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ISSN: 2329-6607
Cardiovascular Pharmacology: Open Access

Opinion Article

Ferroptosis as a Target for Preventing Cardiomyopathy

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DESCRIPTION

Heart disease is the leading factor in global mortality. Heart muscle injury, a structural and functional change in the heart, heart failure and sudden cardiac death are all symptoms of the condition known as cardiomyopathy. The loss of cardiomyocytes is the main pathogenic cause in cardiomyopathy, however the underlying molecular pathways are yet unknown. A recently identified controlled form of cell death known as ferroptosis is characterized by iron buildup and lipid peroxidation. Recent research has demonstrated the crucial regulatory roles ferroptosis plays in the occurrence and progression of numerous cardiac disorders, including myocardial ischemia/reperfusion damage, cardiomyopathy and heart failure. Ferroptosis and cardiomyopathy are known to be associated systemically, however more research is required to clarify this. In this review, we give a general overview of the molecular mechanisms behind ferroptosis and its function in several cardiomyopathies and we emphasize that targeting ferroptosis may one day be a viable therapeutic approach for the treatment of cardiomyopathies.

All living cells in both plants and animals experience cell death as a necessary and inevitable step in the latter stages of their life cycles. The two basic types of cell death apoptosis and necrosis have already been identified. Recent research has revealed that a form of genetically regulated non-apoptotic cell death known as "regulated necrosis," which includes necroptosis, proptosis, parthanatos and ferroptosis occurs. Discovered a new substance called erastin that could specifically kill cancer cells that expressed the RAS gene. However, they discovered that this erastin-related cell death occurred in a different way than what had previously been observed, with no change in nuclear morphogenesis, no DNA damage, and no caspase activation. This process was even resistant to caspase inhibitors. Discovered a new substance called erastin that could specifically kill cancer cells that expressed the RAS gene. However, they discovered that this erastin-related cell death occurred in a different way than what had previously been observed, with no change in nuclear morphogenesis, no DNA damage, and no caspase activation. This process was even resistant to caspase inhibitors.

The most important biological processes to induce ferroptosis are the depletion of the intracellular antioxidant tripeptide glutathione and a drop in activity. A set of cardiovascular disorders known as cardiomyopathies have a poor prognosis and a high fatality rate. Cardiomyopathy is categorized by the American Heart Association (AHA) into main genetic, mixed, or acquired and secondary categories, which segregate it into types of dilated, hypertrophic, and restricted patterns. Several categories, including ischemia, metabolic, infectious, toxic, auto-immunogenic, and neuromuscular, can be used to classify secondary causes. Dilated Cardiomyopathy (DCM) is the most prevalent form of no ischemic cardiomyopathy, while Hypertrophic Cardiomyopathy (HCM) is the most prevalent primary form. Excessive iron overload in bodily organs damages organs. Iron overload cardiomyopathy is the medical term for Iron-Related Cardiac injury (IOC). The most prevalent kind of heart illness, Ischemic Heart Disease (IHD), also known as Coronary Artery Disease (CAD), causes myocardial infarction and ischemic cardiomyopathy.

Heart transplantation is a proven treatment for end-stage Heart Failure (HF) however Post-Transplant Cardiomyopathy (PTCM) is brought on by graft damage and graft failure. The majority of breast and lung malignancies are treated with radiation therapy, although RCM (Radiation-Induced Cardiomyopathy) is brought on by the heart damage Diabetes can cause diabetic cardiomyopathy, a unique type of cardiomyopathy that is unrelated to coronary artery disease and high blood pressure. While chemotherapy drugs like Doxorubicin (DOX) cause cardiomyopathy (DICM) because of their cardiotoxicity Seventy percent of sepsis patients will develop Septic Cardiomyopathy (SCM), a potentially fatal organ failure caused on by infection Cardiomyocytes death is a constant feature of all those multifactorial cardiomyopathies. The injured tissue gradually gives way to fibrotic scar tissue, which is made up of proliferating fibroblasts that lack contractile function. Loss of cardiomyocytes ultimately results in abnormal ventricular remodeling and heart failure as a result.

Cardiomyocytes are lost as a result of cell death during

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Received: 08-Aug-2022, Manuscript No. CPO-22-17910; **Editor assigned:** 11-Aug-2022, PreQC No. CPO-22-17910 (PQ); **Reviewed:** 25-Aug-2022, QC No. CPO-22-17910; **Revised:** 01-Sep-2022, Manuscript No. CPO-22-17910 (R); **Published:** 08-Sep-2022, DOI: 10.35248/2329-6607.22.11.301

Citation: Giorgio C (2022) Ferroptosis as a Target for Preventing Cardiomyopathy. Cardiovasc Pharm. 11:301

myocardial infarction. Additionally, mature mammalian cardiac cardiomyocytes have relatively limited post-injury proliferative and regeneration capacities. Cardiomyocytes are diploid during their developmental stage before changing to polyploidy as they mature. Cardiomyocytes are lost as a result of cell death during myocardial infarction. Additionally, adult brain hearts' cardiomyocytes have relatively limited proliferative and regeneration capacities. Cardiomyocytes are diploid during their developmental stage before changing to polyploidy as they mature, In which pre-existing cardiomyocytes are used to produce new cardiomyocytes.