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Article

Biomechanics of metabolism and energy consumption of college female football players under mechanical force

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Abstract: Research on college women's football in the field of sports mainly focuses on macroscopic performance, with too much emphasis on the analysis of macroscopic sports performance and energy consumption, ignoring how mechanical forces indirectly affect athletes' metabolic processes through the behavior of cells and tissues. This paper takes college women's football players as the research object, combines biomechanical analysis at the cell and tissue level, and explores how mechanical forces indirectly change athletes' metabolic processes by affecting cell and tissue behavior. Through multi-scale analysis, this paper studies how to reveal the transmission and conversion mechanism of mechanical effects in organisms from the molecular, cellular to tissue levels, thereby affecting overall metabolism and energy consumption. Taking mechanical force as the starting point, combined with biomechanical analysis at the cell and tissue levels, this paper systematically explores how mechanical force indirectly changes the metabolic process of athletes by affecting cell and tissue behavior. The study adopted a multi-scale analysis framework, from the molecular, cellular to tissue levels, to reveal the transmission and conversion mechanism of mechanical action in the organism, affecting the overall metabolism and energy consumption. Cell mechanics experiments and metabolic modeling, a comprehensive metabolism and energy consumption analysis model was constructed by combining single-molecule mechanics experiments, verifying the key role of high-intensity mechanical force in improving energy consumption efficiency. The experimental results show that compared with normal training, training under mechanical force intervention can effectively improve the training effect of college women's football. This conclusion also has certain reference significance for other high-intensity athlete groups. Although the training intensity and physical response of different sports may vary, the same mechanical force intervention may also produce significant energy consumption effects in other types of sports. Under the high-intensity training mode, mechanical force intervention training can increase the maximum oxygen uptake, blood lactate concentration and muscle thickness by 6.2 percentage points, 33.9 percentage points and 7.2 percentage points respectively compared with training without mechanical force intervention. The research results provide support for cell and tissue level analysis in sports biomechanics research, as well as theoretical support for optimizing athlete training plans and sports rehabilitation, and reliable technical support for the integrated development of biomechanics and sports science.

Keywords: mechanical force; college female football; biomechanical research; multi-scale analysis; metabolism; energy expenditure

1. Introduction

With the continuous development of sports science and sports physiology, the evaluation of athletes' physical fitness and performance has gradually shifted from macroscopic sports performance to more detailed physiological mechanism research [1,2]. In high-intensity exercise, mechanical force affects athletes' sports

performance and may also indirectly change metabolic processes and energy consumption patterns through reactions at the cellular and tissue levels [3]. In high-intensity sports such as women's soccer, athletes' bodies are exposed to different forms of mechanical forces, and the effects of the transmission and conversion of these forces on cell function and metabolic responses have not been fully understood [4,5]. Traditional sports physiology research usually focuses on the macroscopic performance and energy expenditure of athletes. The studies show the changes in energy expenditure during exercise, but ignore the impact of mechanical effects on metabolism at the cellular and tissue levels [6,7]. The rapid development of cell mechanics and multiscale modeling techniques in biomechanics has provided a new perspective for understanding how mechanical forces affect athletes' metabolic processes through mechanical responses at the molecular, cellular and tissue levels.

Biomechanical studies have shown that mechanical force can have a profound impact on metabolic processes by changing cell structure, regulating intracellular and extracellular signaling pathways, affecting gene expression and protein synthesis, etc. [8]. When muscle cells are subjected to mechanical forces such as stretching and shearing, their cytoskeleton can be reorganized, which can trigger a series of biochemical reactions, helping cells to adapt to changes in the external environment and regulate intracellular energy metabolism pathways [9,10]. The interaction between cells and the extracellular matrix (ECM), the mechanical signal transmission between cells, and the viscoelastic characteristics of tissues can all change the overall energy consumption pattern of athletes under mechanical stimulation [11,12]. In recent years, there has been an increasing number of studies on the mechanical response and metabolic regulation of hepatocytes under different exercise intensities. Mechanical force not only has a significant impact on bone metabolism, but also plays an important regulatory role in the metabolic processes of tissues such as the liver. Studies have shown that during high-intensity interval training, the stress response of hepatocytes leads to significant changes in the rate of glycogen synthesis, and the fatty acid metabolic activity in the liver is also adjusted accordingly [13]. These cross-level studies provide us with a new perspective to understand the complexity of the relationship between mechanics and metabolism, and provide important theoretical support for this study. Although there have been some studies on the relationship between mechanics and metabolism, these studies are often limited to a single level of discussion and lack a comprehensive analysis of mechanical responses from the molecular, cellular to tissue levels. In the study of female football players, how to effectively couple cell mechanics, tissue mechanics and metabolic responses has not yet formed a systematic theoretical framework. Traditional research methods rely more on observational experiments and the measurement of single physiological indicators, but lack a deep understanding of the changes in cell behavior under mechanical action.

This paper aims to explore how mechanical forces affect the behavior of cells and tissues through multi-scale effects from the molecular, cellular to tissue levels, and change the metabolic process and energy consumption pattern of female football players from the perspective of biomechanics, combined with mechanical analysis at the cell and tissue levels. Existing studies are limited to single-scale or simple

mechanical action analysis, ignoring the transmission and coupling of mechanical effects at different scales. This paper adopts a multi-scale analysis framework, combined with single-molecule mechanics experiments, cell mechanics experiments and metabolic analysis, in the molecular dynamics simulation, shear force, tensile force, etc. of different strengths are applied to ensure the diversity and reliability of the experimental results. A research model of coupling between mechanical action and metabolism is constructed to explore the impact of mechanical force on athletes' metabolic processes from the molecular to the tissue level. Through this study, it is expected to provide a new theoretical basis for the personalized training and performance optimization of female football players, and provide an innovative perspective for the development of sports physiology and sports rehabilitation.

