Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Stress, Chronic Inflammation, and Amyloidoses

CALL FOR PAPERS

The inflammatory response, elicited by body tissues as a result of harmful stimuli including oxidative stress, is a set of sequential events attempting to promote healing and shield viable tissue from additional injuries. It results from the action of host biomolecules acting as mediators of inflammation including Damage-Associated Molecular Patterns—DAMPs—and Pathogen-Associated Molecular Patterns—PAMPs—and brings forth the production of proinflammatory cytokines (PCs). In spite of the acute beneficial effects of these PCs, uncontrolled inflammation, reactive oxygen species (ROS), and ROS-induced injuries are central to almost every major disease, including cancer, diabetes, neurodegenerative conditions, and amyloidosis. The latter is a pathologic process in which structural transitions of normally soluble proteins into polymeric aggregates generate poorly soluble fibrillar extracellular tissue deposits with higher incidence in the elderly. Aggregation of antiparallel -pleated amyloid fibrils is observed in the parenchyma and very often in blood vessels of different organs, causing compression, ischemia, organ dysfunction, and cell ultimately death with increasing geriatric age. Originally grouped into primary, secondary, and familial types based on the clinical characteristics, a more modern protein-based classification of amyloid diseases has contributed to a better understanding of the disease pathogenesis. At the present time 30 different proteins and more than 100 genetic variants are known to be associated with systemic and localized forms of the disease in humans. The most frequently diagnosed form of systemic amyloidosis—about 60% of the cases—is associated with the deposition of Immunoglobulin Light chains, generally as a complication of monoclonal gammopathies. Among cerebral amyloidosis, parenchymal and vascular deposition of the amyloid- (A) protein in patients with Alzheimer's disease constitutes the most common form in aging dogs and humans. In recent decades, chronic inflammation and oxidative stress have emerged as crucial players in the disease pathogenesis of many—if not all—of these forms of amyloidosis irrespective of the tissue localization and biochemical composition of the deposits, emerging as an attractive translational target for novel therapeutic interventions.

This special issue is intended to provide an updated view on the role of proinflammatory cytokines and inflammation induced by oxidative stress and its related cellular pathways in the etiopathogenesis and treatment strategies of amyloidosis.

Potential topics include but are not limited to the following:

- Influence of PCs on the secretion and fibrillogenic processing of protein substrates and generation of ROS in amyloidoses
- Identification of the proinflammatory mechanisms influencing amyloidogenesis
- Description of new cellular and animal models aiding in the understanding of the role of inflammation-related mechanisms in different types of amyloid diseases associated with ROS-mediated cytotoxicity
- Neuroinflammation as an emerging factor modulating neurodegenerative diseases associated with amyloid deposition and oxidative stress
- Modalities, future therapeutic strategies, drugs, and prospects for the removal of amyloid deposits to limit harmful effects of ROS

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

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