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# Changes of Serum Endothelial Microparticles in Mice with Septic Lung Injury

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**Abstract**

Sepsis is an inflammatory response syndrome that causes the failure of multiple organs in the body. During the onset of sepsis, lung tissue is most vulnerable to damage due to its own physiological characteristics, thus forming an acute lung injury disease. As a carrier of inflammatory mediators, endothelial microparticles participate in endothelial function damage and may play an important role in acute lung injury. The purpose of this article is to explore the changes and mechanisms of serum endothelial microparticles in mice with septic lung injury. With 50 healthy mice as experimental subjects, the mice were divided into a septic lung injury observation group and a corresponding control group. Mice underwent cecal ligation and perforation to create an animal model of septic lung injury. Twelve hours after the model was successfully created, blood was drawn from the lung tissue of the mouse to detect the concentration of pro-inflammatory factor IL-1β, interleukin IL-6 and tumor necrosis factor TNF in the mouse serum, and detect the content of endothelial cell particles in the mouse serum, and use observe the ultrastructure changes of mouse lung tissue under microscope. The results of the study showed that compared with the control group, the serum pro-inflammatory factor IL-1β concentration level in the septic lung injury observation group increased by 18.4%, and the interleukin IL-6 concentration level increased by 21.5%. The level of tumor necrosis factor TNF increased by 24.8%. The content of endothelial microparticles in the serum of the observation group was (862.3±54.5)/μL, which was significantly higher than that of the control group (378.2±40.6)/μL. At the same time, the ultrastructure of the lung tissue of the observation group was destroyed.

**Keywords:** Septic Lung Injury, Mouse Serum, Endothelial Microparticles, Animal Model

## 1. Introduction

Sepsis is a systemic inflammatory response syndrome caused by infection. It is a common complication of severe trauma, burns, hypoxia, reperfusion injury and major surgical procedures. It is the number one cause of death in ICU patients. The lung is the most vulnerable target organ in sepsis. Acute lung injury occurs earliest and has the highest incidence. In sepsis, endotoxin stimulates inflammatory cells to produce a large number of inflammatory mediators and lipid metabolites, which can promote the response of many inflammatory cells in the chest lung mucosal tissue and the recruitment and oxidative activation of a large number of leukocytes, and further catalyze the production of inflammatory cell response factors.

Microparticles (MP) are tiny membranous vesicles that can be secreted by almost all cells. When cells are apoptosis or stimulated, microparticles can be produced [1]. The diameter of MP is about 0.1-1 microns, it can carry a variety of biologically active molecules, such as protein, DNA, RNA, etc. At present, research has confirmed that MP can be widely involved in the pathophysiological process of inflammation, coagulation, and immunosuppression, and play a wide role in the acute occurrence and early development of a variety of heart diseases, such as acute atherosclerosis, myocarditis, and pre-disease. Acute cardiopulmonary injury and acute chronic nephritis. Endothelial cell microparticles (EMPs) bubbles are a small sac-like bubble active substance normally released by endothelial microparticle cells after cortex activation or apoptosis. They are particles expressing CD31, CD105, and CD144, and their diameter is smaller than lump [2]. It has been

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confirmed that EMPs can be used as effective carriers of inflammatory mediators and adhesion molecules, participate in cell signal transduction, aggravate the damage of endothelial function, and play an important role in lung injury caused by sepsis [3].

In order to explore the changes and mechanisms of serum endothelial microparticles in mice with septic lung injury, this article has consulted a lot of relevant data. Among them, Takei gave a detailed introduction to the causes of sepsis and pointed out that the imbalance of inflammatory factors is the main cause of sepsis, at the same time, he pointed out that sepsis, as an inflammatory syndrome, can cause organ failure and hypofunction [4]. In his article, Giannella pointed out that sepsis is easy to cause acute lung injury, because lung tissue is the primary target organ of sepsis, he analyzed the current research status of septic lung injury and introduced several treatment methods [5]. Singh analyzed the role of endothelial microparticles in the body and pointed out that most endothelial microparticles are produced when endothelial cells undergo apoptosis or stimulation, and emphasized that endothelial microparticles, as a carrier of inflammatory mediators, are related to various diseases [6]. Berezin found through research that severe vascular leakage is widespread in patients with sepsis that is, vascular barrier function decreases and permeability increases, inflammatory factors, complement, lipids and other macromolecular substances in the blood can abnormally pass through the endothelial barrier to reach various tissues and organs [7]. Tian's clinical investigation on 56 patients with septic lung injury found that septic lung injury can cause the increase in the concentrations of pro-inflammatory factors IL-1β, interleukin IL-6 and tumor necrosis factor TNF, and it can also affect the patients, the ultrastructure of lung tissue has caused serious damage, emphasizing that all of this may be caused by the increased content of endothelial microparticles in the serum [8].

