tion to be used as a receptor. The experiment consisted in comparing the binding value of the receptor treated with anti-estrogens or synthetic estrogens. As a result, it was clear that 16β-ethyl estradiol inhibited the binding of [6,7-^{3}H]-estradiol-17β with receptor protein and also U-11,100A, U-11,555A, clomiphene and epithioandrostanol inhibited the binding in some degree. Synthetic estrogens except methallenestril inhibited the binding. While estrone, estriol, testosterone and progesterone have no competitive action with [6,7-^{3}H]-estradiol-17β in the binding with receptor protein.

23. **High affinity binding of glucocorticoids, estrogens and androgens in cytosols of human mammary carcinomas,** F. A. G. TEULINGS, R. E. TREURNIET, J. ALEXIEVA-FIGUSCH, J. BLONK-VAN DER WIJST and H. A. VAN GILSE, Rotterdamsch Radio-Therapeutisch Instituut, Rotterdam-3024, The Netherlands

Soft tissue metastases of patients with advanced mammary carcinomas often respond well to therapy with glucocorticoids. It is not known whether the presence of high affinity binding proteins for glucocorticoids is essential for a response and it has also not been established whether the presence and concentration of glucocorticoid "receptors" in tumor tissue is related to the estrogen and androgen receptor concentrations or to the age of the patients. Low temperature agar gel electrophoresis was used for the quantification of high affinity binding of glucocorticoids in cytosols (tissue–buffer 1:2, w/v). [^{3}H]-dexamethasone was used as the radioligand. Simultaneously estrogen and androgen receptor concentrations were measured using [^{3}H]-estradiol-17β and [^{3}H-5α]-dihydrotestosterone. The ranges (and median values) of the receptor concentrations (pM/l) have been compared between patients under and over 55 years of age:

A slight positive correlation seems to exist between the respective receptors in the group of 21 cytosols: the partial correlation coefficients (Spearman-rank) are: estrogen–androgen 0·50; estrogen–glucocorticoid 0·01; androgen–glucocorticoid 0·43.

In conclusion, high affinity binding for glucocorticoids in mammary carcinomas can be found in low concentrations, independent of the age of the patients and not clearly related to the presence of androgen or estrogen receptors.

24. **Human renal carcinoma and steroid hormone receptors,** G. CONCOLINO, A. MAROCCHI, M. L. MARTELLI, V. GAGLIARDI and F. DI SILVERIO, Instituto di Patologia Speciale Medica e Metodologia Clinica II, and Clinica Urologica, University of Rome, Italy

In previous investigations using agar-gel electrophoresis (Wagner) we demonstrated the presence of estradiol and progesterone receptors in normal human kidney. The study is now extended to six human renal adenocarcinoma. Cytosols (200,000 *g* supernatant) or cytosols treated with a suspension of charcoal in buffer for 18 h were incubated *in vitro* at 0° 1–4 × 10^{-9}M tritiated estradiol and progesterone; the tritiated steroid bound to protein was determined by electrophoresis at low temperature. With this technique estradiol receptor and the estradiol-SHBG complexes were easily discriminated. Presaturation of CBG with cortisol (1 × 10^{-5}M) was necessary to ascertain the presence of progesterone receptors. The specificity of the binding was verified by adding a 100-fold excess of non-radioactive steroids. The presence of a specific receptor for progesterone was demonstrated in all the tumours examined. On the contrary estradiol receptor was found only in three out of the six carcinoma studied. Scatchard plot analysis was done in order to measure the binding capacity and the dissociation constant. Progesterone, which is known to produce a regression of these tumours in animals and has been used for the treatment of renal adenocarcinoma also in the human, in our experience does not compete with estradiol at the receptor level.

25. **Translocation of specific steroid hormone receptors into purified nuclei *in vitro* in Syrian hamster tissues and estrogen-dependent renal tumor,** JONATHAN J. LI and SARA ANTONIA LI, Department of Medicine, SDTU, Veterans Administration Hospital, 55417, Department of Pharmacology, Medical School, University of Minnesota, Minneapolis, Minn., 55455, and Department of Biological Chemistry, LHRRB, Harvard Medical School, Boston, Massachusetts, 02115, U.S.A.

The estrogen-induced and dependent renal tumor in the Syrian hamster is a unique steroid responsive tissue in that specific cytosol estrogen (8S, 8S+4S), progesterone (6–7S), and androgen (8S) receptors reside in the same tissue. The presence of these receptors was detected by incubation of the tumor cytosol at 0°C with 2–5 × 10^{-9}M tritiated steroid *in vitro* and subsequent sucrose gradient analyses after Dextran-charcoal treatment. Receptor specificity was assessed using a competitive binding assay with various steroid metabolites and anti-steroidal agents. The steroid receptors in the renal tumor have properties similar to those found in the hamster uterus and seminal vesicles, respectively. To elucidate the requirements for steroid hormone receptor translocation, we examined the ability of the renal tumor steroid receptors and both hamster and rat uterine receptors to translocate *in vitro* into target and non-target nuclei of hamster tissues. Incubation of uterine cytosols from hamster and rat and renal tumor with purified nuclei (determined by electron microscopy) from hamster renal tumor, kidney, and liver at 28°C for 20 min resulted in nuclear [^{3}H]-estradiol-5S receptor complex in all tissues in 0·4 M KCl gradients. The magnitude of nuclear retention was renal tumor ⩾livers >kidney based on equivalent DNA content. The amount of cytosol translocated into nuclear preparations also depended on the cytosol receptor used, hamster uterus ⩾renal tumor >rat uterus. [^{3}H]-Dihydrotestosterone binding in the renal tumor cytosol translocated into nuclei of all tissues examined and nuclear extracts contained a 3·2S receptor which is clearly distinguishable from that of the nuclear estrogen receptor complex. However, hamster uterine and renal tumor progesterone receptors did not translocate into non-target nuclei. (Supported by National Cancer Institute Grant CA 16854-01 and Research Service, Veterans Administration Hospital, Minn.)

26. **Comparison of the binding of [2,4,6,7-^{3}H]-estradiol-17β (^{3}H-E_2) to the nucleus in the immature rat uterus under *in vivo*, *in vitro* and cell-free conditions,** E. EKKA and R. DE HERTOGH, Endocrinology and Nutrition Unit, Hôpital St. Pierre, University of Louvain, Leuven, Belgium

In order to establish if any direct and close relationship existed between the experimentally induced status of the cytosol receptor and its binding to the nuclear component(s), the following study was undertaken. Crude nuclear pellets, from uteri of immature Wistar R rats (28 days old), after *in vivo* infusion of 180 ng/h of ^{3}H-E_2 for 4 h, or after *in vitro* incubation of whole uteri at 0–4°C