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"Progesterone receptor expression in human prostate cancer:
Correlation with tumor progression."

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BACKGROUND: The recent discovery of the classical estrogen receptor alpha (ERalpha) in metastatic and recurrent prostatic adenocarcinoma suggests that estrogens are implicated in prostate cancer progression.

METHODS: To get more insight into estrogen signaling in prostate cancer tissue, the current study has examined the immunoprofile of the estrogen-inducible progesterone receptor (PR), and evaluated its relation to ERalpha gene expression.

RESULTS: In primary tumors, the PR was detectable in 36% of primary Gleason grade 3 (5 of 14 cases), 33% of primary Gleason grade 4 (5 of 15 cases), and in 58% of primary Gleason grade 5 tumors (7 of 12 cases). None of the 41 primary tumors investigated revealed significant PR expression in more than 50% of tumor cells. Conversely, moderate to strong receptor expression was observed in 60% of metastatic lesions (9 of 15 cases), and in 54% of androgen-insensitive tumors (38 of 71 cases). Irrespective of grades and stages, the presence of the PR was invariably associated with high steady state levels of ERalpha mRNA, whereas the ERalpha protein was undetectable by immunohistochemistry (IHC) in a significant number of cases (58 of 97 cases).

CONCLUSIONS: The progressive emergence of the PR during tumor progression obviously reflects the ability of metastatic and androgen-insensitive tumors to use estrogens through a ERalpha-mediated pathway. The present data provide a theoretical background for studying the efficiency of antiestrogens and antigestagens in the medical treatment of advanced prostate cancer. Copyright 2001 Wiley-Liss, Inc.

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Evaluation of androgen, estrogen (ER alpha and ER beta), and progesterone receptor expression in human prostate cancer by real-time quantitative reverse transcription-polymerase chain reaction assays.

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Steroid hormones can have profound effects on prostate tumor development making it important to define steroid receptor expression in prostate tissues.

For this purpose, androgen receptor (AR) and estrogen receptor (ER alpha and ER beta) expression was quantified in 12 clinically localized and 11 hormone-refractory sporadic prostate tumors, using real-time quantitative reverse transcription-PCR assays.

To gain more insight into hormone-responsiveness, estrogen-regulated progesterone receptor (PGR) and androgen-regulated prostatic acid phosphatase (PAP) mRNA levels were also quantified.

There is a decrease in expression of ER beta in both clinically localized and hormone-refractory tumors relative to normal prostate tissues. Moreover, hormone-refractory tumors display a decreased expression of ER alpha and an increased expression of AR. There is a positive association between ER alpha, ER beta, and PGR expression ($P < 0.0001$) and a negative association between AR and the androgen-regulated gene PAP expression in hormone-refractory tumors.

Taken together, these data indicate that, although increased expression of the AR gene might play a key role in endocrine treatment failure, it cannot be considered as the sole actor of this unresolved dilemma, and abnormalities in ER alpha and/or ER beta expression may also modulate the growth response of prostate cancer to hormone withdrawal.

Our results also suggest that ER alpha and ER beta expression status could be used to identify advanced prostate tumor patients who may respond to antiestrogen therapy.