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Exercise Training on Cardiac Cell Proliferation Induced by HGF and TGF-B1 Expression in Rats

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Abstract: The highest incidence of cardiovascular disease is myocardial infarction and myocardial hypertrophy, these two diseases threaten the lives of patients at any time. The purpose of this article is to solve the problem of how exercise training induces the expression of HGF and TGF-β1 in the myocardium and to explore the effect of different HGF and TGF-β1 expression on the proliferation of myocardial cells. This paper designed a research experiment on rats. The rats were divided into groups and exercise training was carried out. After the training, the expressions of HGF and TGF-β1 in the rat myocardium were analyzed by immunohistochemistry and immunofluorescence detection and the proliferation of rat myocardial cells was studied. The results show that aerobic exercise can reduce the heart rate of rats by 17%, increase the occurrence rate of binuclear cells by 21%, increase the expression of myocardial HGF and TGF-β1 by 25%, and increase the expression of HGF and TGF-β1 in myocardial tissue can promote myocardium. The occurrence of cell proliferation and the upregulation of cardiomyocyte proliferation can effectively improve heart function, proving that exercise training has a good effect on the treatment and prevention of cardiovascular diseases.

1. Introduction

Experts from the World Health Organization predict that human cardiovascular disease will not become the disease with the highest human mortality rate in the world. The main types of cardiovascular diseases include acute coronary heart disease, hypertension, stroke, and peripheral arterial vascular disease, etc., which have major characteristics such as a relatively high mortality rate. According to statistics: at least 1.8 million Chinese people die of acute cardiovascular disease every year in the world, and it is generally recognized as the world 's number one killer.

The huge impact of exercise on the normal proliferation of myocardial cells has been highly concerned by people in the society. Decreased exercise capacity training can directly lead to acute

myocardial hypertrophy and acute cardiac vasoconstriction exercise ability is significantly strengthened, blood pumping and cardiac function increased significantly [1]. For a long time, it has been generally believed that cardiomyocytes are only a terminal and differentiated cell. They will withdraw from the cell cycle shortly after birth and lose their normal proliferative development ability [2]. Some medical researchers have found that rats performing aerobic swimming or walking and running within 4 weeks after the operation of the left myocardial infarction can significantly reduce the expansion of the left ventricular wall, and the left ventricular wall in the non-infarct surgical area can be slightly adapted Sexual hypertrophy, the area of the infarct wall is significantly reduced. Exercise signals can help to upregulate the expression of myocardial leukocyte HGF and TGF-β1, and have an important role in promoting the normal regeneration of cardiomyocytes, proliferation and development, but its specific operating mechanism and exercise signal transmission pathways are not yet clear [3].

In this paper, in order to study the effect of exercise training on myocardial cell proliferation induced by the expression of HGF and TGF-β1 in rat myocardium, we have read a lot of relevant data. Among them, Li made a detailed introduction to the pathogenesis of cardiovascular diseases, analyzed the current problems in the medical system for treating cardiovascular diseases, and elaborated the medical research methods and technologies for cardiovascular diseases [4]. Riley pointed out in his article that in the case of today's medical technology is very limited, sports training is very important for the treatment of cardiovascular disease, and pointed out that sports training is a treatment method without side effects, and also shows that the heart For vascular patients, adherence to drug treatment is also important [5]. In the article, Zhai elaborated on the specific process of exercise training to induce the expression of HGF and TGF-β1 in myocardium, and introduced the relevant principles, and introduced the reasonable application method [6]. Yu proposed that the expression of myocardial HGF and TGF-β1 will have a certain effect on the proliferation of myocardial cells. The specific effect is determined by the expression of myocardial HGF and TGF-β1 [7]. SONG proposed that myocardial cell proliferation ability is an important indicator to measure cardiovascular health, and pointed out that very cardiovascular and cerebrovascular diseases are caused by the inhibition of cell proliferation activity, and the improvement of cell proliferation ability has very important physiological significance [8].

In the study of the effect of exercise training on the expression of HGF and TGF-β1 in rat myocardium on the proliferation of cardiomyocytes, this paper summarizes and analyzes the research experience and achievements of a large number of predecessors. Specific innovations include the following: First, this article uses immunohistochemistry and fluorescence detection for the first time to explore the effect of exercise training on the expression of cell growth factors HGF and TGF-β1, and improves the accuracy of the test results. Second, this article is the first to use the electric shock method to exercise the rats. This method improves the exercise ability of the rats and thus improves the experimental effect. Third, this article is the first to explore the effect of exercise training on myocardial cell proliferation induced by exercise training induced myocardial HGF and TGF-β1 by establishing a rat MI model. This is a more three-dimensional research process.

