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Commentary

A Brief Note on Atrial Fibrillation

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DESCRIPTION

The most prevalent supraventricular arrhythmia is Atrial Fibrillation (AF), which affects up to 1% of the general population. Its frequency rises dramatically with age, reaching as high as 8% in the senior population. The treatment of AF is a complicated problem that is the subject of continuous fundamental and clinical study. Ion channel failure, Ca^{2+} handling problems, and structural remodelling are all factors in atrial fibrillation. Over 100 genetic loci linked to atrial fibrillation have been discovered through Genome-Wide Association Studies (GWAS). The majority of these genes are associated with ion channels, cardiac-enriched transcription factors, and other regulatory genes.

The discovery of post-transcriptional regulatory mechanisms involving non-coding RNAs (especially microRNAs), DNA methylation, and histone modification has recently allowed to better understanding how a healthy heart develops and which modifications are involved in reshaping the processes that lead to arrhythmias. The goal of this study is to provide the current state of the art in terms of identifying and functionally characterising AF-related epigenetic regulatory networks. The most prevalent supraventricular arrhythmia is Atrial Fibrillation (AF), which affects up to 1% of the general population. Its prevalence rises exponentially with age, reaching up to 8% of the senior population (age>80 years), making it one of the world's most serious health problems.

The presence of AF contributes considerably to morbidity and death by lowering the quality of life of patients and raising the risk of embolic stroke and heart failure. Aside from age, a variety of cardiac and medical disorders might raise the risk of atrial fibrillation. Hypertension, cardiomyopathies, obstructive sleep apnea, and valvular dysfunction are examples. For many years, atrial fibrillation was not thought to be a hereditary disorder. The finding of the first lone atrial fibrillation in a family with an autosomal dominant inheritance pattern, on the other hand, suggested a genetic role in the development of atrial fibrillation. Following that, multiple investigations have indicated that atrial fibrillation, particularly lone atrial fibrillation, has a significant hereditary foundation.

Familial studies and population-based Genome-Wide Association Studies (GWAS) have revealed genetic alterations and polymorphisms linked to atrial fibrillation and explaining its heritability in part. Over 100 AF risk loci were discovered in a recent meta-analysis of GWAS for atrial fibrillation. The discovery of risk loci is simply the beginning of a long journey to figure out how these mutations enhance the risk of atrial fibrillation. Genetic variations can affect the pathophysiology of atrial fibrillation through changing the structure, and hence the expression and function, of proteins involved in many cellular functions.

The combination of a variety of environmental variables and individual lifestyles is known to cause variation in gene expression across cells and people, but the relationship between these external risk factors and the internal genetic machinery has remained unclear. The discovery of epigenetics has aided scientists in understanding the relationship between genes and the environment. Changes in gene expression that cannot be explained by changes in DNA sequence but instead come from changes in the packaging and/or translations of genetic information are referred to as "epigenetics."

Multiple epigenetic mechanisms, including as non-coding RNA molecule production, DNA methylation, and histone modification, regulate gene expression, resulting in dramatic changes in cellular structure and function, and so altering the organism's response to illnesses. Epigenetic processes are a means of controlling the cardiovascular transcriptome in a well-coordinated manner during development and illness. They can be acquired or inherited. Although the significance of epigenetic processes in Cardiovascular Diseases (CVDs) has received a lot of attention in recent years, its involvement in the initiation and progression of atrial fibrillation is still unclear. As new epigenetic processes are found, AF is being considered as a much more complicated and dynamic illness.

As a result, crucial new discoveries, such as novel atrial fibrillation biomarkers and treatment targets, are likely to emerge in the near future. The cellular and molecular underpinnings of atrial fibrillation, as well as the role of epigenetics in illness genesis and progression.

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Pathophysiology of atrial fibrillation

In health, the atrial myocardium is more prone to the production of very fast rates with complicated patterns of conduction than the ventricular myocardium due to variations in action potential length and refractory time. These physiological abnormalities promote fast and chaotic atrial activity in atrial fibrillation, which leads to decreased atrial function. Despite decades of research into the disease's activates and sustainer factors, atrial fibrillation mechanisms remain poorly understood, and the condition remains under-treated. The loss of synchronization of atrial contraction, according to a widely recognized mechanism of atrial fibrillation, is caused by randomly propagating waves with occasional blockades, annihilation, and re-generation of discrete waves. On an EKG, the lack of a P-wave and uneven QRS complexes indicate fast and disorganized atrial activity.