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Strain Articular Cartilage and Synovial Tissue Based on Observation Exercise under Microscope Image

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Abstract: A piece of hyaline cartilage between the bones in the joint is called articular cartilage, and the synovial tissue is the inner layer of the joint capsule, which is light red, smooth and soft. Therefore, many studies have focused on the changes in articular cartilage and the possible strain of synovial tissue in the process of osteoarthritis, and the necessity and effectiveness of treating osteoarthritis based on this structure. Applying high-intensity cyclic tensile strain to the monolayer cultured chondrocytes can make the chondrocytes change from polygon to spindle shape and cause changes in matrix proteins and matrix metalloproteinase. The abnormal mechanical load of the articular cartilage may cause the degeneration of the articular cartilage. Based on the above background, the research content of this article is to observe the articular cartilage and synovial tissue under a microscope, and analyze the possible damage of the articular cartilage and synovial tissue. First, the function of articular cartilage and synovial tissue is analyzed theoretically, and the influence of exercise on articular cartilage and synovial tissue is analyzed. Through the rat simulation experiment, the results show that the rats will develop sinusitis after 42 days of continuous exercise, and the histological score of the synovium of the rats is greater than that of the A group. The loss of articular cartilage and long-term high-intensity exercise can cause the degradation and variation of articular cartilage.

1. Introduction

Moderate exercise is necessary to maintain the stability of normal articular cartilage. Excessive exercise will lead to the destruction of articular cartilage morphology and bone metabolism. Cartilage consists of chondrocytes and matrix. When articular cartilage is damaged, the expression and secretion of collagen in cartilage matrix will also change.

In previous studies, the strength of bone and cartilage can depend on the mechanical load [1].

Although physical exercise can increase bone density and bone mass, the adaptive response of bone to exercise and bone maturity [2]. There is an important relationship between exercise intensity and load. Excessive exercise may cause harmful injury [3]. The main reason for the injury of articular cartilage is due to the pathological changes of body function caused by sports trauma. The stress state of body function will affect the biological state and normal function of human organs, tissues and even cells, which has a certain effect on the regulation of the physiological process of articular cartilage [4]. However, the growth and maintenance of articular cartilage will be greatly affected by hand exercise load [5]. In competitive sports like marathon, whether long-term high-intensity running will lead to knee joint injury remains controversial [6]. According to the epidemiological survey, there is no statistical difference between the prevalence of OA in long-distance runners and normal people. The relevant research team found that exercise with certain intensity can inhibit the loss of mucopolysaccharide in cartilage injury, reduce the withering rate of chondrocytes, and reduce the stimulation of inflammation on human body's mechanism, so as to protect cartilage tissue [7]. At the same time, some clinical studies at home and abroad have also found that some sports enthusiasts' knee MRI will have persistent knee pain, which will lead to the probability of abnormal performance of sports enthusiasts' knee MRI [8-9].

Li observed the effect of simple acute hematoma on cartilage and synovium, and the improvement of hyaluronic acid (HA) on its injury [10]. Kim established a co culture method of chondrocytes and mesenchymal stem cells (MSCs) to overcome the dedifferentiation of chondrocytes during the expansion of chondrocytes in vitro. Compared with other mesenchymal stem cells, synovial stem cells (sdscs) have good chondrogenic potential and are easy to obtain without donor site complications. They are promising co culture cell sources. However, the study on co culture of chondrocytes and synovial stem cells is very limited. Kim's aim was to investigate whether direct co culture of human chondrocytes and synovial stem cells could promote cartilage formation. Kim used three different proportions of chondrocytes and synovial stem cells to co culture directly, the proportions were three to one, one to one and one to three respectively [11-12]. Zou observed the expression of hypoxia inducible factor and vascular endothelial growth factor in the synovium of collagen arthritis, and whether PI3K pathway regulated the angiogenesis of RA [13]. Warnock evaluated ten horses for cartilage damage under arthroscopy and received various intra-articular treatments after operation. Three of the ten horses had injuries beyond the reach of surgery. Four horses recovered their health, their pre-operative exercise level and one horse's continuous intermittent claudication. None of the horses that were hard to reach recovered. The time of lameness before operation, the evidence of degenerative arthropathy before operation and the accessibility of cartilage injury to operation had no significant effect on the prognosis. As a main cause of lameness, the articular cartilage injury of distal interphalangeal joint can guarantee the healthy prognosis through surgical treatment [14-15].

The stress and strain around the joint act on the cell and cell matrix and produce a series of biological changes. It not only depends on the stress acting on the joint, but also depends on the neuromuscular environment, physiological state and soft tissue function around the knee joint. Of course, the damaged structure is more sensitive to normal or excessive stress stimulation, and the risk of inducing OA is greater. In this paper, T1rho and T2mapping sequences were used to study the effect of different daily exercise on the biochemical components of the knee joint cartilage that have not changed in morphology and structure, and to explore the significance of biomechanical changes in the mechanism of cartilage degeneration and injury, so as to provide important reference information for various studies.

2. Articular Cartilage and Synovial Tissue Damage

2.1. Articular Cartilage and Synovium

The articular cartilage can reduce the relative friction between the joint surface and the joint surface in the repeated movement. It is an elastic bearing tissue on the human bone joint surface. Articular cartilage can buffer the impact to reduce the damage to human body, and absorb a certain degree of impact. The distribution of chondrocytes and components was uneven. The chondrocytes in the surface layer of human body are irregular, flat and round; the chondrocytes in the middle layer are nearly round. The distribution of proteoglycans is characterized by less shallow layer and more deep layer.The synovium of patients with advanced OA is thickened due to hyperplasia and fibrosis, but compared with it, the synovium of patients with early OA shows higher inflammatory characteristics. Synovitis is an independent risk factor for OA. Synovitis is an independent risk factor in the early stage of OA, which has a threshold effect. When synovitis aggravates to a certain extent, such as the increase of synovium thickness, synovitis itself can be an independent risk factor, leading to structural damage, such as knee joint cartilage and osteoarthritis. MRI is a feasible and reliable method to evaluate synovitis. It is found that chronic synovitis can accelerate the degeneration of knee joint cartilage, promote the occurrence and development of OA, and cause knee joint pain. Spontaneous and disordered energy transfer makes magnetization or spin ordered.The process of diagnosis and evaluation of OA patients is shown in Figure 1:

