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Taurine on Myocardial Mitochondria of Exhausted Rats

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Abstract: Taurine is the most abundant free amino acid in the human body, which can kill cells, stabilize cell membranes, inhibit lipid peroxidation damage, eliminate oxygen free radicals, regulate cell osmotic pressure and stabilize intracellular calcium. There are mechanisms to protect cardiomyocytes, such as maintenance mechanisms. This article mainly studies the protective effect of taurine on myocardial mitochondria of exhausted mice. In the experiment, the rats were randomly divided into two groups, one group was the control group, and the other group was the exercise exhaustion group (n=7). An animal experiment platform was used to create a model of exhaustion of rats. After exhaustion, the rats were immediately selected for experiments. The serum substance content of each group was measured, and the enzyme was used for immunosorbent experiment. The results showed that the taurine group had a heart rate of 359 and the control group had a heart rate of 372, indicating that taurine has a certain protective effect on rat myocardium. The experimental results show that taurine plays a significant protective role in myocardial mitochondria of exercise-exhausted rats, improves cardiac contractile function, and reduces heart rate variability caused by insufficient resistance exercise.

1. Introduction

In recent years, through detailed studies on the structure and function of mitochondria, it has been found that the activity of mitochondria plays an important role in the pathogenesis of various cardiovascular diseases. At the same time, mitochondria are also the target of many kinds of drugs. Myocardial ischemia is a common disease, which can endanger human life clinically and bring many negative effects on the heart. The direct effect is that the metabolism of myocardial cells is weakened, the capacity is reduced, the energy supply for heart activity is insufficient, and even arrhythmia and heart failure are caused. Taurine has a wide range of effects on the cardiovascular system, and its mechanism is complex. Taurine can protect myocardial disorders caused by ischemia, prevent arteriosclerosis, prevent lipid peroxidation, eliminate anaerobic free radicals, adjust cell osmotic pressure, and maintain the stability of intracellular calcium and regulate blood pressure.

Taurine has a protective effect on rat myocardial cells with acute myocardial ischemia and hypoxic disorder under conditions of glucose deficiency, which greatly reduces the death of myocardial cells. Taurine significantly increases the content of taurine in cardiomyocytes, thereby greatly increasing the delivery efficiency of taurine to cardiomyocytes.

Taurine has the function of regulating the normal physiological activities of the body, and can effectively reduce the damage of myocardial cells. Horvath investigated the effects of taurine and β-alanine supplementation on muscle function and expression of muscle taurine transporter (TauT) protein in mdx mice. In wild type (WT) and mdx mice (5 months) supplemented with taurine or β-alanine for 4 weeks, after which in vitro contractile properties, fatigue resistance and strength recovery, as well as TauT protein and those involved in excitatory contraction Protein expression (EC) coupling was examined in fast-twitch muscles. He found no difference in basal TauT protein expression or basal taurine content between mdx and WT muscle. Taurine and β-alanine supplementation can increase and decrease the taurine content in the muscles of wild-type and mdx mice, but has no effect on TauT protein. Taurine supplementation reduces the mass of the body and muscles and enhances the fatigue resistance and strength recovery capabilities of mdx muscles. Supplementation of β-alanine can enhance the anti-fatigue ability of WT and mdx muscles [1]. Although his research has certain reference value, the experimental process is not fully considered. Ren believes that long non-coding RNA (lncRNA) taurine upregulated gene 1 (TUG1) is involved in the occurrence and carcinogenesis of various tumors, indicating the diagnostic potential of TUG1 in these cancers. However, the exact role of TUG1 and its potential mechanism in gastric cancer (GC) are still unknown. In this study, he detected the expression of TUG1 and miR-145-5p in GC cell lines and non-malignant gastric epithelial cell lines by qRT-PCR. He transfected BGC-823 and SGC-7901 cells with si-TUG1, pcDNA 3.1-TUG1, miR-145-5p mimetics or matched controls. By MTT assay, Transwell invasion assay and tumor xenograft experiment, the biological functions of TUG1 and miR-145-5p in GC cell proliferation and invasion in vitro and tumor growth in vivo were studied. The regulatory relationship between TUG1 and miR-145-5 was confirmed by luciferase reporter gene assay [2]. Although his research has certain feasibility, it is more difficult to operate. Zhang believes that isoflurane is a commonly used inhalation anesthetic that may cause neurocognitive dysfunction, especially in elderly patients after surgery. Isoflurane causes ER stress and subsequent neuronal apoptosis in the brain, leading to cognitive dysfunction. Here, he checked whether taurine could prevent isoflurane-induced endoplasmic reticulum stress and cognitive impairment in elderly rats. Elderly rats were treated with low-, medium-, and high-dose excipients or taurine 30 minutes before exposure to 1.3% isoflurane for 4 hours. Old rats without any treatment served as controls. Brains were collected for molecular measurements 6 hours after isoflurane exposure, and behavioral studies were conducted 2 weeks later. Compared with the control group, isoflurane increased the expression of hippocampal ER stress biomarkers, including glucose regulatory protein 78, cleaved ATF-6 and C/EBP homologous proteins, and activated apoptosis pathway, BCL2-related X Decreased protein, increased expression of cytochrome c and cleaved caspase-3. Taurine pretreatment can dose-dependently inhibit isoflurane-induced increase in ER stress biomarker expression (except P-EIF2α and ATF-4), and reverse isoflurane-induced apoptosis-related protein changes. In addition, isoflurane can cause spatial working memory impairment in aging rats, and taurine pretreatment can prevent this situation [3]. Although his research is relatively comprehensive, the focus of the experiment is relatively vague.

