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Research Article

The Effect of Anaerobic and Aerobic Exercise on Mitochondrial Function and Lipid Metabolism in Healthy Young Adults

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Abstract
The intricate interplay between physical exercise, mitochondrial function, and lipid metabolism has garnered considerable attention, particularly in the context of energy balance and overall metabolic health. Studies comparing which exercise has the most beneficial effects on mitochondrial activity and lipid metabolism are still scarce. This study aimed to investigate the effects of anaerobic and aerobic exercise on mitochondrial activity and biogenesis, as well as lipid metabolism, in healthy young adults. Eighteen subjects (young male adults) were randomly divided into two groups: anaerobic exercise group (n=9) and aerobic exercise group (n=9) for 4 weeks. Blood samples were collected before and after training and the whole blood was separated into leucocyte cells and plasma. Lysate from leucocyte cells was used for the measurement of mitochondrial function, including succinate dehydrogenase (SDH) activity, peroxisome proliferator-activated receptor gamma coactivator 1-α (PGC-1α), and ATP levels. Blood plasma was used for the measurement of lipid metabolism, including HDL cholesterol, adiponectin, and free fatty acids levels. This study demonstrated that SDH activity increases significantly after aerobic exercise (122%) compared to before exercise, although it is not supported by the results of PGC-1a and ATP levels. This may be because these parameters are measured in the blood (leukocyte cells). Adiponectin levels increase significantly after anaerobic exercise (43.8%) while free fatty acid levels increase significantly after aerobic exercise (104%) compared to before exercise. The results of HDL cholesterol levels found a tendency to increase both after aerobic (12%) and anaerobic exercise (10%) compared to before exercise. We conclude that aerobic exercise gives the best impact on increasing mitochondrial activity systemically (significantly increasing SDH activity), supported by the significant increase in free fatty acid levels in the blood, which reflects that lipolysis is increased.

Keywords: aerobic exercise; anaerobic exercise; lipid metabolism; mitochondrial function

1. INTRODUCTION

Physical exercise is one of the most effective ways to prevent metabolic diseases (obesity, hyperlipidemia, metabolic syndrome, diabetes mellitus) and cardiovascular disease (hypertension, coronary heart disease, heart failure). There are two main types of exercise, aerobic and anaerobic, which differ in terms of intensity, duration, and the types of muscle fibers engaged. Aerobic exercise involves continuous and rhythmic activities that rely on oxygen for energy production. These exercises are usually performed at a moderate intensity over a longer duration, such as running, cycling, swimming, walking and dancing. Anaerobic exercise is short, intense bursts of activity that don't require oxygen for energy production. The body relies on stored energy in muscles for quick and explosive efforts, for examples weightlifting, sprinting, high-intensity interval training (HIIT) and jumping [1][2].

Mitochondria play a crucial role in energy production (ATP) within muscle cells, and their number and function are crucial for performance in endurance and explosive sports. Aerobic and anaerobic exercise trigger distinct physiological and metabolic responses in our body. It has been reported that endurance training can enhance muscle aerobic capacity by stimulating mitochondrial biogenesis (the generation of new mitochondrial components that improve mitochondrial function and quantity) [3]-[5]. However, studies are still needed to explore the best training exercise to stimulate mitochondrial biogenesis.

Mitochondrial biogenesis is crucial for enhancing muscle function, particularly in terms of endurance and energy production during exercise. The process is regulated by several pathways, most notably through the activation of peroxisome proliferator-activated receptor gamma coactivator 1

