SRIF has been shown recently to be involved in a great number of inhibitory effects totally distinct from its action on growth hormone secretion. For instance, SRIF modulated neuronal firing activity in various brain structures[5,6], counteracted the stimulatory effect of VIP on prolactin release from the pituitary[7] and the action of histamine on cyclic AMP accumulation in gastric glands[8]. Moreover, it has been recently reported[9] that intraventricular administration of SRIF in adult ovariectomized female rats was able to reduce plasma concentrations of LH and FSH and that SRIF could also in some instances reduce LH levels in humans[10]. Our results suggest that the possible direct effect of SRIF on gonadotropin secretion takes place at the hypothalamic level to inhibit LHRH release. This hypothalamic site of action for a direct effect of SRIF is substantiated by the fact that SRIF does not seem to act on the pituitary to affect LH and FSH release[11].

The observation that VIP does not affect LHRH release under our experimental conditions is consistent with other data showing that VIP does not interfere with basal or induced-LHRH release by either dopamine or potassium in various in vitro systems[12,13]. This conclusion is further substantiated by the finding that neither secretin nor glucagon, 2 peptides structurally related to VIP, has any effect on the spontaneous LHRH release.

High doses of neurotensin have been shown to inhibit LH release with no effect on FSH[14]. Our results suggest that, in physiological concentrations, neurotensin does not act directly on the release of LHRH from the MBH.

Lack of effect of various neuropeptides on spontaneous LHRH release from MBH

	Control	10^{-10} M	10^{-9} M	10^{-8} M
VIP	2.4±0.2 (6)	2.6±0.3 (7)	2.3±0.3 (8)	2.4±0.4 (9)
Secretin	2.7±0.2 (7)	2.7±0.3 (7)	2.3±0.2 (7)	2.9±0.5 (6)
Glucagon	2.7±0.2 (7)	2.7±0.2 (7)	2.4±0.3 (7)	2.6±0.3 (7)
Substance P	2.7±0.4 (7)	3.1±0.4 (7)	3.4±0.4 (7)	3.3±0.4 (7)
Neurotensin	2.7±0.4 (7)	3.0±0.3 (7)	3.1±0.3 (7)	2.9±0.3 (7)
AVT	3.0±0.4 (8)	3.3±0.6 (8)	3.8±0.5 (8)	2.7±0.4 (8)

Results are expressed as percent of LHRH released in the medium (pg/MBH equivalent) over LHRH content in the tissue (pg/MBH equivalent). The data are mean±SEM. Number of experimental points in parentheses. The results are a representative example of 1 of 4 different experiments. Data from the other experiments are similar and give no significant differences between groups by analysis of variance.

The lack of action of substance P at the hypothalamic level can be correlated with the observation that the inhibitory effect of substance P on gonadotropin secretion reported in vivo[15] can take place directly at the pituitary level[16]. The ineffectiveness of AVT in affecting LHRH secretion is in agreement with the hypothesis of Pavel et al.[17] suggesting that AVT acts on LHRH through a serotoninergic system. Since we previously reported that serotonin can inhibit LHRH release from the MBH[18], further studies are necessary to confirm this possibility.

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Luteinizing hormone content of the pars tuberalis of the hypophysis of neonatally androgenized female rats[1]

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Summary. The immunoreactive LH content of the pars tuberalis of neonatally androgenized female rats was studied and found to be significantly lower than that of control females at oestrus or of control males. It is concluded that the rat pars tuberalis does secrete LH and that this secretory activity is diminished by neonatal androgenization in a manner that differs from that reported for the LH secretion in the pars distalis.

Several types of secretory cells have been described in the pars tuberalis of the hypophysis[3,4]. The fine structure of one of these cell types is similar to that of gonadotropes from the pars distalis of the hypophysis[4]. Immunocytochemical studies have shown that in the rat there is only cell-type common to both the pars distalis and the pars tuberalis; that the cells secreting luteinizing hormone (LH)[5]. It has also been reported that the immunoreactive LH cells of the pars tuberalis and those of the pars distalis undergo the same modifications after castration or estrogen treatment[6]. The latter results could be taken as an indication that the LH cells of the pars tuberalis could be altered by the hormonal milieu of the animal. In order to investigate this possibility, the LH content of the pars tuberalis of adult female rats that had been neonatally masculinized during the critical period of sexual differentiation of the brain, and that of intact female at oestrus and intact males, were determined.