The innovation of this article is that taurine drug pretreatment can effectively increase the myocardial capillary regeneration, reduce the area of myocardial infarction after I/R injury, and reduce the damage of tissue morphology. Taurine can increase the plasma VEGF production in rats

and promote ischemia Hypoxic myocardial angiogenesis. At the same time, it can also promote the migration of endothelial cells, promote angiogenesis, and then protect the ischemic myocardial tissue.

2. Protective Effect of Taurine on Myocardial Injury

2.1 Taurine

Taurine (Taurine) is a non-protein β-amino acid with high content in the body. Its chemical name is 2-aminoethanesulfonic acid. It is a colorless tetrahedral needle-like crystal, easily soluble in water. Under physiological conditions, taurine is mainly present in excitable tissues such as nerves, muscles, and glands of mammals. Taurine accounts for 50% of the free amino acids in myocardium and plasma [4].

Tau has the functions of anti-oxidation, stabilizing cell membrane, stabilizing Ca2+ and maintaining cell osmotic pressure, etc. It is an important substance for regulating the normal physiological functions of the human body. It can reduce cell damage caused by hypoxia, and adjust the balance of vasoconstriction, and has a certain preventive effect on hypoxic pulmonary hypertension. Tau has the effects of improving heart failure, improving fat metabolism, improving glucose metabolism, resisting hypertension and inhibiting arrhythmia and platelet aggregation. It exerts its biological function by rolling on the membrane, usually Tau is spontaneously transferred to the cell and is present in the cell at a high concentration, while Tau's rotator is on the cell membrane, which regulates the intracellular concentration through uptake and release, And indirectly adjust the osmotic pressure to protect the damage caused by swelling of nerve roots and so on. Many factors can enhance the activity of TAUT through extracellular hypertonicity, EPO, cAMP, etc., but high concentrations of glucose, ethanol, tumor necrosis factor, etc. can inhibit the function of TAUT [5].

Taurine can maintain the normal physiological functions of the human body, especially the brain. It can promote the growth and development of infants and young children, brain development, vision, reproduction and immune function. If it is lacking, it may cause children's abnormalities, visual impairment, increased susceptibility to epilepsy, and even diseases such as cardiomyopathy. It has a wide range of effects on the health of pharmacology and nutrition, including heat-clearing effect, analgesic effect, sedative effect, anti-spasmodic effect, excitatory effect, respiration function, heart function, anti-arrhythmic effect, blood pressure lowering effect, hypoglycemic effect, Antibacterial effect, hyperimmune function, anti-platelet aggregation effect, bile secretion promotion function, liver protection effect, detoxification effect. Moreover, it also involves regulating vascular tone, which may be an inhibitory neurotransmitter with many clinical uses [6].

2.2 Mitochondria

Different strength endurance exercise depends on different energy metabolism pathways, and the types of tendon fibers involved are also different. Mitochondria play an important role in maintaining the normal energy metabolism, structure and function of cells. Mitochondrial synthesis is controlled by both the nucleus and the mitochondria. The mitochondria in the nucleus are in a dynamic process of fusion and dissolution, so the proliferation of cells takes a certain amount of time, and the number, shape, and volume of mitochondria remain high. In the process of mitochondrial proliferation, mainly under the control of nuclear genes, multiple genetic factors were found in the cells, and the amount of newly generated protein accounted for about the amount of all

