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Role of Igf Signaling in Prostate Cancer Cell Line (Pc-3)

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Human prostate carcinoma is one of the most common cancers among men and second leading cause of cancer related death. Prostate cancer cell growth is controlled by several factors including androgen, growth factors and their respective receptors. Growth factors and their receptors are over expressed in advanced prostate cancer including epidermal growth factor (EGF), transforming growth factors (TGF) α and β , fibroblast growth factors (FGF), and insulin like growth factors (IGFs). IGFs are potent mitogenic factors for a variety of cancer including prostate cancer which stimulates cell growth and inhibits apoptosis. The importance of the IGF system in prostate cancer cell growth is underscored by the detection of every molecules of this system, including IGF-I, IGF-II, IGF-IR and as well as IGF binding proteins in normal, hyperplastic and or neoplastic cells and tissues. IGFBP3 binds to IGF-I or IGF-II and blunts their proliferative effects on cells. IGF-I increases proliferation of prostate cancer cells whereas antisense mediated inhibition of IGF-IR expression suppresses *in vivo* tumor growth and prevents prostate cancer cell invasiveness. Quercetin which possesses a wide spectrum of pharmacological properties is found in many fruits and vegetables. It inhibits the proliferation of cancer cells and induces apoptosis. Our previous studies proved that quercetin leads to cell cycle arrest and induces apoptosis in prostate cancer cells. We also studied the effect of quercetin on IGF system and its signaling molecules of prostate cancer. Zinc is an essential trace element present in mammalian prostate gland. Our recent study demonstrated that zinc treatment significantly reduces the cell viability of PC-3 cells. Zinc leads to cell cycle arrest and induces apoptosis by regulating IGF system and signaling molecules. It decreases the protein levels of IGF-IR, IRS-1, IRS-2 and increased the level of IGFBP-3. Thus, our study suggests that quercetin and zinc decreases the survival of androgen independent prostate cancer cells by modulating the expression of IGFs systems, signaling molecules and induces apoptosis which could be useful for the androgen independent prostate cancer treatment.