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Editorial
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Involvement of Transient Receptor Potential Channels in Cardiac Health and Disease

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Abbreviations: TRP: Transient Receptor Potential; RT-PCR: Reverse Transcriptase Polymerase Chain Reaction

Editorial

Calcium is a critical 2nd messenger in cardiac function. It is vital for the cardiac excitation-contraction coupling and relaxation [1], important for the activation of pathways responsible for hypertrophic cardiac remodeling and heart failure [2], as well as for cardiac development, cardiac energy homeostasis, and eventually for cell death [3].

In addition to the pathways associated with fast Ca cycling, transient receptor potential (TRP) proteins have been revealed as cation channels involved in the action of catecholamines or angiotensin II in cardiac cells and as factors controlling cardiac functions [4]. To date, 28 mammalian TRP genes had been identified, that were divided into six associated subfamilies: TRPC (canonical), TRPV (vanilloid), TRPM (melastatin), TRPA (ankyrin), TRPML (mucolipin), and TRPP (polycystin) [5].

TRP channels are activated by many stimuli such as pressure, sheer stress, mechanical stretch, oxidative stress, phospholipids, and their metabolites. In response to these stimuli, TRP channels integrate and transduce their activity to the downstream cellular amplification system via Ca2+ entry and membrane depolarization, thus exhibiting a vital role in regulating essential cell functions such as contraction, relaxation, proliferation, differentiation, and cell death [6]. It has been stated that all TRPs are expressed in the heart and TRP transcripts are detected by reverse transcriptase polymerase chain reaction (RT-PCR), Northern blot analysis, in situ hybridizations, or microarray analysis, while TRP proteins are detected by Western blots, immunocytochemistry, immunohistochemistry, or immunoelectron microscopy [7].

In addition to expression in the healthy heart, TRP transcripts and proteins were found to be differentially expressed in cells and tissues from experimental models inducing cardiac remodeling or dysfunction as well as in myocardial biopsies from diseased human hearts. Increased expression of TRPC1, TRPC2, TRPC3, and TRPC6 has been reported in a rodent model of cardiac hypertrophy [8]. Moreover, by using a microarray screening, increased expression of TRPC1 and TRPC5 in human failing hearts [9], increased TRPC6 mRNA in human hearts with dilated cardiomyopathy [10], and downregulation of TRPC4 in ventricular myocytes in biopsies from patients with ischemic cardiomyopathy [11] were shown.

Additionally, TRPV2 expression is downregulated in rodent models of myocardial infarction [12] and upregulated together with TRPC1 in hearts from patients with dilated cardiomyopathy [13]. Several TRP channels have been shown to be involved in arrhythmogenesis; in patients with atrial fibrillation, TRPM6 mRNA and protein levels in the right atrium were significantly increased [14]. Analysis of mRNA and protein levels showed increased expression of TRPM7 in the left ventricle free wall from patients with ventricular tachycardia [15].

In conclusion, the TRP superfamily consists of various non-selective cation channels characterized by with variable degree of Ca^{2+}-permeability. The 28 mammalian TRP channel proteins can be grouped into six subfamilies: TRPC, TRPV, TRPM, TRPA, TRPP, and TRPML. TRP channels play a critical role in regulating essential cell functions such as contraction, relaxation, proliferation, differentiation, and cell death. Recent studies have revealed that TRP channels are involved in numerous cellular functions and

play an important role in the pathophysiology of many diseases in the cardiovascular system.

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