Preliminary Analysis of Exercise on Endothelial Cells and Its Mechanism
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Abstract
This article investigates the impact of exercise on endothelial cell function and its underlying mechanisms. Endothelial cells play a key role in maintaining cardiovascular health, and their dysfunction is closely associated with various cardiovascular diseases. Research shows that exercise improves endothelial function through various mechanisms, including increasing the production of nitric oxide (NO), reducing inflammation levels, improving lipid metabolism, and promoting angiogenesis. These effects collectively contribute to reducing the risk of cardiovascular diseases. It is hoped to provide references for researchers studying how exercise improves endothelial cell function.

Keywords
Exercise; Endothelial Cells; Cardiovascular Health.

1. Introduction
With the rapid changes in modern lifestyles, cardiovascular diseases have become a major challenge to global public health[1]. Statistics show that the incidence and mortality rates of cardiovascular diseases are continuously increasing, imposing a significant burden on individual health and the socio-economic fabric[1]. Among the numerous strategies for the prevention and treatment of cardiovascular diseases, exercise, as a non-pharmacological intervention, is receiving increasing attention[2]. However, how exercise specifically affects the cardiovascular system, particularly its impact on endothelial cell function, still requires further investigation.

This article aims to review the effects of exercise on endothelial cell function, explore how exercise improves endothelial function through various mechanisms, and discuss the current research limitations and future directions. Through a comprehensive analysis of existing literature, we hope to provide researchers with a deeper understanding of the relationship between exercise and endothelial cell function, and to emphasize the importance of moderate exercise in cardiovascular health management.

2. Overview of Endothelial Cells
2.1. The Role of Endothelial Cells in Vascular Health
Endothelial cells are a unique type of flat cells that form the inner lining of all blood vessels[3]. Morphologically distinct, these cells can adapt based on the type of vessel and the requirements of hemodynamics. Each endothelial cell is tightly joined to others, forming a continuous monolayer. This structure not only ensures the structural integrity of the vessel but also allows for the precise regulation of its function by the endothelial cells[4].

Endothelial cells play a crucial role in maintaining vascular permeability and controlling blood flow. Endothelial cells regulate vascular tension by producing nitric oxide (NO) and other
vasodilatory factors, thereby influencing blood pressure and the distribution of blood flow[5]. Furthermore, endothelial cells are involved in blood coagulation and fibrinolysis, coordinate anti-inflammatory responses in the vessel wall, and regulate the migration of white blood cells and other immune cells, all of which are key to maintaining vascular health and the overall function of the circulatory system.

2.2. The Association between Endothelial Dysfunction and Diseases

Endothelial dysfunction refers to the inability of endothelial cells to perform their physiological functions properly, typically characterized by reduced production of nitric oxide, increased oxidative stress, and heightened inflammatory responses[4]. This dysfunction is an early indicator of the development of various cardiovascular diseases, including atherosclerosis, hypertension, and coronary artery disease. The onset of endothelial dysfunction can be influenced by multiple factors, such as unhealthy lifestyles, chronic health conditions, and aging[4]. Therefore, understanding and improving endothelial cell function is crucial for the prevention and treatment of cardiovascular diseases.

In contemporary medical research, endothelial cells and their dysfunction have become central to the study of cardiovascular diseases[6]. By studying the physiological and pathological processes of endothelial cells, we can better understand the workings of the cardiovascular system and develop effective prevention and treatment strategies.

3. The Mechanism of Exercise-Induced Improvement in Endothelial Function

3.1. Nitric Oxide (NO) Production and Vasodilation

Nitric Oxide (NO) is a crucial vasodilator produced by endothelial cells, essential for maintaining vascular health and regulating blood pressure, playing a key role in modulating vascular tension and hemodynamics. NO is primarily produced by endothelial nitric oxide synthase (eNOS) in endothelial cells. In the presence of NADPH and oxygen, eNOS catalyzes the conversion of L-arginine to NO and L-citrulline. Once released, NO activates guanylate cyclase in vascular smooth muscle cells, thereby increasing the levels of cyclic guanosine monophosphate (cGMP). cGMP acts as a second messenger, causing vascular smooth muscle cells to relax and thus leading to vasodilation. Through this mechanism, NO can effectively regulate vasodilation, responding to physiological demands, such as increased blood flow during exercise or the redistribution of blood flow during tissue hypoxia.

Exercise can enhance the ability of endothelial cells to produce nitric oxide, thereby promoting vasodilation and improving hemodynamics. Studies have shown that exercise can increase the expression and activity of eNOS[7]. The mechanisms mainly involve shear stress (mechanical stimulation of blood vessels by blood flow), changes in calcium ion concentration in endothelial cells, and biochemical signals produced during exercise (such as cytokines and hormones). By increasing the expression and activity of eNOS, exercise maintains normal blood pressure and improves vascular function, playing a significant role in the prevention and treatment of hypertension and other cardiovascular diseases.