2. Related Principles and Functions

2.1. The role and Principle of Cardiomyocyte Proliferation

Regarding the proliferation of cardiomyocytes on the matrix, there have been different theoretical views that cardiomyocytes are no longer just cardiomyocytes that have been differentiated from terminal cardiomyocytes, and are no longer equally important for cardiomyocyte

stromal cell proliferation and cardiomyocyte regeneration. But under the normal working condition of myocardial cell tissue, there will be no abnormal death and apoptosis of myocardial cells, but the number of myocardial cell death will not gradually decrease greatly[9]. Even in the whole myocardial cell tissue under ischemic or hypoxic circulation, although rapid necrosis of myocardial cells and accelerated injury and apoptosis will not occur, the number of myocardial cell apoptosis will not be reduced and increased to a direct effect. The normal integrity of the entire myocardial cell tissue suggests that the myocardial cells may be fully capable of rapid proliferation and rupture and differentiation[10]. However, the number of proliferating cells in this disease is far from being able to effectively compensate for the necrosis of myocardial cells caused by ischemia or hypoxia.The proliferation and hypertrophy of cardiomyocytes have positive significance, which can strengthen the contraction of myocardium and effectively compensate for cardiac function. The amount is far from making up for the amount of myocardial cell damage caused by ischemia and hypoxia. The proliferation and hypertrophy of cardiomyocytes have positive significance, which can strengthen the contraction of myocardium and effectively compensate for cardiac function. Some neurotransmitters can promote cell proliferation. Among them, the research on serotonin (5-HT) is more common. 5-HT promotes smooth muscle cell hypertrophy and fibroblast proliferation by serotonin receptor (5-HT2R), and there are many 5-HT receptors in mammalian heart. It is suggested that in the hypertrophic myocardium caused by pathology, it may play a key role in regulating the pathogenesis of myocardial hypertrophy by promoting the proliferation of myocardial cells.

Cardiomyocytes originate from mesoderm progenitor cells. Mammalian embryonic cardiomyocytes are mononuclear and have the ability to proliferate. However, most cardiomyocytes replicate after birth but do not accompany cytokinesis and become binuclear and diploid. Most cardiomyocytes in the adult human heart are mononuclear, polyploid, and few cardiomyocytes enter the cell cycle. Studies have found that the heart of lower vertebrates has a strong regenerative capacity, even if the heart muscle tissue is missing 20%, it can be completely regenerated. Under normal physiological conditions, some myocardial cells may have chronic death, apoptosis and autophagy, but the number of myocardial cell death will not gradually decrease. Even under normal ischemic or hypoxic survival, the liver cardiomyocytes will not undergo rapid necrosis of the cardiomyocytes and accelerate the rate of apoptosis, but the number of proliferation of cardiomyocytes will not be reduced and cannot affect the liver myocardium. The normal integrity of the cell tissue has even disappeared completely, suggesting that cardiomyocytes still have a certain cell proliferation activity, which is a perspective to maintain a dynamic balance of the number of cardiomyocytes and stabilize liver cardiovascular function, that is, it can be clearly speculated that the performance Cardiomyocytes must also have this proliferative phenomenon.

2.2. Biological Functions of Cell Growth Factor (HGF) and Transforming Growth Factor (TGF-β1)

HGF is an interstitial derived multifunctional immune cell-derived factor with a gene of about 70 kb, composed of a heavy chain (a-chain) with a relative molecular mass of about 69 KD and a light chain of 34 KD such as ß-chain through disulfide bonds. The combined isopropyl dimer is a multifunctional indirect substance-derived immune cell-derived factor widely present in plasma cells in mammals with acute hepatitis B injury and can directly stimulate DNA in acute liver injury cells[11]. Enzyme synthesis can also play an important role in inhibiting the regeneration of liver cell enzymes. HGF plays an important regulatory role in the normal growth and differentiation of