In the study of exploring the changes and mechanisms of serum endothelial microparticles in mice with septic lung injury, this article summarizes and analyzes the research experience and results of a large number of predecessors. In addition, this article has made some innovations in research content and detection methods. There are two specific innovations: First, this article uses a high-power microscope to observe the non-tissue ultrastructure of the modeled mouse, and uses the SPST-620 software to collect and analyze the statistical data of the detection results of the mouse lung tissue cell ultrastructure change. Which greatly improves the accuracy of the research results? Second, this study used cercal ligation and perforation (CLP) to create a sepsis acute lung injury mouse animal model, and observe the changes in the blood endothelium of the sepsis mice and the pro-inflammatory factor IL-1β and interleukin IL.

## 2. Sepsis's Barracks and the Effect of Endothelial Particles

### 2.1 Etiology and Pathology of Sepsis

Sepsis is often one of the complications of inflammatory patients, and it may also induce small septic shock and other multiple organ dysfunction Roche syndromes (Mods). The treatment of patients with sepsis symptoms is developing rapidly. Although there is no good clinical diagnosis and treatment technology and biological monitoring and treatment methods, the incidence of sepsis and the mortality of patients are still high. In the past, it was generally believed that the main cause of acute sepsis was caused by excessive immune response to the body's inflammatory immune response. However, more and more clinical research results have found that the pathological and physiological changes of sepsis are very complicated, including inflammation, disorders of immunity and the function of the body's coagulation system involve various physiological changes in the body's cellular immune function, metabolism and drug metabolism [9]. Early inflammatory cell proliferation factors proinflammatory factors and cell proliferation factors are closely related to the normal occurrence and early development of chronic inflammation, including the necrotic cytokine TN-α, interleukin Il-4, 16 and inflammatory interferon (IFN). Among them, TNF-α factor is the most important proinflammatory factor and cell metabolism factor in the early stage of inflammation, and it can play an important inhibitory role in the body's immune toxin defense response, and it is also an important immune mediator for toxins in the body to damage immunity. The main role of Il-1 in the treatment of acute sepsis is very similar to its TNF-α. Il-1 may trigger a variety of inflammatory chemical reactions through the expression in illβ and through the expression of TN-α Il-6 in plasma, the level of uric acid can be widely used as an important clinical predictor of the severity of sepsis.

In patients with sepsis, the balance between pro-inflammatory cytokines and anti-inflammatory cytokines is usually unbalanced. If not controlled, inflammatory cells cannot be activated effectively, and anti-inflammatory cytokines are insufficient,

and the body is prone to abnormal immune function. In addition, it has been reported that kinase inhibitors (Socks) for signal transduction of cell growth factors can also participate in the synthesis of these anti-inflammatory catalytic reactions [10]. Socks cells can bind to a receptor in a cell growth factor or directly interact with a Janus cell kinase (Jack). Socks cells can negatively regulate the signal transduction pathway between Jack/t's signal transduction and neural transcription cell activator (stat) by changing its phosphorylation response, thereby effectively reducing various inflammations for sepsis. The analysis of a large number of clinical data shows that the anti-hmcb1 antibody combination therapy can effectively and quickly alleviate lactation sepsis and reduce the mortality of mammals. Therefore, Hmgb-1 is widely considered to be a key late-stage cellular inflammatory response factor that may produce lethal suppression in advanced sepsis, and its positive level can be used as an important medical indicator for clinical monitoring of the severity of advanced sepsis and the prognosis of treatment. In addition, coagulate sepsis is always accompanied by abnormal blood coagulation, and may even cause diffuse intravascular coagulation.

Sepsis and central nervous system endocrine regulation immune system network in the early stage of treatment of sepsis, the nervous system quickly and directionally transmits various inflammatory immune signals to the central nervous immune system, so that it can continuously regulate neuroendocrine and regulate the immune system function to reduce the influence to control the normal occurrence and early development of sepsis [11]. A recent study showed that the neuroimmune system can even directly help regulate the recovery process of acute sepsis through its own various neurotransmitters. Hypothalamus adrenal pituitary and left adrenal (Hap) adrenal axis injury is one of the important anti-inflammatory and inhibitory pathways in the nervous system during acute sepsis. Severe injury of the hap adrenal axis will directly promote acute sepsis. Reoccur and continue to develop. The experimental results of a large number of autophagy gene cell knockout technologies indicate that the occurrence of autophagy cell disorder may be mainly due to the change of autophagy cell gene function, the acceleration of cell gene apoptosis and cell necrosis, which may induce acute sepsis. Autophagy is functionally activated in the early stages of some sepsis, and these dysfunctions also occur during the early pathological evolution of some sepsis, especially when it inhibits the formation of autophagy lysosome cells. A large number of clinical studies have confirmed that the direct synthesis of pro-inflammatory leukocyte kinase factors in sepsis is closely related to the protein kinase (MAPK) pathway activated by mitosis, which directly activates a variety of downstream protein transcription kinase factors and upstream proteins. The kinase molecule thus plays a role [12]. Anti-inflammatory chronic cell mediator factor inhibition can effectively antagonize the anti-inflammatory cell mediator and thereby inhibit the continued development of chronic inflammation, including factors il4, il10, transforming growth factor β (tag-β), il-13, etc.

