Effects of Moderate and High Intensity Swimming Exercise on Liver Lipid Metabolism in Rats

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Abstract: The purpose of this paper is to measure the total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) in liver tissue by medium and high intensity swimming intervention. Describe the effects of white mice on lipid metabolism in the middle and high intensity swimming. In this experiment, 30 healthy adult male Sprague-Dawley rats were divided into control group (C group), moderate mild training group (M group) and experimental load group (O group), in which mice were distributed according to the equal proportion. The control group was naturally fed for five weeks according to the established schedule, the middle control group was swimming for five weeks according to normal operation, and the experimental load group was swimming for five weeks according to high intensity. Finally, the TC content and liver tissue HDL levels in the quiet control group and the moderately mild training group and the quiet liver tissue were taken as one of the important judgments of this study, and the difference was statistically significant (P<0.05). It was found that the content of HDL in liver tissue of group C was significantly higher than that of group C, and the difference was statistically significant (P<0.01). Compared with the O group, the HDL content in the liver tissue of the C group also increased, and the difference was extremely significant (P<0.01). Compared with group C, the LDL content of liver tissue in group M was significantly lower than that in group C, and its value was statistically significant (P<0.05). The LDL content in the liver tissue of group M and group O was significantly lower, and the difference was statistically significant (P<0.05). Experiments have shown that moderate-intensity, periodic, and appropriate aerobic exercise is beneficial to the liver's lipid metabolism and is more beneficial to the health of the normal population.

Key words: Liver Lipid Metabolism, Different Intensity Exercise, Animal Experiment, Lipid Metabolism

1. Introduction

As a physical activity that enhances physical fitness and maintains good physical condition, physical exercise also has two sides. High-intensity exercise is harmful to health [1]. A large number of scientific experiments and examples prove this point. In recent years, in the pursuit of “higher, faster, stronger” in competitive sports, there are more and more heart failure, visceral rhabdomyolysis and sudden death caused by excessive exercise, which reminds us of the science of athletes. The potential for exercise and the development of appropriate exercise intensity should be more concerned. Through animal experiments, it has been found that different exercise intensities have different effects on the brain, internal organs, bones, muscles, and the like. Studies have shown that strenuous exercise can aggravate or cause necrosis of rat cardiomyocytes. The causes of aggravation or necrosis include decreased antioxidant capacity, increased free radical production, various enzyme metabolic disorders, cellular blood supply and oxygen supply. Insufficient. Whatever the cause, the end result is cell membrane rupture and cell lysis [2]. Cholesterol plays an important role in all mammalian cell membranes. In addition, membrane cholesterol is strictly controlled at different concentration levels to maintain the homeostasis of the cell membrane. Biochemical and physical properties such as fluidity, initiative, and permeability of biological cell membranes are closely related to cholesterol concentration [3]. Cell membrane cholesterol affects the expression of various cell membrane functions and a variety of key proteins by regulating the activity of various enzymes. Current results indicate that the primary role of membrane cholesterol is achieved by maintaining the integrity and function of lipid regions on the membrane. The interaction between cholesterol lipids promotes lipid formation and small recesses in the membrane [4]. Therefore, studying the cholesterol balance on the cell membrane is important for studying the effects of exercise on cardiomyocytes.

Lack of exercise can lead to many diseases. In 1961, researchers studied the lack of exercise-induced illness. In 1996, the Centers for Disease Control and Prevention (CDC) listed the names of metabolic diseases caused by lack of exercise in its report on physical activity and health. The report also lists possible
complications of these diseases to alert people to the level of daily physical activity and the risk of lack of activity, such as obesity, hypertension and diabetes [5]. The World Health Organization (WHO) lists obesity as a risk factor for health. Obesity is one of the top five risk factors for health. Obesity is extremely harmful to human health. Studies have shown that the number of overweight and obese people in China is increasing year by year, approaching or exceeding a quarter of the national population [6]. Obesity can lead to abnormal lipid metabolism in the body. At present, the incidence of abnormal blood lipid metabolism has increased year by year, which seriously affects people's health. Abnormal lipid metabolism is one of the important risk factors for cardiovascular disease. Epidemiological studies have shown that cardiovascular disease morbidity and mortality are closely related to serum cholesterol, low-density lipoprotein (LDL) levels, and body fat percentage (PBF). Regular physical exercise improves abnormal blood lipid metabolism [7]. Previous studies focused on the lipid metabolism of sports in a single form of change campaign, and long-term training, one-time exhaustive exercise and training combined with one-time exhaustive exercise for a long time in the same study rare reports in animal models. This paper investigates the effects of different intensity swimming exercises on lipid metabolism in the same experiment [8]. By comparing the effects of different intensity swimming exercises on AMPK/ACC signaling pathway, the regulation mechanism of different intensity swimming movements on AMPK/ACC signaling pathway was explored, which provided a theoretical basis for studying the effects of high-intensity and low-intensity swimming exercises on lipid metabolism [9].

