



Calcium Signaling in Mammalian Cells: From Physiology to Pathology

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Message from the Guest Editor

The changes in intracellular Ca^{2+} concentration ($[\text{Ca}^{2+}]_i$) regulate a wide repertoire of functions, such as exocytosis, gene expression, metabolism, proliferation, and cell death. In the case of excitable tissues, calcium signals with different spatiotemporal characteristics modulate and integrate the activity of myriads of cells. Intracellular Ca^{2+} homeostasis is maintained by Ca^{2+} ATPases, exchangers, ion channels, and buffer systems, including calcium-binding proteins, internal stores, and mitochondria. This machinery provides Ca^{2+} inflow and outflow, while G-protein coupled receptors are considered the key modulators of Ca^{2+} dynamics. Taking into account the pivotal role of Ca^{2+} in cellular physiology, it is not surprising that a disruption in Ca^{2+} handling may be the reason behind or consequence of different pathologies. In this regard, pharmacological correction of Ca^{2+} homeostasis may serve as a therapeutic approach for the treatment of several diseases. This Special Issue on “Calcium Signaling in Mammalian Cells: from Physiology to Pathology” welcomes research dealing with any aspects of intracellular Ca^{2+} signaling in mammalian cells in health and diseases.





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