Cardiac Research Team

**Major Research Aims**

My lab has devoted to find the clinical cardiovascular disease especially in the field of cardiac arrhythmia, investigate the probable mechanisms and treatment either in basic research or in clinical applications. We had established a series platform and animal models for research of the mechanism and potential treatment of cardiac disease. The research scope involves both atrial and ventricular arrhythmia and their potential etiologies such as renal failure, electrolytes imbalance, gender, dyslipidemia, degenerated heart (ex. Sinus node dysfunction) and the use of medication (ex. NSAID), or their potential therapeutic agents such as HDAC inhibitor or antioxidant.

**Representative figures of major achievements**

1. We highlighted the therapeutic potential of Histone deacetylase (HDAC) inhibitor and antioxidant on treatment of atrial fibrillation, which may reduce atrial arrhythmogenesis through multiple target modifications.
2. We explored the role of renal failure, electrolytes imbalance, gender, dyslipidemia, degenerated heart (ex. Sinus node dysfunction), and the use of medication (ex. NSAID) in the pathogenesis of atrial tachyarrhythmia. These studies point out the electrical interactions of sinus node and pulmonary vein cardiomyocytes and dysregulated calcium homeostasis in the pathogenesis of atrial fibrillation (Figure 1).
3. We studied the mechanism how epicardial fat, adipocytokines, and fatty acids modulating ionic currents and electrophysiologic characteristics of atria, which may potentiate the atrial fibrillation occurrence (Figure 2).
4. We researched the pathophysiology of ventricular tachyarrhythmia and found that RVOT cardiomyocytes contain distinctive calcium homeostasis with high arrhythmogenesis.

**Potential Mechanisms of Fat Arrhythmogenesis**

Calcium dysregulation with or without electrical and structural remodelings – vital to the pathophysiology of arrhythmogenesis

Proposed mechanism of epicardial fat induced electrophysiological and structural remodeling and potentiated AF occurrence.

**Major relevant publications**


**Staff and contact information**

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