Special Issue

Cellular Mechanisms in Mitochondrial Function and Calcium Signaling

Message from the Guest Editors

Calcium (Ca2+) is a ubiquitous second messenger whose signaling is regulated by compartmentalization and transport across several cellular membranes. Ca2+ controls a wide range of cellular processes, from cellular contraction to exocytosis or gene expression. Mitochondria transform the free energy stored in highly reduced compounds into chemical energy in the form of ATP to support energy-consuming cellular processes across the cell. Beyond this well-known role, mitochondria are crucial in apoptosis, metabolite biosynthesis, and cellular immune response, among other cellular processes. Mitochondrial function is highly integrated within the specific cellular context of each cell, and Ca2+ signals play a key role in maintaining it. Moreover, they frequently also involve other organelles such as the plasma membrane or the endoplasmic/sarcoplasmic reticulum. This Special Issue aims to publish manuscripts that explore the intricate relationships between mitochondria and cellular calcium homeostasis in both health and disease.

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About the Journal

Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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manuscripts are peer-reviewed and a first decision is provided to authors approximately 16 days after submission; acceptance to publication is undertaken in 2.7 days (median values for papers published in this journal in the first half of 2025).