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STEROID HORMONES IN BREAST TISSUES.

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It has been suggested that estrogens may cause breast cancer. According to the window hypothesis, rather the unopposed estrogen than the absolute estrogen excess would be a causative factor in carcinogenesis. Analogously, a close relationship between benign breast disease and an estrogen-progesterone imbalance has been stressed e.g. in the case of luteal insufficiency, that is characterized by reduced progesterone levels in serum. It is however uncertain if progesterone levels in breast tissues would show a comparable decrease. We have measured the concentrations of progesterone (P) and estradiol (E_2) in serum and in different breast tissues of patients suffering from breast cancer or from benign breast disease (BBD). In serum, P levels were significantly decreased in the luteal phase of patients with BBD (4.2 compared to 13.7 ng/ml for normal healthy women). Steroid concentrations in breast tissues could not be compared with control values in healthy women. We observed a considerable accumulation and retention of P and E_2 in breast tissues in both the follicular and luteal phases of the cycle : in glandular tissues : P 6.3 and 19.8 ng/g wet tissue and E_2 0.14 and 0.17 ng/g; in adipous tissues : P 7 and 69 ng/g and E_2 0.20 and 0.35 ng/g. In menopausal patients E_2 and P concentrations in those tissues were comparable to follicular levels. Fibrocystic tissues showed no significant differences in both phases of the cycle. In breast cancer tissues no rise of E_2 concentrations was observed.

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DETERMINATION OF E1, E2, E3 AND DHAS IN THE TISSUE OF BREAST CARCINOMA: RELATIONSHIPS WITH RECEPTOR STATUS

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In women with breast cancer cytosol estrogen and progesterone receptors (ER, PR) are usually determined to predict the response to endocrine therapy. However, receptor status may be linked to hormonal pattern in the tissue. In order to analyze this problem we determined both ER and PR, and levels of estrone (E1), estradiol (E2), estriol (E3) and deydroepiandrosterone solfato (DHAS) in the cytosol of 198 specimens of breast carcinoma. ER and PR were determined by dextran coated charcoal method; steroids were mesured by RIA method after diethyl ether extraction. All parameters were expressed in relation to the concentration of cytosol protein (cp). We considered receptor positive tumors (R+) only cases with 10 fmoles/mg of cp or more of both ER and PR, and receptor negative tumors (R-) those with less than 10 fmoles/mg of cp of both receptors; ER+PR- and ER-PR+ cases were excluded from the evaluation. Furthermore, we divided the patients into 3 groups according to their menstrual status: premenopausal (PRE), perimenopausal (PERI) and postmenopausal (POST). In PRE group, we found significantly higher levels of E1 ($p< 0.01$) and E2 ($p < 0.01$) in R+ then in R- tumors; E3 has a similar pattern althoug without reaching significant differencies; DHAS showed a trend towards higher levels in R- tumors. In PERI group, although a trend towards higher levels of E1, E2, E3, and also DHAS in R+ cases is still appreciable, the small number of R- cases prevents us from making an adequate statistical evaluation. In POST group only E1 is still significantly higher in R- cases ($p< 0.001$). From our data we can draw the following conclusions: 1) E3 is present in the tissue of the tumor in high levels: the meaning of this datum is under study; 2) tissutal concentration of estrogens does not seem the cause of false negative estrogen receptor determination.