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Prostate stem cells and cadmium in the development of BPH to PCa: Revealing the secret

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Aberrant proliferation of prostate stem cells lead to Benign prostate hyperplasia (BPH) and prostate cancer (PCa) which are common prostatic disorders affecting elderly men. Hormonal imbalance, disruption of cell proliferation, apoptosis, chronic inflammation, aging and environmental pollutants are prominent factors contributing to the pathophysiology of the diseases. Environmental pollutants such as cadmium (Cd) with an androgen mimicking activity induce hormonal imbalance and hence are potential carcinogens. Proliferative and carcinogenic effects of Cd on the prostate have been shown earlier *in vitro* and *in vivo*. Cd can induce malignant transformation of non-tumorigenic human prostate epithelial (HPrE) cells into tumorigenic cells *in vitro*. Whether the same effect can be seen in BPH cells leading to the conversion of prostate cancer is not yet known. Hence, this study focused on the dose dependant role of cadmium in BPH to cancer progression. Another key target for tumorogenesis in human prostate is stem cells. Therefore, it is vital to understand the role of cadmium in stem cells in prostate carcinogenesis. As a prerequisite, we isolated stem cell population from BPH patients which expressed basal and epithelial cell markers and formed teratomas when transplanted into balb/c mice along with three germ layers formation. Further evaluation of cytotoxic concentration, morphological assessment and cell cycle analysis confirmed the dose dependant role of cadmium in disease conversion. Current treatment strategy involves removal of hyperplasic region by surgical intervention which is only short term relief from BPH and may revert back in due course of ageing. Thus understanding the etiology of stem cells and intervention of environmental pollutants will help to better control the disease.

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Natural isothiocyanates may aid in metastasis control by down-regulation of HIF-1α in breast cancer cells

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Secondary breast cancer occurs when cancer cells spread from the primary tumor to some distant parts through the lymphatic or blood system. Hypoxia-inducible factor 1α (HIF-1α) plays a crucial role in facilitating tumor progression and metastasis. Hence reducing the levels of HIF-1α might be an important anticancer strategy. It is therefore important to understand the key cellular events involved in HIF-1α activation. Exploration of natural compounds having little or no toxicity in this regard is very important. Phenethyl isothiocyanate (PEITC), a natural isothiocyanate, found in cruciferous vegetables is stud with anticancer properties. Effect of PEITC on the expression of HIF-1α and HSP90 in breast adenocarcinoma cell lines (MCF-7 and MDA-MB-231) under both normoxia and hypoxia had been explored. ROS plays an important role in up-regulation of HIF-1α and HSP90. PEITC induced nuclear accumulation of Nrf2, increased the activities of several antioxidant enzymes and thus reduced the ROS burden of the tumor cells by acting as an indirect antioxidant. This resulted in the down-regulation of HSP90 and thereby HIF-1α expression. HSP90 was also found to be involved in the regulation of HIF-1α. A probable link between down-regulation of HIF-1α with reduction of ROS by PEITC through induction of Nrf2 was determined. PEITC, by virtue of its modulatory effect on HIF-1α could aid in control of metastasis by altering two important metastatic proteins MMP 2 & 9 and VEGF. These series of events culminated in retardation of adhesion, aggregation, migration and invasion of the breast cancer cells.

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