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Replication stress response defect and cancer stemness

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Replication stress response (RSR) is a subset of DNA damage response that safeguards the replication process. It has been known that in normal cells, hyperproliferative activity caused by oncogene activation leads to replication stress and to the activation of RSR. The activation of RSR consequently triggers cellular senescence or apoptosis to prevent cell transformation. Recently, my lab has developed a unique cell model to study how normal cells respond when they are simultaneously challenged with oncogenic stress and the loss of RSR. Intriguingly, it was found that these RSR defective cells exhibited many characteristics of cancer stem cells, including possessing cancer stem cell markers and EMT (epithelial-mesenchymal transition). Present work therefore functionally links RSR defect with cancer stemness during the process of cellular transformation.

Biography

Shiaw-Yih Lin's research efforts are focused within an overall theme of DNA damage response defects in cancer etiology, cancer prevention, cancer detection and targeted therapy. He has worked in these fields for more than 10 years, and has published more than 50 papers. He serves as an editor of International Journal of Cancer Therapy and Oncology and on the editorial boards for *World Journal of Clinical Oncology*, *American Journal of Cancer Research, Frontiers in Molecular and Cellular Oncology, ISRN Cell Biology, World Journal of Translational Medicine, Experimental Hematology & Oncology,* and *Annals of Translational Medicine*. He has served on numerous review committees for NIH, DOD and other funding organizations both nationally and internationally.

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