suggests that the endocrine control of these hormones, in cattle, may be a reciprocal system.

This study describes the first attempt in defining the role of SRIF in the endocrine control of mammotropic hormones of cows. The control mechanism of PRL release is at present unknown, although a consensus prevails that PRL is tonically inhibited by prolatin inhibiting factor (PIF). One might speculate that the action of SRIF on increasing basal serum PRL and potentiating the milking induced rise in serum PRL may be via interaction with PIF release, perhaps by inhibiting it. Alternatively, SRIF may directly promote the release of PRL at the pituitary.

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Anti-androgenic action of cyproterone acetate in the epididymis of the rhesus monkey[1]

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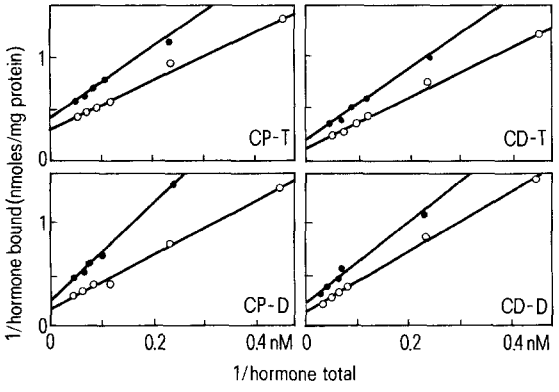
Summary. Cyproterone acetate (1 μM) inhibited the binding of labeled testosterone (T) and dihydrotestosterone (DHT) in the caput and cauda epididymides of the rhesus monkey. The same concentration of the anti-androgen failed to inhibit androgen binding in the ductus deferens, prostate and seminal vesicles.

In a study aimed at the evaluation of androgen action in various mammalian tissues, Prased et al.[3] showed that in the rat, hamster and monkey, the epididymis exhibited a higher androgen threshold requirement in comparison to the accessory glands. The fact that the spermatozoa undergo morphological and physiological changes leading to their maturation within the epididymal canal points up the organ as a potential target site for male fertility control. It was suggested that since the epididymis shows a higher androgen threshold requirement, an anti-androgen at a dose level too low to alter the functional integrity of the accessory glands should be able to cause selective inhibition of the epididymal function. An experimental approach to this concept showed that microquantities of the anti-androgen cyproterone acetate (CA) released from s.c. implanted silastic capsules in the rat inhibited epididymal function while not affecting the physiology of the accessory glands[4]. In a companion report[5], we have shown that the caput epididymidis of the rhesus monkey differed from the accessory glands in molecular mechanisms fundamental to androgen action; namely, the hormone-receptor interaction. The present report is an extension of this study where it is demonstrated that the mode of action of microquantities of CA in the epididymis is different from that in the accessory glands.

Materials and methods: The details regarding experimental animals, sources of labeled androgens, preparation of cytosol fractions and the analysis of radioactivity are given in the companion paper[5]. Equilibrium dialysis, for the study of in vitro hormone binding was carried out as follows: Polypropylene 'wells' (2 cm in height and 1.5 cm in diameter), fixed on a glass plate, were partitioned in the middle by dialysis membranes. Cytosol (0.5 ml) was added to 1 chamber and 0.5 ml of TEM buffer containing labeled T or DHT, with or without 1 μM CA was placed in the opposite chamber. The 'wells' were sealed on top and agitated in a metabolic shaker at 4 °C for 40 h. At the end of dialysis, the volume of the fluid on either side of the

Effect of 1 μM CA, in vitro on testosterone (T) and DHT (D) binding to the cytosol macromolecules of ductus deferens (DD), seminal vesicle (SV) and prostate (PR) (expressed as nmoles/mg protein). Method: equilibrium dialysis

