Changes and Mechanisms of Macrophages Exposure to Exercise Training and Sports Supplement

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Abstract. Macrophages, the first line of defense of body against the pathogenic microbial invasion, are widely distributed in many tissues and are of great significance to improve the athletes’ immune system, exercise capacities and health. However, the real regulatory mechanisms of exercise and combined with supplements on macrophages were still unclear. Furthermore, upon exercise stimuli, the actual changes and associated mechanisms of macrophages and immunoregulation are still insufficient. Moreover, how to improve the functional performance of macrophages by means of exercise supplements during exercise remains unknown. Therefore, in the current study, we made a systematic review of the current research on the relationship between exercise and macrophages by employing literature inquire, with the aim at the theoretical system to sort out the relationship between the two frameworks and the relationship to supply some theoretical basis.

Introduction

Macrophages (Mφ) are widely distributed in the liver, lung, peritoneum, adipose tissue, blood, bone and muscle and other organs and organs. It is the body against the pathogenic microbial invasion of the first line of defense. In recent years, many scholars at home and abroad have done a lot of research in this area. The research results are of great significance to improve the immunity of athletes, especially athletes, and protect their health [1]. In addition, the effects of exercise on macrophages and the mechanism of macrophage immunoregulation are still not deep enough; and how to improve the function of macrophages by exercise supplements during exercise or during exercise. Research is still in the initial stage. In this paper, the literature review method, through access to relevant research frontier and literature, the current people on the relationship between exercise and macrophages research, a systematic review. Aimed at the theoretical system to sort out the relationship between the two frameworks for people to further understand and studies the relationships between the two do some theoretical basis.

Immune Function of Macrophages

Antigen Presentation

Macrophages are innate and acquired immune bridges, which are scattered in all parts of the body, through the inflammatory response and antigen presenting cells in the form of immune
response. In the process of antigen presentation, macrophages engulf foreign microorganisms into phagocytic bodies, and then the foreign protein is digested and processed into antigenic peptides. These peptides are linked to MHC class II molecules and transferred to the surface of macrophages and CD4+ T lymphocytes interact. The interaction between macrophages and T cells produces antigen-specific T cell clones that ultimately recognize and destroy invasive microbes and provide long-lasting immunity [2]. Macrophage antigen delivery capacity is essential for immune function, when this function is impaired; the probability of onset and infection risk of death will rise.

**Bactericidal Effect**

Macrophages are the first line of defense against the invasion of pathogenic microorganisms and can be produced by the "respiratory burst" by the NADPH oxidase-mediated production of reactive oxygen species (ROS), pathogens microbes, which are the main mechanism of macrophage sterilization, play an important role in the prevention of germs. Pathogen invasion, MΦ can produce a large number of intracellular ROS to kill the invasion of pathogenic microorganisms, which is an important way of sterilization.

**Effects of Exercise on Macrophages**

**Effects of Low-intensity Exercise on Macrophages**

**Influence on The Number of MΦ.** Adequate evidence suggests that acute exercise can cause an instantaneous increase in the number of Mo in peripheral blood (50% -100%). Wang Xueqin and others found that low-intensity exercise to increase the phagocytosis of liver macrophages using the experiment [2]. Yao Yucai and so on through the MΦ phagocytic neutral red experimental study found that the number of MΦ in 30min and 1h group increased, the number of value with the MΦ phagocytosis enhanced value, it can be seen from the number of macrophages and exercise intensity of the time closely with the number of MΦ changes [3].

