correlation coefficient for the 2 assay values was 0.89. Previously reported ratios of RIA/PCA were 0.56–1.22[5]. It has been suggested that RIA does not react to all the biologically active 'PRL activity' in pituitary preparations and that the substance responsible for the added response in PCA was ACTH[11]. More recently, using rat mammary gland organ culture bioassay, Leung et al. demonstrated that RIA and bioassay estimates of blood PRL levels showed good correlation, but RIA measured only about 25% of the hormone detected by the bioassay[12]. The differences in the results between our study and the above study could be attributed to the different types of materials tested and the different types of bioassays employed.

Comparison on a relative basis. Regression analysis of RIA and PCA values, in different units, obtained from 58 medium samples with varying PRL potencies gave the equation $Y = 6.53 \times 0.61(X)$, where Y = Reece-Turner units obtained in PCA and X = µg PRL estimated by RIA. The correlation coefficient (r) was 0.87, which was highly significant ($p < 0.01$).

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In vitro conversion of steroid hormones in bovine ovarian follicles

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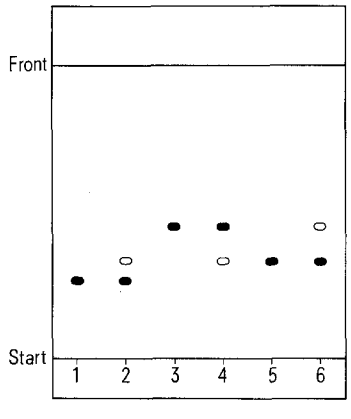
Summary. In bovine ovarian follicles an in vitro conversion of androstenedione and oestrone into oestradiol-17β, and of oestradiol-17β into oestrone was observed. There was no in vitro conversion of pregnenolone, progesterone, dehydroepiandrosterone and testosterone.

The biosynthesis of steroid hormones by the individual cell types in the ovary can be studied only indirectly using in vitro techniques. In short term incubations, or in tissue culture, granulosa cells from various animal species could readily convert androstenedione and testosterone into oestrone and oestradiol-17β[1-3].

However, there was no report in the literature on the in vitro conversion of steroid hormones in bovine ovarian follicles. Before starting our investigation of the steroid hormone concentration in bovine ovarian follicles at various stages of the cycle[4] we checked the stability of these hormones in the follicles.



Autoradiogram after TLC: 1 and 2: ^{3}H-androstenedione after incubation for 1 min (1) or 60 min (2); 3 and 4: ^{3}H-oestrone after incubation for 1 min (3) or 20 min (4); 5 and 6: ^{3}H-oestradiol-17β after incubation for 1 min (5) or 20 min (6).

Ovaries from 44 cows and heifers were collected at the local abattoir and the prooestrous stage of the cycle was selected according to the morphological appearance of the ovary.

5×10^5 dpm (= 0.7 ng) of the tritium labelled steroid hormones in 0.1 ml buffer were injected into the Graaf'ian follicle, kept at 4 °C. 100-µl samples of the follicular fluid were withdrawn at 10-min intervals after the injection.

The samples were immediately mixed with 100 µl methanol to inhibit further in vitro conversion and extracted with 5 ml diethyl ether.

The steroid hormones were separated on a TLC plate (cyclohexene: ethylacetate = 1:1) and the location of the tritium label was determined by autoradiography after 5 days exposure to an X-ray film. In the figure the conversion of oestrone and oestradiol-17β after 20 min incubation and of androstenedione after 60 min incubation is shown.

While the conversion of oestrone occurred in all 9 incubations, that of androstenedione took place only in 11 out of 18 and that of oestradiol-17β in 8 out of 10 incubations.

Androstenedione and oestrone were converted mainly into oestradiol-17β, while oestradiol-17β was converted into oestrone. These conversion products bound to a specific antibody against these hormones after extraction from the follicular fluid.

Quantification or further characterization of the conversion products was not performed, as our aim was just to check the in vitro conversion of these steroid hormones in the ovarian follicle.

For the determination of the exact concentration of oestradiol-17β, oestrone and androstenedione in bovine follicular fluid, our results show that it is necessary to withdraw the samples immediately after slaughter. These findings may well have an impact on experimental data on the steroidogenesis obtained in bovine ovarian follicles cultured in vitro.

