Volume 4, Issue 4, April 2026 ISSN (E): 2938-3765

# THE ROLE OF INNATE AND ADAPTIVE IMMUNITY IN THE DEVELOPMENT OF DILATED CARDIOMYOPATHY IN CHILDREN

Yusupova Gulasar Komiljon qizi
Assistant of the Department of Propaedeutics of
Pediatric Diseases, Uzbek State Medical University, Ph.D.

**Abstract**
Dilated cardiomyopathy (DCM) in children remains one of the most challenging and devastating pediatric cardiac conditions, with implications ranging from chronic disability to death. The disorder is characterized by the dilation and impaired contraction of one or both ventricles, ultimately leading to systolic dysfunction and congestive heart failure. Understanding its multifactorial etiology has been an intense focus of pediatric cardiology, and among the many contributors to the disease, the immune system — both innate and adaptive — has emerged as a crucial determinant in its onset and progression. Delineating how the two pillars of immunity interact, escalate, or fail to control the pathological process is essential to designing effective diagnostics and therapeutic interventions.

**Keywords**: Dilated cardiomyopathy, children, innate immunity, adaptive immunity, myocardium, autoimmunity, immune response, T lymphocytes, B lymphocytes, inflammation.

## Introduction

When considering DCM in children, it is vital first to appreciate the intricate interplay between their still-maturing immune systems and various environmental and endogenous triggers. Children's immune responses can differ markedly from those of adults, not only in their intensity but also in their regulation and in the spectrum of pathogens they most frequently encounter. Innate immunity, the body's first line of defense, is non-specific but rapid and robust. It acts through pattern recognition receptors such as toll-like receptors on cells like neutrophils, macrophages, dendritic cells, and natural killer (NK) cells. Upon the introduction of a viral, bacterial, or parasitic pathogen — or other injury signals — these cells are activated almost immediately. They mobilize to the heart, where they recognize danger-associated molecular patterns and initiate a cascade of events that include the secretion of pro-inflammatory mediators such as tumor necrosis factor-alpha, interleukin-1 beta, and interleukin-6. This cytokine storm, while designed to contain infection, often has the side effect of introducing localized inflammation in the myocardium — a process central to the development of DCM [1].

Such an inflammatory reaction, left unresolved either because of persistent infection or other factors like autoantigen exposure, results in ongoing cardiac tissue injury. Macrophages engulf infected and dying cells; neutrophils release enzymes and reactive oxygen species, which can damage both pathogens and bystander myocardial cells. The process of phagocytosis, while vital, is non-discriminatory at times and can propagate collateral cell death. Dendritic cells in the heart meanwhile



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Licensed under a Creative Commons Attribution 4.0 International License.

pick up cardiac antigens and transport them to lymph nodes, where they present these antigens to T cells — this is the critical moment at which adaptive immunity is engaged. Adaptive immunity is precision-targeted but slower to respond than innate immunity. It centers on lymphocytes — T cells (both CD4+ helper and CD8+ cytotoxic subtypes) and B cells — which retain a form of immunologic memory that shapes future responses to the same antigens. In the context of DCM, the transition to adaptive immunity marks a new phase of the disease. Here, the immune system is supposed to clear residual pathogens or resolve inflammation, but in many cases, especially in genetically susceptible children, the adaptive response becomes dysregulated.On a broader scale, the social and developmental impact of DCM in children must also be considered. Persistent heart failure, frequent hospitalizations, and the side effects of aggressive immune suppression can significantly reduce quality of life. Hence, pediatricians and cardiologists must be vigilant for early signs of immune-mediated cardiac injury and collaborate closely with immunologists to optimize individualized treatment plans. Recent years have also seen advancements in imaging and laboratory assays, fostering earlier detection of subclinical myocarditis and more accurate monitoring of immune activation. This progress increases the prospects for targeted intervention, potentially halting or even reversing the course of DCM in its earliest immune-related stages [2].

In summary, dilated cardiomyopathy in children is not merely a structural heart disease but often the result of complex, ongoing interactions between innate and adaptive immunity. The innate immune system provides the first rapid defense, but if the threat is not quickly resolved, sustained inflammation and tissue damage lead to the engagement and eventual dysregulation of adaptive immunity [3].

This in turn triggers a cycle of autoimmune-directed injury, progressive heart muscle destruction, and eventual cardiac dilation. Advances in immunology now make it possible to more precisely interrogate which immune processes are at fault in each child and offer the promise of therapies tailored to the specific immune derangements at play. As scientific understanding matures, it is increasingly clear that the investigation and treatment of DCM in children will demand an integrative approach at the crossroads of immunology, genetics, infectious diseases, and pediatric cardiology. The hope is that one day, by comprehensively understanding the role of innate and adaptive immunity in DCM pathogenesis, disease progression can be halted or prevented, conferring better cardiac health and improved lifespans for children at risk [4].

## Conclusion

The development of dilated cardiomyopathy in children is shaped fundamentally by the dynamic interplay of innate and adaptive immunity. While the innate immune system is primarily responsible for early containment of pathogens and immediate inflammation, it is the maladaptive or dysregulated adaptive immune response that often leads to ongoing cardiac injury, autoimmunity, and disease chronicity. Future diagnostic and therapeutic strategies will focus on identifying the particular contributions of each immune arm in individual patients and intervening before irreversible cardiac remodeling takes place. Ultimately, advancing the understanding of immunological mechanisms involved in pediatric DCM holds the promise of tailored, effective, and less toxic treatments for affected children.



Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

Licensed under a Creative Commons Attribution 4.0 International License.

**References**

1. Aripov O.M., Uzbekistanova G.S. “Pediatric Cardiology: Etiology and Immunopathogenesis of Cardiomyopathies.” Tashkent, 2015. pp. 112-157
2. Bagdatov M.M. “Immunological Basis and Cardiac Diseases.” Tashkent, 2018. pp. 78-95, 187-201
3. Vengerova T.G., Lakin Yu.A. "Immune System Pathology in Cardiomyopathies." Moscow, 2012. pp. 145-168
4. Yakubova N.L., IslOmov F.A. “Cardiomyopathy: Immune Response and Development in Children.” Republican Scientific-Practical Pediatrics Journal, 2019, No.2(3), pp. 137-151
5. Sagdullaeva B.A. “Immunity in Pediatrics and Cardiac Muscle Diseases.” Tibbiyot, 2020. pp. 97-120.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5



Licensed under a Creative Commons Attribution 4.0 International License.