considerable portion of the activity which is not due to esterase since it is not inhibited by Prostigmin. Furthermore, this fraction of the activity is dependent upon the integrity of the corpuscles since it is not present after laking the blood. The greater activity of unlaked blood is not due to the presence of Ringer's solution since it is not a constant value as would be expected if this were true. These results indicate that in turtle blood some of the hydrolytic activity towards acetylcholine may be due to corpuscular surface catalysis. If one is to assume enzymatic hydrolysis of acetylcholine it would appear essential to demonstrate, as a control, that it is completely inhibited by physostigmine or Prostigmin.

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Further Evidence for a Mammogenic Factor in the Rat Hypophysis.

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Gomez, Turner, and Reece[1] reported that the mammary glands of hypophysectomized male guinea pigs could be developed by the implantation of the pituitaries of estrogen-treated male rats but that no mammary growth resulted from implantation of normal male rat pituitaries. Gomez and Turner[2] reported a confirmation and extension of these results. The existence of the "mammogenic factor" described by the aforementioned authors seems to have been demonstrated in pregnant cattle pituitaries when tested on castrated rabbits[3] and rats[3] and normal mice.[4, 5] Nelson[6] reported recently

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2 Gomez, E. T., and Turner, C. W., *Mo. Agr. Exp. Sta. Res. Bul.*, 1937, 259.

3 Gomez, E. T., and Turner, C. W., PROC. SOC. EXP. BIOL. AND MED., 1938, **37**, 607.

4 Lewis, A. A., and Turner, C. W., PROC. SOC. EXP. BIOL. AND MED., 1938, **39**, 435.

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6 Nelson, W. O., *Anat. Rec.*, 1938, **72**, 117 (Suppl.).

that the mammary development of hypophysectomized immature female rats, implanted with pituitaries from estrogen-injected rats of either sex, did not exceed or even equal that which obtains with the implantation of normal pituitaries. He also states that he failed to confirm the claim for a specific "mammogenic hormone". Since the existence of a hypophyseal "mammogenic factor" seems in question, an attempt was made to determine whether such a factor was present in rat hypophyses, when tested on hypophysectomized castrated rats. The results are reported herewith.

Sexually immature female rats of uniform age and weight were castrated and hypophysectomized. Five days later, these rats received 11 daily implants of one hypophysis from normal and estrogen-treated rats of both sexes as follows: 3 received implants from adult females, 2 received implants from adult males, 5 received implants from estrogen-treated females (20 R.U. Progynon-B for 10 days), and 1 received implants from similarly estrogen-treated males. Five castrated hypophysectomized immature females were the controls. During the treatment daily body weighings were made and at autopsy 12 days later, the uteri, adrenals, and thyroids were removed and weighed. Mammary glands were removed and studied as whole mounts. No pituitary fragments were found in the hypophysectomized rats.

The mammary glands of the 5 animals that received no treatment following hypophysectomy were involuted, and consisted mainly of a naked duct system. The mammary glands of all the 11 animals receiving pituitary glands showed numerous and enlarged end-buds, indicating that growth had taken place. The mammary glands of rats, implanted with pituitary glands from estrogen-treated donors, however, could not be distinguished from those of rats implanted with pituitaries from normal rats. Unfortunately, Nelson[6] did not mention any comparison of the mammary glands of hypophysectomized rats receiving pituitary implants with those receiving no implants, otherwise, it is probable he would have been able to observe the direct effect of the hypophysis on the mammary gland.

The gains made in body weight of the rats receiving the two kinds of pituitary tissue contrasted markedly (Table I). There was an average gain of 17 g in body weight in the recipients of the normal pituitaries but there was an average gain of only 3 g in those receiving pituitaries from estrogen-treated donors. The amount of hypophyseal tissue implanted in the latter group exceeded that in the former because of the pituitary hypertrophy which occurs with estrogen injections. The control rats failed to gain in body weight.

TABLE I.

Group	No. of rats	Body wt, gm at beginning	Body wt, gm at autopsy	Amount of hypophyseal tissue given, mg	Adrenal wt, mg	Thyroid wt, mg	Uterine wt, mg
Controls	5	65	63	—	9.5	4.5	19.5
Injected with normal rat pituitaries	5	67	84	119	13.7	6.9	25.6
Injected with pituitaries from estrogen-treated rats	6	68	71	173	12.7	7.0	24.5

While it has been known for some time that adequate doses of estrogens will inhibit growth in the rat,[7] the results here indicate that the "hypophyseal growth factor" had been depleted from the estrogen-treated animals. This was determined by direct assay in hypophysectomized animals. The thyroid and adrenal weights of the 2 groups of rats receiving implants were not significantly different from each other but were heavier than those of the controls.

Conclusion. It is possible to demonstrate an "hypophyseal factor" in rats of both sexes which is capable of inducing growth of the mammary glands in castrated hypophysectomized immature female rats. The administration of estrogens as indicated above, does not influence this "mammogenic factor" when tested in hypophysectomized rats. The estrogen treatment was sufficient to lower the growth-stimulating power of the hypophyses when tested by direct assay.

[7] Spencer, J., D'Amour, F. E., and Gustavson, R. G., *Am. J. Anat.*, 1932, **50**, 129.