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Short Communication

Mechanism of Heart Failure and Cardiac Remodeling

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DESCRIPTION

A healthy heart goes through considerable remodeling at the organ, cell, and molecular levels as heart failure develops. Mechanistic understanding of heart failure has been produced and is still being produced by research into cardiac remodeling. Changes in cardiac energy metabolism are a crucial part of the remodeling process. The heart is an energy-intensive organ, and the mitochondria's oxidative metabolism accounts for over 95% of the Adenosine Triphosphate (ATP) it produces and uses. [1].

Myocardial inefficiency and mitochondrial dysfunction have long been known to deplete the failing hearts' energy stores. However, the majority of research on cardiac metabolism in heart failure up to this point has been on HFrEF, or heart failure with decreased ejection fraction, which is defined as left ventricular ejection fraction. Patients with heart failure who nevertheless have a significant amount of ejection fraction. The causes of HFpEF seem to be more varied than those of HFrEF, and the disease processes underlying HFpEF are not well known.

There are several comorbidities that have been found that jointly raise the risk of, including hypertension, diabetes mellitus, obesity, and ageing. The role of metabolism in the development of HFpEF has not been fully explored, despite the fact that metabolic derangement and mitochondrial dysfunction are common pathogenic mechanisms for these comorbidities. Information on the metabolic profile in HFpEF heart from studies of myocardial energetics in human patients and available animal models. We hope to emphasize the need of further research on HFpEF metabolism by contrasting these findings with what we now know about metabolic remodeling in HFrEF hearts.

Since examining the energy metabolism of human hearts is technically impossible, animal HFrEF models are the main source of information about cardiac metabolism in heart failure. Nevertheless, it has been difficult to create animal models that accurately represent the clinical characteristics of HFpEF. The clinical diagnosis of HFpEF is made based on the presence of heart failure symptoms and signs with maintained LVEF (at least >40%). The majority of these patients are elderly, obese, and/or hypertensive [2]. Therefore, Cardiac mitochondria can oxidise substrates with a lot of flexibility and capability. To produce energy, every kind of carbon substrate can be oxidised, including glucose, fatty acids, amino acids, and ketone bodies [3]. Fatty acids are the main fuel for oxidative metabolism in normal adult hearts, which produces 95% of ATP. However, pathological situations like hypertrophy and HFrEF cause the heart's substrate usage to change. It is distinguished by diminished.

It has long been known that failing hearts have lower amounts of high-energy phosphate [4]. When heart failure is advanced, this initially shows up as a drop in the energy reserve molecule Phosphocreatine (PCr), which is followed by large drops in the amount of ATP in the myocardium. A 50–70% drop in PCr and a maximum drop of 30% reduction of ATP have been observed in advanced HFrEF hearts. In addition, long-term study in HFrEF patients with dilated. Recent research has discovered mitochondrial processes, including as redox imbalance, oxidative stress, protein modification, altered mitochondrial Ca^{2+} homeostasis, and inflammation, that contribute to the development of HFrEF in addition to energy deprivation. Currently, several of these mitochondrial pathways are being thought of as possible therapeutic targets. Studies used both 3-hit obesity-induced HFpEF models.

CONCLUSION

Evidence for alterations in cardiac metabolism in HFpEF has just recently started to appear, in contrast to the considerable research on metabolic remodeling in HFrEF hearts. Myocardial energetics were shown to be compromised in clinical trials of HFpEF patients as indicated by a reduced PCr/ATP ratio. A small number of investigations in animal models with HFpEF found decreased oxidative metabolism in the heart and compromised myocardial energetics.

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