Mediators of Inflammation

Hindawi

Special Issue on

DNA Repair Enzymes, Oxidative Stress, and Inflammatory Processes

CALL FOR PAPERS

Inflammation is a determining factor for a large number of acute and chronic diseases including cancer, atherosclerosis, asthma, diabetes, COPD, hepatitis, and many others. One of the major components of the inflammatory responses, either as a driving force or as a consequence of the persistence of inflammation, is oxidative stress. It is well established that oxidative stress results in tissue and cell injury by targeting major cellular macromolecules including proteins, lipids, and nucleic acids. DNA damage elicits the response of many DNA repair enzymes that attempt to repair the lesions to ensure cell homeostasis or induce death of cells with extensive DNA damage. Accordingly, DNA repair enzymes have always been regarded as reacting proteins rather than driving factors of inflammation. Accruing evidence from a number of laboratories demonstrates the involvement of these enzymes in inflammation as determining factors of the pathogenesis of the diseases. Many of these enzymes have been shown to regulate inflammation-related cellular processes and signaling pathways. The cumulative effects of persistent inflammation lead to major organ pathologies and/or accumulation of mutations that increase probability of malignant transformation.

The purpose of this special issue is to provide a platform on which a better understanding and appreciation of the roles of DNA repair factors in inflammation is achieved and clarification of the underlying mechanisms by which these factors participate in the processes of chronic inflammation-mediated diseases. We are thus inviting the submission of manuscripts describing original research as well as reviews on the emerging roles of DNA repair in chronic inflammation.

Potential topics include, but are not limited to:

- New developments on the role of oxidative DNA damage in chronic inflammation
- Role of DNA repair proteins such as PARP-1, DNA-PK, ATM, and others in regulating transcription of inflammatory genes
- Regulation of transcription factors and associated signal transduction by DNA repair proteins. The transcription factors of interest include, but not limited to, NF-κB, p53, STATs, and AP1
- The relationship between lipid peroxidation, lipid mediators of inflammation, and oxidative DNA damage
- Are antioxidant enzymes actually anti-inflammatory? If there are over 100 different inflammatory mediators, why are there not any real anti-inflammatory enzymes?
- Clearance of inflammatory cells including efferocytosis and the role of DNA repair in the process
- The roles of DNA repair enzymes in switching cell death from apoptosis to necrosis and how this switch participates to chronic inflammation (e.g., asthma severity)
- The relationship between chromatin modulators and expression of inflammatory genes
- Noncoding RNAs involved in the modulation of DNA repair proteins in response to oxidative stress

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/dnare/.

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