Female Wistar Holzman rats were injected at day 5 of age with 1.25 mg of testosterone propionate (TP) (androgenized rats) or oil (controls). TP was dissolved in vegetable oil and

administered s.c. Males from the same litter were used as normal males. 90 days after treatment all animals were killed by decapitation. The pars tuberalis-median eminence (PT-ME) region from 4 androgenized rats, 4 control females at oestrus and 4 control males were dissected out and processed for morphological studies. That part of the pituitary stalk which had remained connected to the median eminence (ME) was cut off under a dissecting microscope so that only the ME and that part of the pars tuberalis close to it were used to prepare extracts. That part of the stalk attached to the ME was excluded from the extracts because of 2 main reasons. Firstly, we had previously found that when this procedure was not applied there was a large variability in the LH content of this region from one rat to another; this was probably due to the fact that when the brain is pulled out, the pituitary stalk does not always break at the same point and, consequently, the amount of stalk tissue attached to the ME is quite variable. Secondly, we were especially interested in studying the LH cells located in the pars tuberalis close to the ME because of its particular anatomical characteristics[8]. The blocks of tissue were fixed in diluted Bouin for 24 h, dehydrated and embedded in paraffin and serially sectioned. The sections (8–10 µm thick) were processed according to the unlabeled enzyme immunocytochemical method for the demonstration of rat LH[9]. Anti-rat LH serum directed against the whole LH molecule (kindly provided by NIAMDD-NIH) was used, at a dilution of 1:250. Controls were performed by incubating slides with preabsorbed antibody with an excess of rat LH (1 µg/ml), and without first antibody.

When used for radioimmunoassay, the ME-PT of each animal was homogenized separately in 0.1 phosphosaline solution, centrifuged in a Beckman microfuge and the supernatant stored at −20 °C until assayed. LH was determined in duplicate by the double antibody radioimmunoassay as recommended in the directions supplied with the NIAMDD kit with minor modifications. A kit for determination of LH was provided through the NIAMDD–NIH Pituitary Hormone Program. The results are expressed in terms of LH RP-1 standard. The sensitivity of the assay was between 5 and 8 ng/ml (0.5–0.8 ng per tube) and the standard curve was linear between 5 and 180 ng/tube. Intra-assay variation was less than 10% and between-assay variation was less than 12%. The quality of the assay was controlled according to the criteria proposed by Rodbard[10]. Statistical significance was determined with the ANOVAR-1 way test and, when necessary, by analysis of variance, followed by Duncan's multiple range test.

The effect of postnatal TP treatment on the LH content of the PT-ME is shown in figure 1. All androgenized females used in this study were anovulatory, showing a vaginal state of oestrus. Therefore, rats at oestrus were used as control females. The androgenized rats showed a significant decrease ($p < 0.001$) in the content of LH in the PT-ME as compared with that of the control litters injected with oil or with that of intact male rats.

As shown in figure 2, both experimental and control animals displayed immunoreactive LH cells in the pars tuberalis. In the pars tuberalis of both male rats and control rats at oestrus, the number and distribution of these cells were similar. In these animals, most of the immunoreactive LH cells were confined to 2 regions of the pars tuberalis, that surrounding the pituitary stalk and that occupying the tuberoinfundibular sulci. In the pars tuberalis of androgenized animals the immunoreactive LH cells were virtually absent from the region close to the tubero-infundibular sulci, whereas the number of those located in the vicinity of the pituitary stalk was diminished as compared with that of control rats. As judged from the immunocytochemical study, the radioimmunoassayable LH detected in extracts of the PT-ME region is, in all probability, exclusively

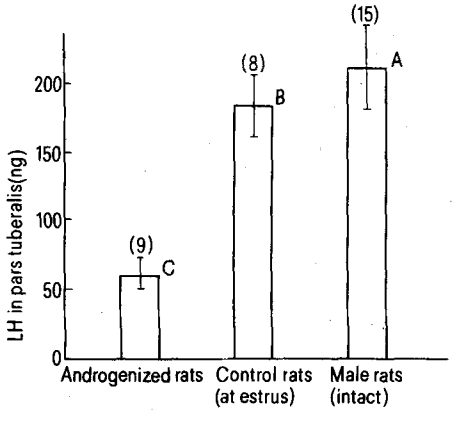

Figure 1. LH content of the pars tuberalis of androgenized rats and control females and males. Vertical lines indicate SE. In parentheses, number of animals. In all cases, results are expressed as RP-1 standard.

