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## JOURNAL ARTICLE

# Suppression of steroid receptor-chromatin interaction by inhibitors present in ammonium sulfate-fractionated rat testicular androgen receptor preparations

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Tritiated androgen receptor complex was partially purified by ammonium sulfate precipitation at 15 to 37% saturation from rat testicular or prostatic cytosols. A greater amount of prostatic tritiated androgen receptor complex bound to rat Sertoli cell chromatin than tritiated testicular androgen receptor complex. However, the testicular androgen receptor complex was more potent in competing for binding of either tritiated androgen receptor complex preparation to the Sertoli cell chromatin. Combining equal amounts of tritiated testicular and prostatic androgen receptor complexes resulted in lower binding than that observed with tritiated prostatic androgen receptor complex alone, while doubling the concentration of tritiated prostatic androgen receptor complex led to a 2-fold increase in binding. It is conceivable that the partially purified testicular androgen receptor complex preparations contain factor(s) that inhibit the binding interaction of tritiated androgen receptor complex with chromatin, while there is no evidence for the presence of such inhibitors in tritiated prostatic androgen receptor complex preparations. Fractionation of testicular cytosol on DEAE-cellulose yielded a flow-through fraction with inhibitory activity and a bound fraction containing tritiated androgen receptor complex that was eluted with 0.3 M KCl and also showed inhibitory activity. These factors also inhibited the binding of tritiated estradiol receptor complex to chromatin. They were non-dialyzable and their inhibitory effect was abolished after heating at 60 C for 30 minutes. It is concluded that ammonium sulfate-fractionated rat testicular androgen receptor complex preparations contain inhibitors of steroid receptor-chromatin interaction. The inhibitory factors can be fractionated into two distinct fractions by DEAE-cellulose column chromatography. They are nondialyzable and heat-labile. The precise chemical nature of the postulated inhibitor(s) remains to be determined.

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