

Transcriptional response of DNA repair genes to DNA damage in cancer cells

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During the last decade, there has been significant progress in our understanding of the mechanism to delay cell cycle progression and to progress to cell death in response to DNA damage. Direct and indirect connections between DNA damage checkpoints and DNA repair have also been revealed as some components of the checkpoints are directly involved in various DNA repair pathways and some DNA repair proteins are indeed terminal substrates for activated DNA damage checkpoint pathways. However, the transcriptional response to DNA damage is less well understood because this response appears more complicated and less conserved among different organisms. In particular, the checkpoint response mediating transcriptional induction of DNA repair genes is not well characterized, with many more details left to be discovered. We observed that checkpoint kinases are directly involved in the transcriptional induction of DNA repair genes in human cancer cells in response to DNA damage. Several transcription factors (e.g., E2F1) were found to mediate transcriptional induction of a group of DNA repair genes after the activation of DNA checkpoint pathways in cancer cells. Increasing knowledge of the DNA damage response (DDR) as a result of our proposed studies will not only enhance our understanding of the DDR functions but will certainly present exciting opportunities for better understanding and managing human cancer.

Biography

Dr. Daekyu Sun completed his Ph.D. and postdoctoral training at the College of Pharmacy at the University of Texas at Austin. He was employed with the Cancer Therapy Research Center (CTRC) at San Antonio, Texas from 1995 to 2002 as a Research Scientist and Assistant Member. He has been employed as an Assistant Professor at the University of Arizona at Tucson, Arizona since 2008. He has published more than 64 papers and has served as a reviewer for several reputed journals, such as the Journal of Nucleic Acids and Journal of Medicinal Chemistry.

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