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THE EFFECT OF SEX HORMONES ON GASTRIC SECRETION IN THE RAT

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Summary

Experiments were performed to study the influence of sex hormones on gastric secretion in response to pentagastrin (130 μg/100 g rat) by Shay's method. Adult rats were subjected to bilateral gonadectomy and treated with sex steroids (estrogen, progesterone and testosterone) in oil suspension from 1 to 14 days. There was no significant change in acid secretory activity between the male and female or in the gonadectomized rats. However, the gonadectomized rats of both sexes treated with estrogen showed a highly and statistically significant reduction ($P < 0.001$) in acid and pepsin secretions. When rats were treated with testosterone and progesterone, the gastric secretory activity did not change significantly from control.

The histamine contents in the gastric mucosa were found to be the same in estrogen treated and normal rats. The number of the parietal cells was reduced, and the remaining showed some degree of atrophy. It was concluded that of all the sex hormones used, only estrogen had a significant effect on gastric secretion but the small reduction of parietal cell number could not totally account for the inhibition observed.

Introduction

It has long been recognised that the incidence of ulceration rarely occurs in human pregnancy and that pregnancy often exerts a beneficial effect on the healing of duodenal ulcers in women. In addition, the nausea and vomiting usually occur in early pregnancy. Naturally, the gastric secretory activity during this period has been widely studied. Attempts to relate pregnancy to gastric activities such as de-

layed emptying, diluted acidity etc., were not conclusive. Arzt[1] suggested that the apparent hypochlorhydria was not caused by deficiency of secretion, but resulted from the neutralization of acid by alkaline materials regurgitated from the duodenum. This was disputed by Strauss and Castle[2] because bile was found in only very few of their gastric juice specimens. There have been reports of diminished secretion of pepsin and hydrochloric acid during pregnancy[2,3] whereas in the dog, no such change was found[4]. Pregnant rats also showed a significant reduction in acid secretion[5].

These observation raised the question of the cause of the reduced acidity of the gastric contents during pregnancy in women. Since a high blood level of female sex hormones occurs in this state, a link between sex hormones and acid secretion has been sought experimentally and clinically. There are reports that estrogen helps the cure of peptic ulcers in man[6-8] and that this hormones also significantly reduce the acid secretion in response to histamine in cats[9] and in guinea pigs[10]. The experimental work on cats has shown that male animals exhibit significantly higher gastric secretory activity when compared with the female[9], and there was also a report that women secrete less acid than men[11] especially in the second half of the menstrual cycle. Therefore it is likely that sex hormones may have a physiological effect on gastric acid secretion in both male and female animals.

In 1954, Shay *et al.*[12] described a simple method for collecting gastric juice in the rat stomach by ligating the pylorus and esophagus (Shay rats). This model has frequently been used for studying the effect of drugs upon gastric secretion. However, there have not previously been studies on the effect of sex hormones upon acid and proteolytic activity of gastric juice in Shay rats. The present investigation was performed in an effort to find the mechanism underlying the effect of sex hormones on pepsin and acid secretion induced by pentagastrin in Shay rats of both sexes.

Materials and Methods

Experimental animals

Adult Fisher rats of both sexes weighing 150-200 g were subjected to bilateral gonadectomy under light ether anesthesia. The female rats were ovariectomized by making lateral incisions above the hip. A ligature was placed below the ovary at the top of the fallopian tube, tied and then the ovary removed. Orchidectomy in the male rat was done through a midline incision at a point above the urinary bladder. Ligature were tied above the testes and the testes removed. The completeness of the ovariectomy and orchidectomy was subsequently determined by vaginal smear and the atrophy of the seminal vesicles, respectively. Five days after gonadectomy, the rats were treated with sex hormones in oil suspension for 1 to 14 days. The animals were divided into groups as follows:

i) Normal sham operated, non-gonadectomized rat receiving corn oil served as control.

ii) Ovariectomized (or orchidectomized) rats not treated with sex steroids.

iii) Ovariectomized (or orchidectomized) rats treated with sex steroids in oil suspension.