many human tissues and plant cells[12]. HGF and its molecular receptor binding c-met are widely expressed in nerve tissues. Cardiovascular endothelium and erythrocytes of cerebral blood vessels including the cardiovascular nervous system have no physiological stimulation and immunomodulatory effects. HGF is widely used to study how the body regulates the normal growth of myocardial cells, the occurrence of tissue and cell morphology, and complex human biological and physiological processes such as local movement. The role of HGF in inhibiting the promotion of normal growth and development of vascular endothelial-forming cells is mainly reflected in that it can significantly inhibit the expression and cellular transcriptional activity of increased ets-1 cells minas, and stimulate endothelial growth factor (VEGF), c- Met and metalloproteinases (mmp-1) in the vascular matrix are expressed between endothelial-forming cells and cells vamps that enter the blood vessel and thus can play an important induction role in the development of vascular endothelial-forming cells in neonates. One study showed that after the HGF gene was transferred to the glomerular vasodilator via dilatation, its location at the endometrial injury site could significantly inhibit the formation of neovascular endometrioma, and it could transfer normal vascular endothelial tissue the cell regeneration function restores normal endothelial cell function. A large number of experiments have proved that HGF can promote the proliferation of cardiomyocytes.

TGIF is widely present in normal transformed tissue cells of human animals and transformed tissue cells of plants, and is a type of growth factor that has multiple cellular biological transformation effects. Transforming growth factor superfamily proteins are a group of versatile cell signal transduction factor proteins, which are highly conserved in the structure and conduction function of cells during the process of cell evolution and development, benign proliferation and redifferentiation in many types of tissue cells, and tissue function damage Cell repair, formation of interstitial cells, regeneration of bone structure, regulation of immune function, and its role in the process of embryonic growth play an important key role in conduction. It has been found that there are three types of cells in mammals in vivo, including TGF-β1, TGF-β2 and TGF-β3. The extracellular ligand binds to the corresponding receptors on the cell membrane, namely type I and type II Ser / Thru kinase receptors, to activate the downstream effector Shads protein, which forms a multimeric complex and enters the nucleus through the nuclear membrane transport mechanism. The regulated target gene binds and regulates the transcriptional activity of the gene. TGF-β1 polysaccharides are expressed in the central cardiovascular nervous system. Cardiovascular cells include smooth muscle cortical cells, endothelial cortical cells, macrophage cells and vascular hematopoietic cells. TGF-β2 is expressed in human epithelial stromal cells and nerve cells, and the main expression in mesenchymal epithelial cells is TGF-β3. TGF-β1 directly stimulates the myocardial cell membrane and secretes fibronectin globulin through the ttsmad3 signal reflection pathway, which increases the extracellular matrix of neointimal and promotes neointimal hyperplasia, which leads to ISR. In addition, TGF-β1 can also stimulate endothelial cells to initiate the process of epithelial-mesenchymal transition (MT) to deform, fibrosis and even transform into endothelial cells, which is not conducive to the proliferation of endothelial cells and hinder the reendothelialization process. Studies have shown that TGF-β1 secretion in cardiomyocytes has a significant effect on improving myocardial function. Therefore, inducing increased expression of TGF-β1 in the early stage of the disease is an effective means to prevent the occurrence of myocardial disease, and is a hot spot in the field of cardiovascular disease research.

3. Experimental Content and Data Processing

3.1. Object Selection and Motion Design

In this experiment, 40 rats with a six-month-old body weight close to each other are selected as the research object, and the body weight is (about 250g). The laboratory that has been disinfected and has no pollution is selected as the experimental site. The laboratory temperature is 20-30 ℃.The mid-water content is about 14.3-15.8%, the exposure time of Yangquan is from 8: 00-17: 00, the lighting rate is good, the laboratory is kept ventilated to ensure that the oxygen content in the air is sufficient, and it is divided according to the national standard sentinel animal feeding specifications Cage feeding, each cage serves 10, free to eat and drink water. Coronary artery ligation was performed on rats in two days. The left anterior descending branch of the heart was used for ligation, which was divided into two types: complete ligation and open thoracotomy without ligation. After 1 week of proper training, the rats with exercise problems were excluded, and 20 rats were randomly divided into two groups according to body weight and exercise ability, one group was a quiet control group, and the other group was an exercise group. Ten rats in the non-exercise control group were divided into three groups with 10 rats in each group.

