

markedly the secretion of gastric juice. Inhibition commenced within 45 minutes and lasted for 3 or 4 hours. The percent inhibition during the 3-hour period following the administration of extract is shown in Table I. It is seen that the extracts of urine from duodenectomized and from gastrectomized dogs also inhibited secretion. The extracts were without effect on blood pressure and their inhibitory effects on gastric secretion were quite independent of the course of body (rectal) temperature.

TABLE I.

The inhibition of gastric secretion during the 3 hours following administration of urine extract is expressed as percent of the secretion in control experiments during the same period.

Nature of urine	No. of experiments	% inhibition
Control (no urine given)	9	0
Human, Normal	7	67.5
Dog, Normal	5	63.0
'' Gastrectomized	7	45.9
'' Duodenectomized	4	56.0
Human, Heat-inactivated	3	1.5

Conclusion. An extract can be prepared from the urine of normal dogs which inhibits gastric secretion. The inhibitory principle is still present in the urine after removal of either the stomach or duodenum.

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Hormonal Inhibition of Lactation.*

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In a previous communication¹ it was reported that the daily injection of 100 r.u. of a gonadotropic principle during the first 5 days of the lactation period in the rat caused no inhibition of lactation. Larger dosages and dosages over a longer period of time re-

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¹ Hathaway, I. L., Davis, H. P., Reece, R. P., and Bartlett, J. W., *Proc. Soc. Exp. Biol. and Med.*, 1939, **40**, 214.

duced the rate of growth of the young 22 to 33%. Earlier work^{2, 3, 4} showed that estrogens were less effective in inhibiting lactation in the ovariectomized rat than in one with ovaries intact. These results stimulated interest in attempting to determine whether or not the effectiveness of estrogens in inhibiting lactation could be augmented through simultaneous administration of a gonadotropic principle and, if possible, the route of the inhibitory action.

Fifty-six lactating rats and their litters were used in this study. The number of young in the litters was fixed at 6, and daily weighings were made. The diet fed was the same as that reported in a previous paper.¹ It was decided to use as a criterion for the inhibition of lactation the failure of a litter to gain weight on 3 successive days. Seventeen lactating rats, designated hereafter as Group I, were injected daily with 200 r.u. of a gonadotropic principle from pregnant women's urine (Antuitrin-S†) plus 100 r.u. of an estrogen (Progynon-B†). Group II contained 17 lactating rats that received daily 100 r.u. of Progynon-B. In Group III 5 lactating rats were injected daily with 200 r.u. of Antuitrin-S. Seventeen lactating rats, Group IV, served as controls. When the young of a lactating rat in Group I failed to gain weight on 3 successive days they were removed, the lactating rat was injected with one gamma of colchicine per gram of body weight and sacrificed 9½ hours later. For comparative purposes a lactating rat from each of Groups II and IV, in the same stage of the lactation period as the rat from Group I, was given similar treatment. No experimental lactating rats received more than 10 injections.

The ovaries and pituitaries were removed and weighed and the latter assayed for their lactogen content by injecting the tissue intradermally over the crop glands of common pigeons. Mammary glands were removed and fixed for study.

In Group I, 16 of the 17 litters failed to gain weight on 3 successive days, 4 pups having died before reaching this stage. Five of the 17 litters in Group II failed to gain weight on 3 successive days, one pup having died before reaching this stage. In Groups III and IV none of the litters failed to gain weight. On the 11th day of the lactation period the average weight of the remaining young in the

² Anselmino, K. J., and Hoffman, F., *Zentralbl. f. Gynak.*, 1936, **60**, 501.

³ Reece, R. P., and Turner, C. W., *Mo. Agr. Exp. Sta. Res. Bul.*, 1937, 266.

⁴ Folley, S. J., and Kon, S. K., *Proc. Roy. Soc., B*, 1938, **124**, 476.

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several groups was as follows: Group I, 9.7 g; Group II, 12.8 g; Group III, 17.3 g; and Group IV, 18.8 g.

The ovaries of the rats in Group I (268.4 mg) were significantly heavier than those of the rats in Group II (73.6 mg) and Group IV (69.8 mg) while the pituitary glands from the rats in Group I (14.0 mg) and II (14.2 mg) did not differ significantly yet they were significantly heavier than the pituitary glands of rats in Group IV (9.4 mg).

