.97	± 2.39	Time Intensity Tme x Inten.	83.402 9.167 78.253	.003 .001 .001	B < E**, C < F** D < E** < F**	.283 .404 .426
		MI (B)	26.41	± 3.78					
		VI (C)	27.89	± 3.26					
	post	LI (D)	26.16	± 2.21					
		MI (E)	29.25	± 1.83					
		VI (F)	33.94	± 3.41					
absolute VO ₂ L·min ⁻¹	pre	LI (A)	1.95	± .23	Time Intensity Tme x Inten.	.630 .978 .438	.002 .001 .006	B < E**, C < F** D < E** < F**	.312 .316 .387
		MI (B)	2.15	± .43					
		VI (C)	2.05	± .38					
	post	LI (D)	1.84	± .23					
		MI (E)	2.40	± .34					
		VI (F)	2.52	± .23					
Ventilation L·min ⁻¹	pre	LI (A)	64.30	± 6.46	Time Intensity Tme x Inten.	59.004 5.125 47.893	.301 .013 .417	ns D < F*	.040 .275 .063
		MI (B)	67.70	± 8.37					
		VI (C)	67.89	± 3.94					
	post	LI (D)	62.93	± 8.72					
		MI (E)	70.29	± 7.62					
		VI (F)	72.62	± 6.53					
Exercise time (sec)	pre	LI (A)	682.00	± 131.5	Time Intensity Tme x Inten.	69632.267 71765.317 18092.117	.031 .036 .277	B < E*, C < F* D < F*	.161 .219 .091
		MI (B)	652.80	± 136.3					
		VI (C)	747.80	± 141.4					
	post	LI (D)	681.10	± 72.8					
		MI (E)	762.10	± 148.2					
		VI (F)	843.80	± 118.5					

** $p<0.01$, * $p<0.05$; ns, no significance; LI, light intensity; MI, moderate intensity; VI, vigorous intensity, ES (effect size) was classified; small <0.05; medium 0.06-0.13; large >0.14 [28]. $VO_2 \text{ max/peak}$, maximum/peak oxygen uptake, absolute VO_2 , absolute oxygen uptake, VE, expired ventilation per minute.

Table 3. Comparison of the plasma SOD & MAD levels.

Variables	Time	Intensity	m	SD	F	p-value	Post hoc	ES	
SOD (U/L)	pre	LI (A)	1.97	± .43	Time Intensity Tme x Inten.	10.337 .034 2.205	.004 .967 .132	A<D** Ns	.301 .003 .155
		MI (B)	2.07	± .44					
		VI (C)	2.09	± .42					
	post	LI (D)	2.30	± .43					
		MI (E)	2.24	± .37					
		VI (F)	2.13	± .29					
MDA (Umol/L)	pre	LI (A)	829.66	±25.34	Time Intensity Tme x Inten.	37.258 2.838 4.360	.001 .078 .024	A<D**, B<E** Ns	.608 .191 .266
		MI (B)	818.00	±46.79					
		VI (C)	825.66	±41.92					
	post	LI (D)	733.55	±48.29					
		MI (E)	740.33	±56.67					
		VI (F)	803.77	±44.67					

**p<0.01, *p<0.05; ns, no significance; LI, light intensity; MI, moderate intensity; VI, vigorous intensity, ES (effect size) was classified; small<0.05; medium 0.06-0.13; large >0.14 [28]. SOD, superoxide dismutase; MDA, malonaldehyde

Discussion

The present study investigated the effects of different intensities of aerobic exercise before and after an 8-week exercise program on cardiorespiratory fitness and the levels of plasma malondialdehyde (MDA) and superoxide dismutase (SOD). Whether you're exercising or not, the oxygen in your body is used to break down glucose and create the fuel for your muscles called ATP. During exercise, body muscles have to work harder, which increases their oxygen demand. Cardiopulmonary fitness (CRF) evaluates the body's response to exercise, involving the pulmonary, cardiovascular, and skeletal muscle systems at rest and during exercise [29]. The intensity of the exercise and the training level of the subjects affect aerobic capacity [22]. In particular, regular aerobic exercise leads to a wide range of important adaptations that improve aerobic capacity, such as increased mitochondrial content and capillary density [30]. In this study, both VO_2 peak/max ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and absolute VO_2 ($\text{L} \cdot \text{min}^{-1}$) significantly increased at moderate and vigorous intensities after 8 weeks of aerobic exercise compared to the levels before exercise. These results are the effects of regular and appropriate exercise. Also, after 8-week of aerobic exercise, there was a significant increase in CRF factors, including ventilation and exercise time, as exercise intensity increased among three intensities. These results are consistent with studies showing the positive impact of regular aerobic exercise on CRF improvement [30]. Oxidative

stress and exercise capacity are the parameters to predict an individual's cardiovascular risk [31]. Maintaining a regular exercise setting the optimal exercise intensity would benefit the body's anti-oxidative potential [23]. However, excessive exercise beyond an appropriate intensity, duration, and form has negative effects of oxidative stress on the human body [5]. Previous research on exercise intensity reported that oxidative stress is increased and antioxidant enzymes decreased at high-intensity exercise or above compared to moderate-intensity exercise [33]. There are other results that the peak VO_2 was positively correlated with serum SOD activity, while negatively correlated with thiobarbituric acid reactive substances [32]. This study showed that the SOD level was increased after prolonged exercise. These results were the effects that regular exercise can significantly increase anti-oxidant defenses, possibly to counteract higher levels of reactive oxygen species caused by exercise [33]. MDA concentration was decreased at both low- and moderate-intensity exercises after exercise. This result is similar to exercise with moderate intensity causes less lipid peroxidation in comparison with high-intensity exercise [35]. But, the previous study is not corresponding that MDA and SOD levels were to increase significantly following high-intensity aerobic exercise [34]. These results support that blood lipid peroxide was decreased in response to prolonged exercise time due to an adaptation effect, and blood MDA amounts appeared to remain somewhat constant for the training [36]. Increased SOD levels and decreased MDA concentration were

possible to explain because of the effect of regular exercise to increase antioxidant defenses. However, although the present result and some studies suggest that aerobic training may increase antioxidant capacity and decrease oxidative stress, the results are inconsistent and varied. This is perhaps because a variety of factors can influence oxidative stress, such as modes of muscle contraction, exercise intensity, and exercise duration. Therefore, further studies will be done to attain consistent information on oxidative stress and antioxidants considering exercise types and participant conditions.

Conclusions

Based on these results, this study concluded significant improvements in cardiorespiratory fitness (CRF) factors such as $\text{VO}_{2\text{peak}}/\text{max}$, absolute VO_2 , and exercise time in both moderate and vigorous intensities exercise groups after exercise of 8 weeks. There were significant increases at three intensities after exercise of 8 weeks in ventilation. SOD levels also showed an increase in the low-intensity exercise group, while plasma MDA decreased in low and moderate-intensity exercise groups.

Conflicts of Interest

The authors declare no conflict of interest.

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