Training method of the exercise group: the quiet control group does not exercise, and normally moves in the cage. The exercise group is divided into three experimental groups: small-intensity group, medium-intensity group, and high-intensity group. Routine feeding exercise small intensity group, slope 5 °, speed 15m / min, the experiment is equivalent to the maximum oxygen consumption 53%, medium intensity, slope 10 °, speed 18m / min, equivalent to the maximum oxygen consumption 63%. In the intensity group, the slope is 15 °and the speed is 20m / min, which is equivalent to 68% of the maximum oxygen consumption. On the first week, the rats were pre-experimented on the animal treadmill with different intensities, at the same time and in the same exercise mode to adapt each intensity group to the training intensity. The specific arrangement of the 7-week formal training test is that the quiet group is routinely fed and does not exercise. The large, medium and small intensity groups are trained for two days, one day of rest, three days of training, and one day of rest. The specific time is training every Tuesday and Wednesday, closed on Thursday, Friday, Saturday, Sunday training and Monday rest. Each training is from 9:00 am to 13:00 pm in the high-intensity group, continuous training for 60 minutes, from 8:00 am to 15:00 in the mid-intensity group, continuous training for 70 minutes, from 7:00 am to 17:00 pm. Train the small-intensity group and continue training for 20 minutes. When the rat stopped exercising, it was forced to move with an electric shock of less than 5mV. During the training, feed each morning, and clean the cages in time to ensure the test environment is clean and hygienic. Weigh yourself once a week and record the relevant data to prepare for the experimental analysis.

3.2. Research Equipment

The main equipment used in this experimental research institute: ACL-V8 animal ventilator, POWER-8 model physiological signal acquisition instrument, LEICB-218 slicer, BX230 electron optical microscope, 725B spectrophotometer (Jiangsu Zhongdu Analytical Instrument Co., Ltd.), 786S Type UV spectrophotometer (Jiangsu Zhongdu Analytical Instrument Co., Ltd.), fluorescent display, PSCX imager, BCL-210A Rongcheng water tank (Jiangsu Keitai Electric Co., Ltd.), slicer (made for German company), electronic balance Produced by Stayer, the room temperature centrifuge is produced by Changsha Xingu Centrifuge Instrument Co., Ltd., and the animal treadmill (made in Tianjin). Other auxiliary equipment and reagents are shown in Table 1.

Table 1. Other auxiliary equipment and reagents

Group	Usage amount	Source
Biological baking machine	1	German Xircom
Embedding machine	1	Sony Group of Japan
Absolute ethanol	200ml	Shanghai Analytical Instruments
N-butanol	500ml	Caused by Jiangsu Feng Hua
Hydrochloric acid	150ml	Gaohu Chemical Enterprise
Sulfuric acid	300mg	Japan Sanwa Kimono
Sodium chloride	750ml	American SGH
Electrophoresis tank	2	Bosch, Germany

The main reagents used in the experiment are as follows: SACD kit, DBB chromogenic kit, rat monoclonal antibody, trichloroacetic acid (200ml), cresol soap solution (1200ml), sodium hydrogen phosphate (400ml), anhydrous Potassium dihydrogen phosphate (AR), n-butanol (600ml laboratory stock), organic ethanol 100ml, ammonium persulfate 200ml, SDS solvent, NC membrane (purchased from Comfort Engadin Energy Co., Ltd.), pre-stained protein 20mg (purchased From NET company), protease inhibitor (laboratory stock), protein phosphatase inhibitor 80ml, paraformaldehyde 20ml, ammonium bicarbonate 40ml, PMSG (purchased from Yunnan Anuran Chemical Co., Ltd.), RIPA lysate 70ml, PLG Bio Tissue solvent (Shanghai Chemical Reagent Factory).

3.3. Detection and Data Processing Methods

Immunohistochemical method for drug detection: This immunohistochemical method uses a method of distinguishing sac, which is strictly implemented according to the specific operation steps given in the product manual of the detection kit (Wuhan Bos hide). Antigen microwave repair: HGF, p53 and p5TGF-β1 microwave antigen repair after washing with pbs; block with 3% h2o2 for 10min, pbs to wash. Add one normal plus amount of rat serum for 20 min; drop two plus one plus three antibodies: HGF, p53 and β1TGF-β1 antibody drop concentration is 1:20, rinse with pbs; drop one plus two plus three antibodies 32 ℃ 20min, pbs for rinsing; drop in second plus third antibody sac for 20min, tween + pbs for 2h for rinsing; dab has no color, rinse with distilled water; hematoxylin counterstaining, differentiation, dehydration, transparent, neutral sulfuric Gum and sealing sheet. Each time the control staining procedure is set as a negative double antibody control, past replaces the primary antibody, and the other staining procedures are the same. Observe with 500 times optical microscope, and use microscopic image analysis system for image analysis.

