occupied by the hormone for at least 24 h. Similar results have been obtained by Conti et al.[10] in luteinized ovaries. The occupancy of the receptors in luteinized ovaries result in the desensitization of the ovary to further hormonal stimulation and apparently in the degradation of the occupied receptors[10,15]. Therefore the reappearance of free receptors in luteinized cells is due to receptor synthesis. However, as we have shown here, in the ovaries of immature rats, where luteinized cells are not present, the reappearance of receptors at the 2nd day after the injection is independent of protein synthesis but due to the dissociation of the receptor-hormone complex. From these observations it can be assumed that the modulation of LH/HCG-receptors in luteinized cells to HCG is different from the receptor modulation in follicles and interstitial tissue of immature rat ovaries. A more detailed description of our results will be published elsewhere.

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Anti-estrogen inhibition of testosterone-stimulated aggression in mice[1]

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Summary. Testosterone-stimulated intermale aggression in castrated mice can be reversibly inhibited by anti-estrogen administration suggesting that estrogen formation and actions in the brain may be required for testosterone's behavioral actions.

Numerous recent studies support the hypothesis that metabolic conversion of testosterone to estradiol in the brain is an essential step in eliciting the behavioral actions of this androgen. For exemple, in rats and mice biochemical studies have clearly established the presence of the aromatization enzymatic pathway in the brain[2,3], while behavioral studies have shown that male sexual behavior can be stimulated by both androgens and estrogens and inhibited by anti-estrogens[4-7]. Since intermale aggression in castrated male mice can also be stimulated by both androgens and estrogens[8-10], the present study was designed to test the effects of the potent anti-estrogen, CI-628 on this behavior. In previous work with both rats and mice, CI-628 has been shown to be very effective in blocking the nuclear binding of estrogen in preoptic and medial-basal hypothalamic brain regions[2,11].

25 individually housed adult CD-1 mice (Charles Rivers) were castrated and started on a daily s.c. injection schedule of 200 µg testosterone dissolved in 0.05 cm^3 benzylbenzoate-oil (20:80, v:v). Beginning 1 week later all males were placed in $12 \times 28 \times 28$ cm glass testing chambers and given their first 10 min behavioral test. All testing was conducted in the dark phase of the lighting cycle. Males were allowed to habituate to the chamber for 10 min prior to each test. Tests were initiated by the introduction of a group-housed nonaggressive male mouse to the chamber with the test male and all biting and other forms of aggression were scored as described in our previous work[9,12]. After 5 min the group-housed male was removed and replaced with another male for an additional 5-min test. Mice were retested at 3- to 4-day-intervals for the next 46 days. Following these baseline tests 10 males which had displayed active biting during the last 4 tests were selected for further study. For the next 2 weeks these males received additional daily injections of 2 mg CI-628 dissolved in 0.05 cm^3 3% ethanol-saline. Males were tested at 7, 10 and 14 days of CI-628 treatment. For the next week males were given vehicle injections (in addition to testosterone) and tested at 3 and 7 days.

As shown in the table the CI-628 treatment produced a dramatic drop in testosterone-stimulated intermale aggression. By the end of the 2-week period only 1 male was still exhibiting biting attacks ($p < 0.005$, Fisher Exact Probability test). Within 1 week after cessation of CI-628 treatment 80% of the males had resumed biting attacks ($p < 0.01$, compared to last CI-628 test). These data are entirely consistent with the recent report that the placental aromatase blocker, 4-androsten-3,6,17-trione, inhibits testosterone- but not estradiol-stimulated fighting[13]. Thus there is increasing support for the hypothesis that testosterone aromatization to estradiol is an important step in androgen-induced aggression in male mice.

Number of testosterone-treated (200µg/day) male mice exhibiting attacks during the last 4 baseline tests. 3 CI-628 (2 mg/day) and 2 vehicle tests

	Baseline tests				CI-628 tests			Vehicle tests	
No. of days of testosterone	35	39	42	46	53	56	60	63	67
No. exhibiting biting attacks	10/10	10/10	10/10	10/10	5/10	4/10	1/10	5/10	8/10

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Thyroid activity in response to some gonadal steroids in methallibure-treated *Heteropneustes fossilis* (Bloch)[1]

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Summary. Methallibure treatment is as effective as hypophysectomy in reducing thyroid activity in *H.fossilis*. Sex steroids (TP and EB) administration restored thyroid activity in methallibure-treated females to normal level, but failed to elicit any response in males. This drug seems to block TSH secretion and thyroid hormone synthesis in *H.fossilis*.

