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JOURNAL ARTICLE

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Evidence for the regulation of prostatic oxytocin by gonadal steroids in the rat

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Oxytocin and its receptor are present in the mammalian prostate, and the peptide has been shown to increase prostatic growth, 5alphareductase activity, and contractility. This study was performed to investigate whether local concentrations of the peptide were regulated by gonadal steroids in order to establish whether oxytocin has a physiological role in the prostate. Both intact and castrated adult Wistar rats were treated daily for 7 days with either testosterone

propionate or the antiandrogen cyproterone acetate. Animals were then killed, and plasma hormone and prostatic oxytocin concentrations were measured. A separate group of rats was treated with the 5alpha-reductase inhibitor finasteride to investigate whether testosterone or dihydrotestosterone (DHT) was involved in regulating oxytocin concentrations. In a further series of experiments, rats were treated with diethylstilbestrol (DES) or the antiestrogen tamoxifen. Treatment with testosterone significantly decreased prostatic oxytocin, whereas reduction of androgens by castration or by administration of cyproterone acetate increased prostatic peptide concentrations without altering circulating levels of the peptide. Treatment with finasteride increased plasma testosterone but decreased DHT concentrations. Prostatic oxytocin concentrations were higher in finasteride-treated animals than in control animals with comparable testosterone levels. The data suggest that both testosterone and DHT are capable of decreasing prostatic oxytocin concentrations. Treatment with DES did not significantly alter prostatic oxytocin, but administration of tamoxifen decreased concentrations of the peptide, suggesting that low levels of estrogen may be necessary for oxytocin production. These data provide evidence that oxytocin is regulated by androgens, and we hypothesize that this regulatory mechanism may be involved in controlling prostatic growth.

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