Cardiology and hemodynamic function indicators are accurately determined: after the training session, rats can be anesthetized with 100% urethane intraperitoneal solution, the electrodes of the limb electrocardiogram detection needles are directly inserted into the subcutaneous limbs of the rats, and advanced limb leads are used. The technology is used to detect the ECG indexes of the limbs. Cut the surface skin of the neck of the whole rat body, separate the right or left common carotid artery, and inject 3 ‰ rat heparin for anticoagulation (0.3ml / 100g), using a polyethylene antihypertensive catheter containing (Filled with active physiological silicate water). Retrograde drainage cannula through the end of the right common carotid artery of the rat and reduce blood pressure to record the blood pressure at the end of the common carotid artery, and then send the end of the reduced pressure cannula to the left ventricle, and the other end through the heart The

pressure motion sensor is used to reduce the pressure and input it to the -rm-6240 physiological polyduct blood pressure recorder. After the left ventricular vein curve reflection wavelength value is stable, the rat hemodynamic detection indicators can be measured. Left ventricular venous systolic pressure (lisp), left ventricle End-diastolic blood pressure (lived), maximum rate of increase and decrease of left ventricular end-extension pressure (+ dip / tax) and maximum rate of decrease of left ventricular end-extension pressure (-dip / tax), etc.

After verification of the research data in this experiment, a metrology database is established, and the data of the metrology data database is expressed by the angle x ±s. Bidet format test should be used to verify the normality of multiple sets of data results. There is no mean comparison between multiple groups using analysis of variance. There is no mean display between multiple groups. Pairwise comparisons are made using p and q tests respectively. The microscope is connected with the computer information library, and the data results are automatically obtained and saved, and then analyzed and discussed.

4. Effects of Exercise Training on Rats

4.1. Effects of Exercise Training on the Expression of HGF and TGF-β1 in Rat Myocardium

Under the light microscope, it can be seen that Ki-67, PCNA, HGF and TGF-β1 are expressed in the myocardium of each group of rats. The expression of Ki-67, PCNA and HGF in cardiomyocytes was found in the nucleus, while TGF-β1 was mainly expressed in the myocardial cytoplasm. Compared with the quiet control group, aerobic exercise can significantly increase the MOD value of Ki-67, PCNA, HGF and TGF-β1 in rat myocardial tissue ($P < 0.05$), respectively increased by 18.8% and 22.2%, 10.8% and 13.0%. High-intensity exercise will reduce the MOD value of Ki-67, PCNA and HGF in rat myocardial tissue, of which HGF is significantly reduced ($P < 0.05$), respectively decreased by 6.3%, 5.6%, 10.8% and TGF-β1MOD increased by 34.8%. The results of exercise training on the expression of HGF and TGF-β1 in rat myocardium are shown in Table 2.

Table 2. Immunohistochemical results of rat myocardium after exercise training

Group	Ki-67	PCNA	HGF	TGF-β1
High intensity group	56±0.133	154±0.317	86±0.318	348±1.356
Medium intensity group	38±0.214	126±0.185	94±0.132	294±3.247
Small intensity group	44±0.127	138±0.248	87±0.196	266±2.465
Control group	71±0.228	161±0.529	92±0.258	312±3.688

In this study, TGF-β1 is marked in red, HGF is marked in green, and light yellow indicates the expression of HGF in the nucleus of cardiomyocytes. In normal myocardial tissue, light yellow or yellow-green cells are rare, occasionally expressing the expression of the myocardial cell nucleus, indicating that the number of Ki-67 positive cells is very small. Compared with the quiet control group, the cardiomyocytes in the aerobic exercise group were pale yellow or yellow. The obvious increase in green indicates that aerobic exercise can significantly increase the number of HGF-positive cells in rat myocardium. In the high-intensity exercise group, the rats had little yellow or yellow-green color, and occasionally the expression of the myocardial cell nucleus, and the number of HGF-positive cells did not change significantly. TGF-β1 is marked in green, and bright green is marked as PCNA. In normal myocardial tissue, occasionally bright green appears in the nucleus of cardiomyocytes, which indicates that the number of PCNA-positive cells in normal

myocardial tissue is very small, and the bright green of aerobic exercise rats increases significantly, mainly on the nucleus of myocardium Cells, indicating that aerobic exercise can significantly increase the number of myocardial PCNA-positive cells.

4.2. Effects of Exercise Training on Myocardial Tissue Structure in Rats

Research results show that aerobic exercise can have a benign effect on the shape and structure of the heart and enhance the function of the heart, while high-intensity exercise does the opposite. In this study, the results of HE staining, hemodynamics, and electrocardiogram tests showed that exercise training can arrange myocardial fibers closely and evenly stain, and the incidence of binuclear cells is higher than that of the quiet control group. The tax and T wave voltages were significantly increased, and both LVEDP and HR were significantly decreased, indicating that aerobic exercise has a benign effect on rat heart structure, adaptive hypertrophy of cardiomyocytes, slowed heart rhythm, increased cardiac output and obvious cardiac function improvement.Compared with aerobic exercise and quiet control group, high-intensity exercise will loosen the arrangement of myocardial fibers in rats. The cardiac coefficient, LVEDP and QT interval were significantly increased, LVSP and ± dip / tax significantly reduced the morphological structure of the rat heart, adversely affected heart rate, accelerated heart rate, prolonged myocardial contraction time, and enhanced cardiac function. Exercise training is very helpful to improve the heart function of rats, and the specific data that can increase the blood output of the heart is shown in Figure 1.

