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Amyotrophic Lateral Sclerosis: From Molecular Mechanisms to Therapeutic Opportunities

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Deadline for manuscript submissions:
closed (31 January 2024)

Message from the Guest Editors

Dear Colleagues,

In recent years, the understanding of ALS has been fundamentally revolutionized: Thus, it is considered a neuromuscular multisystem disease on a neurodegenerative basis which forms a disease spectrum with the frontotemporal dementias. Since the discovery of TDP43 as the major component of cytoplasmic polyubiquitinylated inclusions in 2006, many novel ALS-causing genes have been identified, with both genetic and pathological overlap with frontotemporal dementias. However, the functions or properties of these ALS genes can be grouped into distinct groups, which has had a significant impact on the understanding of pathophysiology. These groups include axon structure and function, protein metabolism (including autophagy and protein quality control), RNA metabolism (regulation transcription, splicing, RNA transport, RNA granule dynamics), as well as cytoplasmic protein mislocalization and phase transition. Thus, newly discovered mechanisms are increasingly being incorporated into novel therapeutic targets and strategies. This Special Issue aims to collect papers discussing such novel aspects of ALS research, from basic science to clinical translation.

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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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