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# Inflammation Drives Premature Coronary Dysfunction Without Lipids

**Stefan Petrov***

*Department of Interventional Cardiology and Structural Heart Disease, University of Belgrade, Belgrade 11000, Serbia*

## Introduction

This research investigates the specific inflammatory transcriptomic patterns observed in individuals with normolipidemic premature coronary dysfunction. It highlights how distinct gene expression profiles, even in the absence of high cholesterol, can underlie the development of early-stage coronary artery disease, offering potential targets for diagnosis and intervention [1].

Explores the role of specific inflammatory cytokines and cellular pathways in the pathogenesis of premature coronary artery disease, particularly in patients without dyslipidemia. The findings suggest that inflammatory processes are key drivers, even when traditional risk factors like hypercholesterolemia are absent, pointing towards novel therapeutic avenues [2].

This study delves into the genetic underpinnings of premature coronary dysfunction by analyzing transcriptomic data from normolipidemic individuals. It identifies key upregulated genes associated with inflammatory responses, offering insights into early disease mechanisms independent of lipid profiles [3].

Investigates the cellular and molecular mechanisms driving premature coronary artery disease in the absence of traditional risk factors like dyslipidemia. The research focuses on the role of immune cell infiltration and pro-inflammatory signaling pathways, as revealed by transcriptomic analysis [4].

This paper provides a detailed analysis of the transcriptomic landscape in normolipidemic individuals exhibiting premature coronary dysfunction. It identifies specific gene networks and biological processes that are dysregulated, offering a molecular basis for the observed pathology [5].

Focuses on identifying novel inflammatory biomarkers from transcriptomic data associated with premature coronary dysfunction in patients without dyslipidemia. The study aims to uncover molecular signatures that can predict or diagnose this condition early [6].

This article investigates the intricate relationship between inflammation and early coronary artery disease development in individuals with normal lipid profiles. Transcriptomic analysis reveals specific immune responses contributing to vascular damage, even without atherosclerotic plaque formation driven by high cholesterol [7].

Examines the distinct molecular phenotypes of premature coronary dysfunction in normolipidemic patients using transcriptomic profiling. The study identifies key inflammatory mediators and signaling pathways that differentiate these patients from those with typical atherosclerosis [8].

This research investigates the contribution of subtle inflammatory changes, detectable through transcriptomic analysis, to the early development of coronary dysfunction in individuals with normal lipid levels. It provides evidence for non-lipid-driven inflammatory pathways being central to this condition [9].

Analyzes the transcriptomic profiles of patients with premature coronary dysfunction who do not exhibit dyslipidemia. The study identifies specific inflammatory gene expression patterns that may play a causative role, offering potential targets for early therapeutic intervention in this population [10].

## Description

This research investigates the specific inflammatory transcriptomic patterns observed in individuals with normolipidemic premature coronary dysfunction, highlighting how distinct gene expression profiles, even in the absence of high cholesterol, can underlie the development of early-stage coronary artery disease and offering potential targets for diagnosis and intervention [1].

Explores the role of specific inflammatory cytokines and cellular pathways in the pathogenesis of premature coronary artery disease, particularly in patients without dyslipidemia, suggesting that inflammatory processes are key drivers even when traditional risk factors like hypercholesterolemia are absent, thereby pointing towards novel therapeutic avenues [2].

This study delves into the genetic underpinnings of premature coronary dysfunction by analyzing transcriptomic data from normolipidemic individuals, identifying key upregulated genes associated with inflammatory responses and offering insights into early disease mechanisms independent of lipid profiles [3].

Investigates the cellular and molecular mechanisms driving premature coronary artery disease in the absence of traditional risk factors like dyslipidemia, focusing on the role of immune cell infiltration and pro-inflammatory signaling pathways as revealed by transcriptomic analysis [4].

This paper provides a detailed analysis of the transcriptomic landscape in normolipidemic individuals exhibiting premature coronary dysfunction, identifying specific gene networks and biological processes that are dysregulated, thus offering a molecular basis for the observed pathology [5].

Focuses on identifying novel inflammatory biomarkers from transcriptomic data associated with premature coronary dysfunction in patients without dyslipidemia, aiming to uncover molecular signatures that can predict or diagnose this condition early [6].

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This research investigates the contribution of subtle inflammatory changes, detectable through transcriptomic analysis, to the early development of coronary dysfunction in individuals with normal lipid levels, providing evidence for non-lipid-driven inflammatory pathways being central to this condition [9].

Analyzes the transcriptomic profiles of patients with premature coronary dysfunction who do not exhibit dyslipidemia, identifying specific inflammatory gene expression patterns that may play a causative role and offering potential targets for early therapeutic intervention in this population [10].

## Conclusion

This collection of research delves into the complex mechanisms underlying premature coronary dysfunction, particularly in individuals with normal lipid levels. Studies highlight the crucial role of inflammation and specific transcriptomic patterns in the development of early coronary artery disease, even in the absence of traditional risk factors like high cholesterol. Researchers have identified distinct gene expression profiles, inflammatory cytokines, cellular pathways, and immune cell involvement that contribute to vascular damage. The findings suggest that inflammation is a primary driver of this condition, independent of lipid profiles. The research aims to uncover novel biomarkers and molecular signatures for early diagnosis and therapeutic intervention, offering new avenues for managing premature coronary artery disease.

## Acknowledgement

None.

## Conflict of Interest

None.

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***Address for Correspondence:*** Stefan, Petrov, Department of Interventional Cardiology and Structural Heart Disease, University of Belgrade, Belgrade 11000, Serbia, E-mail: stefan.petrov@bg.ac.rs



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