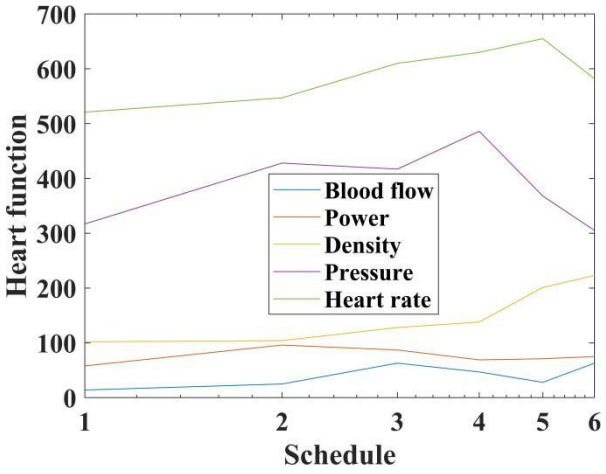

Figure 1. It is helpful to improve the heart function of rats, which can increase the blood output of the heart

From the data in Figure 1, it can be seen that exercise can improve the heart function of rats, how to increase the blood output of the rat heart by 24%, and improve the heart function of rats.

4.3. Test Results of Various Physiological Indexes of Rat Myocardium after Exercise Training

The results of the research analysis showed that compared with the rats in the quiet exercise control group, the average weight of the clinical rats in the aerobic exercise control group was significantly reduced ($p < 0.05$), the weight of the heart tissue was significantly increased ($p < 0.01$), and the cardiac stress coefficient was significant increased ($p < 0.05$); the average body weight of

clinical rats in the high-intensity aerobic exercise control group was significantly reduced (p <0.01), the weight of heart tissue was significantly increased (p <0.05), and the heart coefficient was significantly increased (p <0.05). In summary, aerobic training and high-intensity exercise can make rat heart hypertrophy. Compared with the rats in the quiet training control group, the aerobic training group had significantly higher lisp and lower than ± dip / tax (p <0.05), while lived decreased significantly (p <0.05), indicating that aerobic exercise therapy in rats can significantly improve the cardiopulmonary function of the lungs and heart of this group of rats; the lisp and lower than ± dip / tax of this type of rats in the high-intensity aerobic training group of rats Both were significantly and significantly decreased (p <0.05), lived was significantly and significantly increased (p <0.05), indicating that high-intensity aerobic exercise in rats can significantly increase the heart and lung function and quiet in this group of rats Compared with the rats in the training control group, the heart rate of the aerobic training rats decreased significantly, the T wave voltage increased significantly (P <0.05), and there was no significant change in the QRS interval and QT interval, indicating increased cardiac function; fatigue and aerobic. The pulse heart rate and index QT interval of exercise rats increased significantly linearly (p <0.05), and there was no significant linear change in the QTRS.Interval cardiac voltage interval, indicating that the duration of myocardial elastic contraction in rats was significantly prolonged and the heart function improve. Exercise training can try to reduce the weight of rats and increase the weight of the heart, as shown in Figure 2.

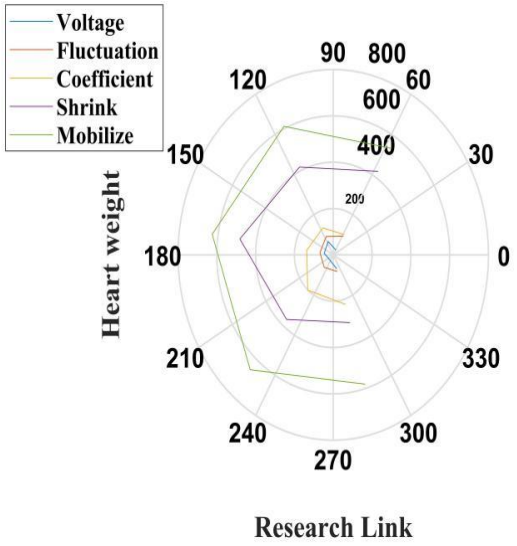

Figure 2. Exercise training can try to reduce the weight of rats and increase the weight of the heart

From the data in Figure 2, it can be seen that exercise training can test the weight loss of the rat, the weight of the heart increases, the weight of the rat decreases by 15%, and the weight of the heart increases by 7%, which is beneficial to improve the heart function of the rat.

