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Determining intracellular localization of cardiac TRPV2 channels, a novel therapeutic target for heart failure

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Management and treatment of heart failure is based on the therapeutic targeting of a limited number of receptors and pathways. Our recent work showed that Transient receptor potential vanilloid 2 (TRPV2) channels are highly expressed in the heart and their activity is associated with normal Ca2+ handling that modulates contractility on a beat-to-beat basis. We have shown that activation of TRPV2 acts as a positive inotrope to increase cardiac output without stimulating the β-adrenergic system, inducing myocardial apoptosis or arrhythmias. Thus, TRPV2 represents a novel therapeutic target for the treatment of heart failure.However, while TRPV2 stimulation may be beneficial to increase cardiac output for the treatment of heart failure, its increased expression during cell stress appears play a contributing role to cardiac hypertrophy. Therefore, to fully realize the translational potential of TRPV2, we need a better understanding of its mechanism of action. The aim of this study was to identify the functional intracellular localization of TRPV2 within cardiac myocytes.We used multiple approaches to demonstrate that TRPV2 may exist in two distinct pools in myocytes with expression on both the cytoplasmic membrane and sarcoplasmic reticulum. Our results also suggest that TRPV2's mechanism of action may depend on its distribution within the cell and which pool of TRPV2 is stimulated. These results increase our understanding of how TRPV2 mediates cardiac function and will facilitate its development as a therapeutic target.

Biography

Michael Tranter completed his PhD from the University of Cincinnati in 2010, and is now an Assistant Professor in the Division of Cardiovascular Health and Diseases at the University of Cincinnati College of Medicine. His work is focused on the role of gene expression in cardiovascular disease, specifically the development of cardiac hypertrophy.

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