Nature of Nuclear Acceptor Sites for Glucocorticoid- and Estrogen-Receptor Complexes*

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SUMMARY

In cell-free systems, glucocorticoid receptor-dexamethasone complexes from hepatoma tissue culture (HTC) cells bind to HTC cell nuclei, and estrogen receptor-estradiol complexes from immature uteri bind to uterine nuclei. The binding is specific in that it is of high affinity (apparent $K_d = 2$ to $3 \times 10^{-10}$ M) and involves a limited number of acceptor sites (about 3850 per haploid genome). Furthermore, nonradioactive receptor-steroid complexes inhibit the binding of the homologous receptors complexed with tritiated steroid. In each case, the maximum number of complexes bound to isolated nuclei is very similar to the number found in the nuclear fraction of intact cells exposed to saturating concentrations of steroid. Receptor-glucocorticoid complexes also bind with high affinity (apparent $K_d = 2.6 \times 10^{-10}$ M) to about 2150 acceptor sites (per haploid genome) in isolated uterine nuclei, even though the immature uterus is devoid of glucocorticoid receptors.

The uterine acceptor sites for glucocorticoid receptors are different from those for estrogen receptors. Thus, the binding of one class of receptor-steroid complex is not inhibited by the other type. The acceptor sites for glucocorticoid receptors in both uterine and HTC cell nuclei are destroyed by DNase, whereas those for estrogen receptors are resistant. Nuclear-bound receptor-dexamethasone complexes are released from both uterine and HTC cell nuclei at a lower concentration of NaCl than is required to release bound receptor-estradiol complexes. Finally, acceptors for estrogen receptors are not found in HTC cell nuclei.

It is concluded that nuclear acceptors for glucocorticoid receptors are not restricted to target tissues and therefore from certain cells are able to bind more than one class of steroid receptor. Where this occurs, however, the results show that each type of nuclear acceptor site binds only one of the two classes of receptor-steroid complex examined. Glucocorticoid and estrogen acceptor sites differ chemically and physically, possibly reflecting differences in the molecular mechanisms of action of these hormones.

Steroid hormones are involved in the regulation of many aspects of cellular development and metabolism (1-4); indeed it appears that all tissues of the body are directly influenced by at least one of the classes of steroid hormones.

In all “target organs,” the initial events in steroid hormone action are broadly similar (5, 6). The steroid readily enters the cell and binds with high affinity and steroid-specificity to cytoplasmic receptor proteins. Conformational changes in the receptor-steroid complex then occur and the complex becomes associated with chromatin in the nucleus. It is thought that the complex acts as a genetic regulatory element, directly or indirectly altering the transcription of specific parts of the genome to produce ultimately the observed biological effects.

Although they have completely different steroid specificities (7-9), sex hormone receptors and glucocorticoid receptors have many features in common (5, 6). It is thus probable that their interaction with the cell nucleus would also be similar.

However, studies on the binding of receptor-progesterone complexes to chick oviduct chromatins have led to the proposal that tissue-specific nuclear acidic proteins are essential for this process (10), whereas a preliminary investigation of the nuclear acceptor sites for glucocorticoid receptors in hepatoma cell nuclei suggests that DNA itself may be the acceptor for these receptors (11). These results taken together suggest that nuclear acceptor sites for different receptor-steroid complexes may have different chemical and physical characteristics.

This communication describes an attempt to gain further insight into the nature of the nuclear acceptor sites for glucocorticoid receptors by comparing their nuclear binding properties in a cell-free system with those of another extensively studied hormone receptor, the receptor-estradiol complex of the immature uterus (5, 6).

Specific binding of the estrogen receptor was observed with
uterine nuclei but not with HTC cell nuclei, whereas the glucocorticoid receptor was bound specifically by qualitatively similar acceptor sites in the nuclei of both cell types. The chemical and physical properties of the uterine acceptor sites for the estrogen receptor differ significantly from those for the glucocorticoid receptors.

**METHODS AND MATERIALS**

**Chemicals and Reagents**—The sources of all chemicals and reagents and the composition of all buffers not mentioned below have been described previously (12, 13).

**Fractionation of Uterus**—Uteri were removed from intact 23- to 27-day-old female Buffalo rats (the strain from which the isolation procedures were at 0°C). The tissue was removed have been described previously (12, 13).