### 2.2 Formation and Function of Endothelial Microparticles (EMP)

Endothelial microparticles (EMP) are endothelial-derived microparticles produced by umbilical vein endothelial cells stimulated by complement complexes. Under this pathological reaction condition, the endothelial leukocytes in the blood vessel are first stimulated by various external hormones, and then automatically produce high-concentration levels of α-amp enzyme and participate in the pathological reaction process of various enzymes. Now, amp hormone, as an important characteristic marker of vascular endothelium and deep vascular tissue function, has gradually increased significantly in the early clinical treatment of related vascular diseases. At present, EMP is mainly released when it receives pathological stimuli or is stimulated to activate white blood cells in the vascular endothelium. When white blood cells in the vascular endothelium are activated by factors, the function of membrane asymmetry disappears and the cell reorganization process of cytoskeletal endothelial protein is the process of vascular endothelium. The important process of leukocyte hormone releasing receptor imp [13]. When the cell particles in the human vascular cortex endothelium are directly stimulated by different human pathological response factors, they will automatically produce a variety of vascular endothelial cell particles with different pathological phenotypes, which are usually used as important markers for pathological detection of Ape. The Imps cell is a white microencapsulated vesicle with a diameter of usually less than 4μm, which is gradually released from the surface of the upper cell membrane of the endothelial cell during the process of cell activation or destruction of apoptotic cells. They not only have the characteristics of being some of the main antigens of endothelial cells, but they can effectively and

accurately reflect the tissue structure and physiological functions and status of human endothelial cells. The sphingomyelin bilayer of the normal phospholipid cell membrane consists of the inner and outer layers of sphingomyelin phosphatidylcholine and the inner layer of sphingomyelin phosphate, while the inner layer is composed of phosphatidylethanolamine and the outer layer of Ps phosphate. Ps cannot be exposed to normal cell membranes. On the outer surface, the cell membrane is difficult to germinate and form phospholipid particles [14].

Studies have shown that endothelial cell particles can not only be a new cycle marker for the discovery of endothelial cell dysfunction, but also play an important clinical biological detection role in skin inflammation, vascular tissue damage, endothelial cell dysfunction and arterial thrombosis. Elderly patients with chronic cardiovascular disease, diabetes, pulmonary vascular disease and other kidney diseases will also increase the level of cortical endothelial cell particulate pigment in their blood cells [15]. Endothelial cell micro particles, especially those camp derived from mature endothelial micro particle cells, can express a variety of adhesion molecules on the surface of other outer membrane cells, such as Cd54 (icam-1), Cd62p (p-cell selecting), Cd62e and cd31. The specific antagonist of NFKB, pyrimidine dithiocarbonate, can inhibit this effect of endothelial microparticles. This result indicates that the inhibition of endothelial microparticle factor can inhibit the kb enzyme and signal transduction in transcriptional cytokines by stimulating endothelial nucleic acid. The enzyme pathway functions to greatly increase its large-scale expression of the inflammatory cytokine Icam-1. Researchers have found that a large amount of Il-6 release is related to a large amount of Il-6 release process, which means that a large number of endothelial particles are formed. It is related to a classic process of mass production of inflammatory endothelial cell micro particles, so that the inhibition of endothelial cell micro particles can promote the mass production of inflammatory cytokines.

