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## Testosterone, estrogen and insulin in the development of prostate cancer

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**Introduction:** Prostate cancer (PCa) is the second most frequently diagnosed cancer in men and the second leading cause of cancer death in the male. This phenomenon could be due to hormonal changes during aging. So in this conference, we will briefly summarize the biochemistry of androgens, estrogens and insulin in relation to prostate cancer development and advanced phenotypes.

**Background:** In the elderly, the androgen levels drop significantly, whereas their estrogen levels remain unchanged or increased, making the estrogen to androgen ratio elevated in the aging prostate. There is also a relationship between different serum testosterone levels and intraprostatic androgen receptor fluxes, signaling efficiency and downstream physiological responses. In addition, we would like to point out that the epidemiological studies indicate a correlation between PCa and circulating estrogen levels, among different ethnic/racial groups with diverse PCa incidence. But some of them emphasize that low testosterone levels may actually be a marker of more-aggressive prostate cancer. The estrogens actions are mediated by estrogen receptors (ERs), alpha ( $\alpha$ ) and beta ( $\beta$ ). The continuing controversy over the function of ER $\beta$ s in prostate carcinogenesis could be due to variable expression levels of different ER $\beta$  isoforms in benign versus malignant tissues during different stages of the process. On the other hand, the insulin and its insulin growing factors had an essential role in prostate cancer carcinogenesis and in prostate cancer aggressive phenotype. Furthermore, the overexpression of insulin receptors (IRs) in prostate cancer and the association between IR expression or IR/IGF1R activation has been found in both stromal and epithelial prostate tissue compartments but changes with cancer progression that becoming an autocrine signal to it.

**Conclusions:** Until now, elucidating mechanisms of paracrine/steroid hormone-induced changes in cellular differentiation, proliferation, and gene expression, and carcinogenesis give us confusing results that crosstalk between the hormonal milieus are essential for prostate cancer development.

**Outcome/Impact:** Outcome is based on the knowledge of the mechanisms involved in prostatic carcinogenesis.

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