2. Related works

In sports training and exercise physiology, studying athletes' metabolism and energy expenditure has become an important direction for optimizing sports performance and improving training effects [14,15]. Previous studies have explored the role of mechanics in cell function and metabolic responses, especially how mechanical force affects cell morphology, gene expression, and protein synthesis [16]. Carneiro et al. [17] found that bone tissue is extremely sensitive to mechanical stress stimulation. Mechanical stress is closely related to the differentiation and formation of osteoclasts and osteoblasts and their bone resorption function, and has an important impact on the bone microenvironment and bone metabolism. Cieri et al. [18] used experimental and clinical data to understand the regulatory mechanism of mechanical force controlling cell death and found that endothelial cell apoptosis can be enhanced, reduced, or reversed with changes in shear stress patterns. Although current research has confirmed that mechanical force has a significant effect on cell morphology and cell regulation, there is still a lack of research on the role of mechanical force in the context of exercise training. The specific regulatory effects of mechanical forces of different intensities and frequencies on overall energy metabolism in a dynamic motion environment are still unclear.

Most existing studies focus on a single level of cells or tissues, and have not yet systematically studied the comprehensive effects of mechanical forces at multiple scales such as molecules, cells, and tissues. The specific mechanism of mechanics in metabolic regulation is still unclear. There is a lack of cross-scale dynamic analysis methods to reveal how mechanics is transmitted from the molecular level to the tissue level and then regulates energy consumption. Existing research has failed to effectively resolve the deep relationship between mechanics and metabolism. The reason for this limitation is the bottleneck in data coupling and model integration. The lack of theoretical framework makes it difficult to effectively implement cross-scale dynamic analysis. Existing research has failed to effectively address the deep relationship between mechanics and metabolism. In order to solve this problem, multi-scale research methods have gradually been introduced into mechanobiology and metabolic research in recent years [19,20]. Combining molecular dynamics simulation, cell mechanics experiments and tissue mechanics models, these studies revealed the transmission and conversion mechanism of mechanical forces in the

body. They studied how mechanics affects protein conformation through molecular dynamics simulation [21] and explored the response of cells to mechanical stimulation through cell mechanics experiments [22] and analyzed the role of mechanics on a larger scale through tissue mechanics models [23]. Another team used patch clamp technology combined with induced nano-electrospray technology [24] to achieve mass spectrometry analysis of single neuronal cells and discovered a new neuro-metabolite molecule, urocanic acid, in the brain. Although these methods can reveal the effects of mechanical forces on metabolism to a certain extent, they still have some shortcomings. There is a lack of a comprehensive framework for effectively coupling these mechanical data at different scales, and the specific effects of different mechanical actions on metabolic pathways have not been fully considered. This multi-scale mechanical analysis framework integrates mechanical data from molecules, cells to tissues, and explores their role in metabolic regulation to address the deficiencies in existing research.

3. Methods

3.1. Construction of a multi-scale analysis framework

In order to comprehensively explore the mechanism of mechanical force in the metabolism and energy consumption of college female football players, this paper constructs a multi-scale analysis framework from molecules, cells to tissues. Through molecular dynamics simulation, cell mechanics experiments and tissue mechanics models, this framework gradually analyzes how mechanical force is transmitted from the molecular level, cell level to the tissue level, and ultimately regulates energy metabolism and consumption.

3.1.1. Molecular dynamics simulation model

At the first level of the multiscale framework, molecular dynamics simulation is used to study the effects of mechanical forces on the conformation and interaction of biomolecules [25,26]. Molecular dynamics simulation uses numerical methods to solve the position, velocity and force interaction of particles in a molecular system, thereby revealing how molecules undergo structural changes under mechanical action. The motion of a molecular system can be described by the classic Newtonian motion Equation (1):

$$m_i \frac{d^2 r_i}{dt^2} = F_i(r_i) \tag{1}$$

m_i is the mass of the ith atom, r_i is the position vector of the ith atom, and F_i is the total force acting on the ith atom. The force F_i is composed of various interactions and can be calculated using a force field model. The force field U is a combination of the Lennard-Jones force field and the Coulomb force field:

$$U = \sum_{i<j} \left(4\epsilon \left[\left(\frac{\sigma}{r_{ij}} \right)^{12} - \left(\frac{\sigma}{r_{ij}} \right)^{6} \right] + \frac{q_i q_j}{r_{ij}} \right) \tag{2}$$

Among them, ϵ is the potential energy depth, σ is the effective diameter between molecules, r_{ij} is the distance between molecules, q_i and q_j are the

charges of atoms i and j respectively.

On this basis, different tensile forces, shear forces or twisting forces are applied to observe the changes in molecular conformation and analyze the effects of mechanical action on the stability, structural rigidity and function of biomolecules. Through simulation results, the specific effects of mechanics on molecular functions such as protein folding and gene expression regulation are quantified, providing necessary mechanical parameters for experiments at the cell level and tissue level.

3.1.2. Cell mechanics model

At the cell level, the finite element analysis (FEA) method is used to construct a cell mechanics model to simulate the response of cells to different mechanical stimuli [27,28]. The deformation and mechanical behavior of cells depend on the elastic properties of the cytoskeleton and the support of the ECM. The cell is modeled as a composite material consisting of three parts: cell membrane, cytoskeleton and extracellular matrix.

The elastic behavior of cells is described by linear or nonlinear constitutive relations. The deformation of cells under stress follows the elastic mechanics equations, and the mechanical behavior of cells is characterized by the stress-strain relationship:

$$\sigma = E \cdot \varepsilon \tag{3}$$

Among them, σ is stress, E is the Young's modulus of the cell, and ε is strain. In order to describe the nonlinear response of the cytoskeleton and cell membrane, an anisotropic and time-dependent material constitutive model is used:

$$\sigma(t) = E_{eff} \cdot \varepsilon(t) + \eta \cdot \frac{d\varepsilon(t)}{dt} \tag{4}$$

Among them, η is the damping coefficient, which represents the viscoelastic behavior of the cell membrane, E_{eff} is the effective Young's modulus, and $\varepsilon(t)$ is the strain at time *t*.

The response of cells to mechanical stimulation is not only mechanical deformation, but also changes in signal transduction. The response of cells to mechanical forces is described by the intracellular signaling pathway model, which is activated by the interaction between receptors and the cytoskeleton. Mechanical signals are transmitted to the cytoskeleton through integrins, thereby affecting gene expression and protein synthesis.

To further simulate the process, a numerical method based on the finite element method (FEM) was used to analyze the deformation process and signal transduction changes of cells when different types of mechanical stress were applied [29]. By tracking the changes in intracellular Ca^{2+} concentration, RhoA activity and other indicators, the regulatory effect of mechanics on cell function and metabolic pathways was studied.