High-intensity exercise is more beneficial to improve the liver's utilization of glucose and oxidation of fat, thereby reducing total plasma cholesterol (TC), triglyceride (TG), and low-density lipoprotein (LDL) [10]. Prolonged exercise reduces levels of TG, TC, LDL, and HDL in the blood. The mechanism by which exercise improves lipid metabolism may be related to increased lipocytase activity, regulation of LDL receptor gene transcription and protein expression. During exercise, the secretion of adrenaline and norepinephrine increases lipoprotein lipase activity, accelerates the metabolism of TG and VLDL, and causes the remaining cholesterol, phospholipids and apolipoproteins to be transferred to the liver and bind to the newly established high density. Lipoprotein particles, thereby increasing the amount of high-density lipoprotein [11]. Changes in high-density lipoprotein, triglyceride, and cholesterol metabolism are closely related to the duration of exercise intensity. Studies have shown that lipid metabolism disorders and imbalances in hormone metabolism are closely related to body movements. With the development of modern society, more and more people suffer from various diseases because fat accumulates in their bodies. Therefore, research to actively promote liver fat metabolism is very important. The purpose of this study was to observe the effects of large and medium-intensity swimming on liver fat metabolism in rats, and to provide evidence and guidance for normal people to exercise weight loss [12].

In this study, 30 male 4-week-old SD rats were randomly divided into 4 groups after one week of adaptive feeding: silent group (Q), long-term training group (LT), one-time fatigue group (OE), long-term training. Combined with a one-time fatigue group (LO group), 10 per group. The LT group was fasted after 24 hours of training. The OE and LO groups collected samples immediately after the last exercise. The levels of hdl-c and ldl-c in serum and liver of rats were determined by COD-PAP method. The levels of hdl-c and ldl-c in serum and liver of rats were determined by copper method. The levels of FFA in serum and liver were determined by copper colorimetry. Western blotting was used to detect the phosphorylation levels of AMPK a and ACC in the liver of four groups of rats.

2. Proposed Method

2.1. Overview of Lipid Metabolism

(1) Lipids

Lipids can be divided into two categories: one is fat, mainly triglyceride, which is the main source of energy; the second is lipid, which is part of the biofilm, including phospholipids, glycolipids, and cholesterol. Triglycerides (TG) are the most abundant lipids in the human body. In mitochondria, TG can be rapidly decomposed to provide energy. TG can be synthesized from adipose tissue such as the liver. TG is mainly stored in adipose tissue. Serum TG is mainly present in the nucleus of chylomicrons (CM) and very low density lipoprotein (VLDL). Triglycerides can be catalyzed by enzymes to produce glycerol and fatty acids and provide energy. Cholesterol (CHOL), also known as 5-choline-3-betaine, is a class of small sterols with a cyclopentane polyhydroxy structure that breaks down large fat granules into smaller granules, making them easier to absorb. CHOL is an important component of biofilm structure and plays a key role in maintaining cell substructure stability and biofilm mobility. It is also the only precursor to the synthesis of sterols such as bile acids and vitamin D, and is involved in many important signal transduction pathways. CHOL can also form a lipid component in the blood together with TG. Current research has found that excessive CHOL is an important cause of hyperlipidemia and a range of metabolic diseases. High density lipoprotein (HDL) is synthesized in the liver and small intestine. It carries CHOL from the surrounding tissue, converts it to bile acids or removes it directly from the intestines through bile. Hdl-c is upregulated to reduce atherosclerosis and prevent coronary
heart disease. Low-density lipoprotein (LDL) is mainly converted from the metabolism of VLDL, which is synthesized by the liver and secreted into the blood. Its role is to transport cholesterol from the liver to tissues throughout the body. When cholesterol levels are too high, cholesterol can easily accumulate on the arterial wall and cause damage to blood vessels.

