

Special Issue on **Redox Control of Vascular Biology**

CALL FOR PAPERS

Reactive oxygen species (ROS) and reactive nitrogen species (RNS) at physiological levels are important signaling molecules maintaining cellular homeostasis. However, excessive ROS/RNS is either a cause or an important mediator of several pathologies. Such a process regulated by ROS is vascular angiogenesis.

The endothelium plays a critical part in the regulation of vascular function. Endothelial dysfunction refers to a pathophysiological disease state in which homoeostatic functions of endothelial cells are perturbed leading to tissue ischemia, atherothrombosis, and infarction.

While excessive ROS produced during ischemia and reperfusion in the vascular tissue cause significant injury to the endothelial cells, ROS also triggers repairing of the vascular tissues that have been damaged during ischemia and reperfusion through angiogenesis in a highly coordinated manner mediated by redox sensing transcription factors and angiogenic genes.

We invite researchers to contribute original research articles as well as review articles that will enhance the ongoing efforts to understand redox signaling in the context of vasculopathy.

Potential topics include but are not limited to the following:

- ▶ Redox signaling influencing angiogenesis and vascular formation
- ▶ Regulators and effects of oxidative stress in vascular endothelial cells (examples include noncoding RNAs, genetic dysregulation, and epigenetic modifications)
- ▶ Influence of redox molecules on cell-cell communication mediated by extracellular vesicles
- ▶ Redox imbalance and treatments targeting oxidative stress in vascular pathology (hypertension and heart failure, wound healing, retinal angiogenesis, diabetes, and tumor angiogenesis)
- ▶ Role of oxidative stress on endothelial cell apoptosis and vascular ageing

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

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

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