3.1.3. Tissue mechanics model

At the tissue level, the continuum mechanics method is used to model the response of soft tissues such as muscles and ligaments under different mechanical effects [30,31]. The mechanical characteristics of soft tissues are usually nonlinear

and anisotropic, and a finite element model based on the Cauchy stress tensor is used.

The stress-strain relationship of the tissue is described by a nonlinear material model. The strain energy density function is set to *W*, and the stress σ is calculated by the derivative of the strain energy density function:

$$\sigma = \frac{\partial \mathrm{W}}{\partial \mathrm{\mathcal{E}}} \tag{5}$$

For anisotropic materials, *W* takes the generalized form:

$$W = \sum_{i=1}^{n} C_i(\mathcal{E}_i - 1)^2 \tag{6}$$

Among them, $\mathcal{E}_i$ is the strain in different directions, C_i is the material constant, and n is the order of the model.

The deformation of muscle tissue after being subjected to force is described by Equation (7):

$$F = JF_{in} = \frac{\partial r}{\partial X} \tag{7}$$

Among them, *F* is the deformation gradient, *J* is the volume deformation rate, and F_{in} is the deformation gradient under the original configuration.

In the simulation of the tissue level, the arrangement of muscle fibers, the anisotropy of tissues, and the characteristics of muscle contraction are taken into account. Through the tissue mechanics model, the mechanical behavior of tissues such as muscles and ligaments during exercise is simulated, and how mechanical force affects the metabolism and energy consumption of tissues is further studied.

This paper adopts a hierarchical simulation method and a data conversion algorithm between different scales to achieve multi-scale coupling from molecules to tissues. The mechanical parameters provided by the molecular dynamics simulation results are transferred to the cell model as input through interpolation and mapping to simulate the behavior of cells in response to different mechanical stimuli. The mechanical information in the cell mechanical model is transferred as parameters to the tissue model as input through the finite element analysis method to simulate the response under the action of overall stress. The coupling of multi-scale models enables mechanical analysis at different levels to verify each other, and the model is tuned through a global optimization algorithm to ensure the true performance of mechanical effects.

3.2. Experiments at the cell and molecular level

3.2.1. Single molecule mechanics experiments

Single molecule mechanics experiments use a variety of mechanical stimuli such as stretching, torsion and shearing to analyze changes in molecular conformation and mechanical properties [32,33]. The experiment uses atomic force microscopy (AFM) and optical tweezers technology to exert controlled mechanical force at the molecular level and observe molecular reactions and mechanical behaviors. Atomic force microscopy and optical tweezers were chosen because AFM

can accurately measure the changes in the morphology and rigidity of molecules under tensile or compressive forces, and is suitable for the study of molecular conformations such as proteins. Optical tweezers technology uses laser beams to apply precise tension or torsion, which can precisely intervene at the molecular or single-cell level and measure the response of molecules to different mechanical stimuli. In the experiment, the accuracy and repeatability of the experimental results are ensured by controlling the probe scanning speed, laser power and the amount of force applied. Changes in external environmental factors such as temperature and humidity will also affect the experimental results, so the experiment needs to be conducted under controlled conditions.

The experiment detected the response of the molecule under different loads and studied the changes in protein structural stiffness and its relationship with function by applying tensile force. The applied force is calculated as shown in Equation (8):

$$F = k\Delta x \tag{8}$$

k is the force constant and Δx is the displacement of molecular elongation.

Change the rate and amplitude of stretching and calculate the energy change of the molecular structure. Use Equation (9) to analyze the effect of mechanical response on molecular folding and conformational change:

$$E_{strain} = \frac{1}{2}k\Delta x^2 \tag{9}$$

Equation (9) shows that the strain energy caused by the tensile force is proportional to the displacement of the molecular deformation. By comparing the energy changes under different mechanical conditions, mechanical force plays a regulatory role in the molecular folding process, thereby affecting its function and reaction speed.

3.2.2. Cell mechanics experiment

Cell mechanics experiments aim to study how external mechanical stimulation affects the metabolic process by affecting cell morphology, movement and growth. To this end, methods such as cell indentation test, single cell tensile test and cell migration test [34] were used. The cell indentation test applies a known force to observe the changes in cell surface morphology and measure the stiffness of the cell membrane and the elasticity of the cytoskeleton [35]. In the experiment, optical tweezers were used to apply tensile or compressive forces of different magnitudes. The magnitude of the force and the duration of application are the key factors in the study. The applied force is calculated by Equation (10):

$$\sigma = \frac{F}{A} \tag{10}$$

F is the applied force and A is the contact area of the cell surface.

The stress value of the cell after being subjected to force is obtained by Equation (10), and the mechanical response of the cell membrane and cytoskeleton is further analyzed. The mechanical properties of the cell under different mechanical environments are obtained through the changes in cell morphology, including cell elastic modulus, deformation capacity and threshold of plastic deformation.

The single-cell stretching experiment uses a microfluidic chip to arrange cells in a single row, applies a stretching force, and observes their morphological changes. As the stretching force increases, the longitudinal strain and volume morphology of the cell change significantly. The deformation speed of the cell is calculated by the strain rate:

$$\dot{\varepsilon} = \frac{\Delta L}{L_0 \Delta t} \tag{11}$$

Among them, ΔL is the cell deformation, L_0 is the initial length, and Δt is the deformation time interval.

As the stretching rate increases, the strain response of the cell increases, the tensile stress of the cytoskeleton also increases, and the signal transduction pathway in the cell is activated, which can further affect the growth, differentiation and metabolic activity of the cell.

The cell migration experiment records the cell migration process by dynamically observing the movement behavior of cells under the action of mechanical forces and combining it with real-time imaging technology [36]. The "wound healing experiment" model can be used, where cells migrate within the wound area to fill the gap. Measuring cell migration speed:

$$v = \frac{\Delta x}{\Delta t} \tag{12}$$

Among them, Δx is the displacement of cell migration, and Δt is the time interval.

The influence of different mechanical stimuli on cell migration behavior is analyzed by cell migration speed. Experiments show that lower-intensity tensile force can promote cell migration, while excessively high-intensity force can inhibit cell migration.

3.3. Energy consumption and metabolic analysis

This section quantitatively analyzes the metabolic processes and energy consumption of college female football players under mechanical forces. The experiment uses a variety of technical means, combined with biomechanical experimental data, to evaluate the metabolic responses of athletes under different mechanical environments, and to study how mechanical stimulation regulates intracellular metabolic pathways and their effects on energy consumption. The analysis steps include monitoring metabolic markers, measuring oxygen consumption and carbon dioxide emissions, and further estimating energy consumption through mathematical modeling.