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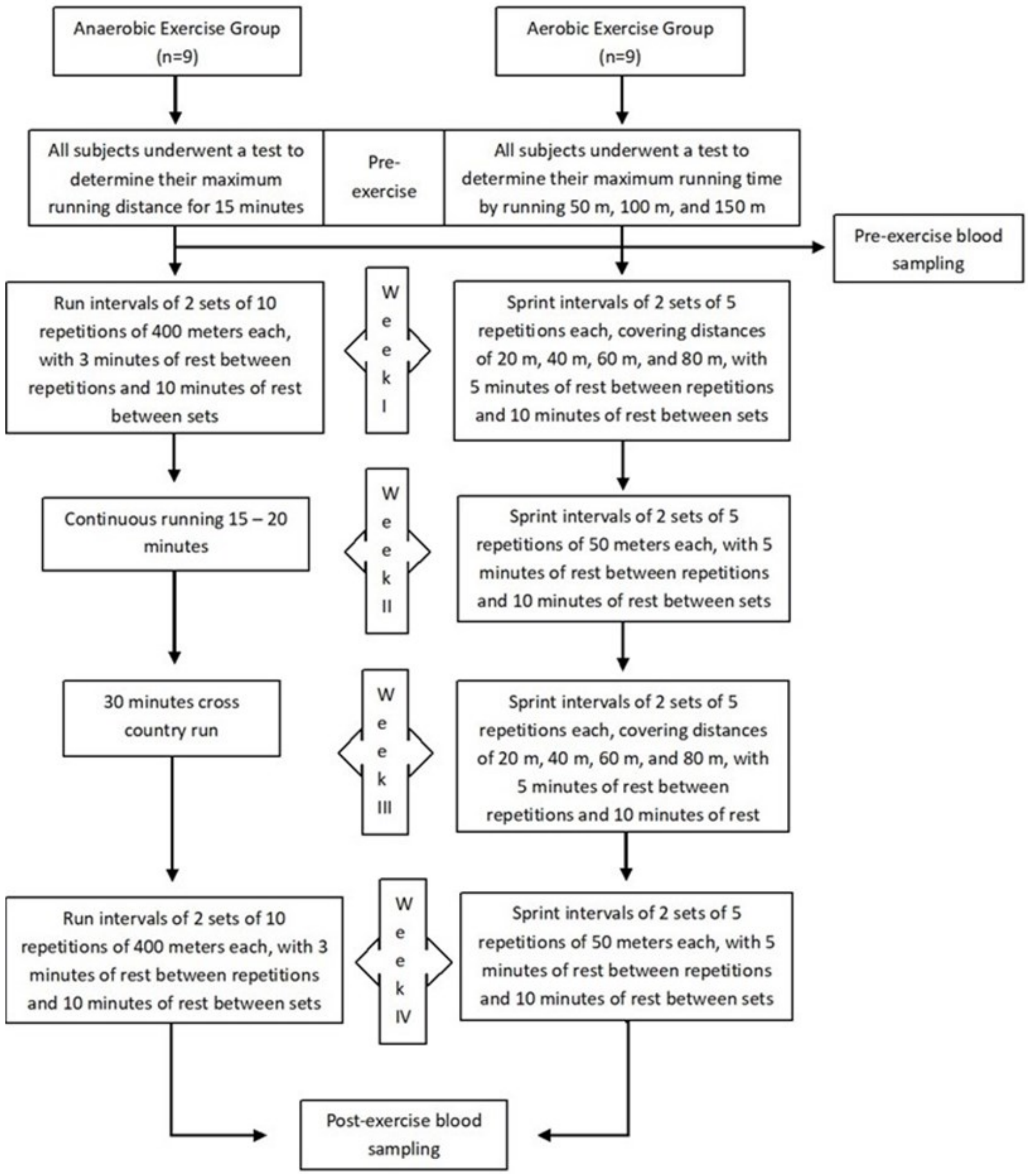

Figure 1. Flowchart anerobic and aerobic exercise treatment to young adults.

-alpha (PGC-1α), a key regulator of mitochondrial function and biogenesis [6]. Besides that, succinate dehydrogenase (SDH), also known as complex II, plays a key role in connecting two important processes within mitochondria: the oxidation of succinate to fumarate as a crucial part of the TCA cycle and the conversion of ubiquinone to ubiquinol in the mitochondrial electron transport chain (ETC). Thus, this is an important molecule that can be used for representing mitochondrial function [7].