Some studies have found that endothelial particles may inhibit the development and stability of atherosclerotic plaques and induce differentiation of endothelial progenitor cells. In the clinical study of primary hypertension (eh) and acute endothelial blood pressure micro particles, it was found that there are positive and negative correlations between the concentration of acute endothelial blood pressure micro particles and the variability of endothelial blood pressure in some patients with eh type. At the same time, the higher the endothelial blood pressure of patients with eh type, the higher the level of amp particles [16]. Animal clinical experiment results show that endothelial mesenchyme micro particle cells can recruit more neutrophil mitochondrial cells and directly release secondary bone marrow cell peroxidase polymerase by triggering a cascade polymerization reaction between bone marrow cell interstitial factors. This may lead to chronic lung injury, which provides new treatment ideas for the treatment of chronic lung injury diseases with drugs. Researchers found that the blood Cd31 concentration of patients with chronic vascular obstructive cardiopulmonary vascular disease (Coped) and acute emphysema in the early stage was increased positively or negatively with the severity of the patient's cop infection, and with the micro vascular blood in the patient's lungs. The perfusion concentration showed a negative or positive correlation. In addition, in the two patients of C and Coped, an auxiliary suppression of T-type white blood cell 18 (th18)/8 adjustable suppression of T-type white blood cell (Tag) autoimmune function imbalance was also found, and endothelial particles increased significantly. There may be a positive correlation between them, which is most likely a sign of the effect of endothelial particles。

## 3. Related Experiments on Changes of Mouse Serum Endothelial Microparticles

### 3.1 Selection of Experimental Animals

In this experiment, 50 mice were selected as the research objects, aged 6 to 8 months, and weighing (60±5.0) g. The laboratory that has been disinfected and pollution-free is selected as the experimental site, and the laboratory temperature is 22-26 ℃, the moisture in the air is about 15.8-20.6%, the laboratory maintains ventilation to ensure sufficient oxygen content in the air, and raises them in separate cages according to the national standard whistle tooth feeding specifications. Check the health of the mice before the experiment and exclude the body mice with abnormal health. The mice were randomly divided into a control group and a sepsis lung injury observation group. The mice in the control group were left untreated, and the mice in the observation group were created as an animal model of acute lung injury in sepsis.

### 3.2 Experiment Related Materials

The main equipment used in this experiment: flow cytometer, magnetic stirrer, -80℃ low

temperature refrigerator, DYG-28A electrophoresis tank, liquid nitrogen biological container, vacuum oven, slice paraffin embedding machine (purchased from Sino pharm Chemical Reagent Co., Ltd. Company). ACL-V8 animal ventilator, POWER-8 model physiological signal acquisition instrument, LEICB-218 slicer. BX230 electron optical microscope, image analyzer (Jiangsu Chengdu Analytical Instrument Co, Ltd.), steam sterilization equipment, surgical instruments, complete Automatic blood analyzer, BCL-210A Ron sheng water tank (Jiangsu Katia Electric Co., Ltd.), slicer (produced by a German company), electronic balance, PH detector, centrifugal acceleration instrument, MP-1600T real-time quantitative instrument. The relevant reagents required for the experiment are shown in Table 1.

*Table 1.* **Related reagents required for the experiment**

| Group | Usage amount | Source |
|---|---|---|
| Annexin V flow cytometry antibody | 460ml | United States, Biosciences |
| CD31 flow antibody | 150ml | United States, Biosciences |
| CD45 flow antibody | 350ml | United States, Biosciences |
| D-Hanks fluid | 720ml | China, Shanghai Sheng gong Company |
| Hydrochloric acid | 450ml | China, Yale Company |
| Penicillin | 300mg | Japan Sanwa Kimono |
| Sodium chloride | 280ml | Fisher Instruments, Germany |
| Calf serum albumin | 520ml | Gibson Corporation United States |
| Fluorescent albumin | 900ml | United States, Costar Corporation |

### 3.3 Establishment of Sepsis Lung Injury Animal Model and Specimen Collection

The cercal ligation and perforation operation were used to create an animal model of septic lung injury. Inject 0.1% sodium pentobarbital (50mg/kg) into the abdominal cavity to observe the anesthesia effect and observe the changes in breathing and heartbeat. The head, limbs, abdomen skin, iodophor cotton balls are routinely disinfected in the supine position and sterile whole towels are laid. Make a 2.0 cm long longitudinal incision in the middle of the midline of the abdomen. Carefully open the abdominal cavity, find the cecum and pull it out of the abdominal cavity, so as not to damage the mesenteric blood vessels. Half of the blind end is ligated with 1-0 silk thread. Puncture the cecum on the other side of the cecum with a 12-gauge puncture needle. Place the cecum back into the cecum after opening the abdominal cavity. At the end of the operation, 3ml of 5% sodium chloride solution was injected subcutaneously, and the animals were kept in cages. After 12h, 24h and 48h, 2% sodium pentobarbital solution (50mg/kg) was injected into the abdominal cavity for anesthesia. The abdomen is disinfected with iodophor and covered with a sterile whole towel. After the abdomen was disinfected, the abdominal cavity was opened, the abdominal aorta was cut, and the mice were killed. The right main bronchus was ligated, and the left lung was flushed with saline with a 5ml syringe, then the right lung was taken out, carefully separated, and the upper lobe of the right lung was stored in a liquid nitrogen tank for total RNA extraction from the tissue. After cleaning the middle lobe of the right lung, weigh the wet weight and place it in an electric furnace at 65°C. After 24 hours, weigh the dry weight and put the lower lobe of the right lung in 8% paraformaldehyde for pathological examination.

