

# Role in Development and Progression of Cardiovascular Disease

Sotiria Liori\*

Department of Cardiology, National and Kapodistrian University of Athens, Athens, Greece

## Abstract

The immune system is a complex network of cells, tissues, and organs that work together to defend the body against harmful pathogens, such as bacteria and viruses. However, the immune system can also play a role in the development and progression of cardiovascular disease, which is a group of conditions that affect the heart and blood vessels. This is due to the fact that the immune system can become dysregulated, leading to chronic inflammation, which is a key driver of CVD. Inflammation is a normal physiological response to injury or infection, but when it becomes chronic, it can lead to tissue damage and the development of CVD.

**Keywords:** Chronic inflammation • Cholesterol levels • Atherosclerotic plaques • Macrophages • Immune system

## Introduction

Chronic inflammation can be triggered by several factors, including high blood pressure, high cholesterol levels, smoking, and obesity. In response to these triggers, immune cells, such as macrophages and T cells, infiltrate the arterial wall and release pro-inflammatory cytokines and chemokines, which further amplify the inflammatory response. One of the main ways that the immune system contributes to the development of CVD is through the formation of atherosclerotic plaques. Atherosclerosis is a condition in which plaques build up in the arterial walls, narrowing the blood vessels and increasing the risk of heart attack and stroke. The formation of atherosclerotic plaques begins with the accumulation of LDL cholesterol in the arterial wall. This LDL is then oxidized by ROS, which are produced by various physiological processes, including inflammation. The oxidized LDL is then taken up by macrophages, leading to the formation of foam cells and the initiation of the atherosclerotic process [1].

## Literature Review

Macrophages within the plaques release pro-inflammatory cytokines and chemokines, which can recruit more immune cells to the site of the plaque. These immune cells can further amplify the inflammatory response and contribute to the breakdown of the fibrous cap, which covers the plaque. If the fibrous cap is breached, the plaque can rupture, leading to the formation of a blood clot and the development of a heart attack or stroke. In addition to promoting atherosclerosis, the immune system can also contribute to the development of other CVD conditions, such as hypertension and heart failure. Hypertension is a condition in which blood pressure is consistently elevated, and it is a major risk factor for CVD. Studies have shown that the immune system can contribute to hypertension by promoting vasoconstriction, or the narrowing of blood vessels, and by reducing the availability of nitric oxide, a vasodilator that helps to keep blood vessels open [2].

Similarly, the immune system can contribute to heart failure, which is a condition in which the heart is unable to pump enough blood to meet the body's

needs. Heart failure can be caused by several factors, including hypertension, diabetes, and coronary artery disease. In response to these factors, the immune system can become activated, leading to chronic inflammation and tissue damage in the heart. This inflammation can impair the heart's ability to contract and relax, leading to the development of heart failure. Several biomarkers of inflammation have been identified as predictors of CVD risk. For example, C-reactive protein is a marker of systemic inflammation that has been shown to be a strong predictor of CVD risk. Similarly, other inflammatory markers, such as interleukin-6 and tumor necrosis factor-alpha, have also been linked to CVD risk. These biomarkers can be measured in the blood and may be used to identify individuals at high risk of developing CVD [3].

## Discussion

The innate immune system is the body's first line of defense against invading pathogens. It is composed of several types of cells, including neutrophils, monocytes, and macrophages, which are able to recognize and respond to foreign antigens. These cells can initiate an inflammatory response, which is essential for the clearance of pathogens and tissue repair. However, in some cases, innate immune responses can become dysregulated and contribute to the development of CVD. For example, studies have shown that activated neutrophils and monocytes can adhere to the endothelial cells lining the blood vessels, leading to the formation of foam cells and the initiation of the atherosclerotic process. Furthermore, activated macrophages can release pro-inflammatory cytokines and enzymes, which can contribute to plaque instability and rupture, leading to myocardial infarction and stroke [4].