5. Discussion on Cell Proliferation

5.1. Comprehensive Analysis of the Effect of Exercise Training on the Expression of HGF and TGF-β1 in Rat Myocardium on the Proliferation of Cardiomyocytes

Exercise training plays an important role in the proliferation of cardiomyocytes by inducing the

expression of myocardial HGF and TGF-β1. Studies have shown that exercise training inhibits the expression of C / EBA, a negative regulator of cell proliferation, and regulates cardiomyocyte proliferation by promoting the expression of HGF and TGF-β1. Athletes can also increase the activity of cardiomyocyte proliferating myocardial cytokines by up-regulating the expression levels of proliferating cells HGF and TGF-β1 in myocardium, and then effectively promote myocardial hypertrophy and csc myocardial cell proliferation. In addition, exercise can effectively promote the normal expression of myocardial cytokines related to the occurrence of myocardial blood vessels, promote the regeneration of myocardial collateral blood vessels, establish myocardial collateral blood circulation, provide a favorable environment for the regeneration of damaged myocardial cells, and improve the myocardium The state of ischemia and hypoxia and the microenvironment of cardiomyocyte regeneration regulate the proliferation of cardiomyocytes. Aerobic exercise proliferates stem cells in various ways so that athletes can play an important role in the proliferation of cardiomyocytes. Stem cells are an important source of myocardial cell proliferation, and the effectiveness of their mobilization is of great significance for myocardial repair after myocardial infarction. Aerobic exercise can effectively increase the expression levels of HGF and TGF-β1 in blood of endothelial progenitor cardiomyocytes, increase the content of HGF in blood serum and TGF-β1 in blood, promote the regeneration of capillaries in myocardial tissue and improve myocardial tissue Blood circulation status. American researchers have found that exercise can effectively increase the expression of HGF in the heart muscle tissue and TGF-β1 in the blood, and it is closely related to the sdf-1 / cxcr-4 signal axis of the human body. Exercise can effectively mobilize CPCS and EPCS and other stem cells to improve their functional activity. By increasing the expression of HGF and TGF-β1 to promote their cardiomyocyte-like differentiation, so as to achieve the effect of improving cardiac function. The study found that exercise training induced the expression of HGF and TGF-β1 in rat myocardium can regulate the proliferation of myocardial cells. The relevant data is shown in Figure 3.

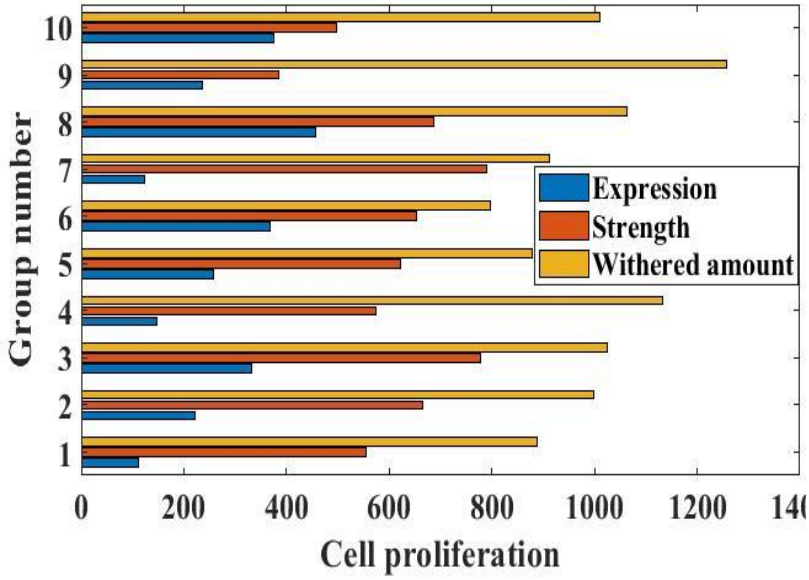

Figure 3. Exercise training induces the expression of HGF and TGF-β1 in rat myocardium can regulate the proliferation of myocardial cells

From the data in Figure 3, it can be seen that the exercise training induced the expression of HGF and TGF-β1 in rat myocardium can regulate the cardiomyocyte proliferative capacity and increase

the cardiomyocyte proliferative capacity by 16.5%.