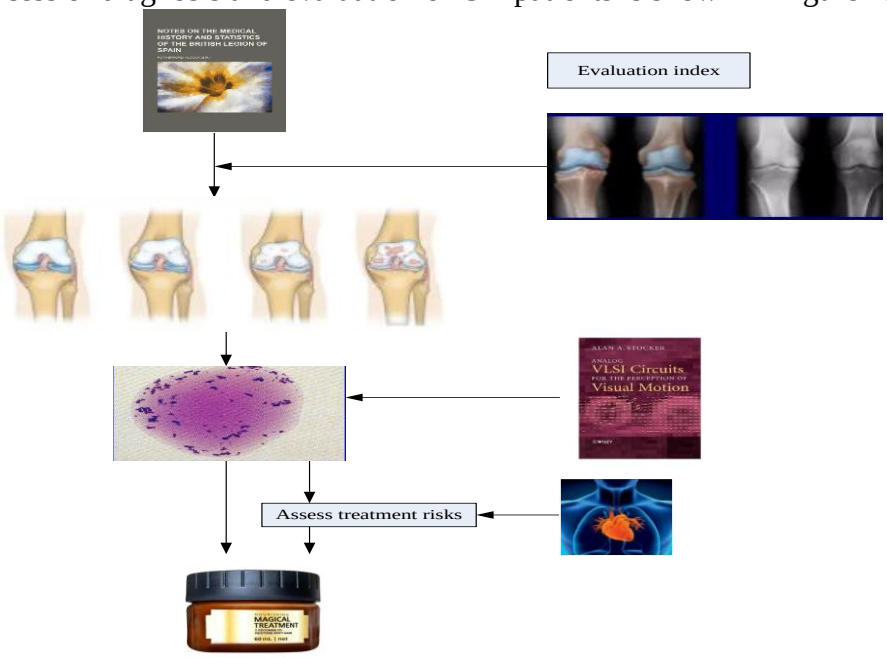

Figure 1. OA diagnosis and evaluation flowchart

Articular cartilage belongs to hyaline cartilage. When the human body bears the mechanical load, it can absorb a certain degree of external pressure through its own deformation, so as to reduce the impact of external pressure on the subchondral bone; at the same time, the physical friction coefficient of articular cartilage is far lower than that of normal bone, so it can reduce the damage of human joints to human bone during movement,Figure 2 is an image of human articular cartilage. The unique chondrocyte and extracellular matrix structure of articular cartilage, the main

components of which are proteoglycan and collagen, determines the biomechanical properties of articular cartilage. Among them, chondrocytes account for only one percent of the total cartilage, but they are very important. Cartilage tissue is responsible for the secretion of components in synthetic cartilage matrix. Collagen of cartilage matrix is mainly type II, accounting for 90% of cartilage collagen. The basic structure of cartilage is constructed by the reticular structure of type II collagen in cartilage. There are horizontal cartilage on the surface of cartilage. This unique structure makes cartilage have the physical characteristics of low friction. Another important role of type II collagen is to combine other macromolecules in the matrix, among which type IV and type VI collagen fibers play a key role. Although the content of type IV and type VI collagen fibers is relatively small, they play a decisive role in maintaining the stability of cartilage; one of the important components of cartilage matrix is proteoglycan, which is composed of a single core protein and a large number of glucosamine chains connected with the core protein. Proteoglycan is a kind of polymer, which exists in the form of polymerized protein. The polypeptide has the characteristics of strong hydrophilicity and low viscoelasticity, and filling in the arched network structure of collagen can make cartilage absorb impact well.

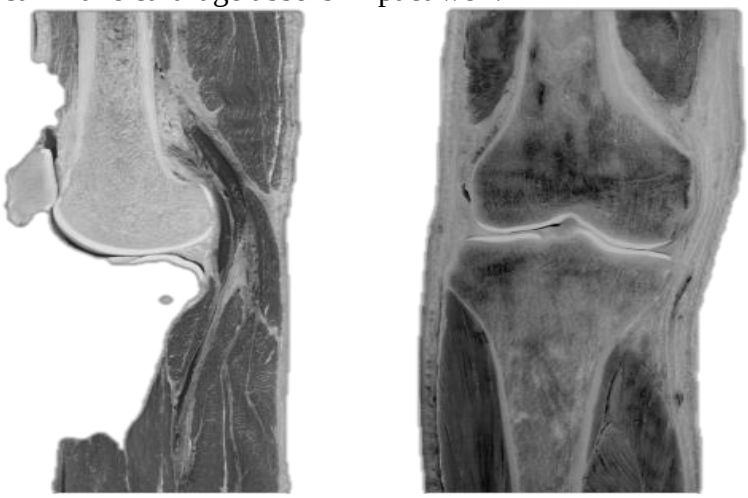

Figure2. Schematic diagram of a specimen of knee joint cartilage

Synovium is a kind of special connective tissue around the joint. It covers the tendon and forms the lining of joint capsule and fat pad. In synovial joints, the synovium separates the fluid in the synovium cavity from the surrounding tissue,the specific synovium diagram is shown in Figure 3.Synovium can secrete synovial fluid and hyaluronic acid to maintain the volume and composition of synovium. Because articular cartilage has no inherent blood vessels and lymph supply, synovium can maintain the nutrition supply of chondrocytes through the produced synovial fluid and hyaluronic acid. The main structure of articular cartilage is extracellular matrix, in which chondrocytes also exist, but the content is very little. ECM includes collagen and non collagen components. In cell matrix and matrix matrix matrix interactions, non collagen components play a very important role. Comp is the main non collagen component of cartilage. In cartilage, the main expression site of comp is located in the cartilage membrane matrix around chondrocytes, so it is not difficult to understand that comp may have direct interaction with chondrocytes. In the early study of collagen synthesis in vitro, comp can organize collagen molecules closely and parallel almost in time, so as to make collagen synthesis and assembly faster and more effective. Compared with that without comp, the number of collagen fibers synthesized by comp increased significantly, indicating that comp plays an important role in the regulation of collagen synthesis.

