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Oxidative Stress, Lipid Peroxidation and Glycoxidation Products in Inflammation, Autoimmunity and Immunity-Driven Inflammation

Guest Editor:

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Message from the Guest Editor

An expanding body of evidence highlights the role of the products of lipid peroxidation (LPO) in the pathogenesis of autoimmunity and immunity-driven inflammation. They represent oxidation-specific epitopes, which act both as: 1) damage-associated molecular patterns, inciting flogosis and activating antigen-presenting cells by binding to pattern-recognition receptors; and 2) neoepitopes derived from the modification of self epitopes, which can be instrumental in breaking the tolerance of autoreactive T and B cells to self antigens. In fact, it has been repeatedly observed in both animal models and humans that the modification of self epitopes by LPO products not only prompts valid responses to modified epitopes, but also promotes their intramolecular spreading to unmodified epitopes of formerly tolerated self antigens. Moreover, the pleiotropic ability of LPO products to form adducts with a broad range of macromolecules may account for the observed intermolecular spreading of autoimmune responses between different protein and nucleic acid antigens. This Special Issue of Antioxidants aims to provide a rigorous update of this intriguing subject.













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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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