

Special Issue on

Interplay between Redox Signaling, Oxidative Stress, and Unfolded Protein Response (UPR) in Pathogenesis of Human Diseases

CALL FOR PAPERS

Endoplasmic reticulum (ER) stress triggers complex adaptive or proapoptotic signaling defined as the unfolded protein response (UPR), involved in several pathophysiological processes. Protein misfolding in the ER triggers the activation of three homologous transmembrane protein kinases, Ire1, Ire1, the PKR-like ER kinase (PERK), and the transmembrane transcription factor ATF6. In previous studies, promising small molecule inhibitors targeting UPR's kinases were developed; however most triggered severe cytotoxic effects.

Protein folding is highly redox-dependent; the relations between generation of oxidative stress and ER stress have become very interesting fields for investigation. Evidence suggests that ROS production and oxidative stress not only are coincidental to ER stress but are integral UPR components. These components are triggered by distinct types of ER stressors and facilitate either proapoptotic or proadaptive UPR signaling. Thus, ROS generation can be upstream or downstream UPR targets. Pathways involved in unfolded protein response are important for normal cellular homeostasis and organismal development and may also play key roles in the pathogenesis of many diseases. Thus, we invite authors to contribute original research as well as review articles that will illustrate and stimulate the growing efforts to understand the implication of ER stress in human diseases. We are interested in articles describing the role of ER stress as the source and effect of the cells redox/oxidative stress.

We are interested in articles describing the role of ER stress, redox signaling, and oxidative stress in the pathogenesis of human disorders, such as cancer and inflammatory and degenerative diseases (including basic mechanistic pathways, translation, and clinical research). The issue is open to novel approach on the field; however, considering nowadays scientific trends and gaps in current state of knowledge, we suggest some aspects to be most beneficial.

Potential topics include but are not limited to the following:

- ▶ Controlling redox homeostasis by unfolded protein response pathways
- ▶ Regulation of redox signaling and antioxidant pathways genes by UPR-dependent transcription/translation regulatory proteins (ATF4, ATF6, eIF2etc.)
- ▶ Correlation between patients clinical parameters and oxidative/ER stress status, application of reliable, and authoritative markers
- ▶ Signaling between three most redox-sensitive organelles: ER, mitochondria, and peroxisomes
- ▶ Discovery of small molecule modulators to target vital players in UPR-redox/oxidative pathways
- ▶ Cross-talk between UPR, oxidative stress, and redox signaling and its implication for crucial cell biology pathways such as apoptosis, autophagy, senescence, and cell cycle regulation
- ▶ Possibilities to take advantage of simultaneous targeting UPR and redox/oxidative pathways in new treatment strategies

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/omcl/tres/>.

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

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