**Fractionation of HTC Cells**—HTC cells were grown and harvested as described previously (12, 13). The washed cell pellet was resuspended in 1 volume of homogenization buffer (0.02 m Tricine (pH 7.4)-0.002 m CaCl2-0.001 m MgCl2). The homogenate was centrifuged at 800 x g for 10 min and the supernatant fraction was removed. Part of the supernatant fraction received 10^-8 m [2,4,6,7,-3H]estradiol (85 to 100 Ci per mmole, Schwarz) and another part received 10^-8 m [3H]desamethasone plus 5 x 10^-10 m nonradioactive estradiol. This was required for the particular experiment, further portions of the supernatant fraction were then treated with charcoal or adsorbed with Sephadex in the presence of the competing unlabeled steroid (nonspecific binding) from the radioactivity retained in its absence.

**Assay of Specific Binding in Cytosol Preparations**—Samples (0.1 ml) of cytosol were assayed before activation (see below) for specific dexamethasone or estradiol binding by the charcoal adsorption method or by gel filtration over Sephadex G-25 (12). The crude homogenate was centrifuged at 800 x g for 10 min and the supernatant fraction was removed. Part of the supernatant fraction received 10^-8 m [2,4,6,7,-3H]estradiol (85 to 100 Ci per min and the supernatant fraction was removed. Part of the supernatant fraction received 10^-8 m [2,4,6,7,-3H]estradiol plus 5 x 10^-8 m nonradioactive estradiol. As required for the particular experiment, further portions of the supernatant fraction were then treated with charcoal or adsorbed with Sephadex in the presence of the competing unlabeled steroid (nonspecific binding) from the radioactivity retained in its absence.

**Activation of Receptor-Steroid Complexes**—The HTC cell receptor-dexamethasone complex does not bind to HTC cell nuclei unless it is “activated” (15). This is achieved conveniently by incubating cytosol containing the steroid at 20°C for 30 min with 15 m NaCl. Uterus cytosol, already prepared in 15 m NaCl, was merely incubated at 20°C for 30 min although it was not determined whether this procedure is obligatory for specific binding of the estrogen receptor to isolated uterine nuclei. After activation, the cytosols were passed through Sephadex (G-25) equilibrated with Tricine-NaCl buffer to remove free steroid. The macromolecular fraction was collected and immediately incubated with nuclei (see below). In each case, the active form of the receptor-steroid complex (capable of binding to nuclei) was estimated by incubating samples (0.05 to 0.1 ml) of the activated cytosol with a large excess of nuclei sufficient to bind all of the active species. Using this assay, it was found that the uterine system and the HTC cell system have many features in common. First, no further nuclear binding occurred when cytosol recovered from such an assay was exposed to fresh nuclei. Second, only part of the specifically bound steroid in activated cytosol was in the form of active receptor-steroid complex, 40 ± 6.5% (S.E.M.) in the case of uterine cytosol containing estradiol. Third, this proportion did not change on subsequent dilution, and, lastly, activated cytosol containing excess competing nonradioactive steroid was devoid of active tritiated receptor-steroid complex.

**Binding of Active Receptor-Steroid Complexes to Nuclei**—Samples (0.5 ml) of the suspension of washed nuclei (see above) were centrifuged (800 x g for 5 min) and the supernatant fractions were stored at 0°C until required (up to 3 hours).

1 The abbreviations used are: HTC, hepatoma tissue culture; dexamethasone, 9a-fluoro-11b,17a,21-trihydroxy-16a-methyl-1,4-pregnadiene-3,20-dione; estradiol, 1,3,5-triandrostane-3,17a-diol; PBS, phosphate-buffered saline; Tricine, N-tris(hydroxymethyl)methylglycine.

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were discarded. To the drained nuclear pellet were added 0.1 ml of sucrose (0.8 M), a variable amount of activated gel-filtered cytosol (see legends), and enough Tricine-NaCl buffer to make the final incubation volume 0.8 ml. The nuclei were resuspended gently with a Pasteur pipette and the suspensions were incubated at 0°C for 2 hours (sufficient for equilibrium binding).

The nuclei were then removed by centrifugation, washed twice with Tricine-NaCl buffer (1 ml), resuspended (Vortex mixer) in 0.5 ml of water, and assayed for radioactivity and DNA (15). In these experiments, the concentration of the active receptor-steroid complex remaining unbound at equilibrium was calculated by subtracting the nuclear-bound active complex from the amount of active complex added at the start of the incubation. The latter was determined by incubating in parallel samples the activated cytosol with excess nuclei (see above).

