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Symposium

The role of TRPM7 in fibrosis associated heart diseases

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Cardiac fibrosis is a hallmark of various heart diseases including hypertrophy, heart failure, and arrhythmia. Cardiac fibroblasts play an important role in fibrogenesis because they differentiate to myofibroblasts under various pathological conditions. Thus, targeting cardiac fibroblast differentiation to attenuate fibrosis represents a new therapeutic strategy for fibrosis associated heart diseases. We have previously used fibroblasts from atrial fibrillation (AF) patients and demonstrated that the Transient Receptor Potential Melastatin 7 (TRPM7) plays an essential role in fibroblast differentiation to myofibroblasts. Here we propose that TRPM7 plays a key role in fibrosis-associated arrhythmia. We used transverse aortic restriction (TAC) induced hypertrophy/heart failure mouse model to generate fibrosis in the hearts. We found that deletion of *Trpm7* (TRPM7-KO) significantly increased survival rate after TAC, and improved heart performance. Moreover, TRPM7 deletion drastically reduced fibrosis in both atria and ventricles. The reduced fibrosis in TRPM7-KO-TAC mice significantly decreased the vulnerability of AF and the duration of induced AF. Thus, TRPM7 plays an important role in fibrosis associated AF, and may serve as a therapeutic target for fibrosis associated arrhythmia.