Appropriate physical exercise stimulates lipolysis, the breakdown of triacylglycerols stored in adipose tissue, leading to the release of free fatty acids (FFAs) into the bloodstream, where they are oxidized in muscles and other tissues. Free fatty acid is a crucial fuel for muscle contraction, both at rest and during physical activity. Triglycerides (TGs), found in adipose tissue and muscle fibers, are the primary source of FFAs that are oxidized during exercise [8]. However, the mechanisms controlling the utilization of these substrates during exercise and what type of exercise is the most effective to induce lipolysis remain unclear.

Exercise can increase the secretion of adipokines, including adiponectin, from adipose tissue. Adiponectin is a beneficial adipokine that has anti-inflammatory effects, improves insulin sensitivity, enhances fatty acid oxidation in the muscles and inhibits the synthesis of lipids and glucose in the liver [9]. During exercise, particularly aerobic activity, the levels of adiponectin in the bloodstream can rise, which may contribute to improved metabolic health [10]. However, the exact mechanisms and the extent of this increase can vary depending on factors such as exercise intensity, duration, and the individual's fitness level. The correlation between mitochondrial biogenesis and lipid metabolism in aerobic and anaerobic exercise is a key area of interest in exercise physiology. Both processes are integral to how the body adapts to different types of exercise, and understanding their interaction can offer insights into optimizing training for performance, endurance, and metabolic health. Therefore, this study aims to analyze the relationship between mitochondrial biogenesis and lipid metabolism in aerobic and aerobic exercise, and to reveal which type of exercise is most effective in increasing mitochondrial biogenesis and lipolysis.

2. MATERIALS AND METHODS

2.1. Materials

Samples were gained from the whole blood of healthy young adult male before and after exercise treatment. Ficoll-Paque PLUS Reagent (GE Healthcare, 190 Chalfont St Giles, UK), Succinate Dehydrogenase Activity Assay Kit (Elabscience®), RIPA lysis and extraction buffer (Thermo Scientific®), PGC-1α ELISA Kit (Elabscience®), ATP Chemiluminescence Assay Kit (Elabscience®), HDL cholesterol Colorimetric Assay Kit (Elabscience®), Human Adiponectin ELISA Kit (FineTest®), and FFAs Fluorometric Assay Kit (Elabscience®) were used in this study.

2.2. Methods

The design of this study is a pre- and post-clinical trial study using healthy young adult male subjects. This study was conducted following the ethical principles outlined in the Declaration of Helsinki. The Ethics Committee of the Faculty of Medicine, University of Indonesia—Cipto Mangunkusumo Hospital granted its approval for the study with approval number KET-1085/ UN2.F1/ETIK/PPM.00.02/2024.

2.2.1. Subjects

This study involved 18 healthy young adult male who were not smokers and had no known medical concerns. They gave signed informed consent after being fully explained the purpose, benefits, and procedures of the study. Participants were randomly allocated to one of two groups: aerobic (n=9) or anaerobic (n=9).

2.2.2. Exercise Protocol

The participants in this study were men between

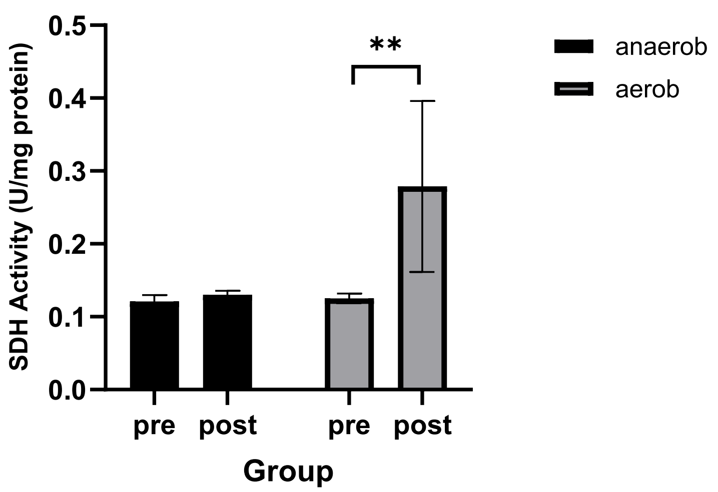

Figure 2. The SDH activity in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks. Statistical analysis used paired-t test with significancy **p<0.001.

