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# T cell mitochondrial function was used to assess the severity and exacerbation risk of coronary artery disease

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# Abstract

### Objective

Exploring the Correlation between Mitochondrial Function of T Cells and the Severity of Coronary Artery Disease.

### Methods

A total of 118 patients with coronary heart disease were divided into 32 cases in the ACS group and 25 cases in the stable angina pectoris (SAP) group. Meanwhile, 61 non-coronary heart disease patients who were hospitalized during the same period were selected as the control group. The expression levels of CD3 + T lymphocytes, CD4 + T lymphocytes and CD8 + T cells, CD4-MM, CD8-MM,CD4-MMP,CD8-MMP in the plasma of the three groups were detected by a flow cytometry instrument and the human lymphocyte mitochondrial function.

### Results

In the SAP and ACS groups, CD4MMP and CD8MMP were significantly lower than those in the control group, while CD4MM was significantly higher than that in the control group($P<0.05$).However, there were no differences in CD4MMP, CD8MMP, and CD4MM between the SAP group and the ACS group($P > 0.05$). CD4MM in the STEMI group was higher than that in the UA/NSTEMI group.

### Conclusion

In the peripheral blood of patients with coronary heart disease, the levels of CD4MMP and CD8MMP are significantly decreased, while CD4MM is significantly increased. These indicators can be used as predictive markers for the occurrence of coronary heart disease.


# Introduction

Cardiovascular disease (CVD) continues to increase in prevalence and death worldwide and remains the leading cause of serious disease burden and death worldwide. Coronary atherosclerotic heart disease, or coronary artery disease (CAD), is one of the most common CVD and the leading cause of death in middle-aged and older adults[1]. CAD is a chronic inflammatory disease that involves many large and medium-sized arteries. Severe coronary artery stenosis and/or unstable AS plaque rupture can lead to vascular embolism, myocardial ischemia, angina pectoris, myocardial infarction, arrhythmia and even sudden death, seriously threatening human life and health. In 2016, about 2.4 million people died from ASCVD, accounting for 61 percent of all CVD deaths and 25 percent of all deaths[2].

CAD is a long-term, progressive disease with multiple clinical manifestations, From asymptomatic to stable angina, acute coronary syndrome (ACS), heart failure (HF), and sudden cardiac death (SCD)[3].

The development of this series is accompanied by changes in the level of inflammation in the body and immune cells.

The results of flow cytometry and single-cell RNA sequencing showed that T cells were the most abundant immune cells in AS plaques[4]. Studies have shown that inflammation and autoimmune diseases caused by immune dysfunction will increase the risk of AS, so the normal metabolism of T cells may be one of the risk factors for CAD and AS. In vivo, the initial CD4 + and CD8 + T cells are activated and differentiated into different subgroups under the stimulation of different cytokines. For example, CD4 + T cells can differentiate into helper T cells (Th) and regulatory T cells (Treg), and CD8 + T cells can differentiate into cytotoxic T lymphocytes. The main characteristic subsets of Th are: 1) Th1 secretes interferon (IFN)-γ; 2) Th2, secreting IL-4, IL-5, IL-13; 3) Th17 secreting IL-17 and IL-22.

Mitochondria are the core of T cell metabolism, and mainly provide energy for T cells in the form of OXPHOS and fatty acid oxidation (FAO), so that they can normally differentiate and proliferate and play an immune role. There is growing evidence that the metabolic status of T cells influences their differentiation, immune response and role in disease progression[5, 6]. Initial CD4 + T cells rely on OXPHOS to efficiently produce ATP, but after activation, in order to produce ATP more quickly, they turn to aerobic glycolysis. Both aerobic glycolysis and OXPHOS are necessary for CD4 + T cell proliferation and survival, while the former is also a Teff cell-dependent metabolic pathway for IFNγ production[6]. acetyl-coA carboxylase (ACC1) is A key enzyme in fatty acid synthesis, and inhibition of ACC1 can lead to the transformation of Teff into Treg cells. Obese patients show abnormal changes in fatty acid metabolism and the ACC1 pathway is activated. In this environment, the production of Teff cells is activated, while the production of Treg cells is inhibited[7]. Studies have shown that the proportion of Th17/Treg cells increased and the proportion of Treg cells decreased in AS patients, and the imbalance in the proportion and function of Teff and Treg cell subsets is an important reason for the occurrence and development of AS plaques[8]. Th17 cells mainly rely on glycolysis and glutamine metabolism pathway for energy, Treg cells mainly rely on FAO pathway for energy. Inhibiting glycolysis and promoting FAO can promote the development and differentiation of Treg cells and inhibit the differentiation of Th17 cells. Studies have shown that inflammatory stimulation can increase the glycolysis rate of Th1 and Th17, increase glucose intake, and promote the proliferation and differentiation of Teff cells[7, 8]. Treg cells mainly rely on FAO, and changes in metabolic pathways determine the fate of CD4 + T cells in the differentiation process and play an important role in maintaining their respective immune functions[9].