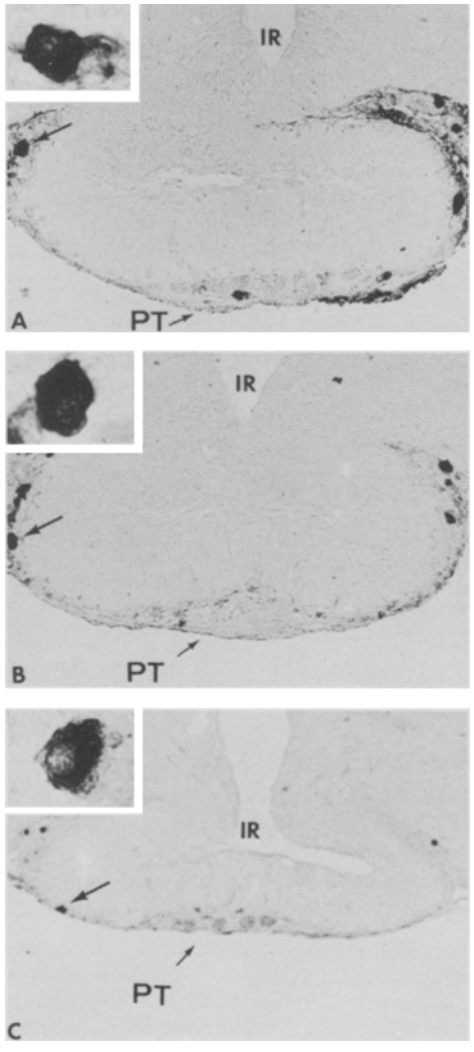

Figure 2. Median eminence-pars tuberalis (ME-PT) region from a male rat (A), a female at oestrus (B) and an androgenized female (C), immunostained for the demonstration of LH. The arrows point to immunoreactive LH cells. PT, pars tuberalis; IR, infundibular recess. A, × 120; B, × 120; C, × 110. The inserts in A, B and C show LH cells at high magnification (× 600).

stored in cells of the pars tuberalis. Therefore, the LH content of the PT-ME region should in actuality be regarded as the LH content of the pars tuberalis.

The present results clearly indicate that the LH content of the pars tuberalis of androgenized female rats is much lower than that of control females at oestrus. On the other hand the LH contents of the pars distalis of androgenized rats and that of the pars distalis of control females at oestrus are not significantly different[11]. Therefore, under the same experimental conditions the LH cells of the pars tuberalis and the LH cells of the pars distalis behave in different way. Furthermore, the LH content of the pars distalis of male rats is much higher than that of the pars distalis of female rats at oestrus. On the other hand the LH content of the pars tuberalis of both males and females at oestrus is not significantly different (fig. 1). These latter results could be taken as an indication that under normal conditions the LH cells of the pars tuberalis and those of the pars distalis do not function as a homogeneous cell population. It is postulated that the LH cells of the pars tuberalis and those of the pars distalis should be regarded as 2 functionally different sources of LH.

It is worth noticing that the LH cells of the pars tuberalis of androgenized female rats do not behave like the LH cells of the male pars tuberalis as judged from the LH content of this pituitary region. A similar finding has been reported for the LH cells of the pars distalis of androgenized female rats[12].

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Why is *Pyrrhocoris apterus* insensitive to precocene II?[1]

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Summary. Precocene II (P II) was applied to the adult females of 2 Pyrrhocorid bugs - *Pyrrhocoris apterus* (insensitive to P II) and *Dysdercus cingulatus* (sensitive to P II) - subjected to allatectomy and intraspecific or interspecific reimplantations of corpus allatum (CA). The failure of P II to inhibit ovarian development in *P. apterus* appears to be caused by both a low sensitivity to P II of the CA itself and unknown 'anti-precocene mechanisms' outside the CA.

Precocene II (P II) has been shown to inhibit the ovarian development in several insect species[2]. The corpus allatum (CA) appears to be a target of P II as juvenile hormone analogues can compensate for the inhibitory effect of P II[2,3]. Histological evidence has been provided that P II selectively destroys the secretory cells of the CA[4-7]. The experiments in vitro suggest that P II inhibits the CA directly[8-10]. In vivo, it has been demonstrated in *Dysdercus cingulatus*[11] and *Oncopeltus fasciatus*[12] that P II inhibits the CA humorally rather than via the nervi allati. However, it is not clear whether the inhibitory effect of P II in vivo depends merely on the specific interaction between CA and P II or whether other physiological processes outside the CA are involved. A similar problem arises when we ask why the P II fails to inhibit ovarian development in *Pyrrhocoris apterus* (Socha, unpublished), although *D. cingulatus* from the same family of Pyrrhocoridae is highly sensitive to P II[2]. We have investigated this question in allatectomized females of *D. cingulatus* and *P. apterus* subjected to intraspecific and interspecific reimplantations of CA and to subsequent treatment with P II.

Materials and methods. Adult females of *P. apterus* and *D. cingulatus* (Heteroptera, Pyrrhocoridae) were used in the experiments. Insects were reared on linden-seed at 26 ± 1 °C and at long-day (18L:6D) photoperiod. The females destined to be donors or recipients of CA were deprived of food within several h after imaginal ecdysis. Next day the CA was excised from each recipient through an incission in

Table 1. Effect of P II on reproduction in unoperated *Pyrrhocoris apterus* and *Dysdercus cingulatus*

Species	n	Oviposited females (%)	Pre-oviposited period (days)	Oviposited period (days)	Post-oviposited period (days)	Egg batches/ female	Eggs/batch
*P. apterus**	9	100	6.4 (6-10)	22.8 (8-29)	2.3 (0-6)	6.9 (3-8)	55.2 (18-69)
P. apterus + P II*	9	100	7.8 (6-8)	21.0 (0-30)	4.4 (0-25)	6.0 (1-9)	57.5 (30-76)
*D. cingulatus***	10	100	9.7 (7-17)	13.4 (8-21)	6.9 (0-16)	4.1 (2-6)	77.1 (12-116)
D. cingulatus + P II**	8	0	-	-	-	-	-

Dose of P II per Petri dish: 600 µg. Experiments lasted 35 days* and 30 days**.