Calcium dyshomeostasis in disease and therapy

Guest Editor

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Message from the Guest Editor

Dear Colleagues,

The cytoplasmic free calcium ion concentration of mammalian cells is generally controlled at 100 ~ 200 nmol/L, while the extracellular and organelle calcium concentration is maintained at the 2 mmol/L level. Calcium homeostasis is essential for regulating almost all biological functions of the body, such as the heart and muscle contractions, neuro-information transmission, learning and memory, embryo formation and development, cell proliferation and apoptosis, cell division and differentiation, cell energy metabolism, protein phosphorylation and dephosphorylation modification, and gene expression and regulation. Therefore, calcium dyshomeostasis can lead to widespread impairment of cellular signaling, subsequently contributing to multiple diseases. Calcium channels, calcium pumps and calcium transporters mediate calcium ions into the cytoplasm and organelles. Abnormalities in these calcium channels, calcium pumps and calcium transporters may cause instability of calcium homeostasis. However, there are still knowledge gaps in the pathophysiology. Elucidating the regulation mechanism is one of the basic links to reveal the regulation rules of calcium dyshomeostasis and human disease, for possible therapeutic strategies for improving treatment. The exact pathophysiological mechanisms of calcium dyshomeostasis are complex. Understanding
disease related downstream pathways detected and identified by using human tissue or animal models, which will take research forward in an important step. The goal of this special issue is to collate articles related to the pathophysiology of calcium dyshomeostasis involved human disease and therapy. We encourage interested investigators to submit case reports, reviews and original research based on cell models, animal models, human samples and clinical cases aiming to promote the research progress of calcium dyshomeostasis.

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