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Oxidative Stress and Cardiac Turnover Capacity

Guest Editor:

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Deadline for manuscript submissions:
closed (31 October 2021)

Message from the Guest Editor

Oxidative stress has been demonstrated to be crucial to the onset and progression of many pathophysiological conditions. The heart is an organ particularly rich in mitochondria, dysregulation of this large mitochondrial mass alters many signaling pathways crucial to conditioning in numerous cardiac dysfunctions. Although an important body of knowledge is being accumulated on mature cells, less clear evidence exists on the impact of oxidative stress on the capacity of the adult heart to self-repair, especially with regard to resident multipotent progenitors and de-differentiating cardiomyocytes.

We invite you to submit original research or review articles to this Issue, which will bring together current research on the impact of oxidative stress on the turnover capacity of the adult heart, both in physiological processes and diseased states. We are particularly interested in in vitro and in vivo studies, in any organism, on any of the following topics:

- net heart turnover
- multipotent resident progenitors
- de-differentiation of mature cardiomyocytes
- cell metabolism and epigenetic regulation
- cellular stress and redox signaling

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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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