(2) Free fatty acids

In long-term training, skeletal muscle energy supply is mainly dependent on the hydrolysis of TG in skeletal muscle and the supplemental consumption of FFA in serum. When the main energy required for muscle activity is consumed by hepatic glycogen, the adipose tissue breaks down the neutral fat into free fatty acids (FFA) for energy use. As the exercise time prolongs, the energy supply ratio of serum FFA increases. Changes in serum FFA can reflect the interaction of glucose metabolism and lipid metabolism. It has been reported that when people are overweight or obese, their fasting FFA levels are significantly higher than those of normal weight regardless of the presence or absence of abnormal glucose metabolism. Many scholars believe that FFA is associated with obesity, insulin resistance and hyperinsulinemia. Some scholars have established a high-fat diet without insulin resistance in obese rat models, and found that serum FFA levels are significantly elevated, suggesting that FFA is an important link between obesity and insulin resistance. It is speculated that the concentration of FFA in serum and liver can be used as a measure of lipid metabolism.

2.2. AMPK/ACC Signal Path

With the deepening of research, the research on the regulation mechanism of cell and molecular signals has gradually increased, the research methods are diverse, and the research content is rich. The theory of cellular and molecular signal transduction mechanisms has been improved. Researchers are beginning to rethink and position the role of mitochondria in cellular energy regulation. Studying the effects of exercise on cellular energy regulation and the adaptation of cells to exercise is a new perspective. The body has many in vivo signaling molecules that are thought to be very sensitive to changes in cellular energy. Regulators or regulatory agencies: AMPK, mTOR pgc-1α and other motor cells have major energy control signals and their entire molecularly mediated signal is stably expressed. The stability and balance of the cellular network of cellular energy play an irreplaceable role. Scholars hope to find more key signaling molecules related to energy metabolism through in-depth exploration. At present, scholars have deeply analyzed the key signaling molecules of lipid metabolism and energy regulation, as well as the signal coupling between the energy metabolism-related pathways and the mitochondrial regulatory network. The ultimate goal is to explain in depth the regulatory mechanisms between exercise adaptation and energy metabolism.

(1) How AMPK is activated

AMPK acts as a protein kinase in the body, catalyzing the phosphorylation of proteins. In mammals, AMPK is a protein complex that binds to alpha, beta and gamma subunits. The AMPK subunit is distributed in various tissues and organs. α-1 is mainly distributed in mammalian tissues such as kidney, liver, heart muscle and brain. α-2 is widely distributed in skeletal muscle, heart and liver. AMPK is activated by a variety of pathways, including the AMP/ATP pathway, the LKB1 pathway, the CaMKK pathway, the transforming growth factor (TAK1) pathway, and other physiological conditions such as starvation, hypoxia, ischemia, and electrical stimulation.

(2) Regulation of AMPK/ACC

The AMPK/ACC signaling pathway regulates the synthesis and breakdown of fat. This pathway is activated by the upstream of AMPK. The alpha catalytic subunit of AMPK contains a kinase domain (KD) at the n-terminus, also known as the catalytic domain, which contains an activation loop. The alpha portion also includes an alpha-hook structure and three important domains of the self-inhibiting sequence (AID) of AMPK alpha. The Ser/Thr kinase region and the threonine (sr-172) site are conserved at the n-terminus of the alpha subunit. Phosphorylation of the threonine site induces activation of the AMPK enzyme, and recent studies have found that phosphorylation at this site is associated with white fat metabolism. The AMPK/ACC signaling pathway is involved in the regulation of lipid metabolism and is involved in the regulation of metabolic diseases such as hepatic steatosis and cardiac hypertrophy. Studies have shown that AMPK phosphorylates the ser79 and ser212 sites of ACC1 and ACC2, thereby inhibiting ACC activity, thereby regulating fatty acid synthesis and the rate of fatty acid entry into mitochondria. A large number of experiments on AMPK signaling have shown that AMPK can prevent and reverse some metabolic diseases. By observing the exogenous intake of baikalin in high-quality mice, some scholars have found that baikalin can significantly inhibit the phosphorylation of AMPK and ACC in the liver, control body weight and reduce the weight ratio of epididymal fat pad, thereby achieving treatment and control of fatty liver influences. In addition, metformin is injected into the lateral ventricle of rats to inhibit AMPK signaling pathway, reduce the phosphorylation level of ACC, reduce appetite, reduce food intake, and thus treat metabolic diseases. The mechanism for regulating the AMPK/ACC signaling pathway is also under investigation.
2.3. Motion and AMPK/ACC Signal Pathway Relationship

