Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Stress in Infection and Consequent Disease

CALL FOR PAPERS

State of the field: viral, bacterial, and parasitic infections comprise a vast group of etiological agents that cause acute or chronic diseases. According to WHO, they represent one of the major causes of human morbidity and mortality. AIDS, lower respiratory tract infections, and diarrheal diseases underlie up to 5 million deaths each year, especially in the middle- and low-income countries. Some of the infections causing chronic disease often lead to the development of an array of deadly pathologies including cancer, autoimmune diseases, diabetes mellitus, and malfunctions of various organs.

During the last two decades, it has been clearly established that many of these infections trigger the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS). It was repeatedly demonstrated for the infections caused by the blood-borne hepatitis viruses (B, C, and D), human immunodeficiency virus (HIV), influenza A virus (HAV), Epstein-Barr virus (EBV), respiratory syncytial virus (RSV), and other viruses. For acute respiratory viral infections, ROS/RNS have been implicated in lung tissue injury and epithelial barrier dysfunction which in turn increased the susceptibility to secondary infections. In case of chronic viral hepatitis, oxidative stress was shown to promote liver fibrosis, cirrhosis, and cancer, as well as metabolic dysfunction. HIV-induced oxidative stress was shown to contribute to neurodegenerative complications which are often observed in AIDS patients. Last but not the least, a virus-induced oxidative burst has been recently associated with the development of the acute childhood lymphoblastic leukemia. In bacterial infections oxidative stress arises, at least in part, from altered metabolic pathways and has also been implicated in organ damage and the development of malignancies. Helicobacter pylori, for example, induces ROS-generating enzymes such as spermine oxidase and upregulates proinflammatory and procancerogenic redox-regulated genes like cyclooxygenase 2.

Our aim is to bring together novel research and in-sight views on the role of oxidative stress in infection and consequent disease and on the translation of the processes occurring in oxidative stress and stress response into cellular longevity.

Potential topics include, but are not limited to:

- Sources and triggers of ROS/RNS in the infected cell
- ROS/RNS and the microbe and redox modifications of the microbial antigens
- Effects of ROS/RNS induced in infection on the host
- Pathogen and host redox system and the impact of the pathogen on the functionality of the cellular antioxidant systems and activity of ROS/RNS-protective enzymes
- ROS/RNS and pathologies
- ROS/RNS and chemotherapy as very little is known about the effect of antimicrobial treatment(s) on the ROS/RNS generation pathways
- ROS/RNS as the organic part of the host immune defense against the microbes
- Other areas of redox biology which may path the way to further investigations of the ROS/RNS interplay in viral, bacterial, and parasitic diseases

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