Journal of Oncology

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Special Issue on

Pharmacological Targeting DNA Repair and Replication, PARP Inhibitors and Beyond

CALL FOR PAPERS

DNA double-strand breaks (DSBs) could be generated from endogenous and exogenous sources, such as replication stress, ROS (reactive oxygen species), ionizing radiation (IR) and genotoxic agents. DNA DSBs are particularly deleterious and could cause cell death. Chemo-radiotherapy kills cancer cells through induction of unrepairable DNA DSBs and is widely used in clinical practice. Thus, DNA DSBs repair capacity is highly associated with cancer therapeutic resistance and clinical outcome, which suggests that modulating DNA repair mechanisms, such as inhibiting DNA repair machinery and DNA damage response, could reverse cancer drug resistance and sensitize the therapeutic efficacy.

Considering the importance of DNA repair in cancer treatment, poly (ADP-ribose) polymerase (PARP) inhibitors have been developed and approved in BRCA1/2 mutant breast, ovarian, pancreatic and prostate cancers. PARP inhibitors are currently the first-line maintenance therapy for patients with advanced ovarian cancer. PARPs are proteins that are involved in single-stranded break (SSB) repair and inhibiting PARPs could lead to DNA DSBs, which are highly dependent on the homologous recombination (HR) mediated pathway to repair. Therefore, PARP inhibitors specifically kill HR-deficient cancer cells, making HR-repair a cancer-specific therapeutic target that induces synthetic lethality to PARP inhibitor treatment. And the success of PARP inhibitor in cancer therapy has made DNA repair and replication mechanisms are hot drug targets for cancer therapeutics.

This Special Issue focuses on publishing original research and review articles focusing on novel DNA repair mechanisms that could be targeted in cancer.

Potential topics include but are not limited to the following:

- Novel DNA repair mechanisms that could be targeted in cancers, including but not limited to HR, NHEJ, BER, NER, MMR
- Novel mechanisms or strategies to exploit replication stress and DNA damage for cancer therapy
- Development and utilization of inhibitors targeting DNA repair and replication machinery in cancer
- Novel mechanisms that contribute PAPR inhibitor resistance and novel strategies that could optimize PARP inhibitor treatment in cancer
- Development of DNA repair and replication-related gene signatures that could predict the prognosis and progression of cancer
- Novel DNA repair and replication mechanisms that could contribute to remodeling the tumor microenvironment
- Novel DNA damage and repair biomarkers for immunotherapy response
- Identification of noncoding RNAs targeting DNA repair and replication machinery, and exploration of their functions and mechanisms

Authors can submit their manuscripts through the Manuscript Tracking System at https://review.hindawi.com/submit?specialIssue=080161.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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