5.2. Discussion on the Effects of Exercise Training Induced the Expression of HGF and TGF-β1 on Myocardial Cell Proliferation in Rat Myocardium

It is generally believed that the proliferation and apoptosis of cardiomyocytes are co-variable phenomena, not a causal relationship. Cell proliferation and apoptosis processes are related by cytokines. However, in recent years, it has been found that the expression of HGF and TGF-β1 induced by exercise training is closely related to the proliferation of myocardial cells. Studies have found that exercise induces HGF expression to form cardiomyocyte complexes, thereby inhibiting apoptosis and promoting the proliferation of cardiomyocytes. The expression of TGF-β1 can induce the expression of regulatory cell proliferation proteins. The release of TGF-β1 not only induces apoptosis but also inhibits the G1 phase of the cell cycle from entering the S phase. Studies have shown that exercise training can effectively increase the expression of HGF and TGF-β1, reduce the release of reactive oxygen species-induced cytochrome C from myocardial mitochondria, inhibit the occurrence of cardiomyocyte apoptosis, and increase the tolerance of cardiomyocytes to apoptosis; At the same time, the expression of myocardial HGF and TGF-β1 can increase the number of bone marrow stem cells in the circulatory system, and can improve the ability of cardiomyocyte migration and promote the proliferation of cardiomyocytes; at the same time, it can promote the expression and level of cytokines related to the proliferation of cardiomyocytes, Activating the signals and pathways of proliferating stem cells in cardiomyocytes allows myocardial stem cells or myocardial bone marrow stem cells to re-enter the cell cycle and promote the proliferation of cardiomyocytes. The study found that exercise training can promote myocardial HGF and TGF-β1 expression, as shown in Figure 4.

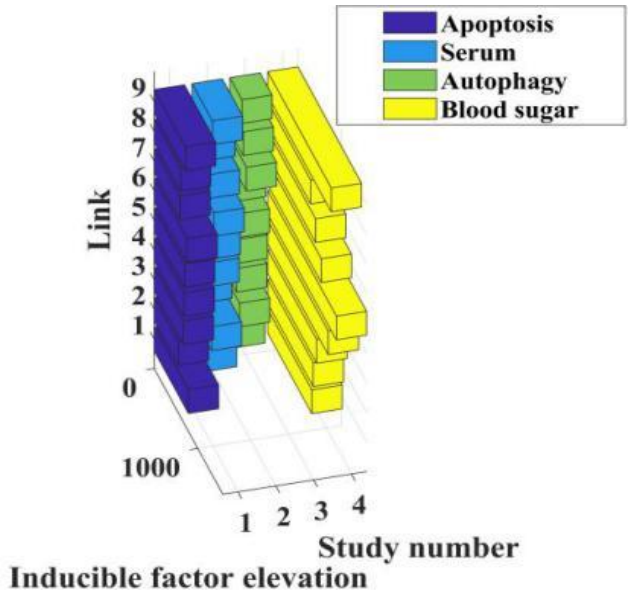

Figure 4. Exercise training can promote myocardial HGF and TGF-β1 expression

As can be seen from Figure 4, exercise training can promote the expression of myocardial HGF and TGF-β1, and increase the expression of myocardial HGF and TGF-β1 by 25%, thereby playing a role in promoting the proliferation of myocardial cells.

6. Conclusion

(1) Cardiovascular disease as a high incidence has seriously affected people's lives and health. Exercise training can affect the proliferation of cardiomyocytes by inducing the expression of myocardial HGF (sensory cell growth factor) and TGF-β1 (transforming growth factor), which can effectively improve the proliferation of cardiomyocytes, and at the same time suppress the apoptosis of cardiomyocytes, and improve the heart function. It is very helpful and can also play a role in improving cardiovascular disease.

(2) Aerobic exercise training can decrease the heart rate of rats by 17%, increase the occurrence rate of binuclear cells by 21%, and increase the expression of myocardial HGF and TGF-β1 by 25%. Proved that exercise training has a good effect on the treatment and prevention of cardiovascular diseases

(3) Studies have shown that exercise can improve the heart function of rats, how to increase the blood output of the heart of rats by 24%, improve the heart function of rats, and at the same time reduce the weight of rats by 15% and increase the weight of the heart by 7%, which is beneficial to improve rat heart function. Exercise training can also induce the expression of HGF and TGF-β1 in rat myocardium, which can regulate the proliferation of cardiomyocytes and increase the proliferation of cardiomyocytes by 16.5%.

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Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

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