proteins. Most of the newly-produced proteins are encoded by nuclear genes. After transcription, translation, and mitochondrial membrane penetration mechanisms, the proteins are classified, fixed in specific nursing rooms, and then assembled to form multiple industrial foundations. Enzyme complexes in the respiratory chain or mitochondrial matrix [7]. Mitochondria are not only the "dynamic field" of cells important for kinetic energy metabolism, but also directly participate in the metabolism and regulation of "exercise oxygen pressure". When mitochondrial respiratory chain synthesizes ATP, it can produce anaerobic free radicals. Superoxygen phosphatase and glutamate dioxygenase remove free radicals and reduce harm. The main antioxidant enzymes present in the body are representative products of lipid peroxides and are sensitive to the measurement of free radical metabolism in the body [8]. It can objectively reflect the level of free radicals produced by the body. During intense exercise, the local oxygen consumption of skeletal muscle increases, and the oxygen consumption increases sharply, which may cause various types of oxidative damage and non-activation. These will lead to the decline of the energy conversion function of the mitochondria characterized by defects and separation of the respiratory chain. It can be seen that changes in the antioxidant capacity of skeletal muscle also affect the activity of mitochondrial respiratory chain enzymes and affect their energy metabolism [9].

Mitochondrial dysfunction may directly lead to cell death, and is an important molecular mechanism for the occurrence and pathogenesis of various cardiovascular and neurological diseases. Under normal circumstances, mitochondria have a double-layer structure to maintain a relatively stable internal environment. If cells die, a lot of cell pressure signals will accumulate around the mitochondria, promoting the transparency of the mitochondrial outer membrane. The transparency of the outer mitochondrial membrane is mainly controlled by Bcl-2 family proteins. The reduction found by Bcl-2 increased the transparency of the outer mitochondrial membrane, brought about the release of tyrosine ester C, and activated the tyrosinase-dependent cascade reaction [10].

2.3 Myocardial Injury

Hcy decreased the viability of cardiomyocytes and resulted in the death of cardiomyocytes. Hcy showed a concentration and time-dependent manner, which affected the adverse reactions of cardiomyocytes. Long term stimulation of a small or moderate amount of Hcy can seriously damage cardiomyocytes and lead to cardiomyocyte death. Most of HHcy patients with mild or moderate increase of Hcy have long-term mild or moderate increase of serum Hcy, which causes significant damage to myocardial cells. Early intervention of patients with mild or moderate increase of Hcy level can prevent myocardial injury. Hhhcy can induce atherosclerosis and left ventricular remodeling. Oxidative stress is widely considered as an important molecular mechanism of disease. Ers plays an important role in cell survival and death. Moderate or short-term stress can activate transmembrane proteins perk, ATF 6 and ire 1 to enhance the processing capacity of intracellular network. It relieves stress and protects cells. Excessive or long-term stress can regulate cell resolution by gradually activating chop, caspase-12 and JNK dependent extinction promoting signals [11-12].

If the blood flow of the myocardium is reduced, the oxygen uptake of the blood cells is directly reduced, the metabolism of the myocardial cells is weakened, and the production of ATP is reduced, which ultimately cannot meet the metabolic needs of the body. Tissue asdosis, calcium overload, ROS accumulation, etc., will lead to myocardial cell damage and reduced cardiac function. The initial damage is relatively small, and ATP is not completely consumed. The remaining ATP can provide the energy needed for cell death. Secondly, if myocardial ischemia cannot be further

improved, ATP will be consumed in large amounts, and myocardial cells will form a necrotic shape.

3. Experimental Study on the Protective Effect of Taurine on Myocardium in Exhausted Rats

3.1 Experimental Object and Exercise Exhaustion Model

14 healthy male SD rats of the same age and weighing 220-250 g were used. It was housed in a constant temperature SPF laminar flow chamber at 25 ℃. Lighting situation: 12 hours of light and 12 hours of darkness alternately. Provide standard pellet feed and drinking water to make it free to eat.

The rats were randomly divided into two groups, one group was the control group, and the other group was the exercise exhaustion group, each group had 7 rats. An animal experiment treadmill was used to establish a rat model of exercise exhaustion.

1) Perform preliminary experiments on mice on a treadmill. Place the mouse on the table regularly every day to adapt to the environment. Use 40 minutes each time for a week.

2) After a week of training the rats on the operating platform, a comprehensive experiment will begin. The electrocardiogram of each rat after exercise was recorded using exercise heart force monitor for 30 minutes and 60 minutes of exercise.

3.2 Instruments, Drugs and Reagents

The main instruments, drugs and related reagents in the experiment are shown in Table 1 and Table 2.

