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Histone Modifications in Cardiovascular Epigenetics

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DESCRIPTION

Congenital Heart Disease (CHD) is the most common birth defect in new-borns worldwide and contributes to significant infant morbidity and mortality. Significant advances in medical and surgical treatment and improvements in prenatal diagnosis have greatly improved outcomes for these children with CHD, and today there are more adults with her CHD than children. Advances in genomic technology have revealed the genetic origins of most of the CHDs, while pointing out that the genetics of CHDs are remarkably complex. For this reason, the complex process of cardiogenesis, controlled by multiple interconnected dose-dependent signalling pathways, is a well-studied process. In addition to genomic sequencing, epigenetic contributions to cardiogenesis are increasingly being recognized. Significant progress has been made in analysing the cardiac epigenome and identifying associations with cardiovascular disease. The role of epigenetic regulation in heart development/cardiogenesis has been well studied using tissue and animal models. Here, we curate the current literature based on human studies that have revealed relevant and/or causative epigenetic factors involved in CHD. With the aim of providing scientists and clinicians with an overview of the aberrant cardiogenic signalling pathways affected by epigenetic mechanisms, and to better understand their implications, we explored epigenetics in cardiac development and various CADs. An attempt was made to summarize current knowledge on the functional role of genetics. To understand the developing fetal heart, especially for readers interested in CAD research.

Cardiovascular Disease (CVD) is a leading cause of debilitation and death worldwide, requiring affordable treatments. Autophagy is a highly conserved catabolic recycling pathway triggered by a variety of intra or extracellular stimuli and plays an important role in development and pathology, including CVD. Therefore, there is great interest in identifying the mechanisms that govern autophagy regulation. Autophagy regulation is highly complex and multifactorial, involving epigenetic pathways such as: B. Histone modifications to regulate autophagy-associated gene expression, decapping-associated mRNA degradation, microRNAs, and long noncoding RNAs. Signal transduction pathways are also known to play a role in cardiovascular disease. A molecular understanding of the epigenetic signalling pathways involved in autophagy and CVD will not only advance our understanding of CVD, but may also provide new therapeutic targets and biomarkers for CVD.

CONCLUSION

Cardiovascular Disease (CVD) is a leading cause of mortality and morbidity across genders, races and ethnicities. Aging and its associated physiological and pathological consequences exacerbate the development and progression of cardiovascular disease, but modulation of biological age through interventions is associated with cardiovascular health. Despite the strong association of aging with cardiovascular disease, surprisingly few studies have directly examined heart failure and vascular dysfunction in older models and subjects. Nevertheless, strong correlations have been found between heart disease, atherosclerosis, hypertension, fibrosis, regenerative efficiency and aging cell burden and its pro-inflammatory consequences. Senotherapeutics have been consistently successful in reducing adverse effects in experimental models of cardiovascular aging and disease. Apart from senotherapy, cellular reprogramming strategies targeting epigenetic enzymes remain an unexplored viable option for reversing or delaying CVD. Epigenetic changes include local and global changes in DNA and histone modifications, transcription factor binding, nuclear lamina disassembly and genomic misfolding, and are hallmarks of aging. Limited studies on the aged cardiovascular system from mouse models or human patient samples have identified strong correlations between the epigenome, age, and aging. Here, we summarize the results of published studies linking epigenetic alterations to his CVD and identify distinct themes of epigenetic deregulation during aging. As direct study of these common mechanisms in aged tissue is pending, this review predicts that future research will establish epigenetic rejuvenation as an effective method of slowing his CVD. It has been.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.