# **Special Issue**

# Autophagy in Neurological Disorders

## Message from the Guest Editors

Autophagy is an intracellular lysosomal degradation pathway by which cytoplasmic cargoes are removed to maintain cellular homeostasis. Dysregulation of this process is linked to loss of cell proteostasis and linked to diverse pathologies. In this Special Issue, we aim to focus on the relevance of the autophagic process in maintaining neuronal health and how it is linked to neurological disorders. Autophagy is essential in neurons and glial cells that need to cope with prolonged and sustained operational stress, and thus autophagy is known to protect against different neurological disorders, such as neuroinfectious diseases, nerve injury diseases, and neurodegenerative diseases. To tackle these pathologies, it is crucial to understand the physiological relevance of autophagy in these cells and how this pathway is linked to pathologies in the nervous system. At the same time, this research remains critical to developing biomarkers and pharmacological agents. In this Special Issue, we welcome any publication related to autophagy, either general autophagy or selective autophagy, or general loss of cell proteostasis or lysosomal-related phenotypes in these disorders.

## **Guest Editors**

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## Deadline for manuscript submissions

30 April 2026



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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.

### **Editors-in-Chief**

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