Sex hormones administration

Gonadectomized rats, both male and female, were given estrogen (Progynon, 1 ml contains 10 mg of estradiol valerate, Schering Co. Ltd.) intramuscularly at physiological dose of 3 μg/100 g rat or at pharmacological dose of 80 μg/100 g rat daily for 1 to 14 days. The same procedure was used in other groups of rats given progesterone (Proluton Depot., Schering Co.) intramuscularly in a daily dose of 2.7 mg/100 g rat or testosterone (Testoviron, Schering Co.) intramuscularly in a dose of 1.3 mg/100 g rat daily. The dilution for injection of the hormones was made with corn oil.

Gastric content analysis

All the animals were tested for gastric acid secretory response to acid secretagogue, pentagastrin (Calbiochem Co.) at the dose of 130 μg/ 100 g rat. The rats were fasted for 48 hours but water was provided *ad libitum*. Under ether anesthesia, the stomach was prepared for the collection of gastric juice according to Shay's method[12]. A longitudinal incision was made about 2 cm below the xiphoid process and the pylorus was ligated. Care was taken not to damage the blood vessels or to disturb the stomach. By means of a polyethylene tube passed orally into the stomach, the content in the stomach was washed free with physiological saline adjusted to pH 5. After the esophagus was ligated at the neck region, the rat was injected intramuscularly with pentagastrin. The animals were then put back into their cages, where they were left for one hour.

All the rats were again operated upon one hour later. The abdomen was opened and the lower part of the esophagus was ligated close to the stomach. After having been isolated with its secretion intact, each stomach was weighed immediately after removal. The stomach was emptied and weighed again. The difference between the weights was used to express the total content of gastric juice. The pH of the gastric juice was measured and the content of hydrochloric acid determined by titration with 0.03 N NaOH to pH 5 using the Beckman automatic titrator. The acid output was calculated as μeq H^+/g stomach/h. The statistical comparison was carried out by the unpaired-t-test using Wang Advanced Programming Calculator.

Proteolytic activities in gastric juice

The gastric juice obtained from either the sham or ovariectomized female rats untreated or treated with estrogen for 7 days in response to gastrin stimulation was also analysed for its proteolytic activity of pepsin.

Total proteolytic activity was determined according to the method described by Miller *et al.*[13] with slight modification. An aliquot (1 ml) of diluted (1:10) gastric juice was incubated with 5 ml of 2% hemoglobin in 0.06 N HCl at 37°C. After 30 min digestion, 10 ml of 5% trichloroacetic acid (TCA) was added and the precipitated protein removed by filtration. The extent of digestion which was the measure

of protease action was determined by mixing 1 ml of filtrate with 3 ml of 0.5 N NaOH and slowly adding 1 ml phenol reagent (diluted 1:3 with water). A blue color, produced by the reaction of phenol with the tyrosine of the protein digestive products in the filtrate, was read with the spectrophotometer against a standard at 660 nm.

A color standard was made with tyrosine dissolved in 0.2 M HCl in place of the TCA filtrate.

An enzyme blank was performed by addition of 1 ml of enzyme aliquot to the TCA before the latter was added to the hemoglobin.

One unit of gastric proteolytic activity in the present study is defined as the amount of enzyme which relases 0.1 mg of tyrosine-like substance from the substrate in 30 min incubation at 37.5°C.

Histamine assay

Histamine content of the rat gastric mucosa was measured by the fluorometric method[14]. The mucosa from the sham-operated and the gonadectomized rats treated with estrogen for 14 days were used in this assay. Rats were anesthetized, the stomach removed and washed free of contents. The gastric mucosa was scraped from the stomach and the accumulated scrapings from two stomachs of the same treatment were weighed (0.3-0.5 g), homogenized with a glass homogenizer and teflon pestle in cold (ice bath) homogenizing medium (10 ml butanol +1.5 ml 0.01 N HCl). The homogenate was centrifuged for 15 min. Four ml of butanol extract was then assayed for histamine by shaking vigorously with 5 ml of 0.1 M borate buffer, pH 10 and saturated with NaCl and butanol. Two ml of the washed butanol fraction was again extracted by shaking with 4 ml *n*-heptane and 2 ml of 0.1 N HCl. One ml of the aqueous layer (HCl) was then assayed for histamine by the addition of 0.2 ml of 1 N NaOH and 0.1 ml of 1% *o*-pthalaldehyde in absolute methanol. Four minutes later, 0.1 ml of 3 N HCl was added and then after another minute the fluorescence developed was measured by the Aminco-Bowman Spectrofluorometer with the activation wavelength at 360 nm and emission wavelength at 450 nm. Thereafter, the histamine content was calculated and expressed in μg/g mucosa.

