Meetings International SciTechnol

J Clin Exp Oncol 2018, Volume: 7
DOI: 10.4172/2324-9110-C3-015

Redox signaling in cell response to radiotherapy

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Redox signaling plays important roles in both normal physiological development and abnormal pathological progression. Malignant tumor cells generally produce the high levels of ROS due to the high rates of metabolism, as well as anticancer treatment like radiotherapy specially generates massive ROS. Virtually, ROS stress-evoked up-regulation of antioxidant enzymes through NF-κB and Nrf2 protective pathways, leading to therapeutic resistance and tumor relapse. Several therapeutic approaches were tested to enhance radiotherapeutic efficiency for advanced prostate cancer by targeting the RelB-activated NF-κB alternative pathway as well as the Keap1-Nrf2 pathway. In particular, natural compounds such as parthenolide and vitamin C selectively sensitize cancer cells to radiation by inhibition of cellular antioxidant defense system, but protect the normal cells from radiotoxicity through adaptive activation of cellular antioxidant defense system. The selectivity of radiotherapeutic effects on cancer cell and normal cell survivals are mediated by differentially modulating cellular redox homeostasis. These results demonstrated that ROS mediates the disparate effects for both cell survival and cell death, suggesting that the regulation of ROS function determines the cell fate.

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