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Extracellular vesicle-mediated regulation of the malignant phenotype in prostate cancer

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Castrate resistant prostate cancer (CRPC) is the second leading cause of cancer-related death in men in the developed world. While androgen deprivation therapy is effective at the onset of treatment, nearly all men develop castrate resistance. Several new therapies, including taxane-based chemotherapy, have improved outcomes for CRPC. New therapeutic regimens and rational targets are needed in order to continue to improve prostate cancer (PCa) patient survival. The role of extracellular vesicles (EVs) in reversing the malignant phenotype and taxane resistance in PCa was studied. Gleason grade 8 EVs significantly induced anchorage independent growth and the malignant phenotype of non-malignant prostate cells. It was identified increased protein levels of putative mediators of the EV-induced changes including 14-3-3 zeta, pRKIP and prohibitin. (log p value <0.05 for all 3 proteins as determined by the Comparison Analysis Tool in the IPA software) in non-malignant cells co-cultured with PCa patient EVs. Knockdown of 14-3-3 zeta lead to partial reversal of soft agar growth, indicating that it is a potential candidate for targeted therapy. Co-culture of malignant PCa cells with non-malignant prostate EVs or EVs isolated from human mesechymal stem cells (hMSC EVs) significantly inhibited tumor xenograft growth and reversed resistance to paclitaxel. The study provides a rational basis for the use of non-cancer cell-derived or hMSC EVs to potentially inhibit CRPC progression and reverse taxane resistance.

Biography

Devasis Chatterjee is currently a faculty member at Rhode Island Hospital and the Alpert Medical School of Brown University, Providence RI, in the Division of Hematology/Oncology. He obtained his PhD in Biochemical Pharmacology/ Molecular Biology and Biochemistry from Brown University. He was a postdoctoral fellow at the Dana Farber Cancer Institute, Boston, MA. His laboratory focuses on experimental therapeutics and signal transduction pathways research. His lab was the first to characterize the apoptosis-inducing and tumor suppressing functions of Raf Kinase Inhibitor Protein (RKIP) in gastric, prostate and breast cancer. His research has been funded by grants from the DOD, NIH and pharmaceutical companies. His lab is currently examining phenotype shifting mediated by extracellular vesicles (EVs) in prostate cancer and the use of EVs as a therapeutic agent.

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