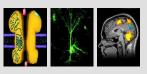


## IGSN/RDN CONFERENCE



## CHARACTERIZATION AND RESCUE OF NEURONAL DYSFUNCTION

April 25<sup>th</sup> - 26<sup>th</sup>, 2023

Session 1 Interfaces of neurological and immunological diseases

## JÖRN STEINERT

Faculty of Medicine and Health Sciences, University of Nottingham, UK

## Neuroinflammation and redox signalling as treatment targets in neurodegeneration

Numerous neurodegenerative diseases associated with protein misfolding (e.g. Alzheimer's and Parkinson's disease) exhibit enhanced oxidative and nitrergic stress conditions following initiation of neuroinflammatory pathways. The underlying activation of microglia within the central nervous system is responsible for the release of pro-inflammatory signaling molecules and the accompanying generation of reactive oxygen species and nitric oxide (NO) which provide a potent contribution to cytotoxic redox signaling. NO and its related nitrergic species in particular, are responsible for inducing post-translational protein modifications, like S-nitrosylation and 3-Nitrotyrosination, both of which may render cellular proteins dysfunctional.

Not only the targeting redox-active biometals, such as copper and zinc, has been suggested as a potential therapeutic strategy in various neurodegenerative conditions but also the suppression of neuroinflammation and boosting of antioxidant systems may provide therapeutic potential. In my presentation, I will review the current understanding of a redox dyshomeostasis in neurodegeneration and the associated targets for treatment.