Histological observation and parietal cell counts

The body area of the stomachs was cut into small pieces, fixed with Bouin's solution, dehydrated and finally embedded in paraffin according to the standard histological technique. The sections were cut 5μ in thickness and stained with hematoxyline and eosin. Random sections from the various blocks of tissues were taken for the parietal cell count using an ocular grid calibated by micrometer and a Leitz Orthoplan microscope.

Results

Gastric acid secretion

Female rats: Fig. 1 and 2 show the gastric acid secretory responses to acid secretagogue, namely, pentagastrin, in normal and ovariectomized female rats, treated

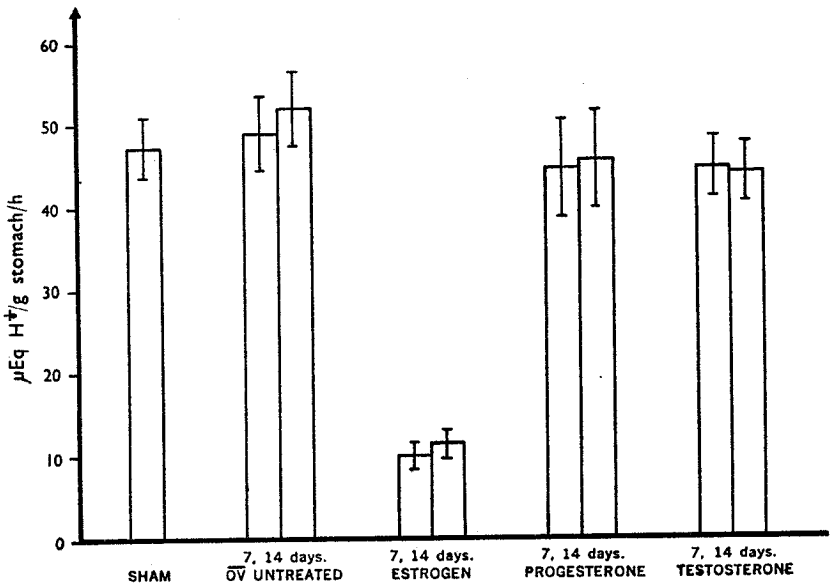

Fig. 1: Rate of gastric acid secretion (μeq H^+/g stomach/h, mean $\pm$ S.E.) in response to intramuscular injection of pentagastrin (130 μg/100 g rat) in ovariectomized ($\overline{OV}$) female rats after treated with various sex hormones (estrogen, 80 μg/100 g rat; progesterone, 2.7 mg/100 g rat; and testosterone, 1.3 mg/100 g rat) for 7 and 14 days. n = 8 in each group.

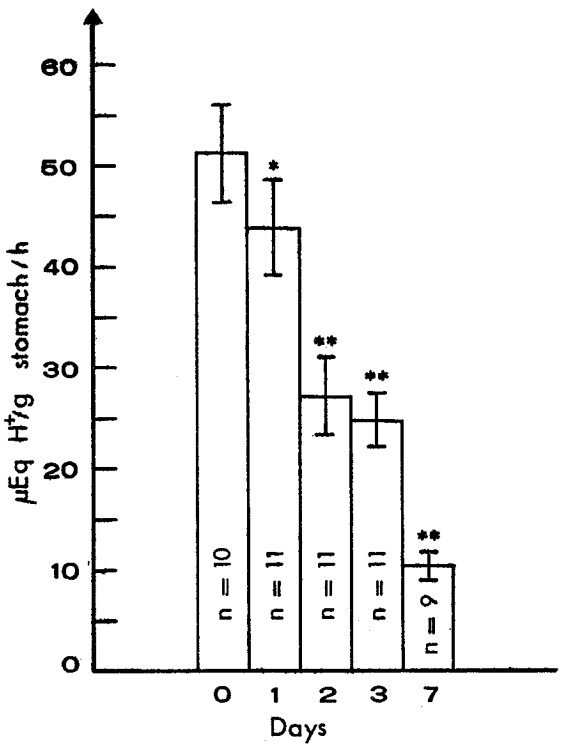

Fig. 2: Rate of gastric acid secretion (μeq H^+/g stomach/h, mean $\pm$ S.E. in response to intramuscular injection of pentagastrin (130 μg/100 g rat) in ovariectomized female rats after treated with estrogen (80 μg/100 g rat) for 1, 2, 3 and 7 days, respectively. *Not significantly different from sham ($P > .05$). **Significantly different from sham ($P < .01$). N = number of rats.

