MEDICAL EFFECT OF SPORTS ON IMPROVING THE MAXIMUM OXYGEN

O EFEITO MÉDICO DOS ESPORTES NA MELHORA DA CAPTAÇÃO DE OXIGÊNIO

EL EFECTO MÉDICO DE LOS DEPORTES EN LA MEJORA DE LA CAPTACIÓN DE OXÍGENO

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Hua Yu[1]
(Physical Education Professional)

1. Jiangxi Vocational College of Industry & Engineering PingXiang JiangXi, China.

Correspondence
Hua Yu
JiangXi, China, 337055.
gjy1213@126.com

ABSTRACT

Introduction: Maximum oxygen uptake is an effective indicator of the level of human cardiopulmonary function and aerobic work capacity. Observing the effects of aerobic training and formulating scientific training plans are of considerable value. Objective: To observe the effect of physical exercise on the human body's maximum oxygen uptake and arterial blood ketone body ratio. Methods: Before and after 4 weeks of physical exercise, the maximum oxygen uptake, blood lactic acid and heart rate changes, and ketone body content in the incremental load exercise experiment was measured in the human body. Results: The subjects' maximum oxygen uptake, maximum exercise load, heart rate, and blood lactic acid levels increased significantly after physical exercise. Conclusion: The human body's maximum oxygen uptake is enhanced under sports. ***Level of evidence II; Therapeutic studies - investigation of treatment results.***

Keywords: *Exercise; Biological oxygen demand analysis; Arterial Pressure; Ketones.*

RESUMO

Introdução: A captação máxima de oxigênio é um indicador eficaz do nível da função cardiopulmonar e da capacidade de trabalho aeróbico humano. A observação dos efeitos de treinos aeróbicos e a formulação de planos de treinamento científicos têm valor considerável. Objetivo: Observar o efeito do exercício físico na captação máxima de oxigênio e a proporção de corpos cetônicos e sangue arterial no corpo humano. Métodos: Antes e após exercícios físicos de quatro semanas, a captação máxima de oxigênio, ácido láctico no sangue e mudanças na frequência cardíaca, além do conteúdo de corpos cetônicos, em um experimento de carga de exercícios progressiva foram medidos no corpo humano. Resultados: A captação máxima de oxigênio, carga máxima de exercício, frequência cardíaca e níveis de ácido láctico no sangue dos indivíduos aumentaram significativamente após o exercício físico. Conclusão: A captação máxima de oxigênio aumenta com a prática de esportes. **Nível de evidência II; Estudos terapêuticos – investigação de resultados de tratamento.**

Descriptores: *Exercício físico; Análise da demanda biológica de oxigênio; Pressão arterial; Cetonas.*

RESUMEN

Introducción: La captación máxima de oxígeno es un indicador eficaz del nivel de la función cardiopulmonar y de la capacidad de trabajo aeróbico humano. La observación de los efectos de entrenamientos aeróbicos y la formulación de planes de entrenamiento científicos tiene valor considerable. Objetivo: Observar el efecto del ejercicio físico en la captación máxima de oxígeno y la proporción de cuerpos cetónicos y sangre arterial en el cuerpo humano. Métodos: Se midió, en el cuerpo humano, antes y después de ejercicios físicos de 4 semanas, la captación máxima de oxígeno, ácido láctico en la sangre y cambios en la frecuencia cardíaca, además del contenido de cuerpo cetónicos, en un experimento de carga progresiva. Resultados: La captación máxima de oxígeno, carga máxima de ejercicio, frecuencia cardíaca y niveles de ácido láctico en la sangre de los individuos aumentaron significativamente tras el ejercicio físico. Conclusión: La captación máxima de oxígeno aumenta con la práctica de deportes. ***Nivel de evidencia II; Estudios terapéuticos – investigación de resultados de tratamiento.***

Descriptores: *Ejercicio físico; Análisis de la demanda biológica de oxígeno; Presión arterial; Cetonas.*

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INTRODUCTION

The redox state of mitochondria plays an essential role in the regulation of aerobic metabolism. Its ratio determines the efficiency of oxidative phosphorylation and ATP synthesis by affecting the electron transfer rate of the respiratory chain, thereby affecting the energy level of the cell-energy charge. When assessing mitochondrial function, obtaining sufficient tissue samples is often the main limiting factor for human application research in sports medicine.[1] Compared with tissue biopsy, the less invasive arterial blood ketone body ratio (AKBR) of sampling can more accurately reflect the redox state and energy charge of liver mitochondria under stress. Clinical medicine has used it to assess the development and prognosis of pathological stress, but this index is rarely used in sports medicine. This study observes the changes of AKBR under the condition that the aerobic exercise capacity of young swimmers changes after endurance training. This provides a basis for assessing the oxidation status and energy charge of liver mitochondria.

