mainly changes reciprocal to these of FT_4. At the beginning, up to the 4th h, the changes were not significant, but at 6 h (maximal FT_4) the TSH level decreased significantly ($p < 0.001$) to 53% of control value, then slowly, but significantly ($p < 0.01$) increased to 80% at 12–24 h and again significantly decreased ($p < 0.05$) after 48 h to 50% of control (the change to the contol value $p < 0.001$). The level of serum total thyroxine was from the beginning of the experiment mainly significantly ($p < 0.001$ at 1, 6, 12, 24 h) decreased to 60% of control, but at 48 h it rapidly increased up to 135% ($p < 0.001$). The levels of T_3 and of AFT_4 from the beginning to the 6th h showed insignificant increase or decrease from the control values, at 12–24 h they were significantly ($p < 0.05$) decreased, but at 48 h significantly ($p < 0.01$) increased.

The maximal changes of TSH and FT_4 temporally coincide at 6 h after SCN^- administration. The levels of other parameters at this time were significantly decreased (T_4) or insignificantly changed (T_3, AFT_4). This suggests that in this period of experiment only the elevated FT_4 was responsible for the inhibition of TSH secretion. In the next period (12–24 h), TSH level increased, but it still did not attain the control level. This increase of TSH could follow from the low concentration of T_4, T_3 and AFT_4, but most probably from rapid decrease of FT_4, which remained, however, increased in comparison with controls. Persistent lower level of TSH could be ascribed particularly to this increased level of FT_4. Reciprocal changes of FT_4 and TSH levels during 24 h are in accordance with the similar changes in the man[7,8]. Moreover, some authors[15,16] also found that in contrast to the inconsistent relationship of AFT_4 and AFT_3 to TSH levels in patients, drug-induced increases in FT_4 and FT_3 appeared consistently to be related temporally to decreases in serum TSH values[16], or to the reduced peak elevation of TSH after TRH[15]. All these findings support the idea that FT_4 may be the important factor regulating the feedback mechanism of the TSH secretion.

48 h after SCN^- administration FT_4 attained control level, while T_4, T_3 and AFT_4 increased vehemently. This unexpected rise of thyroid hormone level could be explained by the increase of hormone biosynthesis (after withdrawal of antithyroid effect of SCN^-) and also by intake of thyroid hormone from intracellular to intravasal space after restoration of binding of protein carriers in blood. Significant decrease of TSH level at 48 h suggests that the other components of circulating thyroid hormones also participate on the feedback regulation of TSH secretion.

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Ovarian serotonin content in relation to ovulation[1]

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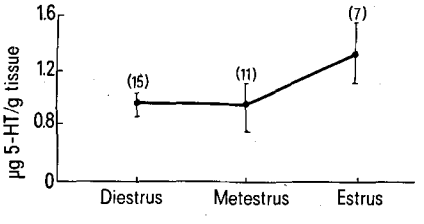
Summary. In the mature cyclic female rat, analysis for ovarian serotonin content reveals comparatively high serotonin content. Fluctuation of serotonin content was observed; peak for serotonin was observed at estrus. In gonadotropin-treated immature rats, there was no detected ovarian serotonin using this procedure. It was concluded that ovaries from gonadotropin-treated immature rats are physiologically different from ovaries taken from mature cyclic rats.

More of the work in the past in relation to serotonin (5-hydroxytryptamine, 5-HT) has focused primarily on the neuroendocrine control of the anterior pituitary function in controlling ovulation. It has been assumed that alterations in the metabolism of amines in discrete parts of the brain influence the secretion of gonadotropines and hence the process of ovulation[4].

It was reported that ovulation can be inhibited in mature adult rats using antagonists to 5-HT such as cyproheptadine, mianserin, and methysergide or LSD[5]. It was also observed physiological and pharmacological differences between the spontaneous ovulating mature rats and the pregnant mare serum gonadotropin (PMS)-induced ovulating immature rats. The present investigation was designed to relate ovarian 5-HT level to ovulation in spontaneously ovulating mature rats and PMS-induced ovulating immature rats.



Levels of ovarian serotonin content in the mature cyclic spontaneously ovulating rats. Each point represents the serotonin content of ovaries ± SE of the mean. Number of animals used to determine each point is indicated in parentheses.

Methods. In the 1st experiment, mature cyclic female Sprague-Dawley rats were used. They were maintained under controlled light (14 h light: 10 h dark) and temperature (23 °C ± 1 °C) in an environmental chamber. Food and water were provided ad libitum. Vaginal smears were taken daily with the establishment of 2 consecutive regular estrus cycles. Animals were sacrificed by over-exposure to ether vapor at stages of estrus, metestrus, and diestrus. The ovaries were removed immediately, dissected free from the bursa and extraneous tissues, weighed and analyzed.