Indirect calorimetry is used to monitor oxygen consumption (VO_2) and carbon dioxide emission (VCO_2) in real time to accurately assess the energy consumption of athletes under mechanical force [37,38]. By measuring the changes in VO_2 and VCO_2, combined with the basic theory of Krebs cycle and oxidative metabolism, the energy consumption per unit time is calculated using Equation (13):

$$K = 3.9 \times \mathrm{VO}_2 + 1.1 \times \mathrm{VCO}_2 \tag{13}$$

In the experiment, the energy consumption of athletes during standardized exercise was dynamically monitored, and the changes under different mechanical forces were recorded.

By collecting blood samples from athletes, the changes in metabolic markers such as lactate, glucose and fatty acids were quantitatively analyzed, and these markers were used to measure the activity of exercise metabolic pathways. The experiment used high performance liquid chromatography and gas chromatography-mass spectrometry to accurately quantify metabolites, and the analysis of blood samples revealed metabolic changes in athletes under different mechanical stimuli. Mechanical force can lead to an increase in lactate concentration, indicating enhanced muscle metabolic activity, and this effect is more pronounced under high-intensity exercise. The glucose metabolic pathway and the ratio of fatty acid utilization are also regulated by mechanical force, further revealing the mechanism by which mechanical force indirectly affects overall energy consumption by affecting the intracellular metabolic network.

In order to better understand the regulatory effect of mechanical force on the metabolic process, the metabolic network modeling method is used to mathematically model the metabolic pathways of athletes under different mechanical environments. Based on metabolic flux analysis (MFA), a dynamic model including major metabolic pathways such as glycolysis, tricarboxylic acid cycle (TCA cycle) and fatty acid oxidation was constructed [39]. The basic equation of the model is:

$$\sum_i (Flux_i \cdot C_i) = 0 \tag{14}$$

Among them, $Flux_i$ represents the metabolic flow rate of each metabolic pathway, and C_i is the coefficient of the reaction. According to the metabolite concentration and reaction rate obtained in the experiment, the flow rate of each metabolic pathway is calculated to obtain the metabolic flow distribution under different mechanical conditions. **Figure 1** is the basic structure diagram of MFA:

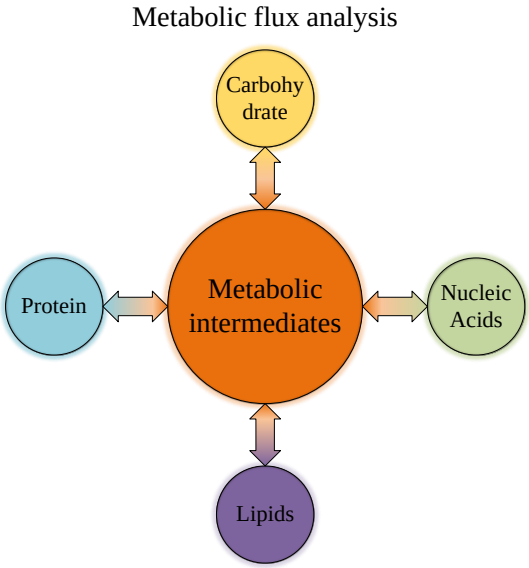

Figure 1. MFA structure diagram.

Based on the above metabolic marker analysis and metabolic pathway modeling results, a comprehensive energy expenditure model is further established to predict the energy expenditure of athletes under different mechanical force stimulation. The model is based on the relationship between metabolic rate and exercise intensity, and takes into account the effect of mechanical stimulation on energy consumption. The basic form of the model is:

$$E(t) = E_0 + \Delta E_{\text{mechanical}}(t) + \Delta E_{\text{metabolic}}(t) \tag{15}$$

Among them, $E(t)$ represents the energy consumption at any time point *t*, E_0 is the basic energy consumption in the resting state, $\Delta E_{\text{mechanical}}(t)$ is the change in energy consumption caused by mechanical force, and $\Delta E_{\text{metabolic}}(t)$ is the change in energy consumption caused by changes in metabolic pathways. The model can simulate and predict the metabolic characteristics and energy consumption trends of athletes under different exercise loads under different mechanical forces.

3.4. Coupling of model and experiment

Combining experimental data with mathematical models, this paper explores the metabolism and energy consumption process of college female football players under mechanical forces through a combination of multi-scale simulation and experimental verification. In order to accurately predict and verify metabolic reactions under mechanical influence, a combination of multiple experimental methods and models is used, and the model optimization is driven by experimental data to achieve a more refined biomechanical analysis.

A metabolic network model based on MFA was constructed, and metabolic indicators such as experimental data oxygen consumption, carbon dioxide emission, and lactate concentration were accurately introduced into the model. The gas exchange data and metabolite concentrations obtained by experimental measurement were used as input variables to fit the reaction rate constants in the metabolic model. The model equation is shown in Equation (16):

$$\sum_{i=1}^{n} v_i \cdot S_{ij} = 0 \tag{16}$$

Among them, v_i is the flow rate of the metabolic reaction, S_{ij} is the reaction coefficient matrix, and n is the number of reaction pathways.

The experimental part measures the oxygen consumption rate (VO_2) and carbon dioxide emission (VCO_2) of athletes under mechanical force, and obtains real-time energy consumption data through indirect calorimetry. The data provides key input for the metabolic model, helps optimize the initial parameters of the metabolic flow model, and ensures that the simulation results are closer to the actual biological reaction. By comparing the error between the experimental data and the model prediction results, the least squares method is used to iteratively adjust the model parameters to achieve a high degree of consistency between the model and the experimental data.

Further multi-scale simulations are carried out to combine experimental data at the molecular, cellular and tissue levels to verify the accuracy and predictive ability

of the model. At the molecular level, molecular dynamics simulation is used to simulate the effect of mechanical force on the cell membrane and the molecular structure within the cell, and calculate the mechanical response at the molecular level under the action of external force. Experimental data at the cellular level, such as changes in cell morphology and intracellular calcium ion concentration, are input into the cell biomechanics model, which further provides feedback for the energy consumption model at the tissue level.