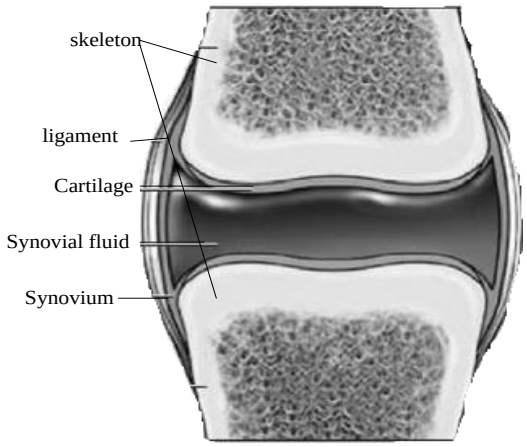

Figure 3. Schematic diagram of synovium in human bones

When observing articular cartilage and synovium, the most common specific methods of calculating phase algorithms are used. The four-step method is a more algorithm used for phase extraction. It needs to measure the intensity information of four images, and the calculation is obtained by simplifying the calculation.

$$\phi(x,y)=\tan^{-1}\left(\frac{I_4(x,y)-I_2(x,y)}{I_1(x,y)-I_3(x,y)}\right) \quad (1)$$

The modulation coefficient can be obtained by the following equation:

$$\lambda(x,y)=\frac{\sqrt{[I_4(x,y)-I_2(x,y)]^2+[I_1(x,y)-I_3(x,y)]^2}}{2I_0} \quad (2)$$

For different phase extraction algorithms, the required interference fringe images are different, but at least three images are required. However, the three-step method only needs to record three interference images. The phase offset here is $\frac{1}{2}\pi$, and α_i is $\frac{1}{4}\pi,\frac{3}{4}\pi,\frac{5}{4}\pi$ respectively. Then the intensity of three measurements is

$$I_1(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)+\frac{1}{4}\pi]\} \quad (3)$$

$$I_2(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)+\frac{3}{4}\pi]\} \quad (4)$$

$$I_3(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)+\frac{5}{4}\pi]\} \quad (5)$$

The phase at this time is

$$\phi(x,y)=\tan^{-1}\left(\frac{I_3(x,y)-I_2(x,y)}{I_1(x,y)-I_2(x,y)}\right) \quad (6)$$

The modulation intensity factor is

$$\gamma(x,y)=\frac{\sqrt{[I_1(x,y)-I_2(x,y)]^2+[I_2(x,y)-I_3(x,y)]^2}}{2I_0} \quad (7)$$

If the phase shift is $\frac{2}{3}\pi$, the intensity information of the three measurements becomes

$$I_1(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)-\frac{2}{3}\pi]\} \quad (8)$$

$$I_2(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)]\} \quad (9)$$

$$I_3(x,y)=I_0(x,y)\{1+\gamma\cos[\phi(x,y)+\frac{2}{3}\pi]\} \quad (10)$$

The phase obtained here is

$$\phi(x,y)=\tan^{-1}(\sqrt{3}\frac{I_3(x,y)-I_2(x,y)}{2I_1(x,y)-I_2(x,y)-I_3(x,y)}) \quad (11)$$

If the phase offset is other than $\frac{1}{2}\pi$ or $\frac{2}{3}\pi$, the phase can be obtained by the following equation

$$\phi(x,y)=\tan^{-1}(\frac{1-\cos(\alpha)}{\sin(\partial)}\frac{I_3(x,y)-I_2(x,y)}{2I_1(x,y)-I_2(x,y)-I_3(x,y)}) \quad (12)$$

The modulation intensity factor is

$$\gamma=\frac{1}{2I_0}\sqrt{\frac{[(I_2-I_3)+(I_1-I_4)]^2+[(I_2+I_3)+(I_1+I_4)]^2}{2}} \quad (13)$$

2.2. Three Dimensional Finite Element Model of Articular Cartilage and Cartilage Imaging Technology

In the numerical simulation of cartilage model, the establishment of cartilage model does not consider the calcification layer and the following parts. According to the physiological structure and stress characteristics of human knee joint, the articular cartilage model considering the effect of calcification layer and subchondral bone under rolling load is established, which makes the model more close to the intact natural physiological structure of cartilage， as shown in Figure 4. The motion of knee joint is the relative motion of tibial plateau and distal femur, which is simplified as a geometric model of rigid sphere acting on planar cartilage. The stress-strain distribution, fluid flow (pore pressure, porosity, flow rate, etc.) of cartilage were analyzed by ABAQUS finite element software. Considering the movement characteristics of flexion and extension, internal and external rotation and internal and external rotation of cartilage, a three-dimensional finite element model of knee joint which can realize composite load during walking was established. To study the

compression, rolling, sliding or rotation of knee joint cartilage under combined load and its numerical analysis. The cartilage stress and its changes were analyzed. Considering the role of calcification layer and subchondral bone, the articular cartilage model is closer to the intact natural physiological structure of cartilage. The motion of knee joint is the relative motion of tibial plateau and distal femur, which is simplified as a geometric model of rigid sphere acting on planar cartilage. Considering the motion characteristics of knee joint flexion and extension, internal and external roll and internal and external rotation, a three-dimensional finite element model of knee joint which can realize walking composite load is established. To study the compression, rolling, sliding or rotation of knee joint cartilage under combined load and its numerical analysis.