(1) Relationship between exercise intensity and AMPK/ACC signaling pathway

Studies have shown that when the body is in low-intensity exercise, the AMPK/ACC signal pathway is not activated. When the body is moving at medium and high intensity, the AMPK activity increases with the increase of exercise intensity. It was also found that as exercise intensity increased, the AMPK/ACC signaling pathway was activated, and AMPK activity was increased, which in turn activated lipoprotein lipase (LPL) and accelerated lipid hydrolysis. It has been reported that after 12 weeks of aerobic exercise intervention, AMPK phosphorylation is increased in APOE rats, fatty acid synthase (FAS) is inhibited, fat synthesis is reduced, and lipid metabolism is improved. Some scholars have found that 6-week endurance exercise can increase the skeletal muscle glycogen uptake ability of rats, and to some extent reverse the impaired AMPK phosphorylation caused by diabetes and improve lipid metabolism. It has also been suggested that high-intensity exercise can promote the AMPK/ACC signaling pathway. Hoffman N et al performed a muscle biopsy of AMPK phosphorylation in four healthy adults before and after high-intensity exercise. Thereafter, a mitochondrial protein akip1 binds as a substrate to a camp-dependent protein kinase PKA (protein kinase A) targeting mitochondria and a kinase such as PKC.

(2) Relationship between exercise time and AMPK/ACC signaling pathway

Exercise regulates the AMPK/ACC signaling pathway, which is involved in the synthesis and transport of fatty acids. Some scholars also designed a combination of human and animal experiments, subjects were divided into voluntary physical activity (VPA) group and endurance training (ET), after 9 weeks of swimming training, weight loss results, etc., visceral fat and fat cell morphology Changes, serum FFA and glycerol levels were significantly reduced, increased fat / CD36, decreased ACC protein expression, and reduced fat synthesis. Some scholars also conducted a 10-week training of rats of different ages to explore the regulation of exercise training in lipid metabolism in aged rats, suggesting that skeletal muscle activity is enhanced by the ACC elderly exercise group, ie exercise training may increase older rats The ability to synthesize fat.

In addition, the AMPK/ACC pathway was activated, and insulin resistance (IR) was significantly lower in the high-fat diet rats after 6 weeks of exercise, and the phosphorylation levels of AMPKα and ACC were significantly increased in the liver, indicating long-term training, plus one-time force Exhaustive exercise can activate the AMPK / ACC signaling pathway and improve lipid metabolism. At the same time, exercise can also up-regulate ACC activity, increase fat synthesis in aged rats, and ensure lipid metabolism balance. As early as 1990, some scholars conducted a random survey of some obese families, and developed a corresponding intervention program, which is to increase walking and skeletal muscle endurance training at home every day, increase the intensity of daily physical activity, and promote the physiological and pathological rehabilitation of patients.

(3) Effect of high-intensity interval training on AMPK/ACC

High-intensity interval training (HIIT) is a group of high-intensity exercises. The total duration of exercise repetition is less than 45s 2min~4min, and the recovery time is less than 10s repeated sprint or 20~30s intermittent sprint. HIIT can cause excessive oxygen consumption after exercise and accelerate metabolism. CasusoRA et al. made a plan to do 50-meter and 200-meter freestyles ten times a day for two weeks, and found that AMPK phosphorylation levels in the triceps were increased after exercise, and ACC activity was inhibited, and recovered after 2 hours, Among them, high-intensity exercise had the greatest inhibition of ACC in the 1990s. Some scholars also found through muscle biopsy that after taking HIIT for 7 days, the ASC phosphorylation level of the lateral femoral muscle increased by 1-3 times, the number of mitochondria and a kinase such as PKC.

