

Special Issue on **Oxidative Stress and Neurological Disorders in Children**

CALL FOR PAPERS

The brain is particularly sensitive to oxidative stress. Under normal physiological conditions, low concentrations of reactive oxygen species are produced as a product of mitochondrial electron transport. If an imbalance between free radical production and antioxidant defenses occurs, oxidative stress arises leading to detrimental effects such as membrane damage, changes in the inner structure and function of proteins, lipid denaturation, and structural damage to DNA. Therefore, oxidant-mediated neuronal damage plays a role in the pathophysiological process of several neurological disorders. During the neonatal period, free radicals can easily overwhelm the antioxidant capacity of immature neurons, resulting in subsequent inflammation and cell death. Studies related to childhood psychiatric diseases have found changes in the oxidant and antioxidant parameters, which are thought to contribute to the development of these pathologies.

The purpose of this special issue is to publish high-quality clinical, basic, and translational original research and review articles that might contribute to a better understanding of the link between oxidative stress and neurological disorders in newborns and children.

Potential topics include but are not limited to the following:

- ▶ Neonatal brain injury: perinatal hypoxia-ischemia, preterm birth, and neonatal stroke
- ▶ Developmental disorders: autism spectrum disorder, attention deficit hyperactivity disorder, and tuberous sclerosis complex
- ▶ New cellular and molecular mechanisms in which oxidative stress is involved and potentially can be targeted for interventions
- ▶ Recent development of oxidative stress biomarkers in the fetus and newborn
- ▶ New therapies with a potential to reduce oxidative stress leading to neuroprotection and/or neuroregeneration

Authors can submit their manuscripts through the Manuscript Tracking System at <http://mts.hindawi.com/submit/journals/omcl/oxisn/>.

Lead Guest Editor

Daniel Alonso-Alconada, University of the Basque Country, Leioa, Spain
d.alconada@ucl.ac.uk

Guest Editors

Walter Balduini, University of Urbino "Carlo Bo", Urbino, Italy
walter.balduini@uniurb.it

Paolo Curatolo, "Tor Vergata" University Hospital, Rome, Italy
curatolo@uniroma2.it

Nicola J. Robertson, University College London, London, UK
n.robertson@ucl.ac.uk

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