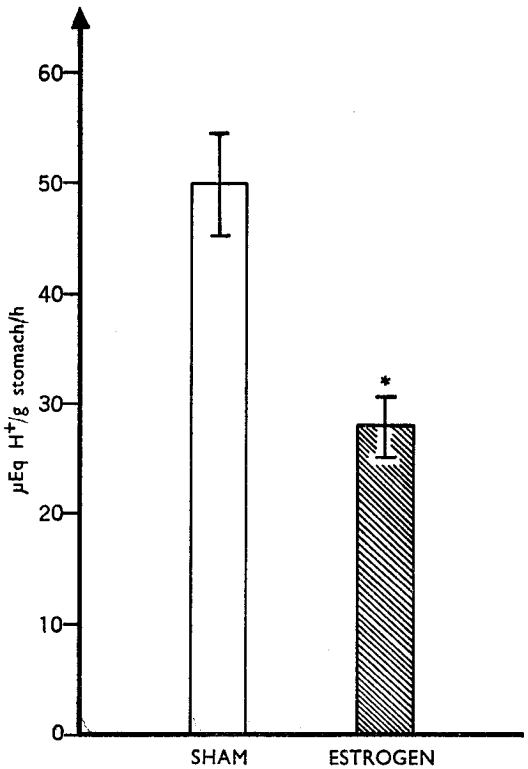

Fig. 3: Rate of gastric acid secretion (mean ± S.E.) in response to intramuscular injection of pentagastrin (130μg/100 g rat) in ovariectomized female rats after treatment with the physiological dose of estrogen (3 μg/100 g rat) for 7 days (n = 10). *Significantly different from sham ($P < .001$). n = 8.

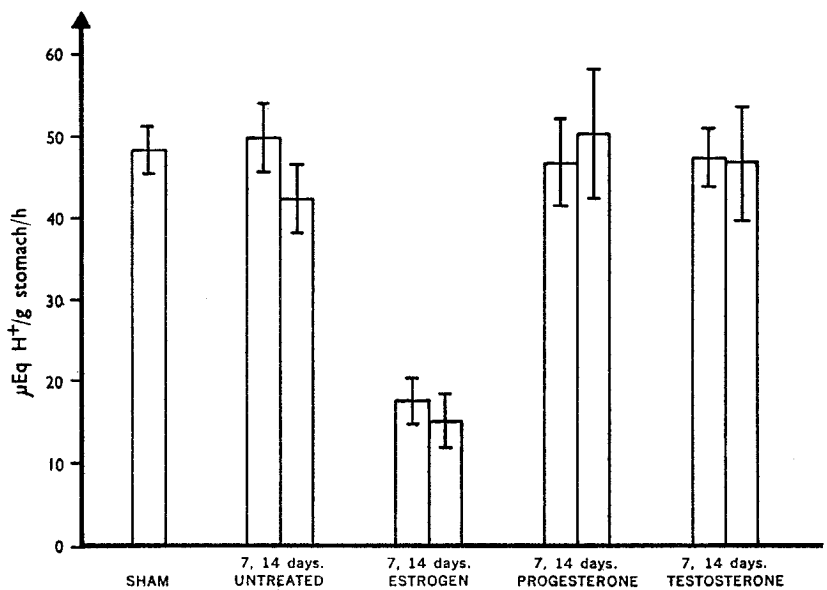

Fig. 4: Rate of gastric acid secretion (mean ± S.E.) in response to intramuscular injection of pentagastrin (130 μg/100 g rat) in orchidectomized male rats after treated with various sex hormones (estrogen, 80 μg/100 g rat; progesterone, 2.7 mg/100 g rat; and testosterone, 1.3 mg/100 g rat) for 7 and 14 days. n = 8.