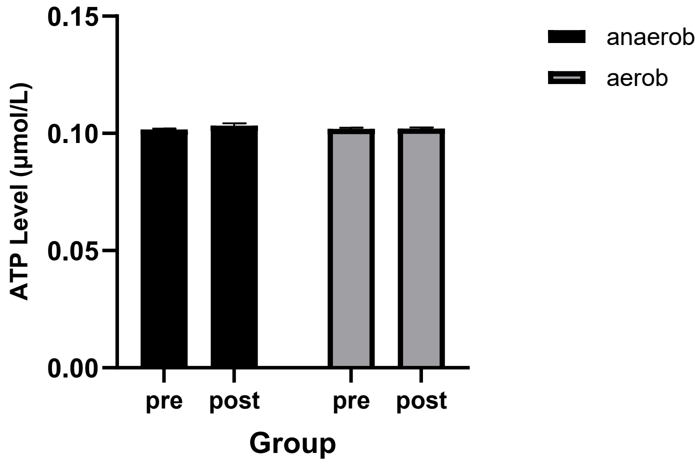

Figure 3. The ATP level in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks. Statistical analysis used paired-t test with no significant result.

the ages of 18 and 23, weighing 56–70 kg, and standing 158–175 cm tall. Following that, blood pressure and VO2max were measured. Eligibility requirements were the systolic blood pressure below 130 mmHg, the diastolic blood pressure below 90 mmHg, and the maximum oxygen consumption (VO2max) of more than 40 mL/kg/min. We described the detail of exercise treatment in Figure 1.

2.2.3. Sample Preparation

Whole blood (5 mL) was diluted with sterile phosphate-buffered saline (PBS) at a 1:1 v/v ratio. We added density gradient medium (Ficoll®) with 1:1 v/v ratio into diluted blood and then centrifuged it at 400 xg for 30 min at room temperature to collect leucocytes/peripheral blood mononuclear cells (PBMC). The leucocytes were lysed using RIPA lysis and extraction buffer (Thermo Scientific®). Blood plasma (the upper part) was transferred into new tube and stored at -20 °C for measurement of HDL, adiponectin and FFA levels. Leucocytes/PBMC (the middle part) were transferred into new tube and lysed it by buffer solution. This lysate was used for measurement of SDH, PGC-1α and ATP levels.

2.2.4. Measurement of Succinate Dehydrogenase, PGC-1α and ATP levels

The activity of SDH level was measured using Succinate Dehydrogenase Activity Assay Kit. PGC-1α level was measured using Human PPARγC1α ELISA Kit, while ATP level was measured using ATP Chemiluminescence Assay Kit. The samples for these measurements were the lysate of leucocyte cells. All measurements were performed according to the manufacturer's guidelines.

2.2.5. Measurement of HDL, Adiponectin and Free Fatty Acids Levels

HDL cholesterol level was measured using HDL cholesterol Colorimetric Assay Kit. Adiponectin level was measured using Human Adiponectin ELISA Kit. FFA level was measured using Free Fatty Acids Fluorometric Assay Kit. The samples for these measurements were the blood plasma. All measurements were performed according to the manufacturer's guidelines.

2.2.6. Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS v.24 for Windows). The normality of data distribution was assessed with the Kolmogorov-Smirnov and Shapiro-Wilk tests. Our data were normally distributed, a paired t-test was used to compare pre- and post-exercise results within the aerobic and anaerobic exercise groups, while an unpaired t-test was employed to compare the pre- and post-ratios between the two groups. Results are presented as mean ± standard deviation (SD). A significance threshold of $p < 0.05$ was set for all statistical analyses. Data visualizations were created using GraphPad Prism.

