

Cardiac Organoids: A Promising Avenue to Model and Heal Heart Failure and Cardiomyopathies

Rawan Salenger*

Department of Surgery, UM St Joseph Medical Center, 7505 Osler Dr, Ste 302, Towson, MD 21204, USA

Introduction

Heart failure and cardiomyopathies represent significant challenges in healthcare, affecting millions of individuals worldwide. Despite advancements in treatment, the complexity of these conditions often hampers effective therapeutic strategies. However, recent developments in tissue engineering and regenerative medicine have led to the emergence of cardiac organoids as a promising tool for understanding disease mechanisms, drug discovery, and potentially, tissue regeneration. This article explores the concept of cardiac organoids, their applications in modeling heart failure and cardiomyopathies, and their potential for therapeutic interventions [1].

Heart failure is a multifactorial syndrome characterized by the inability of the heart to pump blood efficiently, leading to symptoms such as fatigue, shortness of breath, and fluid retention. Cardiomyopathies encompass a group of diseases affecting the myocardium, the heart muscle, leading to structural and functional abnormalities. Both conditions have diverse etiologies, including genetic predisposition, myocardial infarction, hypertension, and viral infections, making them challenging to study and treat effectively [2].

Description

Traditional experimental models, such as animal models and two-dimensional cell cultures, have limitations in mimicking the complex microenvironment of the human heart. Animal models may not accurately recapitulate human physiology, while two-dimensional cultures lack the three-dimensional architecture and cell-cell interactions crucial for understanding disease mechanisms. Thus, there is a pressing need for advanced models that can better emulate the human heart's structure and function. Cardiac organoids, also known as heart organoids or cardiac spheroids, are three-dimensional structures derived from stem cells that mimic certain aspects of the human heart. These miniature organs contain various cell types found in the heart, including cardiomyocytes, endothelial cells, and fibroblasts, arranged in a spatially organized manner resembling native cardiac tissue [3].

Importantly, cardiac organoids exhibit self-organization and maturation properties, making them valuable tools for studying heart development, disease modeling, and drug screening. Cardiac organoids offer a unique platform for studying the pathophysiology of heart failure and cardiomyopathies. Researchers can induce disease-specific mutations in stem cells and

differentiate them into cardiomyocytes to generate diseased organoids. By incorporating patient-derived induced pluripotent stem cells scientists can create personalized organoid models, allowing for the investigation of genotype-phenotype relationships and personalized medicine approaches. Furthermore, researchers can simulate disease conditions in cardiac organoids by subjecting them to mechanical stress, ischemia-reperfusion injury, or exposure to cardiotoxic drugs, thereby recapitulating key aspects of heart failure and cardiomyopathies. By monitoring changes in gene expression, contractility, and electrophysiological properties, scientists can gain insights into disease progression and identify potential therapeutic targets [4].

One of the most promising applications of cardiac organoids is in drug discovery and screening. Traditional drug development pipelines often fail due to the lack of efficacy and unforeseen cardiotoxicity in clinical trials. Cardiac organoids provide a more physiologically relevant platform for assessing drug safety and efficacy. Researchers can test potential therapeutics in organoid models, evaluating their effects on contractility, electrophysiology, and cell viability. This approach not only reduces the reliance on animal models but also increases the likelihood of identifying promising drug candidates for further clinical development. In addition to disease modelling and drug screening, cardiac organoids hold immense potential for tissue regeneration and cell therapy. Researchers are exploring strategies to coax organoids into more mature and functional cardiac tissues, with the ultimate goal of replacing damaged myocardium in patients with heart failure or cardiomyopathies. By optimizing the differentiation protocols and enhancing vascularization within organoids, scientists aim to generate transplantable tissues that can integrate with the host myocardium and improve cardiac function. Despite their promise, cardiac organoids face several challenges that need to be addressed.

These include improving scalability and reproducibility, enhancing maturation and functionality, and better mimicking the heterogeneity of the human heart. Additionally, ethical considerations regarding the use of stem cells and organoid models need to be carefully addressed. Looking ahead, advancements in biomaterials, tissue engineering, and bioengineering techniques hold the potential to overcome these challenges and further enhance the utility of cardiac organoids. Integrating organoids with microfluidic systems and biofabrication technologies could enable more precise control over organoid structure and function. Moreover, collaborative efforts between scientists, clinicians, and regulatory agencies are essential to accelerate the translation of cardiac organoid research into clinical applications [5].

Conclusion

Cardiac organoids represent a promising avenue for modelling heart failure and cardiomyopathies, advancing drug discovery, and exploring regenerative therapies. These miniature organs offer a more physiologically relevant platform than traditional models, allowing researchers to study disease mechanisms, screen potential therapeutics, and develop personalized treatment strategies. While challenges remain, ongoing research efforts continue to push the boundaries of cardiac organoid technology, bringing us closer to the realization of effective therapies for heart failure and cardiomyopathies.

*Address for Correspondence: Rawan Salenger, Department of Surgery, UM St Joseph Medical Center, 7505 Osler Dr, Ste 302, Towson, MD 21204, USA, E-mail: rawnsalenger321@umm.edu

Copyright: © 2024 Salenger R. 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: 01 January, 2024, Manuscript No. jigc-24-132013; Editor assigned: 03 January, 2024, PreQC No. P-132013; Reviewed: 15 January, 2024, QC No. Q-132013; Revised: 20 January, 2024, Manuscript No. R-132013; Published: 30 January, 2024, DOI: 10.37421/2684-4591.2024.8.231

Acknowledgement

None.

Conflict of Interest

None.

References

1. Lopes, Luís R., M. Shafiqur Rahman and Perry M. Elliott. "A systematic review and meta-analysis of genotype-phenotype associations in patients with hypertrophic cardiomyopathy caused by sarcomeric protein mutations." *Heart* 99 (2013): 1800-1811.
2. Kayvanpour, Elham, Farbod Sedaghat-Hamedani, Ali Amr and Alan Lai, et al. "Genotype-phenotype associations in dilated cardiomyopathy: Meta-analysis on more than 8000 individuals." *Clin Res Cardiol* 106 (2017): 127-139.

3. Kimura, Akinori. "Molecular genetics and pathogenesis of cardiomyopathy." *J Human Genet* 61 (2016): 41-50.
4. Teerlink, John R., Rafael Diaz, G. Michael Felker and John JV McMurray, et al. "Cardiac myosin activation with omecamtiv mecarbil in systolic heart failure." *New Eng J Med* 384 (2021): 105-116.
5. Cahill, Thomas J., Houman Ashrafian and Hugh Watkins. "Genetic cardiomyopathies causing heart failure." *Circulat Res* 113 (2013): 660-675.

How to cite this article: Salenger, Rawn. "Cardiac Organoids: A Promising Avenue to Model and Heal Heart Failure and Cardiomyopathies." *J Interv Gen Cardiol* 8 (2024): 231.