Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Mitophagy and Mitochondrial Proteostasis in Cardiovascular Diseases

CALL FOR PAPERS

Cardiovascular disease (CVD) affects the heart or blood vessels. CVD is the main cause of human death. More people die annually from CVD than from any other cause. An estimated 17.9 million people died from CVD in 2019, representing 31% of all global deaths. Of these deaths, 85% are due to heart attacks and stroke. It is widely recognized that mitochondrial malfunction is an early and prominent sign of myocardial damage. Mitophagy is an evolutionarily conserved cellular process that involves engulfing impaired or superfluous mitochondria, which are then degraded by lysosomes. Mitochondrial proteostasis is a dynamic balance in mitochondrial protein synthesis, transport, localization, expression, and degradation.

In response to damage from transient hypoxia or mild oxidative stress, the mitochondrial protein quality control machinery is activated to maintain the diversity and function of mitochondrial proteins through the activity of chaperones and proteases, and induction of the mitochondrial unfolded protein response. When damaged mitochondria cannot be repaired, they are degraded through mitophagy in a receptor-dependent or independent manner. Therefore, it is acknowledged that an efficient mitophagic response and mitochondrial proteostasis are vital for mitochondrial function and concomitant myocardial performance. During CVD, such as myocardial infarction, sepsis-related myocardial depression, heart failure, and diabetic cardiomyopathy, mitochondrial proteostasis is disrupted. The effect is accompanied by the accumulation of unfolded or abnormal proteins within mitochondria, contributing to mitochondrial damage and subsequent cardiomyocyte dysfunction.

However, it remains unclear how mitochondrial proteostasis is fine-tuned during various CVDs. Moreover, clinically useful compounds or drugs that affect the activation or transduction of mitochondrial proteostasis in CVD are thus far lacking. Although most of the literature considers mitophagy to be the guardian of mitochondrial function and cardiomyocyte homeostasis, different adaptors or signaling pathways can trigger mitophagy to varying degrees, promoting either cell survival or cell death. Moderate mitophagy selectively removes damaged mitochondrial subpopulations, an effect followed by an increase in ATP production. In contrast, excessive mitophagy determines cell death by depleting cellular ATP reserves. It still remains unclear to what extent mitophagy activation contributes to mitochondrial dysfunction and cellular energy deficit in CVD.

The aim of this Special Issue is to discuss recent advances in the regulatory roles and molecular mechanisms underpinning mitophagy and mitochondrial proteostasis in the pathogenesis of CVD. Submissions should highlight novel therapeutic approaches targeting mitophagy and mitochondrial proteostasis for the treatment of CVD. Original research and review articles are welcome.

Potential topics include but are not limited to the following:

- Molecular mechanisms underlying impaired mitophagy and dysregulated mitochondrial proteostasis in the pathogenesis of CVD
- Interactive actions between mitophagy and mitochondrial proteostasis during CVD
- Pharmacologic and non-pharmacologic preventive approaches for the treatment of CVD with a focus on mitophagy and mitochondrial proteostasis
- Clinically relevant information on the effects of therapies for CVD through the regulation of mitophagy and mitochondrial proteostasis
- Recent advances in the knowledge and understanding of mitophagy and mitochondrial proteostasis in CVD

Authors can submit their manuscripts through the Manuscript Tracking System at https://review.hindawi.com/submit?specialIssue=872555.

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

Lead Guest Editor
Jin Wang, Chinese PLA General Hospital, Beijing, China
nkuwangjin@163.com

Guest Editors
Sam Toan, University of Minnesota, Duluth, USA
stoan@d.umn.edu

Nan Hu, University of Wyoming, Laramie, USA
nhu@uwyo.edu

Submission Deadline
Friday, 17 September 2021

Publication Date
February 2022