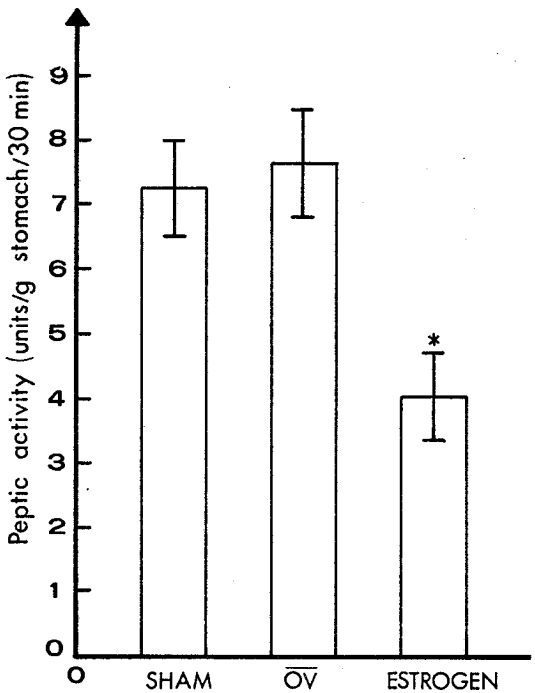

Fig. 5: Total proteolytic activity (units/g stomach/30 min) of gastric juice obtained from sham, ovariectomized female rats ($\overline{OV}$) with and without estrogen (80 μg/100 g rat) treatment in response to intramuscular injection of pentagastrin (130 μg/100 g rat). One unit of gastric proteolytic activity is defined as the amount of enzyme which release 0.1 mg. of tyrosine-like substance from the substrate hemoglobin in 30 min incubation (number of animals, $n = 10$ in each group). *Significantly different from sham ($p < .01$).

and untreated with sex hormones. The sham-operated rats, in all experiments, were used as the controls. The gastric acid secretory response in ovariectomized untreated rats given pentagastrin was not significantly different from the controls ($P > 0.05$). However, ovariectomized rats treated with estrogen (80 μg/100 g rat) from 2 to 14 days (Fig. 1 and 2) show a statistically significant reduction in acid gastric secretion ($P < 0.01$). The acid secretion decreased with the increase in duration of estrogen treatment (Fig. 2) up to 7 days. Additional treatments to 14 days did not further decrease the acid secretion (Fig. 1). Administration of estrogen of physiological dose (5 μg/100 g rat) for 7 days (Fig. 3) also reduced the acid secretory activity from 50 ± 5 to 28.5 ± 2.5 μeq H^+/g stomach/h ($P < 0.001$). In ovariectomized rats treated with progesterone and testosterone (Fig. 1), the gastric acid secretion responses to secretagogue under study did not differ significantly from those in the ovariectomized untreated or sham-operated rats ($P > 0.05$).

Male rats: Bilateral orchidectomized male rats untreated with sex hormones (Fig. 4) did not significantly alter their gastric acid secretory responses to pentagastrin ($P > 0.05$). When the castrated male rats were given estrogen at 80 μg/100 g rats, either for 7 or 14 days, there was a reduction in gastric acid secretion in response to pentagastrin ($P < 0.001$): The treatments of castrated animals with progesterone or testosterone again did not change their secretory response to pentagastrin when compared to the sham operated rats.

Our results show no significant difference in the gastric acid secretory activities between male and female rats. The secretion in response to acid secretagogue is the same in sham-operated rats of both sexes. Both show a highly significant reduction in secretion when estrogens were given, whether at pharmacological dose (Fig. 1, 2, 4) or physiological dose at 3 μg/100 g rat (Fig. 3). Though there seemed to be a greater depression of acid secretion in female than in male rats, the difference was below statistical significance.

Proteolytic activities in gastric juice

Fig. 5 show the gastric pepsinogen secretory activity of ovariectomized female rat, with and without 7 day estrogen (80 μg/100 g rat) treatment. Like its effect upon the HCl secretion, estrogen also effectively reduced the proteolytic activity of the gastric juice (from 7.4 ± 0.77 to 4.12 ± 0.69 units/g stomach/30 min, ($P < 0.01$), whereas, ovariectomy alone did not significantly change this proteolytic activity. It thus indicated that estrogen reduced the general secretory activities of both HCl and pepsinogen in the stomach.

