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9. The effect of antiestrogens on chromatin associated estrogen receptors and egg yolk protein synthesis in the rooster liver, MICHAEL GSCHWENDT, Deutsches Krebsforschungszentrum, Heidelberg, Germany

Recently we have demonstrated and partially characterized estrogen-binding sites on the liver chromatin from roosters. The binding capacity of the chromatin is increased several-fold after estrogen treatment of the roosters (M. Gschwendt and W. Kittstein, 1974, *Biochim. biophys. Acta* **361**, 84–96). Antiestrogens, like nafoxidine (Upjohn) and CI-628 (Parke–Davis) are known to inhibit the uterine estrogenic response. On the other hand they are also weak estrogens in the uterus. Since chicken liver and uterus respond quite differently to estradiol, it was of interest to investigate the effect of antiestrogens on chicken liver. Nafoxidine and CI-628 reduce the stimulating effect of estradiol on the estrogen-binding capacity of the liver chromatin from roosters. They show the ability, however, to increase the estrogen-binding sites on the liver chromatin themselves to some extent. *In vitro* both antiestrogens compete with [^{3}H]-estradiol for the binding sites on the liver chromatin. The relative affinities of nafoxidine and CI-628 are 0·008 and 0·014, respectively. The anti-estrogens inhibit the estrogen-induced synthesis of egg yolk proteins and fail to induce this estrogen-specific protein synthesis by themselves. Thus in the chicken liver antiestrogens are purely antiestrogenic, as far as the specific effect on yolk protein synthesis is concerned, whereas in the uterus an estrogenic response is also observed. Therefore antiestrogens might become a valuable tool for the investigation of mechanistic differences between a rather pleiotypic (uterus) and a specific (chicken liver) estrogenic response.

10. Impaired nuclear translocation and regulation: a possible explanation of anti-estrogenic activity, M. M. BOUTON and J. P. RAYNAUD, Centre de Recherches Roussel-Uclaf, 93230 Romainville, France

The molecular impacts of estrogen action, in particular at the nuclear level, have been investigated in an attempt to elucidate the differences in activity between two stereoisomers: moxestrol (11β-methoxy-ethynyl-estradiol) and RU 16117 (11α-methoxy-ethynyl-estradiol). Moxestrol is a highly potent estrogen (5–10 times more uterotrophic than estradiol in the Rubin test); RU 16117 is an extremely weak estrogen (1/100 EII) and, on the contrary, antagonizes the action of estradiol in a dose ratio of 10:1. Neither distribution nor metabolism explain the differences. Non-specific binding is weak in the plasma and negligible in the uterus; neither compound binds specifically in the plasma. No differences have been detected in the formation of the cytosolic steroid–receptor complex. Both compounds bind to the mouse uterus cytoplasmic receptor with approximately the same affinity ($1/K = 4 \times 10^{-9}$M) as measured by equilibrium dialysis and the association rate constants are the same ($5 \times 10^4 M^{-1} S^{-1}$) as measured by the Dextran-coated charcoal technique. However, the RU 16117-receptor complex dissociates 20 times faster. Both complexes are translocated into the nucleus, but translocation by RU 16117 is slower and quantitatively less. From these results, it would appear that the two steroid–receptor complexes do not have the same capacity to induce a response at the genome level, as has moreover been sub-