## Materials and methods

# Patients and controls

In the Wenzhou People's Hospital, a total of 52 patients diagnosed with coronary heart disease through coronary angiography and having one or more coronary artery stenosis of at least 50% were included. These patients with coronary heart disease were divided into the stable angina pectoris (SAP) group (n = 25) and the acute coronary syndrome (ACS) group (n = 32) (including ST-segment elevation myocardial

infarction (STEMI) (n = 21) and unstable angina pectoris (UA) and unstable ST-segment elevation myocardial infarction (USTEMI) (n = 11)). The definition of SAP was based on the following criteria: typical exertional chest pain symptoms during exercise test and a ST segment depression amplitude greater than 1 millimeter. The definition of ACS was based on clinical manifestations (chest pain within 12 hours), electrocardiogram (ECG) changes (ST segment changes and/or inverted T waves), and positive myocardial troponin detection. 17 patients with a stenosis degree of less than 50% as shown by coronary angiography or coronary CTA were diagnosed with coronary atherosclerosis (CAS). Sixty-one non-coronary heart disease patients who were hospitalized during the same period were selected.

The following exclusion criteria were used: previous myocardial infarction within 6 months, inflammatory conditions likely to be associated with an acute phase response, neoplastic disease, advanced liver disease, autoimmune disease or current use of immunosuppressive agents. The cardiovascular risk factors include the smoking, hypertension and diabetes mellitus. The laboratory tests were measured at Wenzhou People's Hospital: total cholesterol triglycerides, low-density lipoprotein (LDL), highly sensitive C-response protein (Hs-CRP), troponin I (CTn I) and N-terminal pro-brain natriuretic peptide (NT-pro BNP). This work was approved by Ethics Committee of Wenzhou People's Hospital (Approval no. KY-D-2024-234) and was performed in accordance with the Declaration of Helsinki. Written informed consents were obtained from all patients and healthy donors prior to their participation.

## Experimental equipment and reagents

Flow cytometer model: DxFLEX (Beckman Coulter Biotech (Suzhou) Co., Ltd.)

Reagents: CD4FITC/CD45RA PC5.5/CD62L PC7/MitoDye/CD8AC7 detection kit, CD279 antibody, CD28 antibody, mitochondrial probe, hemolysin.

**T cell subsets and mitochondrial function assessment technique.**

Using a flow cytometry instrument to detect fluorescent mitochondrial probes, the original fluorescence intensity is measured. Subsequently, the original fluorescence values are corrected using the human lymphocyte mitochondrial function assessment data analysis software, and the mitochondrial mass (MM) and the percentage of mitochondrial low membrane potential (MMP) are output, which represent the indicators of mitochondrial dysfunction.

# Statistical analysis

Data are expressed as mean 6 standard deviation (s.d.). Comparisons between groups were analysed by two-tailed Student’s t-test or analysis of variance (ANOVA) test when appropriate. For normally distributed data, differences between groups were evaluated using Tukey’s test, and associations were assessed using Pearson’s correlation coefficient. For non-normally distributed data, differences between groups were evaluated using the non-parametric

Mann–Whitney U-test, and associations were assessed using a Spearman's rank correlation coefficient. A value of P < 005 was considered statistically significant. All statistical analyses were performed using SPSS (version 27).

# Results

There were no statistically significant differences in smoking and atrial fibrillation,serum creatinine, BMI among the three groups of patients (P > 0.05). There were significant differences in age, gender, hypertension, diabetes, heart failure, total cholesterol, LDL and HDL, the three groups of patients (P<0.05).Table 1.