The pituitary glands of the rats in Group I contained more lactogen than did those of Groups III and IV and nearly the same as did those in Group II. Pituitary glands from rats in Group II contained more lactogen than did the glands from rats in Groups III and IV while the lactogen content of the glands of rats in Groups III and IV was similar. The results are summarized in Table I.

Microscopic examination of the mammary glands of animals in Groups IV (controls) and III (Antuitrin-S) revealed similar conditions. Mitotic figures were rarely observed and the lumina of the alveoli were distended with milk. Epithelial cells exhibiting arrested mitosis were occasionally found in the mammary gland of rats in

TABLE I.
Hormonal Inhibition of Lactation.

Group*	No. of comparisons	Avg body wt following parturition, g	Avg wt of remaining young on following day of lactation cycle, g			Avg wt of pituitary, mg	Avg. No B.U.† per pituitary	Ovarian wt, mg
			1	6	11			
I }	17	236	5.6	8.2	9.7	14.0	27	268.4
IV }		242	5.6	11.0	18.8	9.4	23	69.8
I }	17	236	5.6	8.2	9.7	14.0	26	268.4
II }		231	5.7	10.4	12.8	14.2	27	73.6
II }	17	231	5.7	10.4	12.8	14.2	28	73.6
IV }		242	5.6	11.0	18.8	9.4	20	69.8
III }	5	245	5.7	10.1	17.3	11.7	17	220.1
IV }		243	5.5	10.9	19.8	10.6	18	70.6
III }	5	245	5.7	10.1	17.3	11.7	15	220.1
I }		242	5.6	8.2	8.5	16.0	23	288.1
III }	5	245	5.7	10.1	17.3	11.7	19	220.1
II }		244	5.8	11.0	12.2	16.1	28	72.6

*Group I received daily 200 r.u. of Antuitrin-S plus 100 r.u. of Progynon-B; Group II injected daily with 100 r.u. of Progynon-B; Group III injected daily with 200 r.u. of Antuitrin-S; and Group IV were the controls.

†B.U.—Bird units.

} Pituitary glands assayed in the same group of birds.

Group II (Progynon-B). The lumina of the alveoli were filled with milk. In marked contrast many mitotic figures were observed in the gland parenchyma of rats in Group I. The lumina of the alveoli usually contained very little milk, and in a few cases they were entirely absent.

Conclusions. The effectiveness of estrogens in inhibiting lactation in the rat can be increased through simultaneous administration of a gonadotropic principle from pregnant women's urine. Many cells in mitoses are observed in the mammary gland parenchyma of rats so treated. It is believed that this is the mechanism of the inhibitory action of these hormones on lactation. That is, one would not expect a proliferating gland to secrete as efficiently as a non-proliferating gland. The administration of an estrogen plus a gonadotropic principle to lactating rats augments the lactogen content of the pituitary gland.

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Influence of Heredity and Environment upon Number of Tumor Nodules Occurring in Lungs of Mice.

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In previous publications it has been shown that the occurrence of lung tumors in mice is influenced by environment and also by heredity¹ and that probably, in the strains studied, a single dominant Mendelian factor is concerned.^{2, 3} We emphasize, however, that our conception as to number of genes may be affected by further study of environmental conditions. The present paper deals with the influence of heredity and environment upon degree of susceptibility among mice with tumor.

The measure used, in this instance, to determine degree of susceptibility, is the number of tumor nodules found in each individual. Although the counts covered only those nodules visible on the surface of the lung and obviously may not represent the total number possessed by the mouse, they are sufficiently accurate for our purpose. In Table I, comparisons are made of the number of lung

¹ Lynch, C. J., *J. Exp. Med.*, 1927, **46**, 917; 1931, **54**, 747; *Occas. Publ. Am. Assn. Ad. Sci.*, 1937, **4**, 22.

² Bittner, J. J., *Pub. Health Rep.*, 1938, **50**, 2197.

³ Lynch, C. J., *Third Internat. Canc. Cong.*, in press.