3. RESULTS AND DISCUSSIONS

This study analyzed the mitochondrial function by measuring SDH activity and ATP level after anaerobic and aerobic exercise treatment in healthy young adults (Figure 2). In addition, PGC-1a levels were also measured as a marker of mitochondrial biogenesis. We found that SDH activity significantly increased (p=0.000, paired-t test) after aerobic exercise (0.2788±0.1172 U/mg protein) compared to before exercise (0.1253±0.0069 U/mg protein). However, it was only slightly increased after anaerobic exercise (0.1300±0.0056 U/mg protein) compared to before exercise (0.1212±0.0085 U/mg protein). We found that the increasing of SDH after aerobic exercise was higher (122%) than after anaerobic exercise (only 7%). The results of ATP levels after both anaerobic and aerobic exercise showed no alteration between before and after exercise (Figure 3).

We demonstrated that after aerobic exercise, the mitochondrial activity increased, proven by the significant increase in SDH activity compared to before exercise. In anaerobic group, the SDH activity was found slightly higher than before exercise but not significant. This result showed that both aerobic and anaerobic exercise stimulate the increasing of mitochondrial activity through stimulating citric acid cycle. Although the measurement of SDH activity was only performed on leucocytes, it can show the systemic effects on mitochondrial activity after exercise as in muscle cells. The previous study reported that endurance training led to a rise in succinate dehydrogenase activity in both younger and older participants, as well as an increase in the number of capillaries surrounding type I muscle fibers [11].

However, if we look at the result of ATP level, there was no increasing of ATP level in both aerobic and anaerobic exercise. This is because ATP levels are measured in the blood, especially in leucocytes, which do not reflect the actual ATP production in muscle cells. Although SDH activity increases significantly during aerobic exercise, ATP levels are not significantly increased. This is also supported by the results of PGC-1α levels which showed a significant decrease after both aerobic and anaerobic exercise. These results indicate that both aerobic and anaerobic exercise treatments do not increase mitochondrial biogenesis even though mitochondrial activity (citric acid cycle) increases. However, this phenomenon needs to be investigated further.

Mitochondrial biogenesis was also measured in this study by analyzing the PGC-1α level (Figure 4). The results of PGC-1a levels showed that there was a significant decrease (p=0.000, paired-t test) both after anaerobic (0.0117±0.0008 ng/mg protein) and aerobic exercise (0.0158±0.0015 ng/mg protein) compared to before exercise (0.0144±0.0024 ng/mg protein for pre-anaerobic and 0.0211±0.0026 ng/mg protein for pre-aerobic).

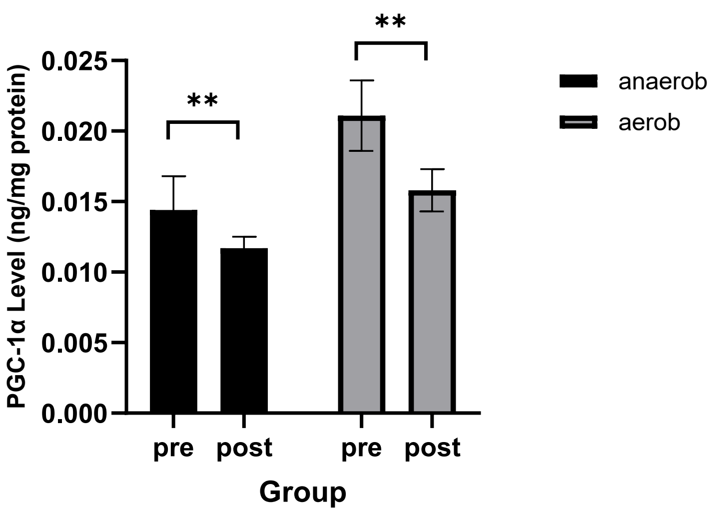

Figure 4. The PGC-1a level in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks. Statistical analysis used paired-t test with significancy **p<0.001.

