Oxidative Medicine and Cellular Longevity

Special Issue on

Hormesis: Autophagy, Ferroptosis, and Other Cellular Mechanisms

CALL FOR PAPERS

Hormesis encompasses the notion that low levels of stresses stimulate or upregulate existing cellular and molecular pathways that improve the capacity of cells and organisms to withstand greater stress. The main hormetic agents identified so far are irradiation, calorie restriction, resveratrol, rapamycin, p53-inducing agents, heavy metals, antibiotics, ethanol, prooxidants, physical exercise, heat shock, and hypoxia. The hormetic response includes the expression of genes that encode cytoprotective proteins such as chaperones like heat-shock proteins, antioxidant enzymes, and growth factors.

Moreover, hormesis mediates a crosstalk between autophagy and cell death. Autophagy, a lysosomal degradation pathway for cellular constituents and organelles, is an adaptive and essential process required for cellular homeostasis. The hormetic capability is to possess both antioxidant and prooxidant properties that are closely related to autophagic and cell death activation processes. On the other hand, ferroptosis is dependent upon intracellular iron, but not other metals, and is morphologically, biochemically, and genetically distinct from apoptosis, necrosis, and autophagy, thereby suggesting that, beyond apoptosis, necrosis, and autophagy, ferroptosis is a fourth programmed cell death. Reactive oxygen species (ROS) are deeply involved in ferroptosis. Thus, whether hormesis also mediates a crosstalk between ferroptosis and cell death is of great interest.

We invite investigators to contribute original research articles as well as review articles that are concerned with the relationships among hormesis, autophagy, and ferroptosis in connection with cell death.

Potential topics include, but are not limited to:

- Hormesis, autophagy, and cell death
- Hormesis, ferroptosis, and cell death
- Hormesis as a phenomenon in redox biology
- Role of ROS in autophagy
- Specific role of iron in ferroptosis

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/omcl/hafo/.

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