Table 1: Main instruments

Serial Number	Instrument Name	Manufacturer
1	AF100 Ice Machine	Scotsman Corporation
2	Millipore Pure Water Machine	France Millipore
3	Pipette	Eppendorf
4	4 ℃ Refrigerator	Baixue Electric Company
5	-20 ℃ Low Temperature Refrigerator	Zhongke Meiling Company
6	-80℃ ℃ Ultra-Low Temperature Refrigerator	American Thermo Forma
7	PH Meter	Tianjin Onuo Instrument Co Ltd.
8	AG135 Electronic Balance	Switzerland METTLER TOLEDO
9	Hitachi-s7500 Transmission Electron Microscope	Hitachi
10	High-Throughput Homogenizer	METTLER TOLEDO Companies
11	Constant Temperature Water Bath	German Edmund Buhler
12	Mini Centrifuge	American Thermo Forma
13	Sukun Sky-200b Constant Temperature Shaker	Shanghai Sukun Industrial Co Ltd.

Table 2: Medicines and reagents

Serial Number	Reagent Name	Batch Number	Manufacturer
1	Taurine	1419568V	Tianjin Biochemical Pharmaceutical Factory
2	Heparin Sodium Injection	20100404	Biochemical Pharmaceutical Factory
3	0.9% Sodium Chloride Injection	2176G4	Tianjin Otsuka Pharmaceutical Co Ltd.
4	Urethane	0808059	Shanghai Zhanyun Chemical Co Ltd.
5	Sodium Dihydrogen Phosphate	821107	Tianjin No. 6 Chemical Reagent Factory
6	Disodium Phosphate	910119	Beijing Chemical Plant

7	Formaldehyde solution	20110708	Tianjin Yingdaxigui Chemical Reagent Factory
8	Tribenzine biological Stain	990113	Beijing Yinghai Fine Chemical Factory

3.3 Experimental Procedure

(1) Immediately after exhausting exercise, draw blood from the rat's lower abdominal vein, centrifuge at 300r/min for 20 minutes, and store in a refrigerator at -80℃. Measure the serum substance content of each group, use enzymes to carry out immunoadsorption experiments, and follow the steps in the kit. The OD value of each well at 450 nm was measured on an enzyme scale.

(2) After anesthesia, weigh the rat and fix its anterior position on the operating table, disinfect the skin of the neck, cut open in the middle of the neck, separate the trachea and insert it into the tube. Isolate the right common carotid artery, pass two silk threads under the common carotid artery, insert a thread in it and tie the distal end. The medial end of the carotid artery closed by the arterial clamp was resected at the medial end of the ligation, and the Millar pressure volume catheter SPR-858 was inserted into the left ventricle of the heart in the direction of reverse flow through the incision. The pressure waveform of the left ventricle of anesthetized rats was recorded in real time, the catheter position was determined by the change of physiological waveform pressure waveform, and the medial end electrode was placed at the tip of the heart. The distal electrode is located on the main valve, and another thread fixes the catheter.

(3) Disinfect the abdomen, cut in the middle of the lower abdomen, apply rapid pressure to the inferior vein to record the changes in pressure and volume waveform when the heart preload changes, and obtain the vena cava occlusion ring. The end systolic inverse volume (Ees) is obtained to measure the contractility of the myocardium itself.

(4) Inject 40μL of NaCl solution from the jugular vein, and record the changes in pressure and wave volume after injection of high leaching saline. It is used to eliminate the parallel volume caused by the resistance (parallel resistance) of the ventricular wall.

3.4 Statistical Analysis

All data are expressed using. The statistical software SPSS 17.0 was used to analyze the experimental data. In order to compare multiple sets of data, elemental analysis of variance was used, and a priori variance consistency test was conducted. When comparing variances using the SNK method, if the variances are inconsistent, use the Dunnett'S T3 method to perform a data difference analysis for each group. $P<0.05$ is statistically significant.

4. Analysis of Protective Effect of Taurine on Myocardium of Exhausted Rats

4.1 Effects of Taurine on Cardiac Structure and Function

The experimental results are shown in Table 3 and Figure 1. It can be seen from the data in the table that the heart rate of taurine group is 359, and that of control group is 372, which indicates that taurine has a certain protective effect on myocardium of rats. Compared with the control group, the cardiac structure (left ventricular systolic / diastolic anterior wall thickness, left ventricular systolic / diastolic posterior wall thickness, left ventricular end diastolic diameter) and function (ejection fraction, shortening rate of short axis) were not significantly changed in the experimental group. In the process of blood microcirculation, the interaction between blood flow force, blood cells and