In both systems, the calculated concentration of the active complex was in close agreement with that determined directly (15).

**Incubation of Nuclei with DNase**—Washed nuclei, resuspended in homogenization buffer with 0.25 M sucrose, were digested with bovine pancreatic DNase I (Worthington, RNase-free). The enzyme was used at 100 μg per ml at 0°C for 1 hour or at 500 μg per ml at 25°C for 20 min. After digestion, the nuclear suspension was centrifuged, the DNA released into the supernatant fraction was assayed, and the ability of the nuclei to bind receptor-steroid complexes was determined (see above).

**Release of Receptor-Steroid Complexes from Nuclei by NaCl**—After binding active receptor-steroid complexes, the nuclei were washed twice and resuspended in homogenization buffer (0.5 ml) containing NaCl (0 to 0.4 M) for 30 min at 0°C. The nuclei were removed by centrifugation and the supernatant fraction was assayed for total radioactivity and receptor-steroid complexes (gel filtration method). At each NaCl concentration, the proportion of the complexes released after 90 min of incubation was the same as after 30 min.

**RESULTS**

**Specific Binding of Estrogen Receptor-Estradiol Complex to Uterine Nuclei**—In whole uterine incubated with estradiol or in intact HTC cells exposed to dexamethasone, the steroid enters the cells and binds to cytoplasmic receptor proteins (9, 10, 19). Subsequently, steroid is bound specifically in the nuclear fraction, the receptor-steroid complex disappearing from the cytosol (20). The results of extensive studies of these reactions in HTC cells and in uteri (20, 21) suggest that it is the receptor-steroid complex itself that becomes concentrated in the cell nucleus.

In a cell-free system, the active HTC cell receptor-dexamethasone complex binds specifically to HTC cell nuclei (15), i.e., it binds with high affinity to a limited number of nuclear acceptor sites. This cell-free binding has a number of features which suggest that it represents the corresponding association in the intact cell (18). Isolated uterine nuclei bind the estrogen-receptor-estradiol complex (5, 6, 22, 23) and this association was estimated ("Methods and Materials"). A, effect of complex concentration on nuclear binding; B, Scatchard (27) analysis of the data.

After intact uteri have been incubated with estradiol at 37°C, an estradiol-protein complex, having in sucrose gradients a sedimentation coefficient of 5 S, can be extracted from the nuclei with KCl (18, 19). A similar 5 S complex can be extracted from uterine nuclei incubated at 25°C in a cell-free system with uterine cytosol containing estradiol (19, 22). In fact, if cytosol containing estradiol is incubated at 25°C, a form of the estrogen receptor-estradiol complex sedimenting at 5 S can be formed in the absence of nuclei (5, 24). Exposure of uterine nuclei to this "activated" cytosol at 0°C results in formation of 5 S nuclear complex (25). It appears that, to facilitate this cytosol conversion and to obtain 5 S nuclear binding at 0°C under cell-free conditions, low ionic strength, divalent cations, and EDTA should be avoided in the preparation of the cytosol (5, 6, 26). Hence in this study, buffer containing 0.15 M NaCl but no divalent cations or EDTA was used for tissue fractionation and nuclear binding of the estrogen receptor. It should be noted, however, that cell-free specific nuclear binding of the active estrogen receptor does occur in the absence of NaCl.

When uterine nuclei were incubated at 0°C with increasing amounts of activated uterine cytosol containing [3H]estradiol, they appeared to have a limited capacity to bind the active receptor-estradiol complex (Fig. 1A). A Scatchard (27) analysis of the data (Fig. 1B) was linear suggesting that there is a single class of nuclear acceptor sites reversibly binding the complex with high affinity. The absence of a component parallel to the abscissa suggests that under the conditions employed the receptor-estradiol complex does not bind nonspecifically. Analysis of six such experiments showed that there were 7700 ± 1400 sites per nucleus and that the Kd is 2.8 ± 0.8 × 10⁻⁹ M (Table I).

The nuclear-bound estradiol is due to receptor-steroid complexes and not to estradiol alone. The concentration of free estradiol in the cytosol after gel filtration, even allowing for some dissociation of the receptor-steroid complex, is too low for nuclear binding of free steroid to account for the observed effect (based on experiments in which nuclei were exposed to a range of estradiol concentrations in buffer). In addition, no nuclear binding of [3H]estradiol occurred when nuclei were incubated with activated uterine cytosol containing sufficient nonradioactive estradiol to prevent the binding of the tritiated steroid to the cytoplasmic receptors.