By comparing simulation results at different levels with experimental data, the mechanical transmission mechanism between cells, tissues and molecules is adjusted to ensure the accuracy of the entire multi-scale framework. Through the iterative optimization method driven by experimental data, the prediction accuracy of the model for actual biomechanical behavior can be gradually improved to ensure that the simulation results can reflect the real process of athletes' metabolic response under different exercise loads and mechanical forces.

4. Experimental design and data collection

4.1. Experimental design

The experimental design of this study aims to explore how mechanical force affects the metabolic process and energy consumption of college female football players, and further reveal how these changes are transmitted and converted at the cellular and tissue levels. The experimental design includes multiple levels, covering exercise load control, mechanical force application scheme, experimental group and control group settings, and the collection and analysis of metabolic and physiological data.

This study selected college female football players as the research subjects, mainly considering the following advantages. Female football players have relatively consistent age, weight, height, and athletic ability, which provides a relatively balanced sample for the study and helps to eliminate the impact of individual differences on the experimental results. Selecting college female football players as the research subjects can fully reflect the metabolism and energy consumption mechanism of the group under the background of high-intensity exercise. Female football players will undergo systematic training, the participants have a high level of health, and have strong physiological adaptability, which can provide stable data support in training and experiments. Considering the statistical significance and the control of experimental errors, 30 athletes were selected as experimental subjects. This sample size can effectively ensure the representativeness and reliability of the experimental results.

The core of the experiment is the effect of mechanical force on athletes' metabolism under different exercise loads, and two training programs with different exercise intensities were designed for this purpose. One is a high-intensity training program, which uses intermittent running training for 10 min, with a running and rest time ratio of 1:1, during which mechanical force is applied. The purpose of high-intensity training is to simulate a situation where a high metabolic load is generated in a short period of time. During training, a mechanical force-applying device is used to apply a cyclical tensile force to the athlete's lower limbs with an

amplitude of 10–15 N and a frequency of 1 Hz to simulate the muscle load generated during actual exercise. The other is a low-intensity training program, which uses stable running training for 40 min, maintaining medium-low intensity (50%–60% of maximum heart rate). The low-intensity training program is used to simulate metabolic consumption under low-intensity exercise and is a control group with the high-intensity group.

In the experiment, a hydraulic device was used to apply mechanical force to ensure the accuracy and repeatability of the load. The hydraulic device applies specific force to the athlete's lower limb muscle groups through a mechanical arm. The force application mode is periodic stretching. In each exercise cycle, the muscles are stretched and relaxed in a short period of time, simulating the actual force state in training.

The experimental group setting is shown in **Table 1**:

Table 1. Experimental group setting table.

Groups	Training intensity	Mechanical force	Number of subjects	Training period	Number of training sessions per week
Experimental group 1	High-intensity training	Yes	10	16 weeks	3 times/week
Experimental group 2	High-intensity training	No	10	16 weeks	3 times/week
Experimental group 3	Low-intensity training	Yes	10	16 weeks	3 times/week
Experimental group 4	Low-intensity training	No	10	16 weeks	3 times/week
Control group	Stationary state	No	10	16 weeks	0 times/week

The basic physical conditions of the athletes participating in the experiment in **Table 1** were similar. The experiment was divided into five groups, each of which contained ten college female football players. The five groups were a high-intensity training group with or without mechanical force, a low-intensity training group with or without mechanical force, and a control group in a static state without mechanical force. The training cycle of each group was 16 weeks, and except for the control group, the other groups trained three times a week. The training period is 16 weeks, which can fully demonstrate the physical adaptation, metabolic level changes and potential physical risks caused by long-term mechanical force intervention. Metabolic and physical fitness tests are conducted every 4 weeks to test athletes' VO_2max, lactic acid concentration, muscle thickness and other levels to observe dynamic changes and adjust training content and intensity in time to ensure the reliability of experimental results and the safety of athletes.

4.2. Evaluation indicators

The data collection in the study was to evaluate the impact of mechanical force on the metabolism and energy consumption of college female football players, and to quantitatively analyze the relationship between exercise load and physiological response. Data collection covers the collection of basic physiological data, as well as biomechanical analysis at the cellular and molecular levels.

This study designed a physiological data collection plan for each subject, including physiological indicators such as heart rate, oxygen consumption, and lactate concentration. The time nodes for data collection include before training,

during training, after training, and the recovery phase within 24 h. To ensure the accuracy and validity of the data, all collected data can be statistically analyzed.

This study focuses on the impact of mechanical force on the metabolism and energy consumption of college female football players, and designs a series of evaluation indicators covering physiological, biochemical and sports mechanical parameters. All indicators are comprehensively monitored from the molecular, cellular to the overall metabolic level to ensure the systematicity and reliability of the data.

Metabolic consumption indicators include maximum oxygen uptake and total energy consumption. Maximum oxygen uptake ($\mathrm{VO_2}$) is as shown in Equation (17):

$$\mathrm{VO_2max} = \frac{\dot{V}_O \cdot (\mathrm{FiO_2} - \mathrm{FeO_2})}{G} \tag{17}$$

Among them, $\dot{V}_O$ represents the minute ventilation, that is, the total amount of gas inhaled per minute. $\mathrm{FiO_2}$ represents the volume fraction of oxygen in the inhaled gas. $\mathrm{FeO_2}$ represents the volume fraction of oxygen in the exhaled gas. G represents the athlete's weight.

Energy consumption is based on the Weir equation, as shown in Equation (18):

$$\mathrm{EE} = \left(3.9 \cdot \dot{\mathrm{V}}_{\mathrm{O_2}} + 1.1 \cdot \dot{\mathrm{V}}_{\mathrm{CO_2}}\right) \cdot \mathrm{Time} \tag{18}$$

Among them, EE represents energy expenditure. $\dot{\mathrm{V}}_{\mathrm{O_2}}$ represents the amount of oxygen consumed per minute. $\dot{\mathrm{V}}_{\mathrm{CO_2}}$ represents the amount of carbon dioxide exhaled per minute. Time represents the duration of exercise.

The relationship between heart rate and energy expenditure is corrected by the metabolic equation:

$$\mathrm{EE} = a \cdot \mathrm{HR} + b \tag{19}$$

Among them, HR represents heart rate. a and b represent the calibrated individual correction coefficients.