blood vessel wall is of great significance for blood material exchange, nutrition and defense function, and maintaining the stability of internal environment. Once one side's function or interaction is abnormal due to some factors, it will cause local and even systemic pathological changes. White blood cells (WBCs) have immune and antimicrobial activities. They perform defense functions in vivo. Normally, they roll from Burmese cells along the surface of postcapillary venule. Generally, leukocytes hardly adhere to the arterioles. However, when the microcirculation disorder is caused by pathological changes in the body's local and even the whole body, the phenomenon of rolling and adhesion between leukocytes and vascular endothelial cells can occur in the retrocapillary venules and venules. In the normal group and sham operation group, the muscle fibers were neat, the nuclear size and morphology were basically the same, the chromatin was uniform, and no hypertrophy, necrosis, atrophy, rupture and interstitial cell proliferation were found. In the model group, the myocardial fibers were hypertrophic, and the broken and atrophic muscle fibers were observed locally, and the transverse diameter of myocardial cells was increased. In addition, in qlqx. H group, only a small amount of myocardial fiber atrophy was observed, and myocardial fiber degeneration and hypertrophy were occasionally seen.

Table 3: Experimental results data

	Heart rate (beats/min)	SBP(mmHg)	Hcy(umol/L)
Control group	372	93.6	8.55
High methionine group	341	104.2	52.3
Taurine group	359	95.2	36

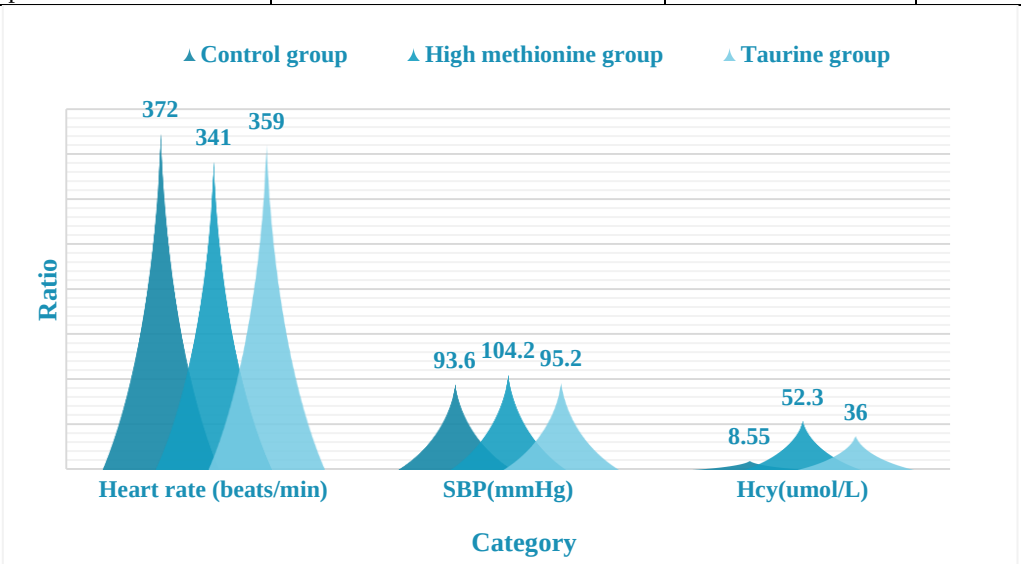

Figure 1: Comparison of experimental data

4.2 Ultrastructural Changes of Rat Myocardium in Each Group

The changes of mitochondrial fusion and division related proteins in rat myocardium are shown in Figure 2. In the control group, the myofilaments were arranged orderly, sarcomere was regular, the structure was clear, the mitochondrial cristae was normal, and the arrangement was dense. In the exhaustive exercise group, the shape of myocardium, the disorder of sarcomere, the widening of perinuclear space, the slight swelling of mitochondria and the fracture or disappearance of cristae were observed. In the experimental group, the number of mitochondria increased gradually and the

