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Smooth Muscle and Endothelial Cells as Pharmacological Targets for Acute Oxidative Stress

Guest Editor:

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Deadline for manuscript submissions:
25 November 2024

Message from the Guest Editor

Dear Colleagues,

Reactive oxygen species (ROS) play an important role in physiological cell signaling. However, excessive levels of ROS damage cells. Acute oxidative stress results from ischemia reperfusion injuries such as stroke, myocardial infarction, and vascular occlusion during surgery in addition to acute injuries such as traumatic brain injury. While great attention has been devoted to limiting damage from acute oxidative stress on the surrounding tissue, the development of strategies to limit smooth muscle and endothelial cell damage to maintain perfusion to the effected tissue is emerging as a promising therapeutic focus.

Damage to smooth muscle and endothelial cells can impair vasomotor control and result in cell death. Recent efforts have begun to evaluate how to ameliorate this damage. In this Special Issue, we seek to bring together research from experts in the field to highlight pharmacological targets to limit vascular cell damage in response to acute oxidative stress. I look forward to receiving your valuable contributions.

Dr. Charles E. Norton
Guest Editor

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