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DNA Damage and Repair in Cancer

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Message from the Guest Editors

Genomic DNA is threaten by DNA-damaging agents. For genomic stability, mammalian cells have DNA damage-sensor mechanisms that induce; apoptosis to eliminate damaged cells; transcriptional response; DNA damage tolerance; activation of DNA damage checkpoints and modulation of cell cycle progression; and/or initiation of DNA repair. To maintain genomic stability, cells detect and repair DNA damage by multiple pathways.

Failure of response to DNA damage can lead to the accumulation of mutagenic lesions associated with ageing, neurological disorders, and cancer. Furthermore, germline mutations in DNA repair genes lead to cancer predisposing syndromes.

Pharmacological targeting of DNA repair can increase the efficacy of treatments and overcome therapeutic resistance. Synthetic lethality, the loss of function of either one of two inter-related genes is not lethal, but loss of both genes results in cell death, provides an exciting new platform for pharmacological targeting of DNA repair.

In this Special Issue, we focus on recent advances in molecular biology of DNA repair, as well as its relevance as a key prognostic, predictive, and therapeutic targets in cancer.

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Message from the Editor-in-Chief

Genes are central to our understanding of biology, and modern advances such as genomics and genome editing have maintained genetics as a vibrant, diverse and fastmoving field. There is a need for good quality, open access journals in this area, and the *Genes* team aims to provide expert manuscript handling, serious peer review, and rapid publication across the whole discipline of genetics. Starting in 2010, the journal is now well established and recognised.

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