THE EFFECTS AND MECHANISMS OF SEX STEROIDS ON GASTRIC ACID SECRETION

ABSTRACT

Experiments were performed to study the effects and the possible mechanisms of various sex hormones on gastric acid secretion in rats. Adult rats were gonadectomized and treated with the steroids for 1 to 14 days before testing for acid secretion in response to secretagogues (Gastrin, Histamine and Carbacholine) by Shay's method.

There was no significant difference ($P > .05$) in acid secretory activity in gonadectomized rats treated with either progesterone or testosterone but a significant reduction ($P < .001$) was observed in gonadectomized rats treated with estrogen. The possible mechanisms of this inhibitory action of estrogen were thus explored. The change in gastric histamine content was ruled out as a causative agent. Furthermore, by means of paired-feeding and forced-feeding methods, it was found that the anorexia effect of estrogen which might indirectly reduce the gastric motor activity and acid secretion could not satisfactorily explain its action. However, the estrogen treated animals showed significantly high level of plasma glucose. Thus, the disturbance of vagal activity by hyperglycemic effect of estrogen could be a factor responsible for the reduction of the gastric acid secretory activity.

The population of parietal cells which are responsible for the acid secretory activity was counted. The number of cells in estrogen treated animals was found to be insignificantly different ($P > .05$) from those of ovariectomized rats but significantly different ($P < .05$) from those
of sham control. However, the reduction in the number of parietal cells was too small to account for the acid reduction. Further electronmicroscopic studies revealed some non-specific alterations in ultrastructure of parietal cells in estrogen treated animals, especially after stimulation. The activities of two H$^+$-transporting enzymes in gastric microsome (K$^+$-stimulated ATPase and K$^+$-stimulated phosphatase) were significantly depressed (P < .05) by estrogen treatment. Studies of both acid secretory rate and enzyme activities performed in vitro also yielded similar results.

In an acute in vitro experiment, estrogen at concentration 10 µg/ml also depressed the acid secretory rate and the enzyme activities of an isolated normal gastric tissue. Kinetic studies of the enzymes showed that the hormone markedly inhibited the maximum activities of the enzymes whereas the apparent affinities to the substrates and activating ion (K$^+$) were slightly affected.

Therefore, it can be concluded that of all the sex hormones used, only estrogen showed a significant inhibition of acid secretion. The inhibitory mechanism of estrogen in vivo might involve both indirect and direct effects on parietal cells, whereas the in vitro mechanism seemed to involve only direct interaction of estrogen with parietal cells.
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