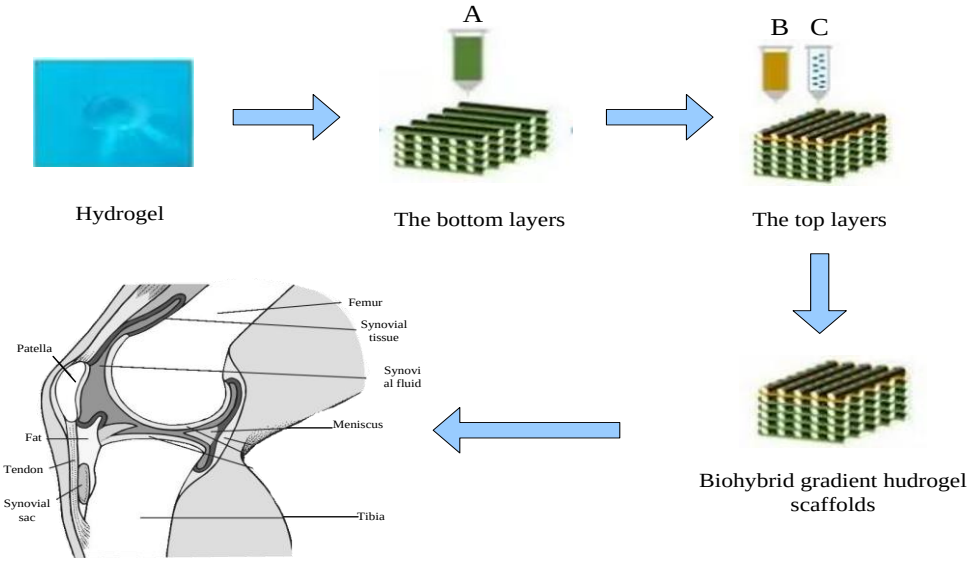

Figure 4. Three-dimensional finite element model of articular cartilage

The finite element method treats all structures as inert structures. It consists of a limited number of interconnected small mechanical units. The mechanical properties of the overall structure are combined with the mechanical properties of each unit. Therefore, the unit displacement can be written as:

$$\{\mathrm{d}\}^e = \{uvw\}^T = [N]\{q\}^e \quad (14)$$

The $[N] = [N_i IN_j IN_m IN_p I]$ in the formula is called the shape function matrix.

Physical equation of element stress matrix

$$\{\delta\} = [B]\{\varepsilon\} = [D][B]\{q\}^e \quad (15)$$

In the formula, [D] is the elastic matrix, and the elastic modulus E and Poisson's ratio D_Dd__[S] are the stress matrix.

From the principle of virtual displacement, the expression of the element stiffness matrix can be obtained as

$$[k]^e = \iiint_V [B]^T [D][B]\mathrm{dxdy}dz = [B]^T [D][B]V \quad (16)$$

After obtaining the stiffness matrix of each element, the total stiffness matrix of the structure is

obtained according to the integration method of the total stiffness matrix

$$[K]=\sum_{e}^{n_c}[k]^e \quad (17)$$

The total equivalent nodal load array generated by various load displacements is

$$\{R\}^{\mathrm{e}}=\{R\}^e_{p_c}+\{R\}^e_{P_x}+\{R\}^e_{P_V} \quad (18)$$

$\{R\}^e_{p_c}$, $\{R\}^e_{P_x}$ and $\{R\}^e_{P_V}$ in the formula are the equivalent nodal load generated after the transplantation of concentrated force, surface force and volume force. The nodal load array of the entire structure is

$$\{R\}=\sum_{e}^{\mathrm{n}_c}\{R\}^e \quad (19)$$

After eliminating the singularity of [K] by adding boundary conditions, the structural stiffness equation

$$\{R\}=[K]\{\mathrm{q}\} \quad (20)$$

The basis and rationality of modeling: on the femoral contact surface, the internal and external surfaces of the joint are approximately elliptical on the horizontal plane; on the anterior plane, the tibial plateau is approximately plane. According to traditional practice, the description of human knee joint movement is based on the assumption that the tibial plateau (in this case, part of cartilage) is completely fixed, and the femoral head rolls, slides, bends / stretches, rotates inward / outward and inward / outward. Therefore, in order to keep the tibial cartilage platform still, the model is simplified as a spherical femur moving in the tibial platform.

The traditional MRI sequence of articular cartilage can clearly show the structure and signal abnormalities of articular cartilage, but it can not show the changes of physiological components and biochemical structure of articular cartilage and the early injury morphology after acute exercise. T1rho imaging is based on the application of high frequency constant pulse RF to lock the effective field B1 in the horizontal direction when the magnetization vector is reversed to the horizontal direction. In the RF field and magnetic rotation framework, different tissues experience their own constant T1rho relaxation time, which avoids the spontaneous and disordered energy transfer of transverse relaxation between macromolecules and makes magnetization or spin order.

The principle of delayed dynamic contrast-enhanced imaging is similar to that of sodium spectrum imaging: glycosaminoglycan in proteoglycan is negatively charged and can adsorb GD dtpa2 + or Na +, indirectly reflecting the change of proteoglycan content. However, intravenous contrast agent is needed for dGEMRIC, and the scanning time is longer. At the same time, the time of image acquisition, registration and post-processing is also very long. Sodium spectrum imaging needs special space transmitting and receiving coils, which takes a long time. Therefore, dGEMRIC and Na spectrum imaging are difficult to be widely used in clinic.

2.3. OA and COMP

Comp is expressed in all joint structures, including ligaments, meniscus, tendons and synovium. In addition, comp can also be expressed in retinal and vascular smooth muscle cells. The biological

function of comp is not clear. Comp is an important component of hyaline cartilage. The C-terminal globular region of comp can connect type I, type II and type IX collagen. Comp may be involved in regulating the fiber structure and maintaining the integrity of collagen network.

After articular cartilage injury caused by various reasons, comp is first released to synovium and then into blood. Articular cartilage is a special structure composed of shallow, middle and deep parts. These regions are formed by regulating the phenotype of epiphyseal chondrocytes during bone growth and maturation. The arrangement of chondrocytes is complex and parallel to the articular surface. This arrangement of chondrocytes is known as the surface chondrocyte space tissue. Because of the continuous arrangement of chondrocytes in the horizontal direction, scso is highly dynamic. At the early stage of OA, chondrocytes of knee joint showed double row connection, and the density of surface cells increased, indicating chondrocyte proliferation. Cell clusters appear in cartilage and eventually develop into scattered cells with irregular arrangement. An important feature of early OA is the proliferation of superficial articular chondrocytes. At the same time, a large number of inflammatory factors participate in cell proliferation, ECM degradation and early inflammatory response.The specific treatment of OA patients is shown in Figure 5:

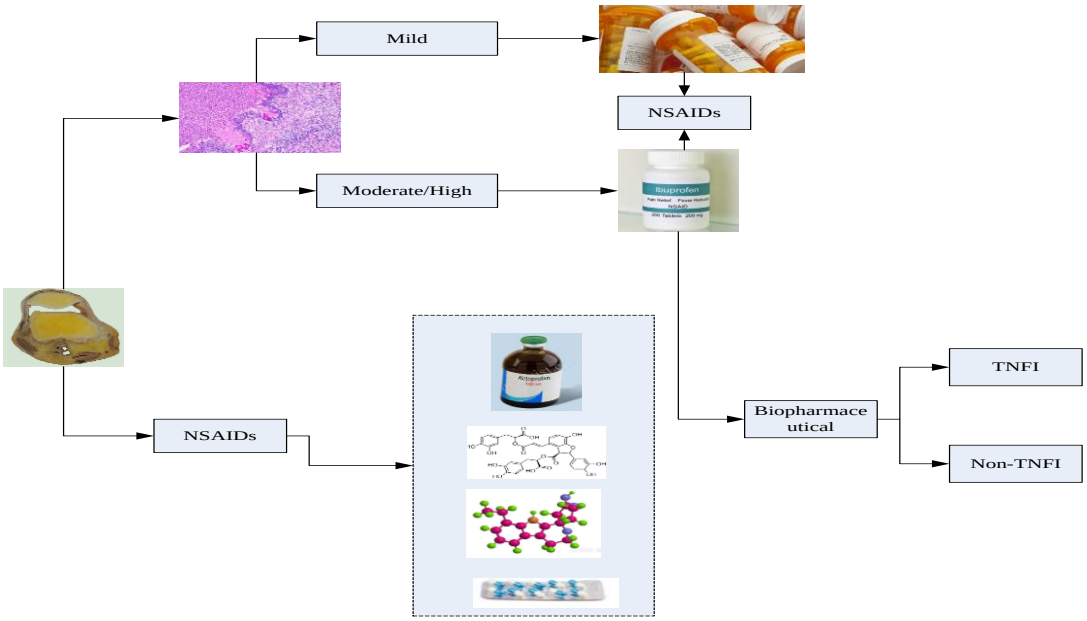

Figure 5. Specific treatment steps for OA patients

3. Research Subjects and Experimental Settings

3.1. Subjects and Environment

Thirty six healthy male clean Wistar rats, aged 16-18 weeks, were purchased from the animal room of experimental research center of a hospital. These animals are kept in separate cages, with three animals in each cage. They are fed with national standard rodent feed and are free to feed. The temperature of the animal room is nineteen to twenty-two degrees Celsius and the relative humidity is forty percent to sixty percent. Before the experiment, all the animals were not running on the treadmill and were not stimulated.

Experimental environment: carbon dioxide incubator; Odyssey dual color infrared laser scanning

system; flow cytometry; Olympus inverted microscope; Olympus vertical microscope; microporous ultra pure water system; autoclave; low speed centrifuge; Rabbit anti mouse comp monoclonal antibody; jx240 treadmill.

3.2. Experimental Setup

The specific image of the optical system of the micro-fluorescence microscope is shown in Figure 6. A microscope is an optical instrument composed of a single lens or multiple lenses. It is a sign that mankind has entered the atomic age, and is mainly used to amplify small objects visible to the naked eye on the instrument. Microscopes: optical microscopes and electron microscopes: optical microscopes were first developed by Jansen in the Netherlands in 1590. Today's optical microscopes can magnify objects 1600 times, with a minimum resolution of 1./2 of the wavelength. A typical household mechanical microscope has a cylinder length of 160 mm, which played an important role in the development of microscopy and microbiology. It is Levenhoek, a Dutch citizen. The miniature fluorescence microscope in this experiment is a new generation microscope developed on this basis, and it plays a very prominent role in this experiment.

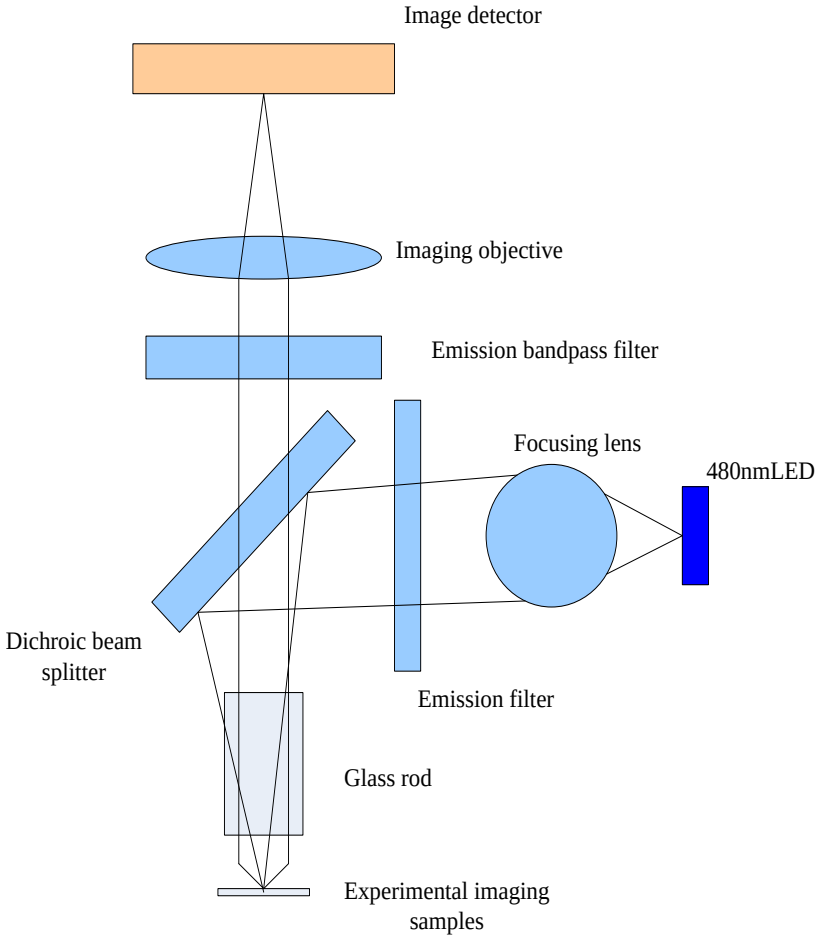

Figure 6. Schematic diagram of the optical path of a fluorescence microscope

The imaging system selects CycloneV series 5CSEMA5F31C6N FPGA chip, and configures NiosII soft-core processor as the control core based on it, and uses the corresponding interface

module and FPGA peripherals on the Avalon bus to complete the system functions shown in Figure 7.