Total hormone in the medium (nM)	Androgen	DD		SV		PR	
		−CA	+CA	−CA	+CA	−CA	+CA
2.22	T	0.540	0.714	0.281	0.481	0.490	0.657
	D	0.709	1.111	0.310	0.396	0.408	0.581
4.35	T	0.909	1.111	0.490	0.809	0.800	0.991
	D	1.087	1.333	0.478	0.725	0.571	0.806
9.09	T	0.990	1.428	0.757	1.186	0.990	1.428
	D	1.369	1.886	0.709	1.052	0.704	1.075
12.50	T	1.250	2.006	0.900	1.250	1.098	1.538
	D	1.562	2.325	0.800	1.250	1.333	2.080
16.70	T	1.333	1.219	1.612	1.333	1.666	2.222
	D	1.624	2.357	0.909	1.408	1.624	2.083
22.22	T	1.428	2.400	1.383	1.718	1.428	1.923
	D	1.685	2.425	0.920	1.416	1.639	2.120



Double reciprocal plots of the data on the binding of testosterone (T) and DHT (D) to the cytosol macromolecules of the caput (CP) and cauda (CD) epididymides, in the presence (−●−) or absence (−○−) of 1 μM CA.

membrane was measured. Protein was estimated according to Lowry et al.[6] and the radioactivity analysed as described[5].

Results and discussion. In the cytosol fractions of the caput and cauda epididymides 1 μM CA inhibited the binding of both ^{3}H-T and ^{3}H-DHT (figure). A double-reciprocal plot of the data showed that the points of intersection of the ordinate by the 2 lines were non-identical, probably indicative of the fact that CA binds to a site on the receptor other than the one occupied by the androgen. Similar results with the cytosol fractions of the ductus deferens, prostate and seminal vesicles showed that in these tissues CA failed to reduce androgen binding (table). On the other hand, there was a marginal increase in hormone binding in the presence of CA.

Whether these results show a qualitative difference between the androgen receptors of the epididymis on the one hand and those of the accessory glands on the other can not be ascertained without additional experimental documentation. However, a gross analysis of the present data does indicate this possibility. According to the currently accepted concepts on steroid hormone action, a saturated hormone-binding to the cytoplasmic receptor protein activates, through an as yet unknown mechanism, the receptor for its migration to the nuclear acceptor sites causing gene activation. The present study shows that an anti-androgen can bind to a site that is not occupied by the androgen and inhibit additional hormone-binding, possibly through a change in conformation of the receptor protein. It may be assumed that this receptor of altered configuration is unable to migrate to the nucleus and interact with the acceptor. However, additional experimental data are required in order to substantiate this hypothesis.

Some of the recent reports on the action of CA upon the reproductive function of male human volunteers[7,8] have documented the following observations. Seminal glyceryl-phosphoryl choline which is a secretory product of the epididymis dropped sharply in all volunteers treated with CA. In vitro sperm migration through mid-cycle cervical mucus (Kremer test) showed that the process was much reduced after CA treatment, again indicating a possible action of the steroid on the epididymis. There were no statistically significant changes in seminal volume, seminal fructose and alkaline and acid phosphatases which are taken as parameters to test the functional integrity of seminal vesicles and prostate[8]. These results, together with the data that we have obtained, emphasize the need for a detailed study of the biological action of CA as a potential male anti-fertility agent and also suggest the use of subhuman primates as better experimental models than the laboratory rodent in such studies.

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In vivo inactivation of denervated corpora allata by precocene II in the bug, *Oncopeltus fasciatus*

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Summary. Transection of the nervous connections between the brain and the corpus allatum (CA) in *Oncopeltus fasciatus* does not alter the susceptibility of the CA to precocene II in vivo.

The discovery by Bowers et al.[2,3] that 2 chromene compounds isolated from plants in the genus *Ageratum* disrupt the development of some hemimetabolous and holometabolous insects, and that these developmental abnormalities can be prevented or reversed by the exogenous application of juvenile hormone (JH), kindled an interest in hormone antagonists as potential 4th-generation insecticides. These chromenes are most active against hemimetabolous species, often causing precocious metamorphosis after exposure of early larval stages or sterility after exposure of adults[3-5]. The most active compound, designated precocene II (P II; 6,7-dimethoxy-2,2-dimethyl-3-chromene), is only effective when the corpus allatum (CA) is synthetically active[6,7]. In addition, treatment of larvae with P II significantly delays molting[4,7]. The inhibition of CA in vitro by P II[8,9], and the histological evidence that P II selectively destroys the parenchymal secretory cells of the CA[10-12], suggest that P II acts directly on the CA rather than on CA control centers in