CALL FOR PAPERS

A large number of human diseases, including Alzheimer’s disease (AD), Parkinson’s disease (PD), type II diabetes and a range of systemic amyloidoses, are generally described as protein misfolding diseases. All of them are associated with the conversion of specific proteins from their soluble states into aggregated species that become deposited within organs and tissues, either as intracellular inclusions or extracellular deposits. These pathological disorders share common risk factors such as oxidative stress, aging, cellular stress, and protein dysfunction. Although misfolded proteins are generally inactive, their accumulation can cause stress responses in cells and organelles such as mitochondria and endoplasmic reticulum. Therefore, maintaining intracellular protein homeostasis by balancing protein folding and misfolding is of fundamental importance.

Two major pathways degrade most cellular proteins in eukaryotic cells and are critical for the maintenance of cellular homeostasis: the ubiquitin–proteasome system (UPS), which degrades the majority of proteins and autophagy, responsible for the degradation of most long-lived or aggregated proteins and cellular organelles. Disruption of these processes can contribute to the pathology of a variety of human diseases, including neurodegenerative disorders. The UPS and autophagy mechanisms were primarily thought to be distinct catabolic pathways and were therefore investigated separately. However, recent advances revealed crosstalk mechanisms involving these pathways, which cooperate in a network that includes chaperones, co-chaperones, and stress-sensing molecules. Mitochondrial proteostasis is also critical for cell survival and its dysfunction can lead to the accumulation of reactive oxygen species, which can be disruptive to overall cellular proteostasis. Another aspect to consider is the long-term activation of the unfolded protein response (UPR), which mediates neuronal dysfunction in neurodegenerative disorders. Understanding crosstalk among the various elements of the UPR, as well as how all of these activities are linked with UPS, mitochondrial function, and autophagy, should provide new treatment options for various pathologies including neurodegenerative disorders, diabetes, and inflammatory diseases.

In this special issue, we invite researchers to contribute original research articles describing novel cellular and molecular mechanisms, linking the disruption of intracellular protein homeostasis with oxidative stress and the development of protein misfolding diseases. Studies characterizing the crosstalk between UPS and autophagy and the implication of UPR in these two systems are particularly encouraged. Review articles describing the current state of the art are also welcome.

Potential topics include but are not limited to the following:

- Proteostasis regulation during redox imbalance in protein misfolding diseases
- Oxidative stress and alteration of proteostasis network as a possible mechanism triggering neurodegeneration
- The link between type II diabetes and Alzheimer’s disease: potential role of oxidative stress and impaired cellular proteostasis
- Disruption of autophagy and impact on cellular oxidative stress in neurodegenerative diseases: from molecular mechanisms to targeted therapies
- A close relationship between ROS and unfolded protein response (UPR) in protein misfolding diseases

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Papers are published upon acceptance, regardless of the Special Issue publication date.