Histamine content in gastric mucosa

The assay of gastric histamine content in normal male and female rats, gonadectomized male and female rats treated with estrogen (80 μg/100 g rat) are shown in Table I. The amount of histamine in the gastric mucosa did not change significantly after the treatment with estrogen ($P > 0.05$) in both male and female rats.

TABLE I: HISTAMINE CONTENT OF NORMAL AND GONADECTOMIZED RATS TREATED WITH ESTROGEN FOR 14 DAYS (n = 7).

Sex	Treatment	Histamine content[a] (μg/g gastric mucosa)	P
Male	Sham	100 ± 6.3	
	Estrogen-treated[b]	105 ± 7.7	> 0.05
Female	Sham	94 ± 2.2	
	Estrogen-treated[b]	96 ± 1.4	> 0.05

[a] Mean ± S.E.
[b] 80 μg/100 g rat

TABLE II: PARIETAL CELL POPULATION IN GLANDULAR AREA OF RAT GASTRIC MUCOSA IN SHAM, OVARIECTOMIZED FEMALE RATS AND OVARIECTOMIZED RATS TREATED WITH ESTROGEN.

Treatment	Cell population (cells/0.3 mm^2)	P
Sham	101 ± 3	
Ovariectomized	98 ± 5	> 0.05[b]
Ovariectomized +	95 ± 4	> 0.05[c]
estrogen-treated[a]		< 0.05[d]

Each figure represents the means of parietal cell counts from 7 random tissue slides taken from 3 animals of the same group.
[a] 80 μg/100 g rat
[b] Not significantly different from sham values.
[c] Not significantly different from ovariectomized untreated values.
[d] Significantly different from sham values.

Parietal cell count in the body area of gastric mucosa

Table II shows the parietal cell count per unit mucosa area (0.3 mm^2) in sham operated, overiectomized untreated and ovariectomized rats treated with estrogen, respectively. It is found that the density of the parietal cells, which are primarily concerned with acid secretion, in ovariectomized untreated rats was not significantly different from that of normal sham rats. But in ovariectomized rats treated with estrogen (80 μg/100 g rat), the number of cells was significantly reduced from 101 ± 3 to 95 ± 4 cells/0.3 mm^2 when compared to the sham rats ($P < 0.05$).

Discussion

The results presented here show that gonadectomy alone does not alter the gastric acid secretory response to pentagastrin in both male or female rats. This

observation contradicts the results of Katz *et al.*[10], who reported that gastric acid secretion decreased after the source of testosterone was removed by orchidectomy for 4 months in the guinea pig. It is likely that the male sex hormone may have a stimulating effect on gastric acid secretion since there was a report that the adolescent male usually secretes more acid than the adolescent female[11]. The above contradiction may be due to the length of time elapsing after gonadectomy before the testing of the acid secretory responses or due to species difference. There may be a gradual decline in the blood concentration of testosterone originating from the testes, and that the decline is insufficient to lower the gastric secretion in our experiment. Therefore, it is not possible to ascertain, from the presently available evidence, whether the testosterone has any stimulating effect and its possible mechanism on the gastric acid secretory activity.

The inhibition of estrogen on gastric acid and pepsin secretions is evident from our results and confirms the previous clinical work[15]. From our findings, however, there was no difference in gastric secretory responses between male and female rats, whether the duration of treatment was 7 or 14 days. However, the secretion seems to be more depressed in female than male rats. Thus, it is similar to the observation of Ojha and Wood[9] who reported a sex difference in secretory responses to estrogen treatment in normal cats and that a higher level of estrogen was required for the inhibitory effect in the castrated male cats.

The results also agreed well with the previous reports that estrogen has a benficial effect in healing peptic ulcers[7]. Parbhoo and Johnston[8] suggested that estrogen controls the symptoms of duodenal ulcers by augmenting the production of mucus in the stomach and not by the inhibition of acid or pepsin secretions. During the course of our investigations, mucus secretion seemed to be normal in the stomachs of estrogen treated rats as observed in our PAS stained gastric mucosa micrographs. However, quantitative measurements of mucus are needed before definite conclusion can be drawn.

