



Special Issue Reprint

Ionizing Radiation, Antioxidant Response and Oxidative Damage: Radiomodulators

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Ionizing radiation (IR) exposure can be deleterious for living tissues and eventually lead to illness or even death. DNA breakdown and the overproduction of highly reactive free radicals, reactive oxygen species (ROS) and reactive nitrogen species (RNS) are considered initiators of IR-induced molecular and cellular damages, whereas NO, TGF-**B** and other proinflammatory cytokines are the primary effectors involved in radiation bystander effects (RIBE). Radiomodulators can reduce (radioprotectors and radiomitigators) or increase (radiosensitizers) IR damage. Past world events have highlighted the urgent need to develop predictive biomarkers of the IR absorbed dose and radiation countermeasures to reduce IR damage. Despite the strong economic and scientific efforts over the last decades, at present, drugs for effective protection against lethal IRs remain an unmet need. Moreover, the development of radiosensitizers that selectively increase IR damage in cancer cells but protecting, or at least do not affect, healthy tissues is also of unquestionable importance to improving patient survival and quality of life. It includes contributions that will help to understand the mechanisms involved in radiation-derived cellular responses and damage, the importance of free radical scavengers and antioxidant cellular defenses in preventing harm, and the relevance of the antiinflammatory response to improve recovery. In accordance, the mechanism of action and effectiveness of relevant radiomodulators are described and discussed, and novel clinical models and IR biomarker technologies are presented.



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