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DNA Damage and Cancer Metabolism: Basic Research to Clinical Translation

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Message from the Guest Editors

This Special Issue is devoted to elaborate the potential molecular mechanisms and translational research connecting DNA damage and cancer metabolism. DNA is vulnerable to damage resulting from various endogenous and exogenous phenomena including endogenous metabolites. Similarly, high levels of ROS stimulate glutathione synthesis to alleviate oxidative stress. Furthermore, various DNA repair pathways require specific metabolic co-substrates to maintain genomic stability. For example, Poly (ADP-ribose) polymerase 1 (PARP1) is an important mediator of DNA repair, and utilizes NAD^+ as a co-substrate. Moreover, indirect action of metabolites in DDR via crosstalk with epigenetic regulation is also widely reported, contributing to the DNA damage repair efficiency or repair method choice. Clinically, the treatment effects of cancer therapeutics such as chemotherapy, radiotherapy, and immunotherapy are highly related to DNA damage and metabolism microenvironment.

This Special Issue welcomes reviews and original research as well as methodologies aiming toward formulating the fundamentals and the clinically relevant translational perspectives about DNA damage and cancer metabolism.

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Message from the Editor-in-Chief

The metabolome is the result of the combined effects of genetic and environmental influences on metabolic processes. Metabolomic studies can provide a global view of metabolism and thereby improve our understanding of the underlying biology. Advances in metabolomic technologies have shown utility for elucidating mechanisms which underlie fundamental biological processes including disease pathology. *Metabolites* is proud to be part of the development of metabolomics and we look forward to working with many of you to publish high quality metabolomic studies.

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