volume became smaller. In the process of oxidative phosphorylation, a large number of positive charges are generated in the mitochondrial membrane space, with positive charges on the outer side and negative charges on the inner side of the inner membrane, which forms the transmembrane potential. However, a large number of negative charges were generated in the mitochondrial matrix, which resulted in the formation of transmembrane potential across the mitochondrial membrane. Normal mitochondrial membrane potential is an important condition to maintain the energy metabolism of cells. The decline of mitochondrial membrane potential is an early indication of mitochondrial function decline and cell apoptosis. The number of apoptotic cells in ISO group was significantly higher than that in control group ($P < 0.001$), suggesting that myocardial ischemia and hypoxia can lead to apoptosis. Compared with ISO group, the number of apoptotic cells in NIC group was significantly lower than that in ISO group ($P < 0.001$). The number of apoptotic cells in 5-HD group was significantly higher than that in NIC group ($P < 0.001$). NADH oxidase, located in the extracellular membrane, can directly oxidize extracellular NADH to NAD + (nicotinamide adenine dinuclear acid) in the presence of oxygen and generate electrons. Under anaerobic condition, proton is the final electron acceptor, and the electron is transferred directly to the intracellular proton through the hydrogenase on the cell membrane, and finally hydrogen is generated. It can be seen that it plays an important role in the glycolysis, the trehalose cycle and the redox reaction in the mitochondrial respiratory chain, which mainly provides the necessary preparation for energy storage and release. After exhaustive swimming, the adhesion between leukocytes and vascular endothelial cells occurred in the microvasculature of different diameters in the mesenteric microcirculation of rats. Because of their large volume and high stiffness, leukocytes adhere to vascular endothelial cells, although they are under the action of shear stress, they are not easy to deform, so that white blood cells often form protuberances on the surface of vascular lumen, which increases the resistance of blood flow in blood vessels. In addition, there is a white blood cell adhering to the surface of endothelial cells in a small artery, and there is also an obvious phenomenon of leukocyte adhesion on the wall of the capillary, which may cause the phenomenon of blood flow obstruction in the capillary, and then cause no reflow phenomenon.

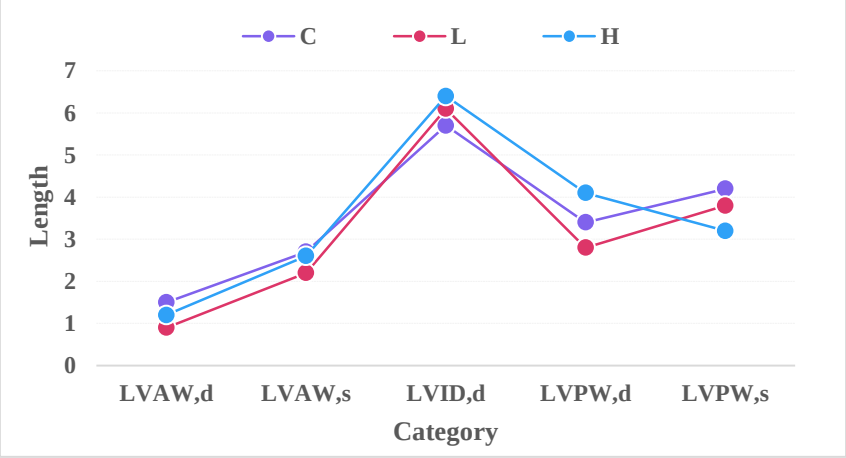

Figure 2: Changes in mitochondrial fusion and division-related proteins in rat myocardium

4.3 Effects of Taurine on Mitochondrial Fusion and Mitotic Protein of Cardiomyocytes

Compared with the control group, the LVEF of rats fed with high methionine diet for a long time was significantly decreased ($P < 0.01$), and the LVEF value of rats treated with taurine was

significantly increased (P < 0.01). Compared with the control group, the lvfs value of rats fed with high methionine diet for a long time was significantly decreased (P < 0.01); compared with the high methionine group, the lvfs value of rats treated with taurine was significantly increased (P < 0.01). GRP78 protein was specifically expressed in ers, and brown granules were observed in the cytoplasm of cardiomyocytes by immunohistochemical staining. In the control group, a small amount of brown yellow particles were observed in the myocardial tissue of rats in the control group; a large number of brown yellow particles were observed in the myocardial tissue of rats in the high methionine group, which was significantly higher than that in the control group (P < 0.01); compared with the high methionine group, the expression of brown yellow particles in the myocardial tissue of taurine group was significantly reduced (P<0.05). Secondly, the regulation of protein expression is not only reflected in the transcriptional level of genes, but also influenced by post transcriptional regulation, translation level and post translational regulation. Therefore, although there is no significant difference in transcriptional level after endurance exercise, it can ultimately show significant differences in protein level. The changes of mRNA contents in rat cells are shown in Figure 3.