In the 2nd experiment, 56 immature female Sprague-Dawley rats were used. They were maintained under the same experimental conditions as in the 1st experiment.

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To induce synchronous ovulation, 4 groups of rats were injected s.c. with 20 IU of PMS (Sigma chemicals). Animals were sacrificed at 0 h, 24 h, 48 h, and 72 h by over-exposure to ether vapors. The ovaries were removed immediately, dissected free from the bursa and extraneous tissues, weighed and analyzed. The procedure utilized for the analysis of ovarian 5-HT was developed by Shellenberger and Gordon[6]. The Aminco-Bowman Spectrophotoflurometer was used at a wavelength of 385 nm for activation peak and 490 nm for emission. The data was subjected to analysis of varience using t-test.

Results. In the 1st experiment, the figure shows the results of assaying for ovarian 5-HT content which reveals comparatively high content, fluctuation of ovarian 5-HT content was observed. The peak for 5-HT was noticed at estrus, which was significantly higher than of that observed during diestrus ($p < 0.05$). No significant difference ($p > 0.05$) was detected between diestrus and metestrus or between estrus and metestrus. In experiment 2, involving immature rats injected with 20 IU of PMS, there was no detected ovarian 5-HT using this procedure. Both experiments were repeated twice and the same results were obtained both times.

Discussion. The increase in ovarian 5-HT level in the cyclic rat during ovulation might indicate the importance of this neurotransmitter in the ovulation process. Injections of 5-HT antagonist in mature animals was found to inhibit ovulation[5]. Meanwhile, large doses of 5-HT inhibit ovulation in the immature PMS-treated animals as well as adult animals[7–9].

Recently it was indicated that the inhibitory effect of 5-HT on ovulation in the adult rat is peripherally mediated rather than centrally[10]. The results presented here indicate a definite physiological difference between the immature induced ovulated animals and the mature cyclic rat. Recently, the pharmacological responsiveness differences have been observed between the 2 different groups of animals[11]. Data from this experiment might indicate the lack of complete innervation in PMS-treated animals which can explain the high number of ova shed in those animals. Follicular innervation might be involved in the control of the number of ova shed to the oviduct. The 5-HT effect on the peripheral level as well as its availability in appreciable amounts in the ovary does not deny its importance in maintaining delicate balance of brain biogenic amines in regulating the release of the hypothalamic gonadotropin releasing factors. This role has been documented previously[9,10]. The present experiment indicates that the mature ovary is probably another site for 5-HT effect. As previously indicated, 5-HT[7–9] and its antagonist[5] can cause the same effect, inhibition of ovulation. It may be that the female rat has 2 5-HT-sensitive areas, one in the brain and the other in the ovary. The pharmacological differences between the spontaneous ovulating mature rats and PMS-induced immature rats may be due to the lack of 5-HT receptors in ovaries of the latter. It can be suggested from this investigation that the 5-HT and serotoninergic fibres of the ovary play an important role in the ovulation process in the cyclic rat, but not in the PMS-induced ovulating rats.

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Effects of precocene II and juvenile hormone III on the activity of neurosecretory A-cells in *Oncopeltus fasciatus*[1]

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Summary. Application of precocene II, topically or by contact method, appears to inhibit the synthetic activity of neurosecretory A-cells of the pars intercerebralis of *Oncopeltus fasciatus*. Treatment of precocene-treated insects with JH III apparently stimulates the secretory activity of these cells.

Treatment of female *O. fasciatus* larvae with the chromene derivative, precocene II[3], induced precocious metamorphosis and sterility[3,4]. In adult females it inhibited egg maturation and corpus allatum (CA) growth[5] and induced degeneration of the CA[6]. The effects of precocene II on ovarian growth can be reversed by the application of exogenous juvenile hormone (JH)[3,4]. It is not known whether precocene II affects the CA directly or whether its effects are mediated through other components of the neuroendocrine system. In *O. fasciatus* the median neurosecretory cells are known to influence the CA growth and reproduction[7]. Therefore we studied the effects of precocene II on the dynamics of the A-cells of the pars intercerebralis of adult female *O. fasciatus* by microspectrophotometry of paraldehyde fuchsin (PF) stained material.

Materials and methods. Newly emerged adult females (0–1-h-old) of *O. fasciatus* were treated topically with 50 µg precocene II (ZR 2448, Zoecon Corporation, Palo Alto, California) dissolved in acetone. Brains from these, and acetone treated control insects were fixed in Bouin's fluid, 2, 4 and 6 days after treatment, and processed for paraffin sections. Another group of newly emerged female insects were treated with precocene II by contact method (15 µg/an²)[6] and then 5 days after were treated topically

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