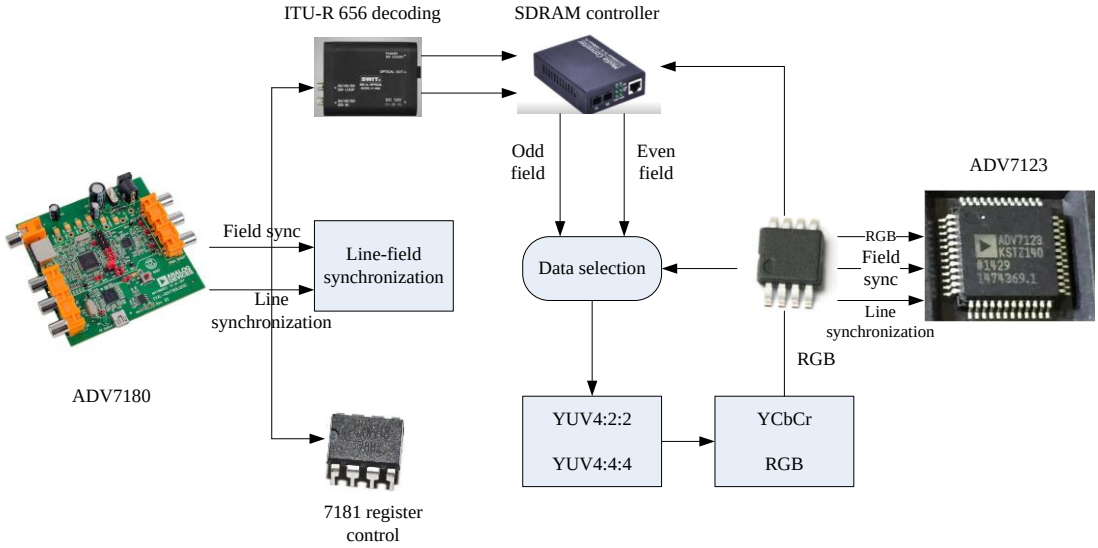

Figure 7. Schematic diagram of the hardware structure of the FPGA system

Control group A: ordinary cage; board group B: zero level board exercise for 15 days, one hour a day, one kilometer an hour; uphill group C: rats in the five degree slope of the running platform for exhaustive exercise. The specific program is as follows: on the first day, do ten groups of training, each group is 12 meters per minute; on the second day, do fifteen groups of training, each group is 15 meters per minute; on the third day, do twenty groups of training, each group is 15 meters per minute; on the fourth day, do thirty groups of training, each group is 18 meters per minute; on the fifth day, do thirty five groups of training, each group is 20 meters per minute; from the eighth day, rats are 12 meters per minute M. after ten warm-up runs, exercise for 50 minutes at the speed of 20 meters per minute. All tests are in accordance with the regulations on the use and protection of experimental animals. The score of synovium histology was t-test ($P < 0.05$).

4. Analysis of Research Results

4.1. Comparative Analysis of Comp Positive Cell Rate in each Group

There was no comp expression in the normal synovium of the control group. See Table 1 for the comparison of comp positive cell rate and synovium histological score in each group.

Table 1. Comparison of synovial histology score and COMP-positive cell rate among rats in each group

Group (N = 18)	Synovial histology score($\bar{x} \pm s$)	COMP positive cell rate ($\bar{x} \pm s$, %)
Quiet control group	1.71 ±0.23	6.83 ±2.25
Exhaustive exercise group	9.86 ±0.32	19.21 ±6.12

The results showed that the expression intensity of synovium comp in the exercise-induced exhaustion group was significantly higher than that in the control group ($P < 0.05$). The comparative analysis of comp positive cell rate and synovium histological score in each group is shown in Figure 8.

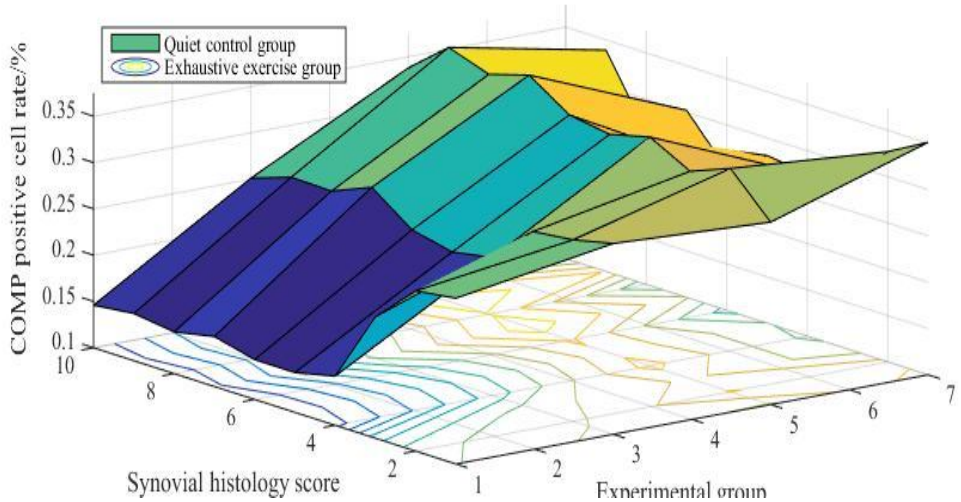

Figure 8. Comparative analysis of synovial histology score and COMP positive cell rate in each group of rats

It's hard for rats to run out every day. Most of the mechanical load is absorbed by the joint, which can lead to articular cartilage degeneration in a short time. And long-term exercise will lead to synovitis of knee joint in rats. Forty two days after exhaustive exercise, synovitis appeared, synovial cells increased, capillary hyperplasia and inflammatory cell infiltration. The histological score of synovitis in rats was higher than that in group A. When synovitis occurs, the synthesis of synovial cathepsin, matrix metalloproteinase, interleukin-1β and other cytokines increases. Synovium cathepsin, matrix metalloproteinase, interleukin-1β and other cytokines enter the articular cartilage through synovial fluid, causing joint damage, which leads to articular cartilage damage.