If uterine nuclei have a limited number of acceptor sites for the active receptor-estradiol complex, it would be expected that activated cytosol containing only nonradioactive estradiol should competitively inhibit the binding of active receptor-[3H]estradiol complexes. This was tested by measuring the amount of re-
HTC Cells Do Not Contain Estrogen Receptors and Immature Uterus Does Not Contain Glucocorticoid Receptors—In contrast to
glucocorticoids which bind specifically to cytoplasmic receptor proteins (9, 12) and induce tyrosine aminotransferase (13) in HTC cells, estrogens, at physiological concentrations, neither induce this enzyme (13) nor are they bound specifically by soluble preparations of HTC cells. 8

The immature rat uterus contains receptor proteins binding estrogens with high affinity (for review see Ref. 6). Two experimental findings make it exceedingly unlikely that this organ also contains receptors for glucocorticoids.

First, in cytosol from immature uteri no specific binding of [3H]dexamethasone could be demonstrated at concentrations in the range 10^-10 to 5 x 10^-8 M. These concentrations are sufficient to saturate specific glucocorticoid receptors in all glucocorticoid-sensitive tissues studied 4 and result in maximal induction of tyrosine aminotransferase in HTC cells (13). The failure of the assay to detect specific binding of dexamethasone was not due to the presence in the cytosol of competing endogenous corticosteroids. 4 Using the same charcoal assay, it was possible, on experiments of 4 such experiments showed that 4350 ± 970 sites per nucleus and that the Kd is 2.6 ± 0.4 x 10^-10 M (Table I).

Considerations similar to those applied to the binding by HTC cell and uterine nuclei of their homologous receptors (see Ref. 15 and above) discount the possibility that the nuclear binding of dexamethasone by uterine nuclei is due to free dexamethasone and not to the glucocorticoid receptor.

Thus binding of the glucocorticoid receptor complex to uterine nuclei is remarkably similar to the binding of the homologous estrogen receptor (see Fig. 1, A and B, Table I) and must therefore also be regarded as specific. There do appear to be fewer uterine acceptor sites for the glucocorticoid receptor than for the estrogen receptor, although the ranges for each class overlap.

The possibility was therefore considered that the uterine acceptor sites for the glucocorticoid receptor might be similar to and even coincide with those for the estrogen receptor.

**Estrogen and Glucocorticoid Receptors Do Not Compete with Each Other for Nuclear Acceptor Sites**—The effect of the glucocorticoid receptor-dexamethasone complex on the binding of the estrogen receptor-[3H]estradiol complex was examined using the same experimental method as was used to show that active receptor-estradiol complex bound with nonradioactive estradiol competitively inhibits the binding of active receptor-[3H]estradiol to uterine nuclei (see Fig. 2).

Increasing amounts of activated, gel-filtered uterine cytosol containing [3H]estradiol were incubated at 0°C with uterine nuclei in the presence and absence of a constant amount of activated HTC cell cytosol bound with nonradioactive dexamethasone. As shown in Fig. 5, binding of estrogen receptors was not affected by glucocorticoid receptors even though in the same experiment receptor bound with nonradioactive estradiol did inhibit the binding of the radioactive estrogen receptor. This suggests that the glucocorticoid and estrogen receptors bind to spatially distinct uterine acceptor sites. This conclusion is reinforced by the results shown in Fig. 6 in which the active estrogen receptor-estradiol complex failed to inhibit the binding of active glucocorticoid receptor-[3H]dexamethasone complex to uterine nuclei.

**Effect ofDNase on Uterine Nuclear Acceptor Sites**—The binding of HTC cell receptor-dexamethasone complexes to HTC cell nuclei is prevented when the nuclei are first digested with DNase, suggesting that DNA is involved in some way in the binding of the receptor complex (11). The sensitivities to DNase of the

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estradiol. Activated uterine cytosol with (●) or without (○) nonradioactive dexamethasone was activated and gel filtered ("Methods and Materials"). Increasing amounts (0.05 to 0.35 ml) of the macromolecular fraction were incubated with uterine nuclei (●) or without (○) nonradioactive estradiol. All cytosols were activated in the same way as the tritiated cytosol.

