

88. PRESENCE OF STEROID RECEPTORS IN C.N.S.:RECEPTORS IN SPINAL CORD TUMORS  
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In order to investigate whether hormones can influence the occurrence and growth of human spinal tumors through a receptor-mediated mechanism, cytoplasmic and nuclear estrogen receptors (ER<sub>C</sub>, ER<sub>N</sub>) and cytoplasmic progesterone receptor (PR<sub>C</sub>) were studied by means of dextran-coated charcoal technique. Sodium thiocyanate extraction-exchange assay (Sica et al. 1981) was used for ER determination. 13 spinal tumors (4 neurinomas, 4 meningiomas, 3 ependymomas, 1 osteochondroma, 1 metastatic papillary carcinoma) were examined: 3 neurinomas were provided of ER<sub>N</sub> (28.57; 32.50; 69.47 fmol/mg DNA) and one of them of PR<sub>C</sub> (7.30 fmol/mg protein); one meningioma had either ER<sub>C</sub> (11.12 fmol/mg protein) and ER<sub>N</sub> (65.47 fmol/mg DNA) or PR<sub>C</sub> (5.40 fmol/mg protein) and one only PR<sub>C</sub> (11.49 fmol/mg protein); ER<sub>C</sub> was present in 2 ependymomas. The osteochondroma was provided either of ER<sub>C</sub> (18.83 fmol/mg protein) and ER<sub>N</sub> (8.83 fmol/mg DNA) or PR<sub>C</sub> (10.73 fmol/mg protein). The metastatic carcinoma was provided of ER<sub>N</sub> (162.00 fmol/mg DNA). These preliminary data could perhaps support an endocrine treatment to prevent recurrences of these tumors. (Work supported in part by CNR grant, PFCGN)

89. INTERRELATIONSHIPS BETWEEN ESTROGEN AND THYROXINE ON PITUITARY LH RESPONSE TO GnRH.  
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The present experiments were designed to study the interaction of estradiol benzoate (EB) and thyroxine (T4) given *in vivo* on the responsiveness of luteinizing hormone (LH) to gonadotropin-releasing hormone (GnRH) *in vitro*. Ovariectomized-thyroidectomized (Ovx-Tx) rats were injected sc with either saline or T4 and either oil or EB for 40 days following a factorial design. The daily doses of EB were 0.01 ug and 0.1 ug in experiment 1 and experiment 2, respectively. The dose of T4 was 2 ug/100g BW/day in both experiments. All animals were sacrificed by decapitation and the blood samples were collected. Anterior pituitaries (APs) were removed and incubated *in vitro* with or without GnRH at 37 C for 4 h. LH levels in the medium and serum were determined by radioimmunoassay. A dose-response relationship was obtained between doses of 0.1 and 50 ng GnRH/ml medium. T4 reduced (P<0.01) serum LH and pituitary LH response to GnRH in Ovx-Tx rats. Administration of EB with 0.01 ug had no effect on serum LH but elevated (P<0.01) LH response to 0.1 ng GnRH. EB with 0.1 ug, however, decreased (P<0.01) serum LH and LH response to GnRH. The interaction between EB (0.1ug) and T4 on serum LH and LH response to GnRH was highly significant (P<0.01). These results suggest that the main effects of estrogen and T4 on the regulation of LH secretion are altered by the presence of the other hormone.

## 9. HYPERTENSION

90. PLASMA LEVELS OF 17 $\beta$  ESTRADIOL, UNCONJUGATED ESTRIOL, ESTETROL AND PROGESTERONE IN THE THIRD TRIMESTER OF WOMEN PREGNANCY INDUCED HYPERTENSION  
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Serial measurements were made of the concentrations of Maternal Serum 17 $\beta$  Estradiol, Estriol, Estetrol and Progesterone in 36 Toxemic mothers during the third trimester period to ascertain relative usefulness of these parameters as indicators of fetal welfare in Toxemic mothers. The Mean Value of 17 $\beta$  Estradiol in normal pregnancy in 39 weeks of Gestation was 25.6ng/ml, in mild Toxemia 23.4ng/ml, in severe Toxemia 14ng/ml. The Mean Value of unconjugated Estriol in normal pregnancy was 12.5ng/ml, in mild Toxemia 10.9ng/ml and in severe Toxemia 6.5ng/ml. The Mean Value of Estetrol in normal pregnancy was 900pg/ml, in mild Toxemia 820pg/ml and in severe Toxemia 500 pg/ml. The Mean Value of Progesterone in normal was 120ng/ml, in mild Toxemia 104ng/ml and in severe Toxemia 71ng/ml. We found nearly the same decrease of the values of these hormones in the Plasma of Toxemic mothers. We did not find any advantage of Estetrol comparing to the other hormones, as an indicator of fetal welfare in Toxemic mothers.