**Influence on the MΦ Function.** Appropriate intensity of exercise can increase the phagocytosis of liver macrophages. Yao Yucai and so on through the MΦ phagocytic neutral red experimental study found that M phagocytes in 30min and 1h group increased, anti-tumor function in 30min group increased, indicating that the following 1h exercise on macrophage phagocytosis and anti-tumor function to promote [3]. (30min / d, 6d / w) of C57BL / 6 mice with swimming training for 3 months). The results showed that the phagocytic and antitumor function of macrophages in the aged group was lower than that in young rats, which indicated that moderate exercise training could enhance MΦ function and play a certain anti-aging effect. In the elderly group of mice (21 months), the appropriate amount of chronic exercise tends to reverse the age-induced changes in macrophage function. Fehr found that mice after 25 days treadmill training, intraperitoneal MΦ intracellular enzymes increased, MΦ phagocytosis enhanced [4]. 3-7d medium intensity could increase the activity of TG induced and propionibacterium acne-activated rat peritoneal MΦ. Xiao Weihua and other studies have found that 25 days after treadmill training mice intraperitoneal macrophages increased enzyme substances, macrophage kinetic energy increased [5]. From the above results can see that the relationship between macrophage phagocytosis and exercise intensity is closed.
Large-intensity Sports Team Macrophages

Influence on the Number of МΦ. With the discovery of Lim CL and Yao Yucai, the activity of macrophages was enhanced with the loss of muscle tissue in the body after a long period of intense exercise, and the phagocytic function of the latter was significantly reduced after 2 hours 2h exercise on МΦ phagocytosis inhibitory effect. Jiang Lu found that over-training will mononuclear Мф system function inhibition, suggesting that over-training may cause immune dysfunction [6]. Chen Shuang and other rats experiment were divided into control group, general training group and intensive training group using incremental load movement, found that general training to improve the ability of МфIL-6, this enhancement in the high-intensity training was significantly inhibited, so МΦ The number also decreases [7].

Influence on the МΦ Function. Strenuous exercise (treadmill exercise to exhaustion) can activate bronchial alveolar macrophages (BAM), improve its phagocytosis, but this increase is temporary, 4 hours after the return to normal levels. But not everyone supports strenuous exercise that can increase the phagocytic function of macrophages. 7 weeks of training and vigorous exercise had a detrimental effect on alveolar macrophage phagocytosis, but strenuous exercise had no effect on phagocytosis of macrophages without systematic training. Long-term vigorous exercise on macrophage phagocytosis of the impact of less reported. The effect of exercise on phagocytosis is related to the animal species, in addition to the nature of the movement. Athletes after a cycle of strenuous exercise, due to muscle tissue damage, the macrophage activity will increase. Yao Yucai and so on through the Мф phagocytic neutral red experimental study found that М phagocytosis in 2h group was significantly reduced, indicating that 2h movement of Мф function damage or inhibition [3], indicating that large Intensity training can inhibit immune function.

Effects of Exhaustive Exercise on Macrophages

Influence on the Number of МΦ (value) of the Impact. Shi Yali and other rats were divided into control group, general training group and intensive training group, the use of incremental load movement, found through the general training to improve the ability of МфIL-6, this enhancement in the high-intensity training was significantly inhibited, high intensity training can inhibit immune function [8]. Exhaustive cycling exercise can improve the activity of TG induced and propionic-bacterium acnes-activated rat peritoneal Мф, exhaustive exercise to reduce alveolar Мф antiviral function. Paulsen G and so on with the greatest degree of centrifugal exercise to study the movement caused by muscle damage and delayed white blood cell relationship between the macrophages found that colony-stimulating factor immediately after the movement reached its peak. From the above results we can see that exhaustive exercise increases the number of cells in Мф.

Influence on the МΦ Function. The available data indicate that the dependence of NOS activity on Ca^{2+} / CaM is different. NOS is divided into two classes, one is the construction of NOS (cNOS); the other is the induction of NOS (iNOS) involved in immune regulation is mainly iNOS catalyzed NO. In this study, we investigated the changes of iNOS activity and the corresponding changes of plasma CD^{4+} / CD^{8+} in peritoneal macrophages by exercise training of different load, and discussed the mechanism of different exercise load on the immune function of SD rats and the health effects of SD rats influences. The release of inflammatory cytokines such as interferon-r (IFN-r) tumor necrosis factor (TNF), interleukin-1 (IL-1) and interleukin-2 (IL-2) promotes macrophages, vascular smooth muscle cells and Cardiomyocytes
in the iNOS gene transcription and expression, thereby promoting a large number of NO syntheses and release of the immune system to regulate.