Table 1
Comparison of general data among the three groups of patients

| | control group(n = 61) | SAP(n = 25) | ACS (n = 32) | F/χ2 | P |
|---|---|---|---|---|---|
| age | 53.13 ± 9.87 | 63.24 ± 10.73 | 60.97 ± 13.65 | 9.484 | <0.001* |
| sex | 33(54.1%) | 11(44%) | 8(25%) | 7.210 | 0.027* |
| Smoking | 45(73.8%) | 18(72%) | 16(50%) | 5.725 | 0.057* |
| Hypertention | 42(68.9%) | 5(20%) | 9(28.1%) | 23.553 | <0.001* |
| Diabetes | 49(80.3%) | 11(44.0%) | 20(62.5%) | 11.283 | 0.004* |
| Atrial fibriation | 58(95.1%) | 25(100%) | 29(90.6%) | 2.563 | 0.278 |
| Heart Failure | 58(95.1%) | 25(100%) | 23(71.9%) | 15.965 | <0.001* |
| Creatinine | 66.70 ± 19.64 | 67.61 ± 12.71 | 79.07 ± 36.13 | 2.764 | 0.067 |
| Total cholesterol | 5.26 ± 1.32 | 4.50 ± 1.09 | 4.49 ± 1.40 | 4.869 | 0.009* |
| LDL-C | 3.22 ± 1.02 | 2.59 ± 0.75 | 2.75 ± 1.00 | 4.604 | 0.012* |
| HDL | 1.37 ± 0.34 | 1.31 ± 0.64 | 1.11 ± 0.34 | 3.958 | 0.022* |
| BMI | 23.92 ± 3.18 | 24.47 ± 3.11 | 24.98 ± 3.73 | 1.090 | 0.340 |
| There were significant differences in age, gender, hypertension, diabetes, heart failure, total cholesterol, LDL and HDL, the three groups of patients *(P<0.05). | | | | | |

There was no statistically significant difference in the levels of peripheral blood T lymphocyte subsets among the three groups (P > 0.05). Except for CD8-MMP□CD4MM(P<0.05).Table 2.

Table 2
Comparison of peripheral blood T lymphocyte subgroups levels among the three groups of patients(□±□, %)

| T lymphocyte | control (n = 61) | SAP(n = 25) | ACS(n = 32) | F | P |
|---|---|---|---|---|---|
| CD3 | 1205.17 ± 494.45 | 1013.85 ± 299.28 | 1142.46 ± 299.28 | 1.761 | 0.177 |
| CD4 | 706.22 ± 292.69 | 606.71 ± 153.24 | 666.65 ± 209.85 | 1.450 | 0.239 |
| CD8 | 402.66 ± 242.65 | 340.89 ± 183.77 | 376.87 ± 222.16 | 0.673 | 0.512 |
| CD4-MMP | 21.19 ± 21.58 | 13.09 ± 4.78 | 15.86 ± 6.24 | 2.649 | 0.075 |
| CD8-MMP | 33.14 ± 13.77 | 23.10 ± 6.04 | 25.08 ± 11.13 | 8.595 | <0.001* |
| CD4-MM | 2.91 ± 1.24 | 3.56 ± 1.14 | 3.54 ± 1.14 | 4.346 | 0.015* |
| CD8-MM | 2.70 ± 3.53 | 3.12 ± 1.03 | 3.09 ± 1.05 | 0.332 | 0.178 |
| CD4-PD1 | 117.16 ± 59.51 | 95.48 ± 28.55 | 106.81 ± 37.89 | 1.812 | 0.168 |
| CD8-PD1 | 83.00 ± 61.77 | 77.28 ± 37.55 | 82.97 ± 43.71 | 0.115 | 0.892 |
| There was no statistically significant difference in the levels of peripheral blood T lymphocyte subsets among the three groups (P > 0.05). Except for CD8-MMP□CD4MM(P<0.05). | | | | | |
| Further analysis revealed that in the SAP and ACS groups, CD4MMP and CD8MMP were significantly lower than those in the control group, while CD4MM was significantly higher than that in the control group(P<0.05)Table 3. However, there were no differences in CD4MMP, | | | | | |
| CD8MMP, and CD4MM between the SAP group and the ACS group(P > 0.05).Table 4 | | | | | |

Further analysis revealed that in the SAP and ACS groups, CD4MMP and CD8MMP were significantly lower than those in the control group, while CD4MM was significantly higher than that in the control group(P<0.05).Table 3–4. However, there were no differences in CD4MMP, CD8MMP, and CD4MM between the SAP group and the ACS group(P > 0.05).Table 5.

Table 3
Comparison between the control group and the SAP group

| T lymphocyte | control | SAP | P |
|---|---|---|---|
| CD4MMP | 21.19 ± 21.58 | 13.09 ± 4.78 | 0.036* |
| CD8MMP | 33.14 ± 13.77 | 23.10 ± 6.04 | 0.001** |
| CD4MM | 2.91 ± 1.24 | 3.12 ± 1.03 | 0.022*** |
| In the SAP group, CD4MMP and CD8MMP were significantly lower than those in the control group, while CD4MM was significantly higher than that in the control group(P<0.05) | | | |