3. Experiments

3.1. Experimental Data Acquisition

Rats in different groups were given the feeding conditions, and they were fed to the feeding environment separately. The rats in each group were fed with high-fat words, and the daily rest and drinking water were freely carried out. Daily feed and water are updated in a timely manner. The daily scheduled study of the rats was carried out while the experimental materials of the rats were collected in time. Feed composition: Ordinary feed: experimental mice grow to maintain granules high-fat diet: This article uses Sun Zhi reported high-fat feed...
slightly improved, its formula: ordinary feed, lard, deer sugar, milk powder, peanuts, eggs, sesame oil, salt. The final data collected will be analyzed by the statistical processing software SPSS22.0 analysis software for actual experimental analysis and comparison, wherein each experimental data is expressed as (mean ± standard deviation); the actual effect between each group of rats One-way analysis of variance was used in the correlation analysis. The difference between the previous data of each group was P<0.05, and the value of P value represented statistical significance to a certain extent. A P < 0.01 was considered to be very statistically significant.

3.2. Experimental Environment

In this study, healthy adult male SD rats weighing about 200 g were selected as experimental subjects. Thirty rats were randomly divided into three groups: group C (quiet control group), group M (medium intensity training group), and group O (overload group). Rats were raised in animal houses and kept in cages, 4 to 5 per cage, free to enter the water (national standard animal feed), relative humidity of about 40% to 60%, room temperature 20 °C ~ 26 °C, good ventilation.

(1) Group C (quiet control group): routine feeding, no intervention.

(2) Group M (medium intensity training group): The rats need to be trained for 6 days per week, and it is necessary to do the daily training time and the swimming in the water for at least five minutes, and then increase the swimming time by 5 minutes every day. At the end of each weekend, the rats need to be intensively trained to do at least 30 minutes of training. In the second week, you also need to increase your swimming time to one hour, and maintain this amount of exercise until the end of swimming for five weeks.

(3) O group (experimental load group): The rats need to be trained for 6 days per week, and it is necessary to do the daily training time and the swimming in the water for at least five minutes, and then increase the swimming time by 5 minutes per day. At the end of each weekend, the rats need to be intensively trained to do at least 30 minutes of training. In the third week, the daily high-intensity swimming time needs to be increased to ten minutes, and the third week of swimming time needs to be increased to two hours. After that, follow this training session until the fourth week to start weight-bearing swimming training. The negative weight at week 4 was 1% of the rat's own weight, the negative weight at week 5 was 3% of the rat's own weight, and the negative weight at week 6 was 5% of the rat's own weight.

3.3. Experimental Steps

After the rats were sacrificed by cervical dislocation, the liver tissues were dissected out. Liver tissue total cholesterol (TC), triglyceride (TG), high density lipoprotein (HDL) and low density lipoprotein (LDL) were determined by enzymatic methods. Detection of total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL) and low-density lipoprotein (LDL) levels in liver tissue using an automated biochemical analyzer to quantify liver tissue homogenate and extract lipids After tissue fluid, centrifugation, and centrifugation tests, the results showed levels of liver TC, TG, high-density lipoprotein, and low-density lipoprotein. Total cholesterol (TC) content was determined by COD-PAP method, 96-well plate method and colorimetric method. Triglyceride (TG) content was determined by apo-pap enzyme labeling, 96-well plate manipulation, and microplate chromogenic assay. Direct determination of high-density lipoprotein (hdl-c) content, 96-well plate manipulation, ELISA-based colorimetry. Determination of low density lipoprotein (ldl-c) by direct method, 96-well plate operation and colorimetry.

4. Discussion

4.1. Changes in Liver Mass in Rat Liver Tissue after Exercise Intervention

(1) As shown in Table 1, the levels of TC and TG in the liver tissues of group M and group O were lower than those of group C after exercise intervention. The liver TC of group M was significantly lower than that of group C, and the difference was statistically significant (P<0.05). The liver tissue TC of group O was higher than that of group M, which was lower than group M, but there was no significant difference. The TG of the liver tissue of group O was lower than that of group M and group C, but there was no significant difference. The TG of liver tissue in group M was lower than that in group C, and there was no significant difference, as shown in Figure 1.

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<th>Weekly times</th>
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(2) It can be seen from Table 2 that the HDL content of the M group is higher than that of the C group and the O group, and the difference is extremely significant (P<0.01). The HDL of group O was lower than that of group C and group M, and the difference was not statistically significant. The LDL content in group M was lower than that in group C and O, and the difference was statistically significant (P<0.05). O group LDL was lower than group C, but the difference was not statistically significant, as shown in Figure 2.