METHODS

General information

The training level of 18 male students in the particular swimming class is level 1 to level 2. The subject's age was 13.9±0.9 (12-16) years old, and the height was 1.68±0.65 (1.56-1.84) m. The weight is 57.7±9.3 (38-68) kg. The subjects underwent 4 weeks of aerobic endurance training during the study period.[2] The daily amount of water exercise is 6±2 (3-10) km, the duration is 3 to 4 hours, and the training is 6 days a week.

Athletic Ability Test

We conducted incremental and constant load exercise experiments at the beginning and the end of the 4-week aerobic endurance training. The incremental load exercise experiment is arranged one week before each constant load endurance experiment to avoid the mutual influence between the two exercise experiments. Keep the time, temperature, and humidity of the two measurements before and after each experiment as consistent as possible.[3] One day before the experiment, the subjects were asked to reduce their training intensity.

The incremental load exercise experiment

The subject maintained the pedal speed of the Monark power bicycle at 60r/min. Volunteers first performed a 2min warm-up exercise with 0 loads and then increased the load by 15W every minute until fatigued according to their age. The fatigue criterion was that the subjects could not maintain the pedaling speed of 50 or 60r/min after repeated encouragement.4 Ear blood was collected before and after exercise and during exercise to determine blood lactic acid concentration. We use VO_2 to collect and analyze VO_2max.

Constant load endurance exercise experiment

The subjects used 90% of the individual lactic acid threshold intensity determined in the first incremental exercise experiment. Equivalent to 65±4(58-72) %VO_2max for 60min of power cycling exercise.[5] The blood lactic acid concentration of 45-50 minutes of exercise was 4±1.32mmol/L. After exercise, AKBR was measured after femoral artery puncture to obtain arterial blood.

Test method

Determination of blood lactic acid

Collect ear blood and dissolve it with a lactic acid hemolytic agent, mix it and inject it into the sample room of the YSL-23L lactic acid analyzer, and multiply the measurement result by the dilution factor.[6] According to the obtained lactic acid value, the blood lactic acid-time curve was drawn to determine the subject's lactic acid threshold (ILAT).

Determination of arterial blood ketone body ratio

After the constant load exercise experiment, the subjects walked into the sterile arterial blood sampling room from the exercise laboratory. After the iodophor disinfects the femoral fossa surgery area, spread a sterile hole towel. We collected femoral artery blood samples by inserting a needle perpendicular to the femoral fossa and arterial pulsation.[7] The blood sample acquisition time was 3.96±1.73min after exercise.

The concentration of acetoacetate (ACAC) or β-hydroxybutyrate was determined by using a colorimetric method to measure the decrease or increase of NADH in the reaction catalyzed by β-hydroxybutyrate dehydrogenase. AKBR is the ratio of acetoacetic acid/β-hydroxybutyric acid.

Statistical processing

The experimental data are expressed as mean ± standard deviation. We use SPSS10.0 statistical software for a paired t-test or rank-sum tests. This article assumes that the initial position y_0 of the moving target is obtained through particle filtering, and the similarity between the candidate target y_0 and the target model is:

$$\rho[p',q'(y_0)]=\sum_{u=1}^{m}\sqrt{p'_u q'_u(y_0)} \tag{1}$$

The similarity is:

$$\rho[p',q'(y)]=\sum_{u=1}^{m}\sqrt{p'_u q'_u(y)} \tag{2}$$

In this way, the problem is transformed into an optimization problem for solving $\max\ \{\rho[p',\ q'(y)]\}$. We use the Taylor expansion of formula (3) at the point y_0 to approximate $\rho[p',\ q'(y)]$:

$$\rho[p',q'(y)]\approx\frac{1}{2}\sum_{u=1}^{m}\sqrt{p'_u q'_u(y_0)}+\frac{C_h}{2}\sum_{i=1}^{hn}w_i k(\left\|\frac{y-x_i}{h}\right\|^2) \tag{3}$$

Among them:

$$w_i\sum_{u=1}^{m}\sqrt{\frac{p'_u}{q'_u(y_0)}}\sigma[b(x_i)-u] \tag{4}$$

To maximize (4), the following formula must be maximized.

$$\sum_{i=1}^{hn}w_i k(\left\|\frac{y-x_i}{h}\right\|^2) \tag{5}$$

To solve (5), let us assume that $\{xi\}_{i=1,\dots,nh}$ is a set of samples. y_0 is the initial cluster center of this set of samples. w_i is the fuzzy membership function of each sample point x_i. We use the method of solving fuzzy clustering to solve the cluster center point y':

$$y'=\frac{\sum_{i=1}^{hn}x_i w_i g(\|y_0-x_i\|^2)}{\sum_{i=1}^{hn}w_i g(\|y_0-x_i\|^2)} \tag{6}$$