Table 4
Comparison between the control group and the ACS group

| T lymphocyte | control | ACS | P |
|---|---|---|---|
| CD4MMP | 21.19 ± 21.58 | 15.86 ± 6.24 | 0.131 |
| CD8MMP | 33.14 ± 13.77 | 25.08 ± 11.13 | 0.002* |
| CD4MM | 2.91 ± 1.24 | 3.54 ± 1.14 | 0.015** |
| In the ACS group, CD4MMP and CD8MMP were significantly lower than those in the control group, while CD4MM was significantly higher than that in the control group(P<0.05) | | | |

Table 5
Comparison between the SAP group and the ACS group

| T lymphocyte | SAP | ACS | P |
|---|---|---|---|
| CD4MMP | 13.09 ± 4.78 | 15.86 ± 6.24 | 0.519 |
| CD8MMP | 23.10 ± 6.04 | 25.08 ± 11.13 | 0.532 |
| CD4MM | 3.12 ± 1.03 | 3.54 ± 1.14 | 0.956 |
| there were no differences in CD4MMP, CD8MMP, and CD4MM between the SAP group and the ACS group(P > 0.05) | | | |

We divided the ACS group into the NSTEMI/UA group and the STEMI group based on whether ST was elevated. Then, we compared the T lymphocytes of the SAP group, the NSTEMI/UA group, and the STEMI group. They have no differences except for CD4MM(P<0.05)Table 6,Moreover, it was found that the CD4MM in the STEMI group was higher than that in the UA/NSTEMI group(P<0.05).Table 7.

Table 6
Comparison of peripheral blood T lymphocyte subgroups levels among the three groups of patients

| T lymphocyte | SAP(n = 25) | NSTEMI/UA(n = 11) | STEMI(n = 21) | T | P |
|---|---|---|---|---|---|
| CD3 | 1013.85 ± 299.28 | 1257.88 ± 400.89 | 1082.00 ± 359.57 | 1.939 | 0.154 |
| CD4 | 606.71 ± 153.24 | 673.79 ± 183.61 | 662.92 ± 226.63 | 0.718 | 0.492 |
| CD8 | 340.89 ± 183.77 | 480.36 ± 290.46 | 322.67 ± 159.21 | 2.469 | 0.094 |
| CD4-MMP | 13.09 ± 4.78 | 15.25 ± 6.67 | 16.18 ± 6.15 | 1.754 | 0.183 |
| CD8-MMP | 23.10 ± 6.04 | 24.05 ± 9.79 | 25.62 ± 11.97 | 0.419 | 0.660 |
| CD4-MM | 3.56 ± 1.14 | 2.83 ± 1.25 | 3.92 ± 0.90 | 4.026 | 0.023* |
| CD8-MM | 3.12 ± 1.03 | 2.48 ± 1.06 | 3.41 ± 0.92 | 3.138 | 0.051 |
| CD4-PD1 | 95.48 ± 28.55 | 101.96 ± 29.77 | 109.35 ± 41.99 | 0.093 | 0.401 |
| CD8-PD1 | 77.28 ± 37.55 | 96.54 ± 43.07 | 75.86 ± 43.36 | 1.062 | 0.353 |
| ACS group into the NSTEMI/UA group and the STEMI group based on whether ST was elevated. They have no differences except for CD4MM(*P = 0.023). | | | | | |

Table 7
Comparisons between each pair of the SAP group, the UA/NSTEMI group, and the STEMI group

| T lymphocyte | SAP | UA/NSTEMI | P |
|---|---|---|---|
| CD4MM | 3.56 ± 1.14 | 2.83 ± 1.25 | 0.055 |
| | SAP | STEMI | |
| | 3.56 ± 1.14 | 3.92 ± 0.90 | 0.247 |
| | UA/NSTEMI | STEMI | |
| | 2.83 ± 1.25 | 3.92 ± 0.90 | 0.006* |
| CD4MM in the STEMI group was higher than that in the UA/NSTEMI group(*P = 0.006). | | | |

# Discussion

Coronary artery disease caused by atherosclerosis is one of the common diseases that lead to death among the elderly. Currently, there are approximately 300 million people suffering from cardiovascular diseases in China. Among the patients who die from heart disease each year, 10% to 20% are caused by coronary heart disease[10]. The mechanism of coronary heart disease occurrence and development is rather complex. Among them, the three main aspects that have been confirmed include lipid infiltration,

thrombosis formation, and endothelial cell damage. Studies have shown that there is immune cell infiltration and immune complex deposition in the atherosclerotic plaques of coronary arteries in patients with coronary heart disease[11]. The local or systemic inflammatory responses that occur during the course of coronary heart disease patients indicate that the pathological process of coronary atherosclerosis is closely related to the immune response (both humoral immunity and cellular immunity)[10, 11]. Therefore, by monitoring the immune cells, we can determine the level of inflammation in the patient's body, understand the differentiation and proliferation of different immune cell subgroups, and thereby be able to assess whether there is an increased risk of disease progression in the patient.

