Special Issue

Genetics and Pathomechanisms of Amyotrophic Lateral Sclerosis (ALS)

Message from the Guest Editor

Amyotrophic lateral sclerosis (ALS) is a fatal motor neuron disease characterized by the degeneration of upper and lower motor neurons, leading to muscle atrophy, paralysis, and death from respiratory failure. Advances in technology, including genome-wide association studies and next-generation sequencing, have identified several ALS-linked genes. Among the 50 potential causative or modifying genes, pathogenic variants in SOD1, C9ORF72, FUS, and TARDBP are most common in familial ALS. Mechanistic studies reveal that disruptions in axonal trafficking, ER proteostasis, and autophagy contribute to motor neuron degeneration. Stress in the endoplasmic reticulum (ER) and mitochondria triggers the unfolded protein response (UPR), while disruptions in Ca2+ handling generate reactive oxygen species, causing cellular stress. Impaired glutamate neurotransmission and glial glutamate uptake also contribute to motor neuron degeneration. Targeting ER stress, mitochondrial responses, and modulating neuronal excitability offers promising therapeutic strategies for ALS.

Guest Editor

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