

## Special Issue on **Targeting Redox Mechanisms for Novel Cardiopulmonary Therapeutics**

# CALL FOR PAPERS

Extensive research in recent years has suggested that low levels of reactive oxygen species (ROS) are essential for redox signaling and normal physiological health, while supraphysiological levels can lead to disrupted signaling and cellular injury. Many human diseases are associated with or exacerbated by oxidative stress and novel approaches to mitigate oxidative stress are needed for prevention and/or treatment. Roles for redox mechanisms have been defined in the pathophysiology of pulmonary diseases such as asthma, bronchopulmonary dysplasia (BPD), acute respiratory distress syndrome (ARDS), pulmonary hypertension, and cardiovascular diseases, such as atherosclerosis and ischemia/reperfusion injury, making redox-related processes a logical target for prevention and therapeutic development.

We invite authors to contribute original research articles as well as review articles that will illustrate and stimulate the rapid advances that are taking place in the field of preventative/therapeutic development to address redox mechanisms and human diseases. We are particularly interested in manuscripts describing studies targeting of redox signaling pathways for the treatment and prevention of cardiopulmonary diseases using animal or in vitro models.

Potential topics include but are not limited to the following:

- ▶ Pulmonary diseases such as asthma, BPD, ARDS, and pulmonary hypertension
- ▶ Cardiovascular diseases such as atherosclerosis and ischemia/reperfusion injury
- ▶ ROS and redox signaling in myocardial ischemia-reperfusion injury and cardioprotection
- ▶ Age-related cardiovascular disease using redox stress markers

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

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

### **Lead Guest Editor**

Bhagavatula Moorthy, Baylor College of Medicine, Houston, USA  
[bxmoorth@texaschildrenshospital.org](mailto:bxmoorth@texaschildrenshospital.org)

### **Guest Editors**

Lynette K. Rogers, Ohio State University, Columbus, USA  
[lynette.rogers@nationwidechildrens.org](mailto:lynette.rogers@nationwidechildrens.org)

Jason Richardson, Florida International University, Miami, USA  
[jarichar@fiu.edu](mailto:jarichar@fiu.edu)

### **Submission Deadline**

Friday, 3 April 2020

### **Publication Date**

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