The inhibition of acid secretion in response to secretagogue can occur at various steps along the course of the gastric secretory mechanism such as the change in enzymes responsible for the production of acid. Some previous findings capable of explaining the reduced gastric acid secretion after estrogen administration seem to be of value. Sawyer and Everett[16] reported an elevation of serum cholinesterase in castrated rats after estrogen treatment. The role of acetylcholine in the production of acid gastric juice is well known[17], and an increased serum cholinesterase level would be expected to inhibit gastric secretion. However, treatment with estrogen was also effective in depressing gastric secretory activity in gonadectomized rats when gastrin is given. Therefore, the reduction in secretory activity due to the lower level of acetylcholine is untenable because gastrin which normally is released by acetylcholine during the sequence of stimulation[18] could not restore the acid secretion to a normal level.

An increased histaminase concentration was observed in the stomach of pregnant rats[5] but this change in enzyme concentration could not, according to these authors, explain the concurrent inhibition of gastric secretion. However, Murray *et al*[19]. could not confirm the increase of the enzyme histaminase in their experiments.

It is unlikely that the mechanism of inhibition of estrogen is by elevated histamine destruction by enzyme histaminase in the stomach since our results indicated no significant change in histamine content in the gastric mucosa after the rat was treated with estrogen.

It is generally accepted that the secretory capacity of the stomach is proportional to the parietal cell mass[20]. In the present result, the reduction in parietal cell number per unit area of estrogen-treated animal was found to be significantly different from sham ($P < 0.05$) but not significantly different from ovariectomized animal ($P > 0.05$). This was in striking contrast to the reduction in acid secretion of which the estrogen/treated group was very much different from the ovariectomized and sham groups. Concerning the actual number of parietal cells, the difference was vary small and could not account for a marked depression of acid secretion by estrogen. The inhibitory effect would rather be at the reduction of cell capacity to secrete acid. It may be possible that, after estrogen treatment the effective secretory capacity of parietal cells in response to stimulus was reduced probably because of the progressive hypofunction of the cell or cell atrophy. These effects were frequently observed after vagotomy and X-irradiation of the stomach. A marked depression in acid secretion without the significant reduction of parietal cell population after vagotomy was observed[21]. Similarly, after irradiation, the lowering of the secretory function of parietal cells was very pronounced, the same number of parietal cells produced 15 times less acid than in the controls three weeks after irradiation[22]. Therefore, it was of interest to investigate the possibility that the reduced acid secretion after estrogen treatment is due to a change in the function of individual cells such as gastric microsomal enzymes which are responsible for H^+ transport instead of changes in cell number.

Finally, it can be concluded that of all the sex hormones used, only estrogen showed a significant reduction in acid secretion. Though the mechanism of inhibition might involve, to a small extent, the reduction of parietal cells population, the main inhibitory effect of estrogen may be related to the reduction of parietal cell capacity.

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บทคัดย่อ

ในการศึกษาผลของฮอร์โมนต่อการหลั่งของกรดในกระเพาะอาหาร พบว่า 7 วันหลังจากฉีดฮอร์โมนเพศอีสโตรเจน โปรเจสเตอโรนและเทสโทสเตอโรนให้กับหนู (rats) แล้ว มีอีสโตรเจนตัวเดียวเท่านั้นที่มีผลยับยั้งการหลั่งของกรดและน้ำย่อย (pepsin) ในกระเพาะอาหารอย่างเด่นชัด ($P<0.05$) ปริมาณของฮิสตามีนในกระเพาะของหนูเหล่านี้ซึ่งมีความสำคัญต่อการหลั่งกรดไม่ได้เปลี่ยนแปลงไปจากหนูปกติ จากการนับจำนวน parietal cells ซึ่งเป็นเซลล์ที่ทำหน้าที่หลั่งกรดพบว่าจำนวนเซลล์ต่อพื้นที่ลดลงเล็กน้อย แต่จำนวนที่ลดลงไม่มากพอที่จะเป็นสาเหตุสำคัญของการลดการหลั่งกรด