**Influence on Macrophages**

Studies have shown that the effect of exercise on macrophage function is comprehensive and profound, most studies suggest that the mechanism of macrophage effects of exercise has eight points: (1) Long-term moderate exercise can promote peripheral blood mononuclear cells to M2 macrophages Cell differentiation, reduce adipose tissue, atherosclerotic plaque, tumor tissue macrophage infiltration, but also affect the number of peritoneal macrophages, muscle macrophages phenotype. (2) Exercise can increase macrophage chemotaxis. (3) Long-term high-intensity exercise can reduce the ability of macrophage antigen presentation, and the ability to reduce the ability of this antigen may be due to antigen processing capacity defects. (4) Long-term low-intensity training, short-term strenuous exercise can increase macrophage phagocytosis, and long-term strenuous exercise on macrophage phagocytosis function less information. (5) The impact of exercise on the secretion of macrophages reported different, by the exercise intensity, exercise time, irritant species, whether the pathological model and so on. (6) Exercise to change the anti-virus ability affected by a variety of factors, such as: sports attributes (exercise type, intensity and duration), gender, age and tissue parts, and diet nutrition. Most studies suggest that moderate exercise can enhance macrophage antiviral ability, and exhaustive exercise can inhibit macrophage anti-virus ability. (7) Moderate exercise and exhaustive exercise can increase the anti-tumor activity of macrophages in vitro. (8) Different forms, intensity and duration of exercise have different effects on macrophage glucose and glutamine consumption, hydrogen peroxide production and enzyme activity.

**Advances in Exercise Complement and Macrophages**

**Effects of Supplemental Sugar On Macrophages**

**Effects of Supplemental Monosaccharide’S on Macrophages.** Exercise can affect macrophage antiviral ability has been a large number of experiments confirmed that this process is affected by nutritional supplements. Monosaccharide it is the body most likely to absorb and absorb the fastest of a carbohydrate, when the human discomfort, the most commonly used supplements is glucose, often referred to as energy. Monosaccharides are the basic components of polysaccharide. Shi Yali study shows that long-term high-intensity exercise of mice gavage of Ganoderma lucidum polysaccharide (GLP), the study of high-intensity mouse macrophage function, polysaccharide on the body's immune function obvious [8]. Thus it is concluded that monosaccharide have a potent effect on the function of macrophages.

**Effects of Supplemental Polysaccharides on Macrophages.** At home and abroad in the field of pharmacology on polysaccharides on the mechanism of MΦ done a lot of reports, and in the field of sports medicine reported particularly small. (YP) can significantly increase the ability of MΦ to secrete NO and IL-1β in high-intensity exercise mice and improve its phagocytosis. Song Yajun and other research results show that Lycium barbarum polysaccharide (LPB) can enhance the phagocytosis of MΦ. Xie Jun and other studies have shown that high-intensity environment, high-intensity exercise will significantly reduce the M phagocytosis, taking military solid beverages (military solid drinks mainly by traditional
Chinese medicine ginseng [9]. Astragalus, Rhodiola and other extracts and more anti-A) improve the phagocytic function of MΦ. Ganoderma lucidum polysaccharide (GLP) can antagonize the respiratory burst of MΦ, M Φ to reduce the production of reactive oxygen free radicals. Ganoderma lucidum polysaccharide peptide (GLPP) can increase the mouse peritoneal MΦ NO production. (PPS) had a significant effect on the production of NO in the myocardium of MCC, and the concentration of glutathione (GSH) decreased with the increase of NO concentration. It is suggested that GSH in MΦ may play a role in regulating NO production in MΦ and protecting host cells from NO-mediated cytotoxicity. Huang Dinan and other studies have shown that Polyporus umbellatus polysaccharide (PPS) can make M Φ iNOS mRNA expression increased and iNOS activity increased. (GPP), it was found that GPP and its components could prolong the swimming time of mice in excessive exercise and improve the immunity ability of mice, especially the ability of phagocytosis of MΦ in the abdominal cavity, The study suggests that GPP and its main components enhance the phagocytic capacity of MΦ in mice may be one of the reasons for prolonging the time of mouse exhaustion. The report suggests that the use of polysaccharide components against hyperactivity on the negative effects of macrophages is an effective means.

