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Ca^{2+} Signaling and Mitochondrial Function in Neurodegenerative Diseases

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Message from the Collection Editors

Dear Colleagues,

Ca^{2+} is a ubiquitous second messenger that regulates numerous cell processes, such as synaptic function, memory formation, bioenergetics, and gene transcription. Substantial evidence accumulated over the last three decades shows that in several neurodegenerative illnesses, including Alzheimer's, Parkinson's, and Huntington's diseases, neuronal and glial Ca^{2+} signaling is irreversibly disrupted at all these spatiotemporal scales.

The purpose of this Special Issue is to overview the current status of the field and highlight the new findings about the role of impaired Ca^{2+} homeostasis in neurodegenerative diseases and the novel experimental and computational approach now available for their investigation. Special focus will be placed on the role of intracellular oligomeric peptides and intracellular organelles in Ca^{2+} signaling impairments, Ca^{2+} mediated changes in mitochondrial function, and the feedback loop between Ca^{2+} disruptions, intracellular organelles, and amyloidosis.

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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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