3.2. The Impact of Exercise on Inflammatory Responses

Inflammation's impact on endothelial cells is a key factor in the onset and progression of cardiovascular diseases. This process is primarily associated with an increase in oxidative stress and a decrease in the bioavailability of nitric oxide (NO). Studies have found that in states of inflammation, immune cells (such as macrophages and neutrophils) are activated and subsequently release inflammatory mediators. This also activates NADPH oxidase, leading to an excessive production of reactive oxygen species (ROS), thereby inducing oxidative stress.
Normally, there is an antioxidant defense system within cells, including enzymes (such as superoxide dismutase (SOD) and glutathione peroxidase) and non-enzymatic antioxidants (such as vitamins C and E), to neutralize excess ROS and maintain redox balance. However, numerous studies have found that inflammation downregulates antioxidant levels, leading to a disruption of the antioxidant defense system and a reduction in NO bioactivity. This could lead to endothelial lesions and plaque formation, increasing the risk of atherosclerosis. This indicates that inflammation not only causes acute damage to endothelial cells but also affects their self-repair and regenerative capacities, leading to long-term structural and functional damage to the vascular wall. It is a significant factor in the development of atherosclerosis and other cardiovascular diseases.

Exercise helps reduce inflammation by lowering the production of inflammatory cytokines and improving the function of immune cells. Studies show that regular exercise can significantly decrease levels of inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), thereby aiding in the alleviation of the body’s inflammatory state. This anti-inflammatory effect is crucial for protecting endothelial cells from inflammatory damage and helps prevent the development of cardiovascular diseases such as atherosclerosis. Furthermore, exercise can reduce oxidative stress and enhance the antioxidant capacity of cells, maintaining the bioactivity of nitric oxide (NO) and thereby improving vascular dilation ability[8, 9]. Therefore, as a non-pharmacological therapy, exercise plays an important role in maintaining cardiovascular health by reducing inflammatory responses and oxidative stress, and improving endothelial function.

3.3. Exercise and Lipid Level Regulation

Dysregulation of lipid metabolism is a key factor leading to endothelial cell dysfunction and atherosclerosis[10]. Exercise positively impacts endothelial cells by improving lipid metabolism, lowering levels of low-density lipoprotein (LDL), and increasing levels of high-density lipoprotein (HDL). Numerous studies confirm that aerobic exercise, in particular, effectively reduces LDL levels in the blood and increases HDL levels[11], helping to prevent the development of atherosclerosis and damage to endothelial cells[12].

3.4. Angiogenesis and the Improvement of Tissue Blood Supply

Angiogenesis, the formation of new blood vessels, is essential for maintaining tissue blood supply and oxygenation[13]. Exercise can promote angiogenesis, particularly through the increased expression of growth factors such as vascular endothelial growth factor (VEGF) [14]. This process aids in improving blood circulation, especially in muscles and other active tissues. Studies indicate that exercise not only promotes angiogenesis but also enhances tissue efficiency in utilizing oxygen and nutrients[13], which is of significant importance for the rehabilitation of cardiovascular disease patients and overall cardiovascular health.

4. Conclusion and Outlook

This review comprehensively explores the impact of exercise on endothelial cell function and its mechanisms, highlighting the critical role of physical activity in maintaining vascular health and preventing cardiovascular diseases. Exercise improves endothelial function through multiple mechanisms, including increasing the production of nitric oxide, reducing inflammatory responses, improving lipid metabolism, and promoting angiogenesis. These mechanisms work together to reduce the risk of cardiovascular diseases and improve overall cardiovascular health. As a simple, low-cost, and widely accessible intervention, exercise is crucial for individuals of all ages and health statuses. Regular and moderate exercise not only improves endothelial function but also enhances overall health and quality of life. Therefore,
incorporating exercise into daily life as a vital component of preventing and treating cardiovascular diseases is essential.

In summary, the positive effects of exercise on endothelial cell function underscore its significant role in cardiovascular health management. Future research should continue to explore the optimal forms and mechanisms of exercise to maximize its potential application in the prevention and treatment of cardiovascular diseases. By promoting the health benefits of exercise and providing targeted exercise guidance, the cardiovascular health of the public can be more effectively improved.

Although current research has made certain progress, there are still limitations in methodological design, sample size, and population diversity. Many studies rely on small samples and short-term interventions, which may not fully reflect the long-term effects of exercise on endothelial function. Moreover, research on specific populations (such as children and pregnant women) is relatively scarce, leading to a lack of sufficient scientific support for exercise recommendations for these groups. Given the limitations of existing research, future studies should focus more on evaluating the long-term impacts of exercise on endothelial function and how to develop personalized exercise plans based on individual differences. Additionally, with the advancement of technology, utilizing advanced measurement techniques and data analysis methods to assess the impact of exercise on endothelial function is an important direction for future research. This will help to more accurately assess the benefits of exercise on cardiovascular health and provide strong support for making evidence-based exercise recommendations.

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References


