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Calcium Signalling in Alzheimer's Disease: From Pathophysiological Regulation to Therapeutic Approaches

Guest Editor:

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Deadline for manuscript submissions:
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Message from the Guest Editor

Dear Colleagues,

In recent decades, several studies have reported calcium dysregulation in AD, affecting different cellular compartments, such as mitochondria, endoplasmic reticulum, lysosomes, and several microdomains within the plasma membrane, and occurring through a broad intervention of several calcium signaling "tool-kits" (receptors, channels, binding proteins, etc.). The obtained results depict calcium signaling dysregulation as a common proximal cause of dysfunctional neurons and also glial supporting cells. The objective of this Special Issue is to gather the newest results and advances on: i) calcium signaling deregulation mechanisms in AD, ii) how they are linked to other players involved in AD pathogenesis, and iii) potential therapeutic approaches to correct calcium alterations to treat AD.

Keywords: Alzheimer's disease; aging; amyloid β; amyloid precursor protein; calcium; Ca^{2+} signaling; Ca^{2+} channels; Ca^{2+} receptors; Ca^{2+} binding proteins; endoplasmic reticulum; mitochondria; lysosomes; synaptic plasticity; IP3R; RyR; SOCE; presennilin; glutamate receptors; AMPA receptors; neurons; astrocytes; microglia; neurodegeneration

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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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