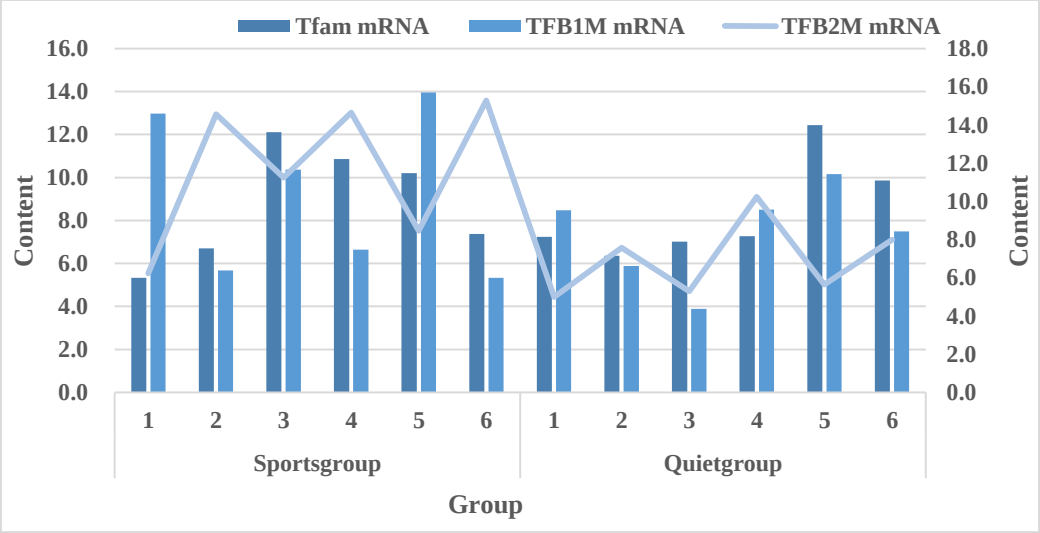

Figure 3: Changes in various mRNA levels in rat cells

GRP78 and CRT are specific expression proteins related to ers. In the control group, the expression of GRP78 protein was detected by Western blot; compared with the control group, the expression of GRP78 protein in the high methionine group was significantly increased (P < 0.01); compared with the high methionine group, the expression of GRP78 protein in the taurine group was significantly decreased (P<0.01). Compared with the control group, the expression of CRT protein was significantly increased in the high methionine group (P<0.01); compared with the high methionine group, the expression of CRT protein in the taurine group was significantly decreased (P<0.05). Chop is an ers related specific Pro apoptotic protein. In the control group, a small amount of chop protein was detected by Western blot; compared with the control group, the expression of chop protein in the high methionine group was significantly increased (P<0.01); compared with the high methionine group, the chop protein expression in the taurine group was significantly decreased (P < 0.05). Compared with the control group, the expression of Bcl-2 protein in the high methionine group was significantly decreased (P<0.01); compared with the high methionine group, the expression of Bcl-2 protein in the taurine group was significantly increased (P<0.05). Compared

with the control group, the expression of Bax protein in the high methionine group was significantly increased (P<0.01); compared with the high methionine group, the expression of Bax protein in the taurine group was significantly decreased (P<0.05).

4.4 Effect of Taurine on Hemodynamic Changes in Exhausted Rats

The effects of taurine on the dynamic changes of myocardial injury site in rats are shown in Table 4 and Figure 4. The change of neuroendocrine system is the key link of stress. Adrenaline and norepinephrine are important hormones in the regulation of cardiovascular system. Its content reflects the activity of sympathetic adrenal medullary system. The levels of NE and E in serum of exhausted group were significantly increased, which indicated that the sympathetic nerve was over excited by exhaustive exercise and stimulated by nerve endings. Adrenal medulla leads to the increase of NE and e release and the increase of catecholamine in blood, which directly affects the heart and blood vessels. The effects of E on adrenergic receptors on myocardial cell membrane cause cardiac hemodynamic changes. Both E and NE can enhance energy metabolism, increase myocardial oxygen consumption, destroy the balance of myocardial oxygen supply and demand, cause myocardial hypoxia necrosis and even energy depletion, leading to heart failure. NE and a large amount of E also mainly bind to receptors on vascular smooth muscle, leading to coronary spasm and further aggravating myocardial ischemia injury. Masson staining showed that the myocardial fibers were red, the nuclei were blue and black, and the collagen fibers were blue. In the normal group and sham operation group, the myocardial fiber bundles were arranged tightly and orderly, and no obvious collagen fiber proliferation was found; in the model group, large area of collagen hyperplasia was observed locally, and spread among the myocardial fiber bundles, showing a network distribution; each drug administration group could inhibit the proliferation of collagen fiber and reduce the area of myocardial tissue and collagen fiber around blood vessels to varying degrees. MDA is the final product of lipid peroxidation between oxygen free radicals and polyunsaturated fatty acids in biofilm. The content of MDA reflects the level of oxygen free radicals and the severity of lipid peroxidation in vivo. The enzyme antioxidant system of the body, including SOD and glutathione peroxidase, can clear oxygen free radicals in the body and form a dynamic balance. The results showed that the oxidative metabolism rate of brain tissue was high, the amount of reactive oxygen species metabolites was large, the antioxidant capacity was relatively low, and the repair ability was poor. Neurons could not be duplicated and the ratio of cell membrane to cytoplasm was high. The concentration of polyunsaturated fatty acids in the cell membrane lipid of brain tissue was high, which became an important source of lipid peroxidation, and it was easy to produce free radicals to damage the adjacent molecules. The long-term existence of moderate HHcy can induce apoptosis, destroy myocardial structure and lead to cardiac dysfunction; the molecular mechanism of HHcy on cardiomyocytes may be related to the excessive activation of ers and mitochondrial dysfunction. Taurine may antagonize the toxic effect of HHcy on cardiomyocytes by inhibiting ers and mitochondrial stress, promote cell survival and improve cardiac function.