T lymphocytes are the most important regulatory cells in the cellular immune response.

CD3 + T lymphocytes are divided into two main subgroups, namely CD4 + and CD8 + T lymphocytes. CD4 + T lymphocytes have the functions of promoting the formation of antibodies by B lymphocytes and stimulating the activation of macrophages. CD8 + T lymphocytes can exert an opposite inhibitory effect in the immune response through self-regulation and inhibition of inflammatory factors. Mitochondrial membrane potential (MMP) is an important component of the energy storage process generated during OXPHOS and is an important indicator of cell health and functional status. As an intermediate form of energy storage in cells, MMP is produced by REDOX transformations associated with the tricarboxylic acid cycle. MMP also plays a key role in maintaining mitochondrial homeostasis and overall healthy function of organelles[12, 13]. The study found that alterations in mitochondrial function, especially MMP, are critical in the development of AS. At present, MMP and MM detection based on flow cytometry combined with fluorescent probes has been widely used in the study of mitochondrial function. Mitochondrial substance (MM) is regulated by mitochondrial quality control (MQC) and is essential for regulation of mitochondrial metabolism. MQC ensures proper mitochondrial function through pathways related to protein folding and degradation, as well as processes involving entire organelles, such as biosynthesis, kinetics, and mitochondrial autophagy. Within the normal physiological range, the higher MM and MMP are, the more ATP is produced by mitochondria per unit time. MM and MMP regulate and affect mitochondrial ATP production[14, 15]. Through mitochondrial function index MM, MMP can reflect the differentiation and immune effect ability of different lymphocytes. The results of this study show the levels of CD4-MMP and CD8-MMP in the SAP group and the ACS group were significantly lower than those in the control group, while the level of CD4MM was significantly higher than that in the control group, Moreover, there was no difference between the SAP group and the ACS group. The decrease of CD4-MMP and CD8-MMP may lead to the occurrence of coronary heart disease, but it is not related to the severity of the disease. However, an increase in CD4MM is likely to cause coronary heart disease. Further, we found that CD4-MM was higher in the STEMI group compared to the UA/NSTEMI. This suggests that patients with higher CD4-MM are more likely to suffer from acute ST-segment elevation myocardial infarction.

In conclusion, we believe that mitochondrial-related indicators may be independent factors contributing to the increased risk of CAD patients, and can provide more sensitive biomarkers for clinical use to

adjust treatment plans. Therefore, by dynamically monitoring the mitochondrial function of T lymphocytes in CAD patients, a more comprehensive understanding of the disease progression of CAD patients can be achieved, guiding the adjustment of medication doses. When T lymphocytes exhibit different types of functional disorders (exhaustion, excessive activation, low effector ability, inhibition, etc.), their metabolic levels and characteristics will also show different abnormal manifestations. Through mitochondrial quality (MM) and mitochondrial membrane potential (MMP) indicators, their immune pathological types and degrees can be reflected, achieving immune typing (domestically exclusive clinical approval). Currently, MM and MMP are mainly detected using specific molecular probes such as MitoTracker® Green, Mitochondrial Damage® probes based on flow cytometry. By analyzing the MM and MMP of T lymphocyte subsets in CAD, Researchers can quantitatively assess the metabolic changes of T lymphocytes and determine the risk of coronary heart disease, providing a basis for subsequent prevention.

# Conclusion

In the peripheral blood of patients with coronary heart disease, the levels of CD4MMP and CD8MMP are significantly decreased, while CD4MM is significantly increased. These indicators can be used as predictive markers for the occurrence of coronary heart disease.

# Declarations


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Not available.

### Funding

NO


### Data availability

The data are available from the corresponding author on reasonable request.

### Authors' contributions

Hao Chen and Shiwei Huang made substantial contributions to the conception and design of the work. fanlu Guan made substantial contributions to the acquisition, analysis and interpretation of data for the work, and drafted the manuscript. All authors revised the manuscript critically for important intellectual content. All authors approved the final version of the manuscript. All authors were agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work were appropriately investigated and resolved.

### Ethics approval and consent to participate

The patients/participants provided their written informed consent to participate in this study. The human study was conducted according to the guidelines of the Declaration of Helsinki and approved by the ethical committee of Wenzhou People's Hospital (Approval no. KY-D-2024-234).

### Patient consent for publication

Not applicable.

### Conflict of interest

The authors declare no conflicts of interest.

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