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Abstract 1429: Ubiquitin specific peptidase 37 facilitate prostate cancer oncogenesis by regulation replication stress FREE

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Prostate cancer (PC) is one of the second most common cancer diagnosed and the second most common cause of cancer death in men worldwide. Currently combinations of tumor surveillance, surgery, radiation, hormonal, and chemotherapies are used in the management and treatment of prostate cancer. The molecular basis of disease is complex and mutations in many different oncogenes and tumor suppressor genes have been found including TP53, RB1, EZH2, BMI, and PTEN. The oncogenesis of prostate cancer appears to be caused by dysregulation of many different cellular signalling pathways especially those involved in cell survival and apoptosis. Protein deubiquitination controls many intracellular processes, including cell cycle progression, transcriptional activation, and signal transduction. Ubiquitin specific peptidases (USPs) remove ubiquitin tags from target proteins to alter protein configuration and function. Recent studies have revealed that Ubiquitin specific peptidase 37 (UPS37) regulates replication stress by regulating important protein involved in several important cellular functions. Analysis of TCGA data indicated that overexpression of USP37 correlated with reduced progression free survival (PFS) in prostate cancer patients. Mass spectrometery analysis of Prostate cancer cells (DU145) indicated that distinct set of genes were altered on overexpression or knockdown of USP37. Our data indicate that USP37 overexpression confers survival advantage while its depletion enhances sensitivity for cell killing in PC cells. USP37 overexpressing cells were able to resolve DNA damage foci much more rapidly than the control cells or cells in which USP37 was depleted in response to genotoxic stress. USP37 depletion results in reduced resolution of γ H2AX and 53BP1 DNA damage foci which indicates the reduced ability of cells to carry out constitutive DNA replication. USP37 was found to interact with different replication factors as also seen in our mass spectrum analysis. We further correlated our data with archived tissue blocks of PC

mechanistic details for the regulation of replication stress by USP37 which merits the development of targeting strategies to explore its therapeutic potential in PC by inducing synthetic lethality.

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