Among them: $g(x) = -k'(x)$. The following are the calculation steps of the mean shift algorithm:

1) Calculate the similarity between the initial target and the target model: $\rho[p',q'(y')]=\sum_{u=1}^{m}\sqrt{p'_u q'_u(y')}$. 2) Calculate the new candidate target y' according to formula (6). 3) Calculate the similarity $\rho[p',q'(y_0)]=\sum_{u=1}^{m}\sqrt{p'_u q'_u(y')}$ between the candidate target y' and the target model. 4) When $\|y' - y_0\| < \varepsilon$, stop the iteration. Otherwise, let $y_0 \leftarrow y'$ go to step (2).

RESULTS

Aerobic capacity

The subject's aerobic exercise capacity and individual lactate threshold before and after training

It can be seen from Table 1 that the absolute and relative values of the subjects' maximum oxygen uptake after 4 weeks of aerobic endurance training were significantly higher than those before the training.[8] The maximum load, maximum heart rate, and maximum lactic acid during incremental exercise also increased significantly. The subjects' lactate threshold only increased (3.60±0.65mmol/L before training vs. 3.88±0.58mmol/L after training, P=0.10).

The kinetic curve of blood lactate, heart rate, and oxygen uptake

Track the changes of blood lactate, heart rate, and oxygen uptake during incremental exercise and draw their dynamic curves, respectively. After training, these three curves all shifted to the right (Figure 1, 2).

Arterial blood ketone body level and AKBR

There were no significant changes in arterial blood total ketones and 3HB levels during the study period after constant-load exercise. ACAC and AKBR levels were significantly higher than before training (Table 2).

Table 1. Changes in aerobic exercise performance indexes of subjects at maximum exercise after 4 weeks of endurance training.

Maximum load (Watt)	Before training	After training
Maximum heart rate (beats/min)	217±39	234±41
Maximum lactic acid (mmol/L)	189±7	193±8
Absolute maximum oxygen uptake (ml/min)	10.39±2.02	13.35±2.10
Relative value of maximum oxygen uptake (ml/kg/min)	2908±565	3023±481
Maximum load (Watt)	49.3±5.4	52.6±6.7

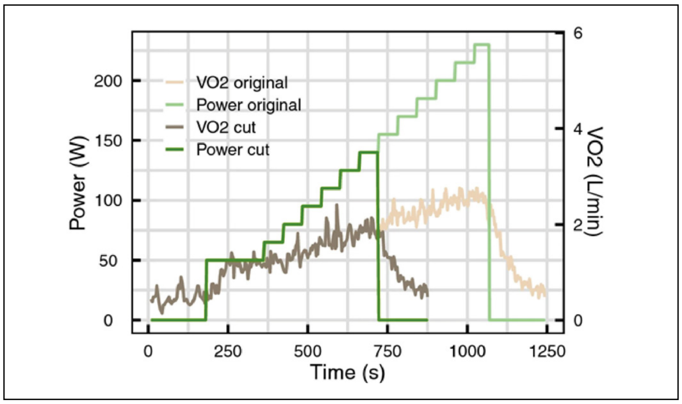

Figure 1. Blood lactate and heart rate dynamics curve during incremental exercise..

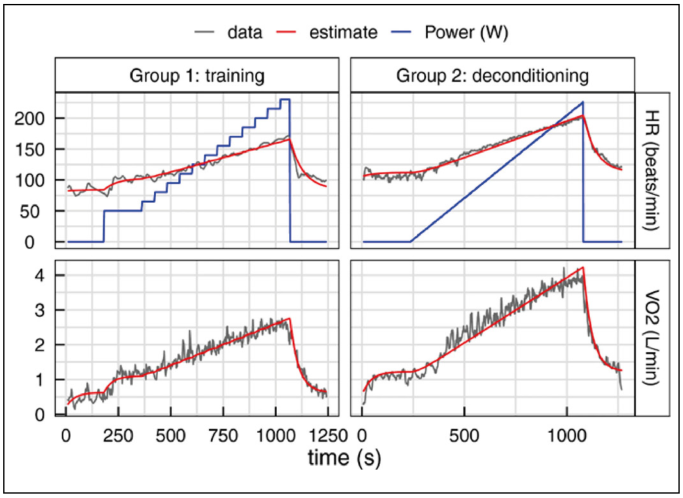

Figure 2. Oxygen uptake dynamics curve during incremental exercise..

