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Oxidative Stress Induced DNA Damage in Cancer Treatment

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Message from the Guest Editors

Anticancer therapies involving the generation of oxidative stress have gained considerable attention in recent years. In cancer cells, current therapies target this effect to induce cellular death and stop cancer growth. However, in healthy cells, different outcomes can be obtained. In general, repair mechanisms will be activated and the damage will be reduced; otherwise, apoptosis will be initiated, leading to cellular death. In some cases, DNA can be excessively damaged, and this can lead to the formation of cancerous cells or cellular damage. When ROS cannot be neutralised after a period of accumulation, this results in DNA damage and can contribute to tumour proliferation, survival, chronic inflammation, angiogenesis, and different organ dysfunctions.

Contributions to this Special Issue may cover all research aspects related to pathways involving ROS-induced DNA damage during chemotherapy or radiation therapy. We aim to specifically discuss cellular mechanisms that are involved in anticancer-therapy-induced DNA damage via oxidative stress pathways in both healthy and cancer cells.

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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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