**Effects of Glutamine Supplementation on Macrophages**

Glutamine is an important source of energy for macrophages. It is still a controversial issue to supplement glutamine when exercising, indicating that over-training supplementation of glutamine and cannot change due to over-training caused by inhibition of ROS, macrophages ROS-mediated physiological function of no improvement. After 1 week of exacerbation, over-training supplemented the glutamine group of macrophages in the ROS returned to the quiet group level [10]. However, another study reported that rats with 85%.VO_{2}max intensity for 1h treadmill exercise on neutrophil ROS production had no effect, and exercise and supplementation of glutamine ROS production increased this may be with the exercise intensity, duration and Cell type is different.

**Effects of Supplemental Antioxidants on Macrophages**

The latest research results show that macrophages and obesity, atherosclerosis and other diseases are closely related to macrophages can infiltrate adipose tissue, atherosclerotic plaque, etc., to improve the level of oxidative stress, the release of inflammatory factors, Leading to the body's chronic inflammatory response, aerobic exercise is to help improve the symptoms of the disease, the main effect of exercise is to reduce adipose tissue and atherosclerotic plaque oxidative stress levels and inflammation levels, so that the symptoms can be alleviated, which A process can be closely related to the antioxidant effect of NO. It is generally believed that ROS overproduction can induce apoptosis by indirectly damaging DNA, lipids and proteins, or directly through ROS-mediated activation of the relevant signaling pathways. Increased levels of ROS in tissues or organs indicate that the level of oxidative stress in the organism is a negative effect. The development of common diseases such as atherosclerosis, obesity, diabetes mellitus and hypertension is closely related to ROS. It is speculated that exercise can increase the level of oxidative stress in the body, so as to improve the ROS generation. However, Xiao Weihua found that overtraining reduced the production of ROS in macrophages, which was significantly lower than the physiological level, resulting in negative effects, which may be high-frequency high-intensity repeated exercise stress to the body in immunosuppression performance [10]. After 1 week of exacerbation, ROS levels in the macrophages returned to a
quiet group level in the overtraining group. Over-training supplemented with antioxidant DPI reduced the amount of ROS produced significantly, significantly lower than the over-training group, and significantly reduced compared with the quiet control group, indicating that the use of DPI over-training will make ROS generation inhibited further deterioration of the situation, Normal physiological function [11]. This is consistent with the literature reported that DPI inhibits the production of ROS in macrophages. After 1 week of training, the amount of ROS in the macrophages supplemented with hypermedia supplemented DPI was also restored to normal levels.

Conclusions

The results show that appropriate moderate and small intensity exercise can enhance macrophage phagocytosis, and high-intensity exercise and over-training on macrophages have damage or inhibition of macrophage phagocytosis function, that is, different intensity of exercise Training caused macrophage function changes are inconsistent. Moderate exercise can enhance macrophage anti-virus ability, while exhaustive exercise is to inhibit macrophage anti-virus ability; exercise effects of macrophage capacity in addition to exercise intensity, duration and frequency of movement, but also by sex, age and Nutritional effects of macrophages may play a role in influencing hormone levels, macrophage phagocytosis, intracellular reactive oxygen production and nitric oxide synthesis.

References