Table 2. Changes in subjects' arterial blood ketone body levels and AKBR after quantitative load exercise before and after four weeks of endurance training.

Before training		After training
ACAC (mmol/L)	0.12±0.05	0.15±0.04
3HB (mmol/L)	0.10±0.07	0.09±0.08
Total ketone body (mmol/L)	0.21±0.08	0.25±0.12
AKBAR	1.82±1.05	2.62±1.60

DISCUSSION

This experiment showed that after 4 weeks of endurance training, the subjects' aerobic exercise capacity improved. The AKBR of subjects immediately after 1 hour of constant load exercise was significantly higher than before training.

Mitochondrial redox state can indirectly reflect the degree of oxidative phosphorylation coupling and oxygen utilization during exercise. It plays an essential role in the regulation of mitochondrial function and aerobic metabolism. However, needle aspiration biopsy is often used when assessing mitochondrial function in normal healthy humans. Such as percutaneous liver puncture, percutaneous myocardial or skeletal muscle puncture to obtain samples. The difficulty of recruiting subjects limits its application in the field of sports human science research. Therefore, looking for non-invasive or less invasive, indirect, and sensitive indicators of mitochondrial function is the key to solving this difficulty.

Ketone bodies are mainly synthesized in liver mitochondria. When the ketogenic effect is inhibited between acetoacetic acid and β-hydroxybutyric acid reaches equilibrium. The ketone body conversion side is mainly determined by the ratio of liver mitochondria $NAD^+/NADH$.[9] The vascular system itself hardly consumes ketone bodies, and the peripheral arterial blood ketone body ratio (AKBR) can accurately reflect the hepatic vein blood ketone body ratio. Therefore, changes in liver mitochondrial redox status and liver energy charge can be reflected by AKBR.

Clinical medicine has confirmed that a significant decrease in AKBR levels is highly related to metabolic disorders such as blood lactic acid accumulation, blood branched-chain amino acid/aromatic amino acid ratio, ketamine, and increased insulin resistance. This can be used to evaluate the redox state and energy charge of liver mitochondria under pathological stress or metabolic disorders and more accurately reflect the failure of multiple organs such as the heart, lung, brain, and systemic energy metabolism under severe pathological conditions. This is an essential indicator for treating liver, heart, lung, brain, and multiple organ system failures and the prognosis of the disease. Because the increase in catecholamine levels and the decrease in insulin levels during exercise limit the mass production of ketone bodies, AKBR should be able to reflect the liver mitochondrial function during exercise.

Some scholars use AKBR to evaluate liver mitochondrial function during acute exercise. Scholars observed that AKBR was negatively correlated with blood lactic acid concentration during incremental exercise with bicycle ergometer (r=-0.41, P<0.01). With the prolonging of exercise time, AKBR gradually decreases from the basic level of the quiet state. After the appearance of the ventilation threshold, its decreasing trend intensified, and AKBR reached its lowest point at the end of the exercise. After that, continue to slowly decrease to 6 minutes after the end of the exercise, and then slowly recover to a quiet level. The author also observed changes in the levels of hormones and aerobic metabolic substrates that affect energy metabolism. It was found that blood catecholamines levels increased significantly during exercise, and insulin levels decreased significantly. At the same time, the blood sugar level was significantly reduced, and the free fatty acid level was significantly increased. Some scholars have found that with the participation of catecholamines and insulin, the reduction state of liver mitochondria during exercise is enhanced with the increase in glycolysis product lactic acid.

CONCLUSION

In this study, AKBR evaluated the human liver mitochondrial function after endurance training and aerobic capacity changes. We found that after 4 weeks of aerobic endurance training, the subjects' aerobic exercise capacity improved. The incremental load exercise experiment observed that the oxygen uptake, heart rate, and blood lactate dynamics curves after training shifted to the right compared to before training. This shows the energy savings of subjects under quantitative load exercise. Mainly the kinetic curve of blood lactate shifts to the right. This indicates that the accumulation of blood lactic acid decreases under the same exercise load intensity. The oxidation and gluconeogenesis of lactic acid in the liver is the leading way for blood lactic acid metabolism during exercise. Lactic acid, mainly from exercise skeletal muscle, is converted into pyruvate and enters the liver mitochondria to synthesize acetyl CoA. This meets the needs of mitochondria for oxidative phosphorylation substrate NADH under strenuous exercise. This may be an increase in the AKBR level when the subject's aerobic exercise capacity improves. Under the same exercise load, the oxidation state of liver mitochondria increases, and the energy charge of liver cells increases.

The author declare no potential conflict of interest related to this article

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