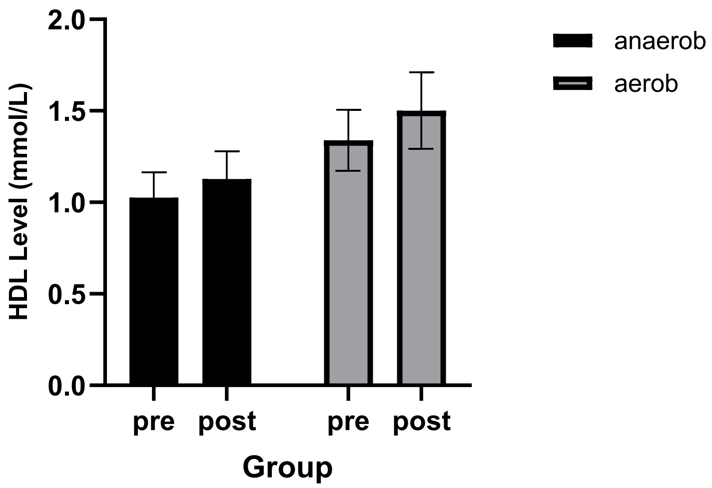

Figure 5. The cholesterol HDL level in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks.

We found the decreasing of PGC-1α after aerobic exercise slightly higher (25%) than after anaerobic exercise (19%). PGC-1α is a crucial transcriptional coactivator that plays a central role in regulating energy metabolism, particularly mitochondrial biogenesis and function, and is involved in various metabolic processes [12]. Some studies reported that exercise strongly induces PGC-1α expression in skeletal muscle, leading to increased mitochondrial oxidative metabolism and improved endurance [4][13][14]. The other studies showed that regular aerobic exercise stimulates mitochondrial biogenesis through the activation of PGC-1α [15][16]. In this study found the opposite results from the previous study, because it might occur due to several factors related to how the body regulates gene expression and the metabolic processes during recovery, beside that this study measured PGC-1α levels in the blood, especially leucocyte because it is a transcription factor inside cells. Exercise, especially intense or prolonged activity, can cause an acute stress response in the body. Initially, there might be an increase in PGC-1α expression, but after exercise, the body shifts towards a recovery mode where the initial upregulation of genes like PGC-1α may be downregulated as part of the normalization of cellular activity [17]. Beside that after exercise, inflammatory markers increase, and the immune system may focus on resolving inflammation rather than supporting processes like mitochondrial biogenesis, which PGC-1α is involved in [18].

Another study using the same sample as this study measured oxidative stress status and revealed a significant decrease in malondialdehyde (MDA) levels after both aerobic and anaerobic exercise. Furthermore, there was a significant increase in superoxide dismutase (SOD) enzyme activity and a significant increase in total antioxidant capacity after aerobic exercise [19]. The data corroborate the study's findings, demonstrating an upregulation of antioxidant enzyme activity (SOD) alongside a reduction in oxidative stress biomarker (MDA), indicative of a good mitochondrial integrity and function. In contrast, elevated oxidative stress is known to induce mitochondrial damage and functional impairment [20]. Optimal mitochondrial function is a critical factor in the regulation and promotion of mitochondrial biogenesis [21].

In this study also analyzed the alterations in lipid metabolism that were measured from HDL cholesterol, adiponectin and FFA levels in the blood after anaerobic and aerobic exercise in healthy young adults. The results show that HDL cholesterol levels tended to increase after both anaerobic (10%) and aerobic exercise (12%), although not statistically significant. The increasing in HDL levels in the aerobic group was greater than in the anaerobic group (Figure 5).

This result indicated that both aerobic and anaerobic exercises can lead to an increase in HDL levels, although the changes were not statistically significant. This finding aligns with the previous studies that highlight the positive effects of physical activity on lipid profiles, particularly HDL. Aerobic exercise has been shown to significantly improve HDL levels in individuals with overweight or obesity [22]. Some studies suggest that the overall impact of exercise on HDL may be modest, with only a small proportion of participants experiencing significant changes, the degree of HDL increase may vary based on individual factors such as exercise intensity, duration, and frequency [23][24].

