Distinctive electrophysiological characteristics and arrhythmogenesis in thoracic vein cardiomyocytes

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A trial fibrillation (AF) is the most common sustained arrhythmia, which can cause cardiac dysfunction and stroke in clinical medicine. Thoracic vein cardiomyocytes play a critical role in the pathophysiology of AF. Through experiments from pulmonary vein and superior vena cava myocardial cells, we found distinctive electrophysiological characteristics and high arrhythmogenesis in thoracic vein cardiomyocytes. These research data also have a critical influence on cellular mechanism of AF-mediated by large thoracic veins. Since calcium regulations play a critical role in AF genesis, we look into the calcium regulation in thoracic vein cardiomyocytes and found abnormal calcium homeostasis in pulmonary veins and left atrial post wall cardiomyocytes, which may induce the occurrences of AF. We created several AF cellular and animal models to link the calcium regulations and AF triggers. In addition, our group studied the epigenetic regulations on calcium homeostasis in cell and whole animals. We found hypermethylation can down regulate sarcoplasmic reticulum ATPase and methylation inhibition can be a potential treatment for heart failure and arrhythmia. Besides, histone deacetylases inhibition also improve heart failure and calcium dysregulation. Therefore, the modulations of methylation and histone deacetylases would be potential treatment strategies for heart failure and arrhythmia.

Biography

Yi-Jen Chen has completed his M.D. at the age of 25 years from Taipei Medical University and his Ph.D. at the age of 34 years from National Yang-Ming University. He is the director of Clinical Research Center, and the chairman of Council for Research and Development at Taipei Medical University Wan-Fang Hospital. He is also the professor of Graduate Institute of Clinical Medicine at Taipei Medical University. He has published more than 100 papers in reputed journals and serving as an editorial board member of repute.

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