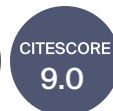




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## Calcium Signalling in Alzheimer's Disease: From Pathophysiological Regulation to Therapeutic Approaches

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Deadline for manuscript  
submissions:

**closed (31 August 2020)**

### 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  $\beta$ ; amyloid precursor protein; calcium;  $\text{Ca}^{2+}$  signaling;  $\text{Ca}^{2+}$  channels;  $\text{Ca}^{2+}$  receptors;  $\text{Ca}^{2+}$  binding proteins; endoplasmic reticulum; mitochondria; lysosomes; synaptic plasticity; IP3R; RyR; SOCE; presenilin; glutamate receptors; AMPA receptors; neurons; astrocytes; microglia; neurodegeneration



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