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Advanced Evolution of Pathogenesis Concepts in Cardiomyopathies.

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Abstract

Cardiomyopathy is a group of heterogeneous cardiac diseases that impair systolic and diastolic function, and can induce chronic heart failure and sudden cardiac death. Cardiomyopathy is prevalent in the general population, with high morbidity and mortality rates, and contributes to nearly 20% of sudden cardiac deaths in younger individuals. Genetic mutations associated with cardiomyopathy play a key role in disease formation, especially the mutation of sarcomere encoding genes and ATP kinase genes, such as titin, lamin A/C, myosin heavy chain 7, and troponin T1. Pathogenesis of cardiomyopathy occurs by multiple complex steps involving several pathways, including the Ras-Raf-mitogen-activated protein kinase-extracellular signal-activated kinase pathway, G-protein signaling, mechanotransduction pathway, and protein kinase B/phosphoinositide 3-kinase signaling. Excess biomechanical stress induces apoptosis signaling in cardiomyocytes, leading to cell loss, which can induce myocardial fibrosis and remodeling. The clinical features and pathophysiology of cardiomyopathy are discussed. Although several basic and clinical studies have investigated the mechanism of cardiomyopathy, the detailed pathophysiology remains unclear. This review summarizes current concepts and focuses on the molecular mechanisms of cardiomyopathy, especially in the signaling from mutation to clinical phenotype, with the aim of informing the development of therapeutic interventions.

KEYWORDS: apoptosis; cardiac remodeling; cardiomyopathy; fibrosis; genetic mutationsPMID: 30995779 DOI: [10.3390/jcm8040520](https://doi.org/10.3390/jcm8040520)

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