The biochemical index is the change in blood lactate concentration. The change in lactate concentration before and after training is used to analyze the adaptation of aerobic and anaerobic metabolism during exercise.

The sports mechanics index uses muscle strength assessment, and the torque data is collected using an isokinetic muscle tester. The equation for calculating the maximum muscle force output is as shown in Equation (20):

$$F_{\max} = \frac{T}{r} \tag{20}$$

$F_{\max}$ is the maximum force generated by the muscle. T is the maximum torque measured by the tester. r is the length of the knee joint force arm.

Gait parameters are recorded in real time by treadmill sensors.

Cell metabolic activity is calculated based on absorbance (optical density value), which is obtained by the colorimetric reaction of the MTS kit. The changes in muscle tissue thickness were measured by ultrasonic muscle imaging technology.

4.3. Experimental results and discussion

This study conducted a quantitative analysis of the metabolism and energy consumption of college female football players under different training intensities under mechanical force through multiple groups of experiments. **Figure 2** shows the changes in maximum oxygen uptake before and after training:

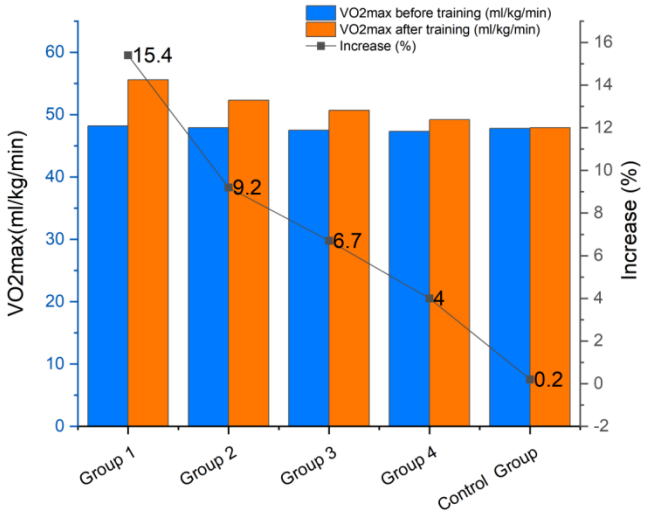

Figure 2. Changes in maximum oxygen uptake before and after training.

Figure 2 shows that the VO_2max of different experimental groups increased to varying degrees after training, while the increase in the control group was negligible (0.20%). This shows that mechanical force has a positive effect on improving athletes' maximum oxygen uptake, regardless of whether it is high-intensity or low-intensity training. The increase in experimental group 1 was the highest, indicating that the combined effect of high-intensity training and mechanical force has the most significant effect on improving athletes' metabolic capacity. Mechanical force produced a synergistic effect on VO_2max by enhancing muscle oxygen utilization efficiency and improving blood circulation. Compared with experimental group 1, experimental group 2 also adopted high-intensity training, but due to the lack of mechanical force intervention, the increase in VO_2max was reduced by about 6.2 percentage points. This indicates that there are certain limitations in improving metabolic capacity only through high-intensity training, and the role of mechanical force cannot be ignored. The improvement of low-intensity training combined with mechanical force is more obvious, but significantly lower than that of the high-intensity training group. This indicates that mechanical force can make up for the lack of training intensity to a certain extent in low-intensity training, but the effect is relatively limited. The improvement in Experimental Group 4 was the lowest among all experimental groups, further proving that low-intensity training itself has limited effect on promoting VO_2max, especially without mechanical assistance. The control group did not undergo any training intervention and had almost no change in VO_2max. This suggests that in a static state, the metabolic capacity of athletes is maintained at a relatively stable level, further highlighting the importance of training and mechanical intervention. In both high-intensity and low-intensity training, mechanical force significantly increased the magnitude of

VO_2max improvement, especially in high-intensity training.

Figure 3 shows the changes in blood lactate concentration of athletes before and after training:

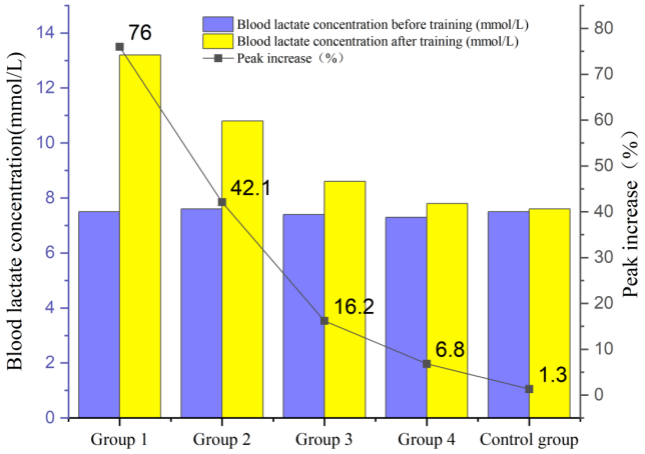

Figure 3. Changes in blood lactate concentration before and after training.

Figure 3 shows that the blood lactate concentration of each group increased after training, but the peak increase in different groups was significantly different. Among them, the peak increase in blood lactate concentration of high-intensity training combined with mechanical force (experimental group 1) was the largest, while the control group had almost no significant change. This indicates that both mechanical force and training intensity have an important impact on post-exercise lactate metabolism. The increase in blood lactate concentration in experimental group 1 was the highest, reflecting the synergistic effect of high-intensity training and mechanical force that significantly enhanced metabolic activity. Mechanical force increases the rate of lactate production by increasing muscle contraction intensity and oxygen utilization, while the lactate clearance capacity fails to keep up, resulting in a significant increase in peak concentration. Experimental Group 2 also used high-intensity training. Due to the lack of mechanical force, the increase in blood lactate concentration was significantly lower than that of Experimental Group 1. This shows that mechanical force can accelerate the production of lactate, further amplifying the effect of high-intensity training on lactate metabolism. Low-intensity training combined with mechanical force has a relatively mild effect on blood lactate concentration. Mechanical force has a certain effect in promoting lactate production in low-intensity training, but the magnitude is much lower than that in the high-intensity training group. The lactate concentration in experimental group 4 changed little, indicating that low-intensity training itself does not stimulate lactate metabolism enough, especially in the absence of mechanical force intervention. The blood lactate concentration in the control group was almost unchanged, reflecting the low metabolic demand in the resting state, and the balance between lactate production and clearance. By comparing the data of experimental group 1 with experimental group 2 and experimental group 3 with experimental group 4, it can be clearly seen that mechanical force has a promoting effect on blood lactate concentration. The mechanical force in the high-intensity training group increased

the peak blood lactate concentration by 33.90 percentage points, and the mechanical force in the low-intensity training group increased the peak blood lactate concentration by 9.40 percentage points.

