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Stromal androgen receptor in prostate cancer

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The androgen receptor (AR) in stromal cells significantly contributes to the development and growth of prostate during fetal and pubertal stages as well as during prostate tumorigenesis and progression. During prostate carcinogenesis and progression, the stromal cells begin to lose AR expression as early as in *in-situ* cancer of the prostate, high grade prostatic intraepithelial neoplasia (HGPIN). The extent of loss of stromal AR is directly proportional to the degree of differentiation and progression of prostate cancer. Co-culture studies suggested that stromal AR inhibits the growth of malignant epithelial cells, possibly through expression of certain paracrine factors in the presence of androgens. This functional reversal of stromal AR, from growth promotion at fetal prostate development stage to growth inhibition in cancer, explains to some extent the reason that loss of AR expression in stromal cells is important for prostate cancer progression during androgen ablation treatment. From a translational perspective, it creates the need to re-identify current treatment options and opens a new direction for therapeutic interventions, especially in advanced prostate cancer.

Biography

Peng Lee is Professor in the Department of Pathology and Urology at New York University School of Medicine and Director of Molecular Pathology at New York Harbor Healthcare System and co-Director of Genetic Program, Center of Excellence of New York University, Urological Disease. He obtained his MD degree from Peking University School of Medicine and PhD from the State University of New York, Downstate Medical Center. Lee was trained as a postdoctoral fellow with Dr. Robert Roeder at the Rockefeller University. Following his residency in Pathology at New York University School of Medicine, he completed a Surgical/Oncologic Pathology fellowship at the M. D. Anderson Cancer Center. He is specialized in surgical, oncologic, genitourinary and molecular pathology.

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