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Sommario/riassunto	Neurodegenerative disorders are characterized by the progressive loss of specific populations of neurons with consequent deterioration of brain’s function and dramatic impact on human behavior. At present, there are no effective cures for neurodegenerative diseases. Because unambiguous diagnosis is possible only after manifestation of symptoms, when a large proportion of neurons has been already lost, therapies are necessarily confined to alleviation of symptoms. Development of cures halting the disease course is hampered by our rudimentary understanding of the etiopathology. Most neurodegenerative disorders are sporadic and age-related and - even for those of known genetic origin - the mechanisms influencing disease onset and progression have not been fully characterized. The different diseases, however, share important similarities in the mechanisms responsible for neuronal loss, which is caused by a combination of endogenous and exogenous challenges. Trophic deprivation, oxidative stress, accumulation of abnormal protein aggregates, and bioenergetics defects have been described in most, if not all,

neurodegenerative disease.To counterbalance these noxious stimuli cells deploy, at least during the initial pathogenic states, intrinsic neuroprotective responses. These are general compensatory mechanisms, common to several neurodegenerative conditions, which reprogram cellular physiology to overcome stress. Adaptation includes strategies to optimize energetic resources, for instance reduction of rRNA synthesis to repress translation, suppression of transcription, and bioenergetics and metabolic redesign. Additional mechanisms include potentiation of antioxidant capacity, induction of endoplasmic reticulum (ER) stress, and activation of protein quality control systems and autophagy. Ineffective execution of these compensatory strategies severely threatens cellular homeostasis and favors onset of pathology. Therefore, a better understanding of these “buffering” mechanisms and of their interconnections may help to devise more effective therapeutic tools to prolong neuronal survival and activity, independently of the original genetic mutations and stress insults.This Research Topic focuses on the initial compensatory responses protecting against failure of those mechanisms that sustaining neuronal survival and activity. The collection intends to summarize the state-of-the-art in this field and to propose novel research contributes, with the ultimate goal of inspiring novel studies aimed to contrast progression of neurodegenerative diseases.