To determine the lipolysis process that occurs in the subject, adiponectin and FFA levels in the blood were measured. The results of adiponectin levels showed a tendency to increase after anaerobic and aerobic exercise (Figure 6), especially after anaerobic exercise the elevation of adiponectin level (6.9277±2.0313 µg/mL) was statistically significant ($p=0.003$, paired-t test) compared to before exercise (4.8180±0.7210 µg/mL). The adiponectin after anaerobic exercise was only slightly increased than before exercise. FFA levels in the blood also supported the results of adiponectin, which showed a tendency to increase after anaerobic and aerobic exercise (Figure 7). However, a significant increasing ($p=0.048$, paired-t test) was observed after aerobic exercise (620.947±161.324 mmol/L) compared to before exercise (304.023±84.559 mmol/L). The increase of free fatty acids after anaerobic exercise was not statistically significant compared to before exercise.

Adiponectin is a hormone secreted by fat tissue that plays a crucial role in regulating metabolism, including glucose and fat metabolism. Adiponectin enhances lipid metabolism by promoting high-density lipoprotein cholesterol (HDL-C) formation and triglyceride catabolism [25]. A previous study revealed that aerobic exercise is associated with increased adiponectin levels, which is beneficial for metabolic health and insulin sensitivity [26]. Another study comparing aerobic and anaerobic exercises found that both types of exercise led to significant increases in adiponectin levels among non-athletic men [27]. The increasing of adiponectin also stimulates lipolysis in adipose tissues, which is proved by the elevation of free fatty acid after aerobic and anaerobic exercise.

Some studies have reported physical exercise stimulates the breakdown of stored fats (triglycerides in adipose tissues). Exercise increases the release of catecholamines, which stimulate lipolysis through hormone-sensitive lipase, leading to a higher rate of fat breakdown [28][29]. We demonstrated that aerobic exercise has a significant increase of free fatty acids between before and after exercise. FFA will be transported to the mitochondria, where they undergo β-oxidation to produce acetyl-CoA, which enters the Krebs cycle and is used to generate ATP. Another study also reported that during aerobic activities, the body shifts its energy source from carbohydrates to fats, enhancing the activity of lipoprotein lipase (LPL)

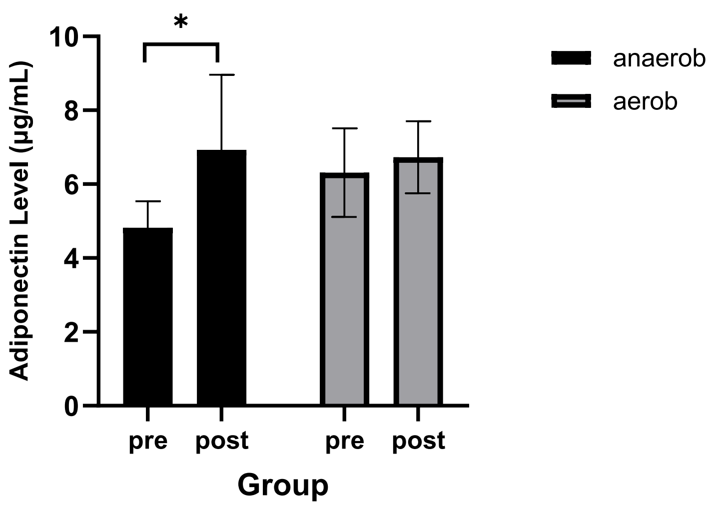

Figure 6. The adiponectin level in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks. Statistical analysis used paired-t test with significancy *$p<0.05$.

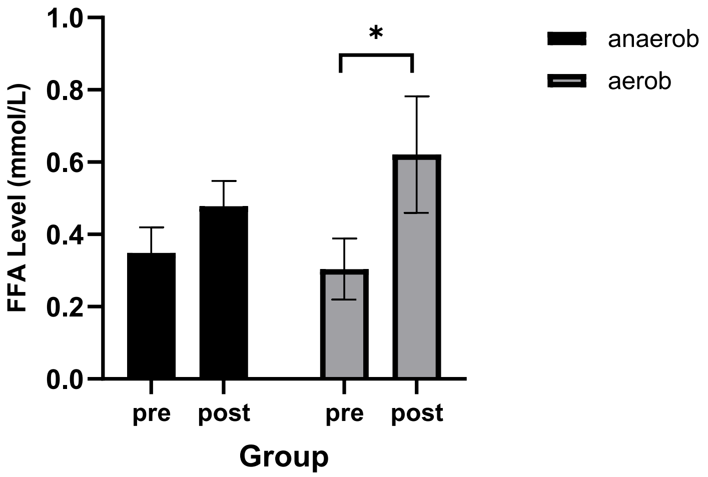

Figure 7. The free fatty acid level in the blood of healthy young adults before and after anaerobic and aerobic exercise for 4 weeks. Statistical analysis used paired-t test with significancy *p<0.05.