Fig. 6 (right). Binding of active receptor-[3H]estradiol complex to uterine nuclei; effect of active estrogen receptor bound with nonradioactive dexamethasone. H1C cell cytosol containing [3H]dexamethasone was activated and gel filtered ("Methods and Materials"). Increasing amounts (0.05 to 0.35 ml) of the macromolecular fraction were incubated with uterine nuclei (●) in Fig. 4. The effects on the binding of the active receptor-[3H]estradiol complex to uterine nuclei are shown of adding 0.35 ml of active uterine cytosol with (●) or without (○) nonradioactive estradiol. All cytosols were activated in the same way as the tritiated cytosol.

**Table II**

| Cytosol | Nucleus | Receptor-steroid complex bound by nuclei |
|---------|---------|----------------------------------------|
|         |         | Control | Treated | Treated, % of control |
|         |         | cpm/sample |        |                      |
| A*      | HTC + dexamethasone | HTC | 3,886 | 1,167 | 30 |
|         | HTC + dexamethasone | Uterus | 1,159 | 487 | 42 |
|         | Uterus + estradiol | Uterus | 13,864 | 25,073 | 158 |
| B*      | HTC + dexamethasone | HTC | 4,327 | 1,132 | 20 |
|         | HTC + dexamethasone | Uterus | 1,391 | 414 | 30 |
|         | Uterus + estradiol | Uterus | 19,144 | 29,498 | 154 |

* Values are mean of duplicate determinations (within 10%). Note that specific activities were 12 Ci per mmole for dexamethasone and 100 Ci per mmole for estradiol.

**Discussion**

Nuclei from both the estrogen-responsive uterus and the glucocorticoid-responsive HTC cell each contain a single class of specific acceptor sites for their own cytoplasmic receptor-steroid complexes. These sites bind the homologous complexes with almost identical affinities and, in both cases, the cell-free binding of steroid has features in common with that observed in intact cells: the receptor is required, the affinity for the interaction is high and the number of acceptor sites are similar.

Glucocorticoid receptors from HTC cells also bind with high affinity to a limited number of uterine nuclear acceptor sites. These acceptor sites are indistinguishable from the acceptor sites...
for glucocorticoid receptor complexes in HTC cell nuclei in that they bind the receptor-dexamethasone complex with the same affinity, they have the same DNase sensitivity and the elution of receptor-dexamethasone complexes from them occurs at similar NaCl concentrations.

These results confirm the previous proposal that DNA is a major component of the nuclear acceptor sites for glucocorticoid receptors. Indeed it is possible that these acceptor sites consist of specific base sequences in the DNA. Regardless of their chemical nature, these sites probably occur in the nuclei of all cells. First, very many tissues contain cytoplasmic receptors for glucocorticoids and, second, at least one tissue, the immature uterus, which is devoid of these receptors nonetheless has glucocorticoid-specific nuclear acceptors. From these considerations, it seems possible that these acceptor sites might be essential elements in all nuclei, but are susceptible to modulation by glucocorticoid receptors only in tissues containing such receptors. Alternatively, it is conceivable that at some time during uterine development, cytoplasmic receptors for glucocorticoids, and thus sensitivity to these hormones, might be present.

In contrast, several lines of evidence show that the nuclear acceptor sites for glucocorticoid receptors are different from those for estrogen receptors. First, estrogen acceptor sites are absent from HTC cell nuclei. Second, estrogen receptors and glucocorticoid receptors do not compete for nuclear acceptor sites in uterine nuclei. Third, treatment of uterine nuclei with DNase destroys the acceptor sites for glucocorticoid receptor complexes but not those for estrogen receptor complexes. Lastly, bound receptor-dexamethasone complexes are eluted from uterine nuclei at lower NaCl concentrations than are required for the release of receptor-estradiol complexes. However, other investigators have shown that the nuclear acceptor sites for estrogen receptors are destroyed by DNase (34, 35). At present it is not clear how their results can be reconciled with those reported here but obviously more detailed investigations of the chemical nature of the nuclear acceptor sites are required. If DNA is involved in the nuclear binding of estrogen receptors, the interaction must be quite different from that with glucocorticoid receptors.

These studies were undertaken to gain further insight as to how steroid hormones evoke a biological response. It has been assumed that the binding of receptor-steroid complexes to nuclear acceptor sites influences transcription of the genome. If this is the case, the results may imply that the molecular mechanisms of action of estrogens and glucocorticoids are dissimilar.

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