



## Oxidative Stress and Antioxidants in Neurodegenerative Disorders

Guest Editor:

**Prof. Dr. Marco Tulio Núñez**  
Faculty of Sciences, Universidad  
de Chile, Santiago, Chile

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### Message from the Guest Editor

It is possible that neuronal death is mediated by ferroptosis, an iron-dependent cell death pathway that differs from other cell death processes at the morphological, biochemical, and genetic levels. Ferroptosis is characterized by iron-mediated lipid peroxidation, depletion of the endogenous antioxidant glutathione, and altered mitochondrial morphology.

Indeed, iron overload has been reported to associate with alterations of mitochondrial function as well as increased oxidative stress, autophagic defects, and diminished lysosomal function. Similarly, a link between iron, the endolysosomal system, and neuroinflammation was recently proposed.

The understanding of how these different players—iron accumulation, ferroptosis, mitochondrial dysfunction, inflammation, and lysosomes—interact to generate neurodegeneration is an open field of huge importance. Equally relevant is the design of therapeutic strategies based on multifunctional drugs that target all these players.





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## Editor-in-Chief

### Prof. Dr. Alessandra Napolitano

Department of Chemical  
Sciences, University of Naples  
"Federico II", Via Cintia 4, I-80126  
Naples, Italy

## 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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*Antioxidants* Editorial Office  
MDPI, Grosspeteranlage 5  
4052 Basel, Switzerland

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