Studies on thyroid-gonad interrelationship in teleosts have yielded debatable results. Some workers[3-6] have reported heightened thyroid activity associated with gonadal development and spawning, while others[7,8] have observed diminished thyroid activity in that sexual phase. Increased thyroid activity during spawning may also be related to variation in physical activity rather than gonadal function[4,9]. A direct augmenting effect of hormonal steroid therapy on thyroids activity has also been recorded[10-13]. Methallibure (a derivative of bis-thiourea) has been successfully used to alter the secretion of gonadotropin and to prevent the action of gonadotropins on ovary[14-16] and testes[17,18]. Except for some histological evidence[17], no work has apparently been done to investigate the effect of methallibure on thyroid physiology of teleosts. In the present experiments, an attempt has been made to compare the effect of methallibure on thyroid activity with that of hypophysectomy. In methallibure-treated fish, the response of thyroid after sex steroid therapy has also been evaluated.

For this project 84 adult specimens (42 of each sex) of *H.fossilis* with average weight 70 g (60–80 g) and length 22.5 cm (20–25 cm) were collected from ponds around Varanasi. They were fed on minced liver on alternate days. Aquarium temperature was not controlled, the variation was uniform in all the aquaria and ranged from 23 to 25 °C. Specimens were divided into 2 groups for 2 experiments. The details of treatment, time interval, number and sex of specimens are given in tables 1 and 2. The techniques already published for hypophysectomy[13] and for estimation of thyroidal activity[11,19] have been used in this project. All injections were given i.p., and the volume of each injection was 0.2 ml. p-values for significance were calculated by Student's t-test.

Response of thyroid after hypophysectomy and methallibure treatment in both sexes is given in table 1. 2 weeks treatment of methallibure at the dose of 100 µg/g thrice a week reduced the thyroid activity to the level comparable to that which resulted after hypophysectomy. Both sexes gave similar response after hypophysectomy, as well as after methallibure administration (table 1). Results of sex steroid therapy in specimens pretreated with methallibure are given in table 2. In females, lost thyroid activity was restored almost to the normal level within 2 weeks of testosterone/propionate (TP), estradiol benzoate (EB) and thyroid stimulating hormone (TSH) administration. But in males except TSH, both the sex steroids tested failed to induce any increase in thyroid activity (table 2). TSH treatment was partially effective. Findings clearly indicate that methallibure treatment, like hypophysectomy, effectively reduced thyroid activity. It seems this drug blocks normal thyroid functioning, probably either by inhibiting TSH secretion or preventing the action of circulating TSH on thyroid or impairing thyroid hormone synthesis. Reduced thyroid activity in response to methallibure treat-

Table 1. Comparison of thyroid activity in hypophysectomized specimens with that of methallibure-treated ones in *H.fossilis*

Batch*	Sex	Treatment	Maximum thyroidal ^{131}I uptake in % (mean ± SEM)	CR**
1	Female	Hypophysectomized	3.77 ± 0.50	8.10 ± 0.65 ($p<0.01$)
2	Female	Methallibure 100 µg/fish thrice a week for 2 weeks	2.84 ± 0.24	10.18 ± 1.06 ($p<0.05$)
3	Female	Sham operated given 0.6% saline injection	15.80 ± 1.22	20.00 ± 1.36
4	Male	Hypophysectomized	2.00 ± 0.72	9.15 ± 0.87 ($p<0.05$)
5	Male	Methallibure 100 µg/fish thrice a week for 3 weeks	4.06 ± 0.74	8.00 ± 0.50 ($p<0.01$)
6	Male	Sham operated given 0.6% saline injection	16.00 ± 1.45	21.08 ± 2.30

*Each batch had 6 specimens; **$CR = \frac{PB^{131}I\ CPM}{^{131}I\ CPM + PB^{131}I\ CPM} \times 100$; p-values in batches 1 and 2 are against batch 3 and in 4 and 5 against batch 6.