Table 2 shows the comprehensive impact data of training energy consumption and metabolic indicators, including energy consumption, lactate dehydrogenase (LDH), creatine kinase (CK) and cell metabolic activity:

Table 2. Comprehensive data table of energy consumption and metabolic indicators.

Groups	Energy expenditure per training session (kcal)	Increase in LDH concentration	CK concentration increase	Increased cell metabolic activity
Group 1	682.5 ± 25.3	23.50%	18.20%	27.40%
Group 2	603.4 ± 21.1	15.60%	12.70%	20.60%
Group 3	480.2 ± 18.7	14.80%	10.90%	18.30%
Group 4	459.8 ± 17.3	8.30%	5.70%	12.50%
Control group	No training	0.00%	0.00%	0.00%

The data in **Table 2** show that the energy consumption of a single training session, the increase in LDH and CK concentrations, and the increase in cell metabolic activity are all significantly correlated with the training intensity and the effects of mechanical force. All indicators of experimental group 1 reached the highest level, while all indicators of the control group remained unchanged. The energy consumption of single training in experimental group 1 was the highest (682.5 ± 25.3 kcal), followed by experimental group 2 (603.4 ± 21.1 kcal). The energy consumption of experimental groups 3 and 4 was significantly lower than that of the high-intensity group, which were 480.2 ± 18.7 kcal and 459.8 ± 17.3 kcal, respectively. Under the same intensity training conditions, mechanical force significantly increased energy consumption. As a biochemical indicator of metabolic activity, the increase in LDH reflects the acceleration of lactate metabolism. The increase in LDH concentration in Experimental Group 1 was the highest, at 23.50%, followed by Experimental Group 2 (15.60%), while the increase in LDH in the low-intensity group was smaller (14.80% in Experimental Group 3 and 8.30% in Experimental Group 4). The LDH data showed that mechanical force plays an important role in promoting lactate metabolism and muscle activity, especially in high-intensity training, which can more significantly amplify the metabolic load. CK concentration reflects muscle metabolic load and damage. The CK concentration of experimental group 1 increased by 18.20%, and that of experimental group 2 was 12.70%, while that of the low-intensity groups (experimental groups 3 and 4) was 10.90% and 5.70%, respectively. Mechanical force promoted the increase of CK concentration by increasing the mechanical load and micro-damage of muscles, but it also suggested that high-intensity training plus mechanical force intervention may lead to a higher risk of muscle damage. The cell metabolic activity of experimental group 1 increased the most, by 27.40%, while that of experimental group 2 was 20.60%, and that of experimental group 3 and experimental group 4 was 18.30% and 12.50%, respectively. This indicates that mechanical force activates the metabolic pathways of muscle cells, enhances their metabolic activity, and thus promotes the efficiency of energy metabolism at the molecular level. Experimental group 1

performed best in all metabolic and biochemical indices, indicating that high-intensity training combined with mechanical force is an effective strategy to increase energy consumption and metabolic activity.

Figure 4 shows the effect of training on muscle thickness:

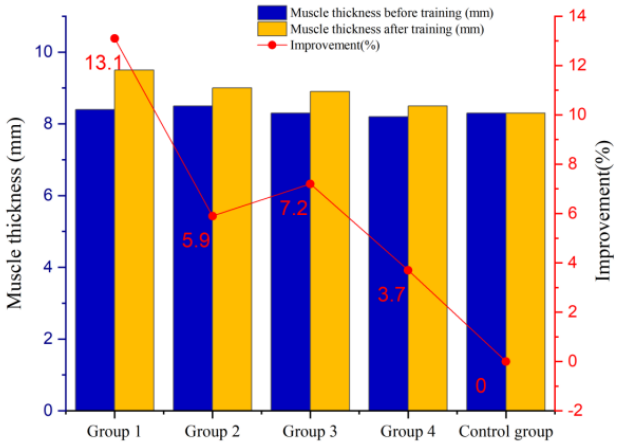

Figure 4. Muscle thickness change.

Figure 4 shows that the increase in muscle thickness varies significantly among different groups, affected by training intensity and mechanical force. Experimental group 1 has the largest increase (13.10%), while experimental group 4 and the control group have the smallest changes, and the control group has almost no change. This shows that mechanical force and high-intensity training have an important role in promoting the growth of muscle thickness. The muscle thickness of experimental group 1 (high intensity + mechanical force) increased by 13.10% after training, the highest among all groups. The muscle thickness of experimental group 2 (high intensity + no mechanical force) increased by 5.90%, which was significantly lower than that of experimental group 1. Under high-intensity training conditions, mechanical force increased muscle thickness by 7.20 percentage points. The muscle thickness of experimental group 3 (low intensity + mechanical force) increased by 7.20%. The muscle thickness of experimental group 4 (low intensity + no mechanical force) increased by 3.70%. Under low-intensity training conditions, mechanical force increased muscle thickness by 3.50 percentage points. Low-intensity training has limited effect on increasing muscle thickness, but mechanical force can still significantly improve its effect, indicating that mechanical force also has a synergistic effect on low-intensity training. The muscle thickness of the control group remained unchanged before and after training, with an increase of 0.00%, indicating that there was no significant change in muscle thickness at rest. Training can significantly increase muscle thickness, and the addition of mechanical force further amplifies this effect. The improvement of experimental group 1 was significantly higher than that of experimental group 2, indicating that mechanical force can stimulate the growth of muscle fibers by increasing muscle load and metabolic demand.

Figure 5 shows the effect of training and mechanical force on the peak force of knee extensors:

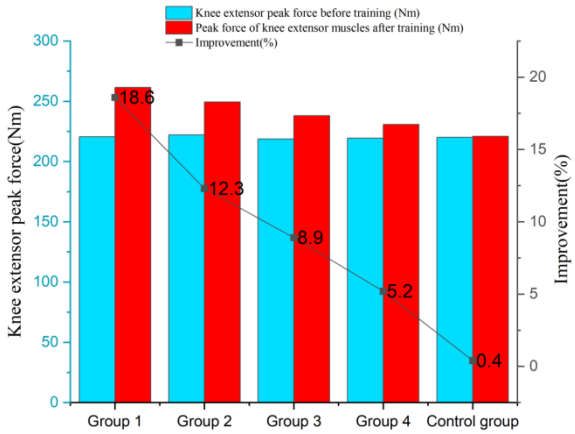

Figure 5. Changes in peak force of knee extensor before and after training.

