



NADPH Oxidase–Mediated ROS in Pulmonary Arterial Hypertension

Guest Editor:

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Message from the Guest Editor

Pulmonary arterial hypertension (PAH) is a progressive disease. How PAH develops remains incompletely understood, but evidence for production of reactive oxygen species (ROS) in pulmonary arteries (PA) has been well documented. The NADPH oxidase (Nox) family is comprised of seven structurally related transmembrane NADPH-dependent. Accumulating evidence strongly suggests that ROS derived from NADPH oxidase play an important role in pulmonary arterial remodeling.

Suppressing pathophysiological levels of ROS by manipulating NOX enzymes, their regulators, and effector ROS targets appears to be attractive therapeutic strategies to treat PAH and other related pulmonary vascular diseases. We invites original research and review articles on the subject of NADPH-oxidases in the context of pulmonary vascular pathophysiology and all related topics indicated below:

Reactive oxygen species; Inflammation; Pulmonary arterial hypertension; Redox signaling; Endothelial cell; Pulmonary arterial remodeling; Adventitia; Fibrosis





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Message from the Editor-in-Chief

It has been recognized in medical sciences that in order to prevent adverse effects of "oxidative stress" a balance exists between prooxidants and antioxidants in living systems. Imbalances are found in a variety of diseases and chronic health situations. Our journal *Antioxidants* serves as an authoritative source of information on current topics of research in the area of oxidative stress and antioxidant defense systems. The future is bright for antioxidant research and since 2012, *Antioxidants* has become a key forum for researchers to bring their findings to the forefront.

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