### 3.4 Detection of TNF-α, IL-6, IL-1β Concentration Levels

At 6, 12, and 24 hours after the successful creation of the sepsis lung injury animal model, two corresponding live mice of different groups were selected in the time axis node. After anesthesia, the eyeballs of the mice were successively removed and then a drop of blood was taken, centrifuged (5000r /min) 20min, then takes a drop of supernatant and put it in the refrigerator at -30℃ for low-temperature storage. The Elisa method is used to detect the serum tumor inflammatory necrosis factor mouse TN-α (malignant tumor inflammatory necrosis factor-α). Jill -6 (tumor leukocyte necrosis interleukin-6), pro-inflammatory response factor Il-1b (tumor leukocyte necrosis interleukin-1β) and then take out the cell tissue of the upper lobe of the lung of living mice stored in a low-temperature refrigerator, and filter. The test paper quickly absorbs part of the exudate and local blood on the surface of the tissue, puts a small amount of liquid nitrogen in a mortar, stirs and grinds, adds a small amount of physiological phosphate water 0.7ml, stirs well, centrifuges 6000r/min×10min, and takes the supernatant. The solution is stored in the refrigerator at -30℃ and used for the analysis and detection of the tumor inflammatory necrosis factor of the upper lobe of the mouse lung.

**3.5 Determination of Serum Endothelial Microparticle Concentration Level**

Take 400 mL of blood from non-tissue parts of mice, centrifuge at 360×g for 20 minutes to obtain rich platelet combined plasma, and then centrifuge at 2000×g for 10 minutes to obtain acute anemia and white plate combined plasma. Take 30ml of the acute anemia egg plate and place it in the plasma, add 10ul of phycoerythrin (Pea) or use a fluorescent agent to fully combine the labeled cell antibody with the Cd144 antibody and mix well. Just avoid sunlight at room temperature and incubate for 35min. The absolute concentration of 100/ml counts cell microspheres 60l and 2000ml phosphate buffered saline (Pubs), and the flow cytometer can be used after mixing. Use a blank group without fluorescently labeled antibody to determine the fluorescence intensity threshold before each test. The endothelial micro particles are defined as being less than 5um in diameter and CD144 positive. Read 20,000 absolute counting microspheres and count the number of CD144-positive endothelial micro particles.

**3.6 Pathological Observation of Mouse Lung Tissue**

Take a mouse with 0.7cm×0.7cm×0.7cm in the upper layer of the lower lobe of the left chest and right lung of a mouse and place it in 5% paraformaldehyde for fixation. Routinely perform dehydration, paraffin embedding, histopathological section, and hematoxylin-eosin. He was stained, and the histology and morphology of the left lower lobe of the left lung were observed carefully under a light microscope. In addition, a case of mouse left liver and right lung lower lobe cell tissue 2mm×2mm×2mm was selected, placed in 4.5% glutaraldehyde fixative solution, stored continuously at 0-8°C, washed with saline, fixed, and immersed in ethanol and acetone gradient for dehydration, acetone gradient immersion, sectioning, then paraffin-embedded film polymerization, urinal acetate and white lead oxide citrate, then ultra-thin cell sectioning can be performed, and the ultrastructure can be observed under a transmission electron microscope.

**4. Results and Correlation Analysis of Changes of Various Indexes in Mouse Serum**

**4.1 Analysis of the Detection Results of Serum Pro-Inflammatory Factor Concentration and Endothelial Microparticle Content in Septic Lung Injury Mice**

In order to understand the changes of lung edema in mice after septic lung injury, this article calculated the wet-to-dry ratio W/D of lung tissue. The results of the study showed that the non-tissue wet-to-dry ratio of the septic lung injury observation group was significantly higher than that of the control group. The W/D value of lung tissue observed 6 hours after septic lung injury was 6.36, the W/D value of the control group was 3.27, the W/D value of lung tissue observed 12 hours after the toxic lung injury was 8.55, and the W/D value of the control group was W/D. The D value was 3.51, the W/D value of lung tissue observed 24 hours after toxic lung injury was 10.48, and the W/D value of the control group was 3.76, indicating that septic lung injury can increase the wet-to-dry ratio of lung tissue in mice. The detailed data of the wet-to-dry ratio of lung tissue of the observation group and the control group after septic lung injury, as show in Table 2.