Figure 5 shows that there are significant differences in the increase in peak force of knee extensor before and after training in different groups, and the results are affected by both training intensity and mechanical force. The peak force increase in experimental group 1 is the highest (18.60%), while the control group has almost no change (0.40%), indicating that mechanical force and training intensity play a key role in the increase of knee extensor peak force. Under high-intensity training conditions, mechanical force intervention increased the increase in peak force by 6.30 percentage points. High-intensity training combined with mechanical force significantly enhanced the power output of the knee extensors. Mechanical force stimulated stronger adaptive growth by increasing joint force and muscle load. Under low-intensity training conditions, mechanical force intervention increased the increase in peak force by 3.70 percentage points. The effect of mechanical force under low-intensity training is limited, but it can still increase the power output of the knee extensor muscles, indicating that the synergistic effect of mechanical force on light training is still significant. The peak force of the knee extensor muscles in the control group was almost unchanged before and after training, indicating that the joint muscle strength can not change significantly in the static state. The data of the control group emphasizes the necessity of training and mechanical force intervention to improve joint muscle strength.

Table 3 shows the change data of different training groups in terms of the increase in cadence, the increase in stride length and the reduction in impact force on landing:

Table 3. Gait data change table.

Groups	Stride frequency improvement	Step length improvement	Landing impact force reduction
Group 1	4.20%	2.80%	−11.60%
Group 2	3.10%	1.90%	−8.20%
Group 3	2.70%	1.50%	−6.50%
Group 4	1.80%	0.90%	−3.20%
Control group	0.00%	0.00%	0.00%

Table 3 analyzes the effect of mechanical force training on the gait

characteristics and sports economy of athletes by comparing the data of different experimental groups. The step frequency of experimental group 1 increased by 4.20%, which was the highest among all groups, indicating that high-intensity training combined with mechanical force significantly improved the athletes' ability to regulate step frequency. The improvement of experimental group 2 was 3.10%, which was slightly lower than that of experimental group 1, but still significantly higher than that of other groups. Experimental groups 3 and 4 increased by 2.70% and 1.80% respectively, indicating that low-intensity training has limited effect on cadence optimization, and there is a significant difference between the presence and absence of mechanical force. The cadence of the control group did not change (0.00%), indicating that training intervention is needed to improve the cadence. Both experimental groups 1 and 2 showed a higher increase in cadence and step length, especially under the action of mechanical force intervention, the gait adjustment ability was significantly improved. This is because high-intensity training enhances the strength of the quadriceps and hamstrings and improves neuromuscular control ability. The improvement in experimental groups 3 and 4 was small, indicating that low-intensity training was not enough to significantly improve gait parameters. The mechanical force intervention groups (experimental groups 1 and 3) performed better than the corresponding non-mechanical force groups, suggesting that mechanical force may further improve gait adjustment ability by optimizing muscle contraction efficiency and nerve conduction velocity. Experimental group 1 performed best in reducing the impact force of landing (−11.60%), indicating that mechanical force intervention can significantly improve the distribution of ground reaction force during running, which may be related to the enhancement of muscle synergy and gait economy. Comparing the data of experimental group 1 and experimental group 2, and experimental group 3 and experimental group 4, mechanical force significantly reduced the impact force of landing, which reduced the risk of joint injury, especially the knee and ankle joints. The simultaneous improvement of step frequency and step length indicates that athletes can run in a more economical way, reduce energy consumption, and reduce impact force, which helps reduce the risk of soft tissue and bone injury and improve sports life. The high-intensity combined with mechanical force training mode is an effective means to improve gait parameters. Mechanical force intervention can effectively relieve the stress on the knee and ankle joints and reduce the incidence of injuries.

The difference in experimental results is not only due to changes in training intensity, but also involves deep physiological mechanisms and mechanical principles. Mechanical force activates metabolism-related signaling pathways and promotes the efficiency of energy consumption by affecting the reorganization of the cytoskeleton. When muscle cells experience mechanical force, their metabolic pathways will be reshaped, enhancing the energy utilization efficiency of muscles and optimizing the clearance process of lactic acid. These changes reflect the profound impact of mechanical force on metabolism at the cellular and tissue levels. In actual training, the use of mechanical force intervention training can effectively promote athletes' metabolic efficiency and energy consumption, thereby improving training effects. During the high-intensity training stage, the appropriate addition of mechanical force intervention can not only improve the physical performance of

athletes, but also optimize the training plan and help athletes better adapt and recover.

5. Conclusions

This study explored the biomechanical mechanisms of metabolism and energy consumption of college women's football players under mechanical force through a multi-scale analysis framework. The study confirmed the key effects of mechanical force on metabolism at the molecular, cellular and tissue levels, constructed an energy metabolism model, and verified the role of high-intensity mechanical force training in significantly improving metabolic efficiency and optimizing energy consumption. The innovative combination of molecular mechanics experiments, cell mechanics analysis and metabolic modeling promotes the application of biomechanics in sports science and provides theoretical support for athlete training plans and rehabilitation optimization. The results show that high-intensity mechanical force training plays an important role in improving training effects, optimizing energy consumption and reducing training risks, and has reference value in fields such as sports rehabilitation and sports nutrition. Future studies can expand the sample range, include athletes from different regions, sports levels, age groups and genetic backgrounds, optimize metabolic models, consider individual differences, and use personalized models to improve prediction accuracy. Explore the synergistic effects of mechanical force and other biochemical signals to reveal their comprehensive effects on metabolic regulation. Long-term follow-up studies are also very important. They can examine the long-term effects of high-intensity mechanical force training, promote the development of personalized training equipment and intelligent monitoring systems, apply research results to actual training, and provide a more scientific theoretical basis for personalized sports training and rehabilitation programs.

Ethical approval: Not applicable.

Data availability: The experimental data used to support the findings of this study are available from the corresponding author upon request.

Conflict of interest: The author declares no conflict of interest.

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