assay were applied to observe the effect of the TRP channels on cell migration. Cell proliferation assay was made with MTT and ^{3}H-thymidine incorporation approaches.

Results: A small background current was inhibited by the TRPC channel blocker La^{3+}. Removal of Mg^{2+} of pipette solution or bath solution induced a Mg^{2+}-sensitive TRPM7 current, and the current was suppressed by the TRP channel blocker 2-aminoethoxydiphenyl borate. RT-PCR revealed significant mRNA expression of TRPC1, TRPC3, TRPC4, TRPV2, TRPV4, and TRPM7 channels in human preadipocytes. Western blot analysis confirmed the protein expression of these TRP channels. ShRNAs targeting TRPV2, TRPV4 and TRPM7 suppressed the corresponding gene and protein expression. Interestingly, TRPV2-shRNA and TRPM7-shRNA significantly reduced proliferation of human cardiac c-kit$^+$ cells. Migration of human cardiac c-kit$^+$ cells was reduced by TRPV2-shRNA, TRPV4-shRNA.

Conclusion: Our results demonstrate for the first time that multiple TRP channels, TPC1/3/4, TRPV1/2/4, and TRPM7, are present in human cardiac c-kit$^+$ cells. TRPV2, TRPV4 and TRPM7 channels participate in regulating migration and proliferation in human cardiac c-kit$^+$ progenitor cells.

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PATCH-CLAMP STUDY OF SINGLE RYANODINE RECEPTOR CHANNELS IN THE OUTER NUCLEAR MEMBRANE

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Modulation of cytoplasmic free calcium (Ca^{2+}) concentration is a universal signaling pathway that regulates numerous cellular processes. Ubiquitous intracellular Ca^{2+} release channels – inositol 1,4,5-trisphosphate receptor ($InsP_3R$) and ryanodine receptor (RyR) channels – localized in the sarco/endoplasmic reticulum (ER) play a central role in this pathway in all animal cells. Electrophysiological study of the single-channel conductance and gating properties of these Ca2+ release channels with conventional patch-clamp approach has been hindered by their intracellular localization. To overcome this limitation, patch-clamp electrophysiology has been applied on isolated nuclei where these Ca^{2+} release channels are found abundantly in the outer nuclear envelope. We have successfully uterlized this nuclear membrane electrophysiology to study the gating properties of single $InsP_3R$ channels in several cellular systems. Whereas, all the current single channel data, including channel conductance, permeation properties, and ligand regulation, of the RyR channels were done exclusively by reconstituting the channels into artificial planar lipid bilayers. To gain insights into the single channel properties of the RyR in its native membrane milieu, we applied nuclear membrane electrophysiological study on isolated nuclei from stable-inducible mouse RyR2 HEK-293 cells. Using potassium as charge carrier, caffeine activated single channel current with conductance of ~750 pS in isolated nuclei. This caffeine activated current showed a linear current/voltage relationship under symmetrical ionic conditions and was sensitive to non-specific RyR inhibitor, ruthenium red. Furthermore, the single RyR channels recorded from the outer nuclear membrane exhibited bi-phasic Ca^{2+} regulation. In conclusion, we demonstrated, for the first time, that single RyR channels recordings from isolated nuclei and our results suggested that the nuclear membrane electrophysiology could be a sensitive and robust technique to study the gating properties of intracellular channels, including the $InsP_3R$ and RyR.

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ROLES OF FUNCTIONAL ION CHANNELS IN HUMAN CARDIAC C-KIT+ PROGENITOR CELLS

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Background and objectives: Cardiac progenitor cells play an important role in cardiac repair and regeneration; however, cellular biology and electrophysiology are not understood. The present study was to investigate the functional ion channel expression in human cardiac c-kit$^+$ progenitor cells and the