<table>
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<tr>
<th>Weekly times</th>
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<tr>
<td>Swimming load</td>
<td>1% of one's own weight</td>
<td>3% of one's own weight</td>
<td>5% of one's own weight</td>
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4.2. Changes in Lipid Metabolism Related Proteins

(1) The effect of exercise on the level of AMPK alpha phosphorylation in rat liver is shown in Figure 3. There was no significant difference in liver AMPK alpha phosphorylation between the LT group and the Q group (P>0.05). Compared with the Q group, the AMPK a phosphorylation level in the OE group was significantly lower (P<0.05). Compared with the Q group, the phosphorylation level of AMPK a in the LO group was significantly increased (P<0.05). Compared with the LT group, the AMPK a phosphorylation level in the OE group was significantly lower (P<0.05). Compared with the LT group, the phosphorylation level of AMPKα in the LO group was significantly increased (P<0.05). Compared with the OE group, the phosphorylation level of AMPKα in the LO group was significantly increased (P<0.05).
(2) The effect of exercise on the expression of ACC phosphorylation in rat liver is shown in Figure 4. Compared with the Q group, the liver ACC phosphorylation levels were significantly increased in the LT group, the OE group, and the LO group (P<0.05). Compared with the LT group, the ACC phosphorylation level in the OE group was significantly lower (P<0.05). Compared with the LT group, the liver ACC phosphorylation level was significantly increased in the LO group (P<0.05). Compared with the OE group, the phosphorylation level of ACC in the liver of the LO group was significantly increased (P<0.05).

5. Conclusions

Cholesterol (TC) and triglyceride (TG) act as two types of fat and play an important role in the human body. Cholesterol stores energy, which is the main component of biofilms and participates in signal transduction between cells. Low density lipoprotein (LDL) is the main carrier of cholesterol. Low-density lipoprotein cholesterol (ldl-c) is an independent risk factor for atherosclerosis. It can be seen from Table 2 that after medium and high intensity swimming training, the contents of TC and TG in rat liver tissue are significantly reduced, which fully demonstrates that medium and high intensity swimming training has a good effect on reducing TC and TG contents in rat liver tissue. As can be seen from Figure 4, in this experiment, the increase of HDL in the liver tissue of rats with moderate-intensity aerobic training was more obvious, while the overload training was slightly lower than that of the quiet control group. This fully demonstrates that moderate-intensity swimming can effectively increase the level of high-density lipoprotein in rats. After moderate-intensity swimming training, the low-density lipoprotein levels in rat liver tissue decreased significantly, and over-load training, although the level of low-density lipoprotein was also reduced, but the scale of reduction was small, not statistically
significant. It is indicated that the moderate-intensity swimming exercise in the last five weeks can effectively reduce the content of low-density lipoprotein.

Medium-intensity aerobic exercise, TC content decreased significantly. TG content decreased slightly, high-density lipoprotein (HDL) increased significantly, and low-density lipoprotein (LDL) decreased significantly. Excessive load and aerobic exercise lead to abnormal fluctuations in the fat metabolism index in the liver, so over-loading is not desirable and is not worth promoting. In this experiment, high-load swimming training was not as effective in reducing liver tissue cholesterol (TC) as in moderate-intensity swimming training. However, it is more effective in reducing the triglyceride (TG) content in liver tissue. Medium and high load swimming training can reduce the content of triglyceride in rat liver tissue, but the reduction effect is not obvious, and there is no statistically significant. This may be related to other factors such as the subject used in the protocol, exercise patterns, exercise intensity and duration.

Long-term high-intensity exercise is not conducive to regulating myocardial cell membrane cholesterol balance and maintaining cell membrane stability. It is not a good method for people to exercise. Therefore, it is recommended to perform medium and small intensity aerobic exercise in daily exercise. For professional athletes, high-intensity exercise training should be combined with moderate-intensity exercise training. Long-term high-intensity exercise can lead to disorder of myocardial cell membrane cholesterol metabolism, affect the stability of myocardial cell membrane, and even destroy the function of cardiomycocytes. Especially in the stage of growth and development of young athletes, it is an important means to promote the physical development of adolescents and ensure the sustained and stable development of important organs such as the heart. At present, the academic community is still exploring the cholesterol metabolism pathway of cell membrane, and the reverse transport process of cholesterol is still in the stage of further exploration and exploration. The factors affecting apolipoproteins are unclear, and the effects of gene expression, inheritance and environment on apolipoproteins are unclear. Therefore, there is still a lot of gap to be filled in the field of cell membrane cholesterol balance. This experiment is expected to provide a valuable reference for further research in this field.

References