4.2. Analysis of the Mean Optical Density of MMP-1 Immunohistochemical Sections of Synovium in each Group

The average optical density of MMP-1 immunohistochemical sections of synovium in each group is shown in Table 2.

Table 2. The average optical density of MMP-1 immunohistochemical sections of rat synovial tissue in each group

Group	N	Mean	Standard deviation
A	10	0.0103	0.00335
B	10	0.0425	0.00887
C	10	0.0434	0.00586

The expression of MMP-1 in synovium of control group A was negative, and that in plate group (uphill group C) was positive. The expression of MMP-1 in group B and C was significantly higher than that in group A ($P < 0.05$), but there was no significant difference between group B and group C ($P > 0.05$). The average optical density analysis of MMP-1 immunohistochemical sections of synovium in each group is shown in Figure 9.

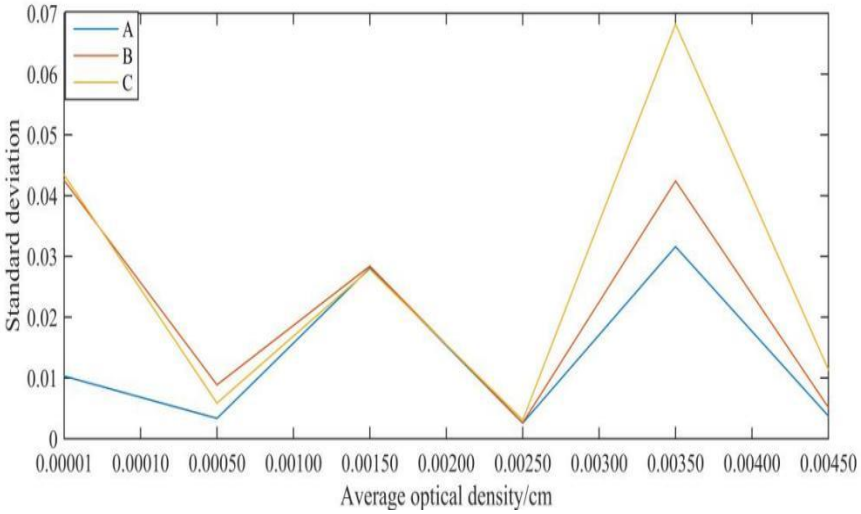

Figure 9. Analysis of the average optical density of MMP-1 immunohistochemical sections of rat synovial tissue in each group

MMP-1 belongs to fibroblast collagenase, which can degrade type II collagen in articular cartilage matrix directly. Type II collagen provides tension and bearing capacity for articular cartilage and forms collagen network structure of articular cartilage. It is a trimer composed of three identical α chains. There is a cleavage site of MMP-1 on the trimer, with which MMP-1 is combined to degrade into fragments, thus losing its original function. Type II collagen is the most abundant and characteristic collagen structure in articular cartilage. The degradation and destruction of type II collagen will directly damage the cartilage layer and cause joint diseases. The expression of MMP-1 was negative in synovium a of control group, positive in synovium B of plate group and positive in synovium C of uphill group, but there was no significant difference between the two groups. There was no significant difference in MMP-1 expression in synovium between B plate group and C plate group, which was related to the difference of synovium movement angle between B plate group and C plate group and the lag of expression in synovium.

On the diffusion tensor imaging of 100 cases of normal knee cartilage and 20 cases of grade 0, mild, moderate, and severe osteoarthritis groups, except for the anisotropy (FA) values of the grade 0 group and the normal group, each group There are significant differences between them ($P<0.05$). As shown in Table 3, different conditions of osteoarthritis were obtained in different degrees.

Table 3. Anisotropy measurements of different degrees of osteoarthritis

Grouping	n	Anisotropy value(FA)
Normal	120	0.612+0.203
0	40	0.604+0.081
Mild	40	0.541+0.06*#
Moderate	40	0.475+0.058*#&
Severe	40	0.416+0.052*#&

On the diffusion tensor stratified imaging of 100 normal knee cartilage and 20 grade 0, mild, moderate, and severe osteoarthritis groups, the anisotropy (FA) values of the surface, middle, and deep layers, except for the normal group In addition to the anisotropy (FA) value of each layer of the 0-level group, there are significant differences between each layer of each group ($P<0.05$). Table 4 shows the anisotropy of different levels of osteoarthritis (FA) value.

Table 4. Anisotropy values of different levels of osteoarthritis(FA)

Measuring part	Normal	0	Mild	Moderate	Severe
Surface	0.595+0.042	0.581+0.045	0.524+0.031	0.455+0.040	0.396+0.027
Middle	0.615+0.054	0.598+0.056	0.537+0.047	0.477+0.041	0.416+0.038
Deep	0.624+0.06	0.620+0.051	0.559+0.054	0.495+0.045	0.442+0.042

4.3. Enhancement of Autophagy Induced by IL-1β by High Concentration Insulin

FLS cells were induced with IL-1β of 10 ng / ml for 24 hours, and FLS, IL-6 and MMP9 were significantly increased by RT-PCR ($P < 0.05$). The analysis results of high concentration insulin on autophagy enhancement induced by IL-1 β are shown in Figure 10.