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Effect of cyproterone acetate on the testis and epididymis of the lizard, *Psammophilus dorsalis* (Gray)

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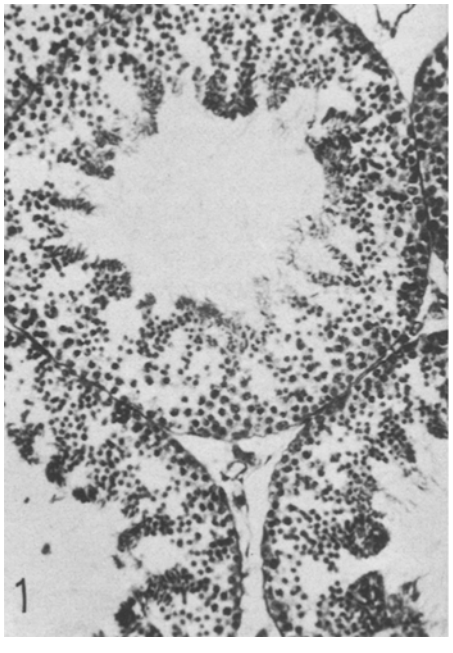
Summary. The effect of cyproterone acetate (CPA) on the testis and epididymis of the lizard, *Psammophilus dorsalis* has been studied. Treatment with CPA affects spermatogenesis and steroid metabolism in the testis. It also causes regression of the epididymis and a decrease in steroidogenic enzyme activity.

Several drugs which suppress spermatogenesis, sperm maturation or sperm transport through accessory ducts have been investigated in a large number of laboratory mammals including man[2]. Cyproterone acetate (6-chloro-17-acetoxy 1α,2α-methylene-4,6-pregna-diene-3,20 dione) (CPA) is one such drug, known more as a potent antiandrogen which interferes with the actions of androgens and estrogens at their target tissues[3,4]. Recent biochemical studies[5] on female rats, mice and gerbils suggests the estrogenic nature of CPA. In the rhesus monkey and hamster CPA selectively alters the epididymal function[6,7]. There are only a few studies using CPA in lower vertebrates and one single report of its use in reptiles. In the male lizard, *Lacerta sicula* it causes regression of the epididymis, cloacal glands and femoral pores[8]. The present study reports the effect of CPA on the histophysiology of the testis and epididymis of the lizard, *Psammophilus dorsalis.*

Materials and methods. Sexually mature male lizards weighing 35-45 g were collected in and around Mysore city. Lizards were maintained in the laboratory under the same conditions of light and temperature as those to which they are normally exposed in their natural habitat. Lizards thus acclimatized to laboratory conditions were used for this experiment. The lizards were randomly divided into groups A, B and C each consisting of 5 animals, and were housed in clean glass cages. They were fed on cockroaches and water was given ad libitum. Group C was injected s.c. with 250 µg CPA in 0.1 ml olive oil on alternate days for a period of 28 days. Group B was injected with the vehicle only and group A served as non-treated controls. All lizards were weighed and killed by decapitation 24 h after the last injection. The testes and epididymes from all the groups were dissected out and the weights were recorded. 1 testis and 1 epididymis from each group was immediately frozen at $-20\,^{\circ}C$ and 16 µm thick sections were cut in a cryostat maintained at $-20\,^{\circ}C$. Air-dried cryostat sections were incubated in a serological water bath at 37 °C for 1 h in appropriate incubation media containing different substrates, co-factors and tetrazolium salt. Δ^5-3β-hydroxysteroid dehydrogenase (Δ^5-3β-HSDH) and 17β-hydroxysteroid dehydrogenase (17β-HSDH) were localized according to the method of Baillie et al.[9], by using pregnenolone



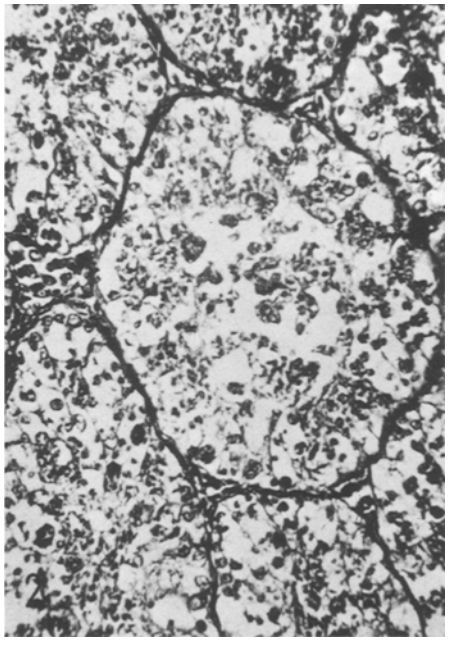


Fig. 1. Testis of olive oil-treated lizard showing active spermatogenesis. Haematoxylin-eosin. × 166.

Fig. 2. Testis of CPA-treated lizard showing disorganized germ cells, Haematoxylin-eosin. × 166.