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Suppression of TPP1 resulted in telomere dysfunction and enhanced radiosensitivity in cancer cells regardless of telomerase status

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Radiotherapy is one of the major therapeutic strategies in can¬cer treatment and identifying new factors that predict radioresistance could be of great value in the treatment of cancers. Telomere homeostasis is now emerging as an effective and important factor in modulating cellular sensitivity to ionizing radiation. The telomere-binding protein TPP1, an important component of the shelterin complex at mammalian telomeres, is an important regulator of telomere homeostasis. In this study, we investigated the role of TPP1 expression in regulating cellular radiosensitivity and telomere homeostasis in both telomerase positive (HCT116) and alternative lengthening of telomere (ALT) cell lines (U2OS). We found that TPP1 deletion lead to a significant increase of radiosensitivity to X-rays in both telomerase positive (HCT116) and alternative lengthening of telomere (ALT) cell lines (U2OS). TPP1 mediated radiosensitization was correlated with increased telomere dysfunction and apoptosis rate after IR exposure. Moreover, TPP1 deletion slowed down the repair kinetics of total DNA damage and telomere dysfunction induced by ionizing radiation. Together, our study demonstrated that TPP1 plays a vital role in telomere maintenance and cellular response to ionizing radiation and may be a potential target in the radiotherapy of cancer regardless of telomerase status.

Biography

Lei Yang has completed his PhD/MD in 2014 from Wuhan University, China. He has worked in the Department of Radiation Oncology & Medical Oncology at Zhongnan Hospital, Wuhan University for 1 year. His main research field is the role of telomere and telomerase in anti-cancer therapy.

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