Abstract of Presentation

Presentation Title:

Molecular components supporting ryanodine receptor-mediated Ca^{2+} release:
roles of junctophilin and TRIC channel in cardiac Ca^{2+} release

Abstract:

Ca^{2+} mobilization from intracellular stores is mediated by Ca^{2+} release channels, designated ryanodine and IP_3 receptors, and directly regulates important cellular reactions including muscle contraction, endo/exocrine secretion, and neural excitability. In order to function as an intracellular store, the endo/sarcoplasmic reticulum is equipped with cooperative Ca^{2+} uptake, storage and release machineries, comprising synergic collaborations among integral-membrane, cytoplasmic and luminal proteins. Our recent studies demonstrate that junctophilins form junctional membrane complexes between the plasma membrane and the endo/sarcoplasmic reticulum in excitable cells, and that TRIC (trimeric intracellular cation) channels act as novel monovalent cation-specific channels on intracellular membrane systems. Knockout mice provide evidence that both junctophilins and TRIC channels support efficient ryanodine receptor-mediated Ca^{2+} release in muscle cells. We would like to focus on cardiac Ca^{2+} release by discussing pathological defects of mutant cardiomyocytes lacking ryanodine receptors, junctophilins, or TRIC channels.