The adaptive immune system is a more specific response that is activated when the innate immune system is unable to clear an infection. It is composed of several types of cells, including T cells and B cells, which are able to recognize and respond to specific antigens. Several studies have shown that adaptive immune responses also play a role in the development of CVD. For example, studies have shown that T cells can infiltrate the arterial wall and contribute to plaque formation and instability. Furthermore, B cells have been shown to produce antibodies against oxidized LDL cholesterol, which can contribute to the formation of foam cells and the initiation of atherosclerosis. Inflammation is a normal physiological response to injury or infection. However, chronic inflammation can lead to tissue damage and contribute to the development of CVD. Several studies have shown that inflammation is a key factor in the development and progression of atherosclerosis [5].

Inflammatory cells, including neutrophils, monocytes, macrophages, T cells and B cells, can infiltrate the arterial wall and contribute to plaque formation and instability. Furthermore, activated macrophages can release pro-inflammatory cytokines and enzymes, which can contribute to plaque rupture and the development of acute cardiovascular events. C-reactive protein is a biomarker of systemic inflammation and has been shown to be a predictor of cardiovascular risk. The immune system plays a critical role in protecting the body from infections

\*Address for Correspondence: Sotiria Liori, Department of Cardiology, National and Kapodistrian University of Athens, Athens, Greece, E-mail: Sotirialiori6@gmail.com

Copyright: © 2023 Liori S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Received: 05 May, 2023, Manuscript No. jigc-23-103666; Editor assigned: 06 May, 2023, PreQC No. P-103666; Reviewed: 19 May, 2023, QC No. Q-103666; Revised: 24 May, 2023, Manuscript No. R-103666; Published: 31 May, 2023, DOI: 10.37421/2684-4591.2023.7.190

and diseases. However, in some cases, the immune system can become dysregulated and contribute to the development of various diseases, including cardiovascular disease. Once the atherosclerotic plaques are formed, the immune system continues to play a role in their progression and destabilization. The immunologic basis of CVD is complex, involving both innate and adaptive immune responses that can lead to inflammation and tissue damage [6].

## Conclusion

Several studies have shown that elevated levels of CRP are associated with an increased risk of cardiovascular events, including myocardial infarction and stroke. Statins are a class of drugs that are commonly used to lower cholesterol levels and reduce cardiovascular risk. However, they also have anti-inflammatory properties and have been shown to reduce the levels of pro-inflammatory cytokines, including interleukin-6 and tumor necrosis factor-alpha. Several studies have shown that statins can reduce the risk of cardiovascular events, including myocardial infarction and stroke, even in patients with normal cholesterol levels. In addition to statins, several other drugs have been developed to target the immune system and reduce cardiovascular risk. For example, monoclonal antibodies that target pro-inflammatory cytokines, such as TNF-alpha and interleukin-1 beta have been shown to reduce the risk of cardiovascular events in patients with rheumatoid arthritis and other inflammatory.

## Acknowledgement

None.

## Conflict of Interest

None.

## References

1. Berntson, G.G., Bigger J.T.J., Eckberg D.L. and Grossman P., et al. Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiol* 34 (1997): 623–648.
2. Soares-Miranda, Luisa, Jacob Sattelmair, Paulo Chaves and Glen E. Duncan, et al. "Physical activity and heart rate variability in older adults: The Cardiovascular Health Study." *Circulation* 129 (2014): 2100-2110.
3. Israeli-Mendlovic, H., Mendlovic J., Zuk L. and Katz-Leurer M. Reproducibility of 24-h heart rate variability measures in preterm infants born at 28–32 weeks of gestation. *Early Hum Dev* 148 (2020): 105117.
4. Selig, Fabio Augusto, Emanuele Renata Tonolli, Érico Vinicius Campos Moreira da Silva and Moacir Fernandes de Godoy, et al. "Heart rate variability in preterm and term neonates." *Arq Bras Cardiol* 96 (2011): 443-449.
5. Takatani, Tsunenori, Yukihiro Takahashi, Ryota Yoshida and Ryuko Imai, et al. "Relationship between frequency spectrum of heart rate variability and autonomic nervous activities during sleep in newborns." *Brain Dev* 40 (2018): 165-171.
6. Cardoso, Sandra, Marta Joao Silva and Hercília Guimarães. "Autonomic nervous system in newborns: A review based on heart rate variability." *Childs Nerv Syst* 33 (2017): 1053-1063.

**How to cite this article:** Liori, Sotiria. "Role in Development and Progression of Cardiovascular Disease." *J Interv Gen Cardiol* 7 (2023): 190.