S07-4 Function of TRIC channels in intracelular Ca2+ store

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Cytoplasmic Ca^{2+} is a major second messenger that controls important cellular functions including muscle contraction, transmitter release, gene transcription, cell growth, and cell death. Two principle sources provide Ca^{2+} to the cell: channels in the plasma membrane that open to allow external Ca^{2+} to enter the cell, and ryanodine receptor (RyR) and IP_3 receptor (IP_3R) in the endoplasmic/sarcoplasmic reticulum (ER/SR) that release Ca^{2+}. In particular, Ca^{2+} release from ER/SR was mediated by RyR or IP_3R. Ca^{2+} release via RyR and IP_3R takes advantage of overwhelming Ca^{2+} concentration gradient. Efficient Ca^{2+} release events would not be possible if only Ca^{2+} moved across the membrane; the rapid movement of the cations would quickly bring the ER/SR membrane potential to the Ca^{2+} reversal potential and should inhibit following Ca^{2+} release. Several K^+ and Cl^- selective channels on the ER/SR, which were previously reported by several investigators, could also participate in neutralizing the transient negative membrane potential generated by Ca^{2+} release, whereas the molecular identity of putative counter-ion channels remains unknown.

This symposium focuses on functional analysis of TRIC (trimeric intracellular cation) channels using with mutant mice lacking TRIC channels. Ubiquitously expressed TRIC channels seem essential for physiological Ca^{2+} release that regulates important cellular functions, and therefore could be unique targets in pharmaceutical or agrichemical applications.