*Table 2.* **Detailed data of the wet-to-dry ratio of lung tissue of the observation group and the control group after septic lung injury**

| Group | 6 hours later | 12 hours later | 24 hours later | 36 hours later |
|---|---|---|---|---|
| Control group | 6.36±1.2 | 8.55±1.4 | 10.48±1.5 | 12.51±1.9 |
| Observation group | 3.27±0.8 | 3.51±1.1 | 3.76±0.9 | 4.07±1.3 |
| P | 0.0247 | 0.0214 | 0.0185 | 0.0153 |

The results of the study showed that the serum pro-inflammatory factor IL-1β levels in the observation group after septic lung injury were significantly higher than those in the control group, and the serum pro-inflammatory factor IL-1β levels in the observation group 12 hours after septic lung injury. The concentration level was higher than 6 hours, and the serum pro-inflammatory factor IL-1β concentration in the observation group was higher than 12 hours after 36 hours after septic lung injury, and the serum pro-inflammatory factor IL-1β concentration in mice 42 hours later. The level no longer changes. The average levels of serum-negative leukocytes and interleukin 5 and IL-6 concentrations in the treatment observation group of two mouse patients after sepsis lung injury were significantly different than those in the experimental control group, and the mean difference had important statistical significance ($p<0.05$) ). The elevated levels of flavin-5 and il-6 in leukocytes gradually increased with the continuous passage of time, and no longer changed after 37

hours. After septic lung injury, the treatment observation group found that the average level of tumor cell necrosis-related factor ten concentration in the infusion serum of the experimental mice was significantly higher than that of the experimental control group. The mean difference has important statistical significance ($p<0.05$). The average concentration of cell necrosis-related factor ten in mice showed a significant upward and downward trend with the passage of observation time, and the concentration no longer changed 35 hours after septic lung injury. The results of the study show that septic lung injury can increase the serum levels of pro-inflammatory factors IL-1β, interleukin IL-6 and tumor necrosis factor TNF. The relevant data are shown in Figure 1.

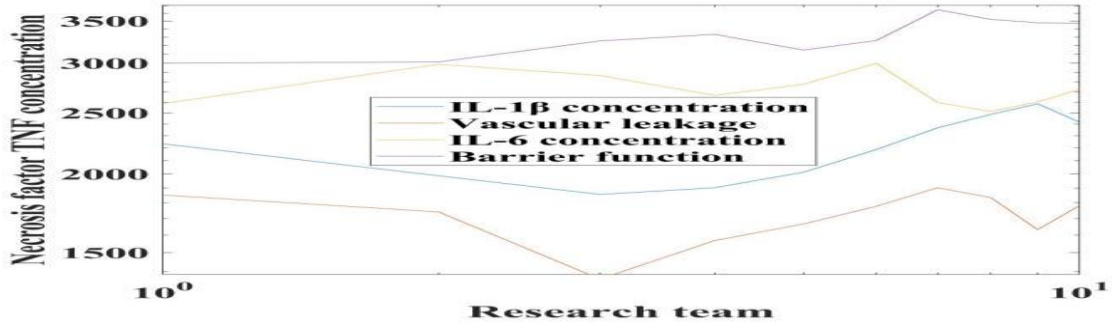


Figure 1. **The effect of septic lung injury on the serum levels of pro-inflammatory factors IL-1β, interleukin IL-6 and tumor necrosis factor TNF**

From the data in Figure 1, it can be seen that sepsis lung injury will increase the serum levels of pro-inflammatory factors IL-1β, interleukin IL-6 and tumor necrosis factor TNF. Compared with the control group, sepsis in the lung injury observation group, the serum pro-inflammatory factor IL-1β concentration level increased by 18.4%. The interleukin IL-6 concentration level increased by 21.5% and the tumor necrosis factor TNF concentration level increased by 24.8%.

The experimental results showed that the serum endothelial micro particle content of the observation group after septic lung injury was significantly higher than that of the control group, and there was a statistical difference between the two groups ($P<0.05$). Six hours after the sepsis lung injury model was established, the content of endothelial micro particles in the serum of the observation group was (662.3±54.5) pieces/μL, and the content of the control group was (369.5±32.8) pieces/μL. The serum endothelial microparticle content in the observation group was (862.3±54.5)/μL 12 hours after the establishment of the sepsis lung injury model in the observation group, and (378.2±40.6)/μL in the control group, and the serum endothelial microparticle content in the observation group was 36 hours later (1047±75.6)/μL, while the data of the control group was not significantly different from 12 hours ($P>0.05$). The results of the study show that septic lung injury can increase the content of endothelial microparticles in the serum of mice. The specific data are shown in Figure 2.