and facilitating triglyceride uptake by muscle cells [30]. Our findings indicate that lipolysis has a greater impact on aerobic exercise, although adiponectin levels increase more during anaerobic exercise. Aerobic metabolism relies on oxygen to efficiently oxidize fat for energy, beside that aerobic training enhances mitochondrial activity, which can be seen at the increasing of SDH activity.

Previous studies demonstrated that aerobic exercise increases the activity of enzymes involved in fat oxidation, such as carnitine palmitoyltransferase I (CPT-1), which facilitates the transport of fatty acids into mitochondria for oxidation [31][32]. This leads to an improvement in the body's ability to use fats as an efficient energy source during prolonged activity. Anaerobic exercise primarily uses stored glycogen for energy, because low oxygen state shifts metabolism pathway into glycolysis as source of energy instead of fat oxidation to supply sufficient ATP [32]. However, while anaerobic exercise relies less on fat as an immediate fuel source during activity, it can still influence lipid metabolism indirectly. While anaerobic exercise does not directly emphasize lipid oxidation during the activity itself, it can improve lipid metabolism at rest [33]. This occurs through enhanced mitochondrial capacity, which allows for greater fat oxidation during recovery periods, when the body is replenishing energy stores and repairing muscle tissues [34]. Therefore, our findings reveal elevated levels of free fatty acids in the blood following both aerobic and anaerobic exercise.

Although adiponectin supports fat metabolism but doesn't immediately drive lipolysis. Adiponectin activates AMP-activated protein kinase (AMPK), which promotes fatty acid oxidation and enhances insulin sensitivity, but does not directly trigger lipolysis [35]. Aerobic exercise maintains a prolonged fat-burning state. We found adiponectin elevates more in anaerobic exercise, nevertheless the body still prioritizes glycogen in anaerobic conditions. Anaerobic exercise primarily promotes muscle adaptations rather than direct fat oxidation [36]. While it enhances metabolic efficiency (including increasing adiponectin), it does not create the same fat-burning conditions as aerobic exercise. While anaerobic exercise can increase adiponectin levels, which are associated with improved metabolic health, the overall fat-burning effect is less pronounced than that of aerobic exercise [26]. Anaerobic training significantly enhances the oxidative capacity of fast-twitch muscle fibers, which can lead to improved energy production during recovery phases [37]. This causes the anaerobic exercise, which primarily utilizes glycogen, can increase in mitochondrial activity will lead to more efficient fat oxidation post-exercise, thereby promoting better metabolic health. This study revealed that both after aerobic and anaerobic exercise, the mitochondrial function and lipolysis increased, but the significant results can be found in aerobic exercise.

4. CONCLUSIONS

Based on the results, it can be concluded that both aerobic and anaerobic exercise enhance systemic mitochondrial activity, as evidenced by increased SDH activity, likely driven by elevated lipolysis indicated by higher circulating free fatty acid levels. However, the significant effects were observed primarily after aerobic exercise. No significant changes in PGC-1α or ATP levels were detected, possibly due to measurements being limited to blood samples rather than muscle tissue. Furthermore, both exercise modalities elicited an increase in blood HDL cholesterol levels. Nonetheless, the findings of this study provide a basis for investigating the direct effects of aerobic and anaerobic training on mitochondrial function and biogenesis within muscle cells, which can contribute to enhancing athletic performance.

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Conflicts of Interest

The authors declare no conflict of interest in this study.

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DECLARATION OF GENERATIVE AI

Not applicable.

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