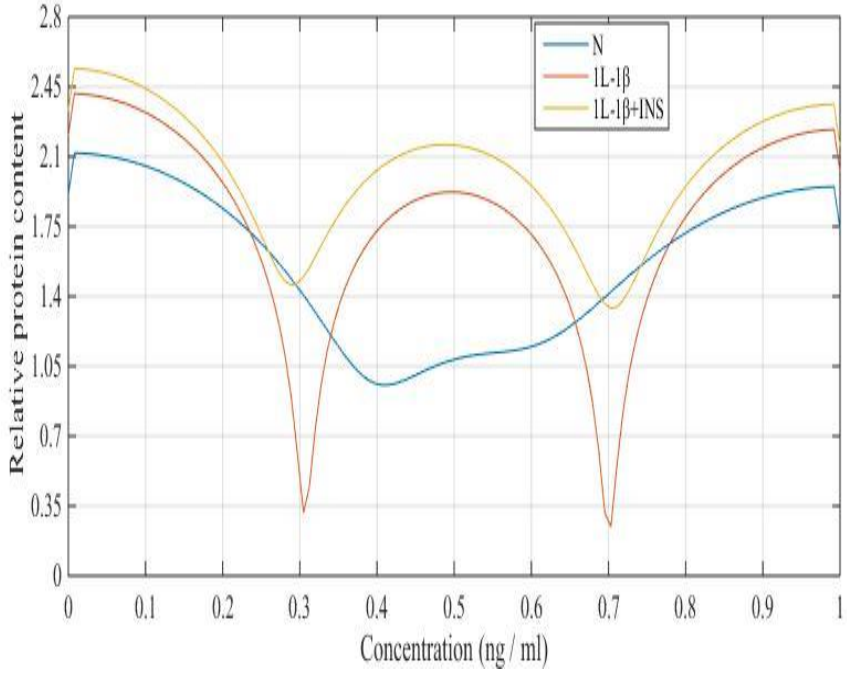

Figure 10. Analysis of the enhanced effect of high concentration insulin on IL-1β-induced autophagy

Western blotting was used to detect the effects of insulin (1000 nmol / L) and interleukin-1β (10 ng / ml) on autophagy related protein LC3 in synovial cells. In IL-1 group, FLS cells were induced by IL-1 β alone for 24 hours. In il-i + ins group, FLS cells were treated with insulin and IL-1β for 4 hours. Quantitative analysis of relative expression of LC3 protein in each group. Compared with the control group, the relative content of LC3-II in IL-1 + ins group was significantly higher than that in IL-1 group ($P < 0.05$). Conclusion high concentration insulin can inhibit autophagy induced by IL-1β.

4.4. Analysis of Finite Element Simulation Results of Moving Knee Joint Cartilage under Different Compression

After pure rolling loading, the percentage analysis results of Mises stress increase with the change of compression, as shown in Figure 11.

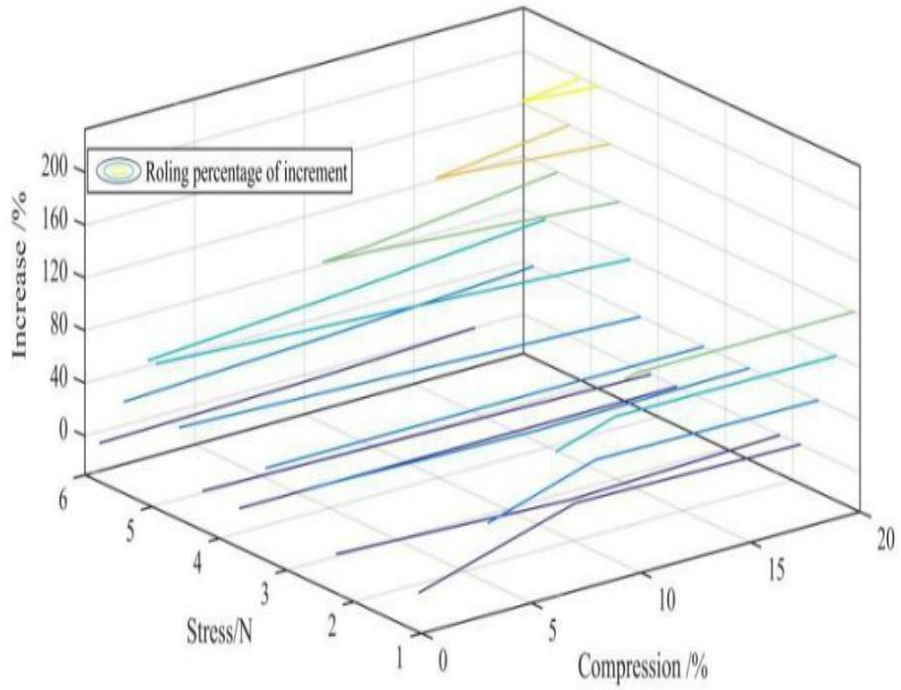

Figure 11. Percentage of Mises stress increase with rolling load under rolling load

Combined with the data in the figure, it can be seen that the stress of articular cartilage is continuous in the process of movement. After loading, the stress is rapidly concentrated. The larger the elastic modulus is, the smaller the deformation is, and the easier the cartilage is to deform, the easier it is to play a protective role. In the rolling process, when the compression of articular cartilage increases from 1% to 19%, the Mises stress increases with the increase of compression, and decreases gradually, and finally tends to be stable. The results show that the change of stress and strain of articular cartilage is not related to the movement state, but to the change of compression force, and the increment gradually becomes small and nonlinear to adapt to the surrounding mechanical environment.

5. Conclusion

High-intensity exercise will increase the thickness of the articular subchondral bone plate, reduce the porosity, harden the subchondral bone plate, and reduce the mechanical shock absorption ability, resulting in cartilage damage and possible strain of the synovial tissue; subchondral cancellous bone The looseness, a large number of plate-like structure changes, further increase the rigidity of the subchondral bone.

In terms of composition, due to the increase in bone remodeling, a large amount of new bone tissue appears, resulting in a decrease in bone mineralization. At the same time, it is found that the hardness of the articular subchondral bone increases, which may reduce the viscoelasticity of the articular subchondral bone and the absorption of impact force. The strain of the synovial tissue may be based on some daily sports activities, and irregular sports can lead to the strain of the synovial tissue. Moderate-intensity exercise has a certain shaping effect on cartilage. By increasing the

content of matrix in cartilage, it shows a protective effect on cartilage.

Comp plays an important role in the process of organizational remodeling, and it plays a key role in our daily life and movement. By measuring the amount of compound produced by different intensities of exercise, we can prevent the early degradation of articular cartilage and possibly avoid unnecessary strains of synovial tissue. The quantitative relationship and stability of serum COMP and arthritis development need to be further studied. There are many factors that affect the secretion and expression of comp, so it is necessary to correct the influencing factors so that comp can truly become an early diagnostic indicator of OA.

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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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