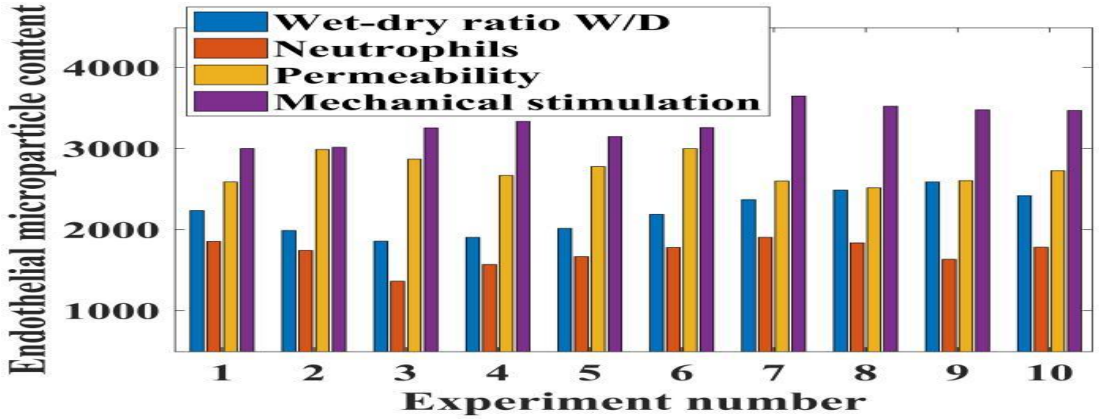


Figure 2. **The effect of septic lung injury on the content of endothelial microparticles in serum of mice**

From the data in Figure 2, it can be seen that septic lung injury can increase the content of endothelial microparticles in the serum of mice. After 12 hours of modeling, the index of endothelial microparticles in the serum of the observation group was (862.3±54.5)/ μL. Significantly higher than the control group (378.2±40.6)/μL.

**4.2 Analysis of the Effect of Septic Lung Injury on the Ultrastructure and Function of Lung tissue in Mice**

The experimental results showed that after the sepsis-induced lung injury model was established, the lung tissue of the control group was pink, with no damage capsules and good elasticity. In the septic lung injury observation group, lung tissue congestion was observed 3 hours later. At 6, 12, and 24 hours, not only the lung tissue was obviously congested, but also edema and bleeding spots were found. Some mice have spotted hemorrhage and intrapleural hemorrhage. Microscopic observation revealed that the lung tissue structure of the control group was normal, there was no exudate from the alveolar cavity, the interalveolar septum was slightly enlarged, and the bronchial mucosal epithelium was not damaged. In the observation group, there was no obvious change within 5 hours, but the pathological changes of lung tissue became more serious over time 6. 12 hours later, alveolar pus rock exudate, individual trough costal emphysema, various degrees of trough wall expansion, excessive lung interstitial, formation of telangiectasia, most neutrophils and a few. The infiltration of mononuclear cells, the expansion and bleeding of interstitial venues, the above symptoms are especially heavy near the pleural cavity, bronchial epithelial cilia are inverted, most of the cell fragments in the lung intracellular group, and the endothelial cells of the pulmonary capillaries thicken. The results of the study show that septic lung injury can cause a large number of cell debris and thickening of capillary endothelium in the lung tissue of mice. The specific data are shown in Figure 3.

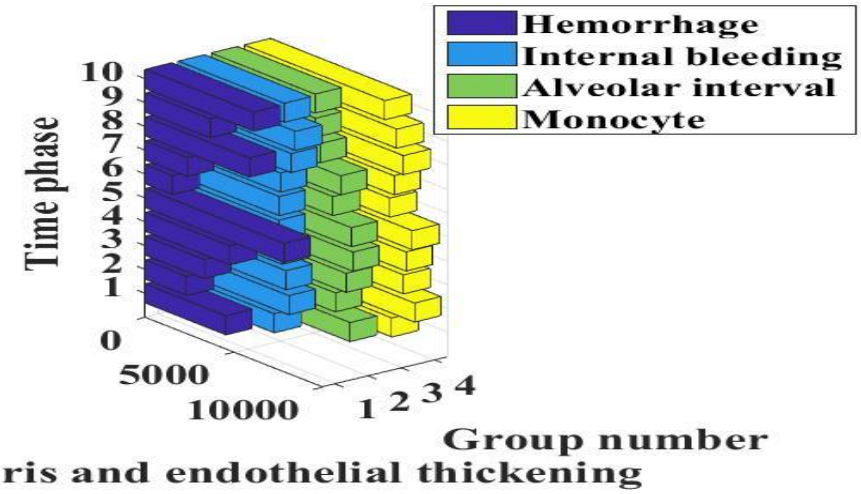


Figure 3. **Effects of septic lung injury on cell fragments and capillary endothelial thickness in mouse lung tissue**

From the data in Figure 3, it can be seen that septic lung injury can cause a large number of cell debris and capillary endothelial thickening in the lung tissue of mice. According to calculations, the cell debris in the lung tissue of the observation group is 4.25 times that of the control group. The thickness of vascular endothelium increased by 16.7%.

