Oxidative Medicine and Cellular Longevity

Special Issue on

Oxidative Stress and Inflammation Interaction in Ischemia Reperfusion Injury: Role of Programmed Cell Death

CALL FOR PAPERS

Oxidative stress and inflammation are interactive and play critical roles in ischemia reperfusion injury in multiple organs. During prolonged ischemia, tissues are energetically deficient. When restoring blood flow, reperfusion not only introduces abrupt energy shift to tissues, but also paradoxically exacerbates tissue injury by inducing oxidative stress and inflammation. These molecular events can lead to different types of programmed cell death (e.g., apoptosis, autophagy, necroptosis, pyroptosis, and ferroptosis) depending on the stimulation (i.e., oxidative stress, or inflammation, or their combination). For example, oxidative stress can induce cell apoptosis and ferroptosis by increasing proapoptotic signal (e.g., increase of caspases 3) and lipid peroxidation; further, it can prompt cell necroptosis by enhancing necroptosis-related protein receptor-interacting protein kinase 3. On the other hand, inflammation can elevate proinflammatory cytokines and chemokines release inducing inflammasome-mediated cell pyroptosis. Each of these types of cell death can solely or jointly disrupt autophagy, resulting in ischemia reperfusion injury. However, the molecular mechanism(s) of the interaction of oxidative stress and inflammation and their interplay with different types of programmed cell death in ischemia reperfusion injury are unclear.

We invite investigators to contribute original research articles as well as review articles that will stimulate the continuing efforts to understand these molecular mechanism(s). Knowledge on the interaction of oxidative stress and inflammation, and their interplay with programmed cell death (e.g., apoptosis, necroptosis, autophagy, pyroptosis, and ferroptosis) in ischemia reperfusion injury under normal and diseased conditions will help to develop strategies in combating these pathological conditions.

Potential topics include but are not limited to the following:

- Role and mechanism of the interaction of oxidative stress and inflammation in ischemia reperfusion injury in different organs (heart, lung, brain, liver, kidney, and/or intestine) under normal and diseased conditions (e.g., diabetes and aging)
- Roles of different programmed cell death in organs ischemia reperfusion injury under normal and diseased conditions
- Cellular protective interventions targeting programmed cell death (apoptosis, necroptosis, and autophagy) that contribute to cellular repairing during ischemia reperfusion injury
- Recent advances in preventing ischemia-reperfusion injury with a focus on oxidative stress and inflammation-mediated programmed cell death

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

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

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