

Preface

The genome integrity requires rigorous maintenance to ensure normal operation of all cellular activities and long-term survival of individuals. Nonetheless, human cells are constantly exposed to genotoxic assaults from both endogenous and exogenous sources which lead to various types of DNA lesions. Among all forms of DNA damage, DNA double-stranded breaks (DSBs) are the most dangerous as unrepaired DSBs results in apoptotic cell death or senescence whereas improper proceedings of DSBs causes chromosomal aberrations, genome instability, and loss of heterozygosity. The significance of DSB repair can be further illustrated by the increased cancer frequency as well as accelerated aging characters in mice and humans carrying germline mutations in DSB repair genes. For example, mutations in BRCA genes promote breast cancer due to ineffective DSB repair through homologous recombination pathway. On the other hand, induction of DSBs is also the common mechanism in cancer treatments including ionizing radiation and many chemotherapeutic agents. It became even more obvious in recent years that many DNA repair enzymes have become therapeutic targets for cancer treatment. This is taking account that cancer cells often carry imperfect DNA repair and addict to the remains of DNA repair mechanism to thrive. Intervention of DNA repair alone or in conjunction with conventional treatment strategy would result in synthetic lethality which has proved to be effective in eliminating cancer cells without significant cytotoxicity to normal tissues.

In this special issue of *Translational Cancer Research*, we have a collection of review articles from renowned experts in DNA repair and cancer biology to examine current understanding of DSB repair mechanisms and their clinic implication. It is conceivable that our in-depth knowledge is essential to translate into clinical benefits in cancer treatment.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer Review: This article was commissioned by the editorial office, *Translational Cancer Research* for the series “DNA Damage and Repair”. The article did not undergo external peer review.

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at <http://dx.doi.org/10.3978/j.issn.2218-676X.2013.06.01>). The series “DNA Damage and Repair” was commissioned by the editorial office without any funding or sponsorship. BPC and DJC served as the unpaid Guest Editors of the series and serves as the unpaid editorial board members of *Translational Cancer Research*. The authors have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Submitted: 20 June 2013; Accepted for publication: 25 June 2013
doi: 10.3978/j.issn.2218-676X.2013.06.01