Studies have found that when sepsis occurs, the body releases a large number of inflammation-inducing and anti-inflammatory mediators, which interact to form a waterfall chain reaction. Excessive inflammatory mediators often lead to dysfunction of remote organs. Under the stimulation of a large number of nan-x and IL-1β hormones, eosinophilia neutrophil mitochondrial cells continue to accumulate in the micro vessels that enter the alveoli, continuously activate it and release a series of damaged cell mediators, causing various. This kind of diffuse late-stage alveolar membrane damage may eventually lead to acute late-stage lung injury. According to this clinical study, the typical lung injury of sepsis is based on the lung injury of the capillaries and mucous membranes in the alveoli, and the severe lung is mainly characterized by pulmonary angioedema with osmotic hypertension and microtubule pulmonary vasodilation injury. Infectious disease,

trauma and pulmonary shock were initially confirmed. The clinical features are mainly acute pulmonary mucosal edema of neutrophils rich in a variety of proteins and enzymes, diffuse alveolar mucosal damage, and vitreous mucosa edema formation, pulmonary aeration, severe hypoxemia and acute respiratory failure. One of the important links that affects the pathological and physiological diagnosis mechanism is due to the severe damage of the blood flow barrier of the lung (the lung, that is, the capillary protective membrane in the alveoli) and the increase in the permeability of the pulmonary capillaries. This may lead to severe acute pulmonary angioedema and affect the normal pulmonary gas-energy exchange physiological function that enters the lungs. The results of the study show that sepsis lung injury can greatly reduce lung function the specific data is shown in Figure 4.

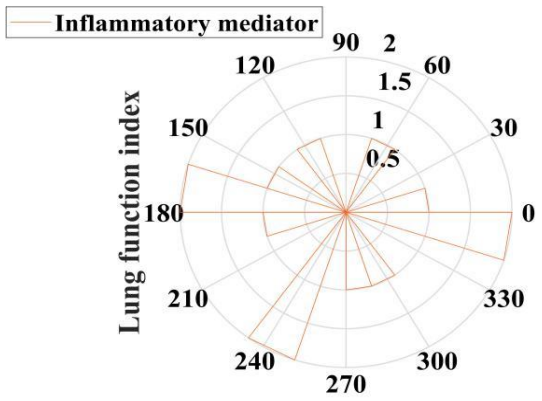


Figure 4. **Decreasing effect of septic lung injury on lung function index**

It can be seen from Figure 4 that septic lung injury can greatly reduce lung function, and pulmonary edema caused by septic lung injury can reduce the lung function index of mice by 35.8%.

**5. Conclusions**

(1) Sepsis is an inflammatory response syndrome, which can lead to the failure of multiple organs of the human body, which is extremely harmful to the life and health of all human beings, and the number of patients suffering from it is increasing year by year. During the onset of sepsis, lung tissue is most vulnerable to damage due to its own physiological characteristics, thus forming acute lung injury disease. As a carrier of inflammatory mediators, endothelial microparticles are involved in endothelial function damage and may play a role in acute lung injury. Important role, this article uses this as a starting point for research.

(2) The results of the study showed that compared with the control group, the serum pro-inflammatory factor IL-1β concentration level in the septic lung injury observation group increased by 18.4%, the interleukin IL-6 concentration level increased by 21.5%, and the tumor necrosis factor. The content of endothelial microparticles in the serum of the observation group was (862.3±54.5)/μL, which was significantly higher than that of the control group (378.2±40.6)/μL. At the same time, the lung tissue of the observation groups the ultrastructure is destroyed.

(3) Septic lung injury can cause a large number of cell debris and capillary endothelial thickening in the lung tissue of mice. It is calculated that the cell debris in the lung tissue of the observation group is 4.25 times that of the control group. At the same time, the thickness of the capillary endothelium increases by 16.7%. Toxic lung injury can greatly reduce lung function, and pulmonary edema caused by septic lung injury can reduce the lung function index of mice by 35.8%. The summary of the full text shows that septic lung injury can cause changes in endothelial microparticles, and the increase in endothelial microparticle concentration is one of the main causes of septic lung injury.

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