Table 4: Dynamic changes of myocardial injury sites in rats

Group	IS(mg)	AAR(mg)	IS/AAR(%)
I/R	85.61	241.36	0.412
Low	61.87	221.03	0.305
Middle	81.52	194.55	0.215
High	51.69	257.23	0.146

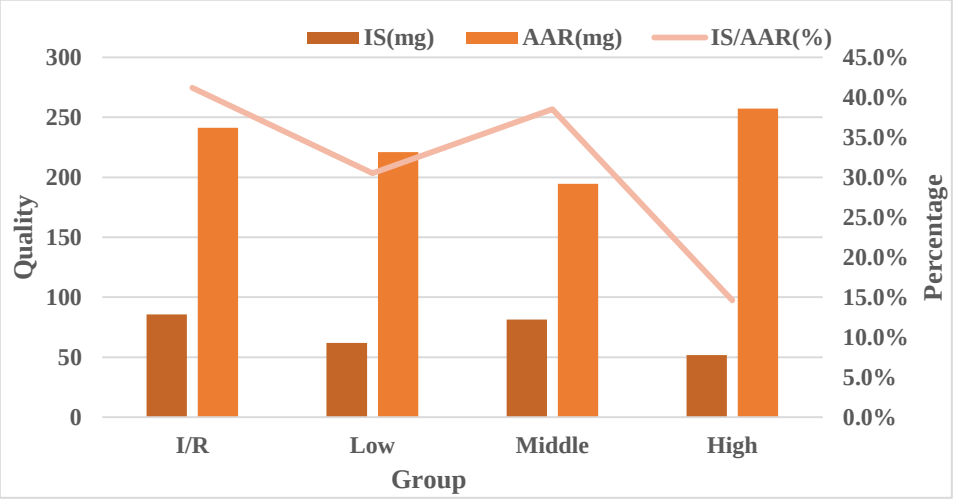

Figure 4: Dynamic changes of myocardial injury site in rats

5. Conclusions

This article mainly studies the protective effect of taurine on myocardial mitochondria of exhausted mice. The experimental results show that taurine can effectively reduce myocardial damage caused by exhaustion, improve the contractile function of the heart, and improve myocardial ischemia. When free radicals undergo excessive violent exercise or weaken the body's ability, the balance between pre-oxidants and antioxidants will be disturbed. However, it will cause changes in the structure and energy of the biofilm, increase the permeability of the biofilm, deviate from the cell content, reduce the fluidity of the mitochondrial membrane and destroy the function. Oxidase activity tends to decrease, indicating that oxidative damage affects the rate of electron transfer in the respiratory chain of the mitochondria and reduces the ability of the mitochondria to utilize oxygen.

Different tissue cells may have different types of TAUT, so their ability to absorb TAU is also different. As a result, in different tissues, the proportion of intracellular and extracellular TAU concentrations is very large. The increase of taurine content in cardiomyocytes increased by exogenous low-dose taurine may increase the tonebp of cardiomyocytes.

Mitochondrial health depends to some extent on mitochondrial kinetic balance. Mitochondria are in the process of continuous division and fusion, which is of great significance for maintaining the stability of mitochondrial morphology and promoting the transmission of chemical and electronic signals. Some pathological lesions disrupt the balance of mitochondrial fusion and division, leading to changes in mitochondrial morphology and reduced mitochondrial function. Mitochondrial division leads to an increase in the number of mitochondria, leading to an increase in ATP content. With the increase of drug dose, the mitochondrial damage is greater than the compensation effect, and the ATP content is significantly reduced.

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