Oxidative Medicine and Cellular Longevity

Special Issue on

Redox Status and Proteostasis in Ageing and Disease

CALL FOR PAPERS

Proteins are the most versatile and structurally complex biological macromolecules. Specifically, proteins must achieve and maintain their native conformation in order to ensure optimal operation of most (if not all) cellular functions. Cells possess an extensive network of components that safeguard the functionality and integrity of proteins, thus maintaining homeostasis of the proteome (proteostasis). The proteostasis network (PN) is comprised of several modules that control protein synthesis, folding, trafficking, and degradation, in order to finely orchestrate both stability and functional properties of the proteome. Deficiencies in PN have been shown to facilitate onset or progression of several diseases, such as neurodegeneration, type 2 diabetes, cancer, and inflammatory and cardiovascular disease, as well as a number of other age-related pathological conditions. PN functionality and protein quality maintenance can be intracellular redox-regulated at many levels. Experimental evidences suggest that production of reactive oxygen species (ROS) or reactive nitrogen species (RNS) and oxidative/nutritive stress can be both the cause and the effect of a complex adaptive signalling that aims to maintain cellular ROS/RNS levels within physiological levels and restore proteostasis. These mechanisms include (among others) the cellular antioxidant responses (e.g., the Keap1/Nrf2 pathway), the unfolded protein response (UPR) of the endoplasmic reticulum (ER), and an armada of intra- and extracellular chaperones, as well as the degradation modules, namely, the ubiquitin-proteasome system (UPS) and the autophagy-lysosome system (ALS). Particularly, chronic ER stress and activation of the UPR through endogenous or exogenous insults may result in impaired redox and calcium homeostasis, resulting in oxidative stress that may ultimately impact mitochondrial functions. Conversely, ROS generated through inflammation or mitochondrial dysfunction may accelerate ER malfunction. Focused research and in-depth investigations of this field are needed to establish a strong rationale for the development of new therapeutic strategies.

In this special issue, we aim to supply an updated overview of the aforementioned PN regulating pathways. Also, this special issue will focus on the cross talk between ER stress-induced inflammatory pathways, oxidative stress, and mitochondrial signaling events, which may negatively impact proteostasis, thus representing a major contributor to the pathogenesis of many chronic or age-related diseases.

We invite investigators to contribute original research articles and review articles as well as clinical studies that will stimulate the continuing efforts to understand the connections between failures in the maintenance of the cellular redox balance and the impairment of the protein quality control system. Studies on the dysfunction of proteostasis in chronic and age-related diseases are also encouraged.

Potential topics include, but are not limited to:

- Oxidative stress pathway and the UPR: the role of ER in health and disease
- Mechanisms and implications of ROS/RNS generation during the UPR
- Redox signaling and proteostasis
- Role of molecular chaperones in the maintenance of proteostasis
- Chronic diseases, redox balance, and loss of protein quality control
- Current strategies to reduce the impact of oxidative stress on the proteostasis network
- Impact of the main degradation modules (i.e., UPS and ALS) on redox signalling, ageing, and age-related diseases

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/omcl/rspa/.

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