TABLE IV.
Neutralizing Antibody Titers in Monkeys Injected with Poliomyelitis Virus Combined with Paraffin Oil and Arlacel A. Dilution of sera before adding equal volumes of virus dilution.

Rhesus No.	Before immunizing inj.	4 wk after 1st inj.	8 wk after 2nd inj.
17	0†	4.1	3.6
18	0	3.3	3.9
19	0	2.4	3.9
20*	2.5	4.6	3.9

* Inapparent infection following intracerebral inoculation.
† Undiluted serum did not neutralize the virus.

bacilli and our own in guinea pigs using either species of acid-fast bacilli(8). In the present experiment, the *Myco. butyricum* was used since it may be a slightly more effective potentiating agent(5,9) than tubercle bacillus. A relatively small amount was injected, 0.1 mg per monkey with the hope of diminishing its untoward effect.

In the present experiments, the "booster" injections were of antigen in saline. With rabies virus high antibody titers were obtained by one injection of antigen in water-in-oil emulsion followed by a "booster" injection of antigen in saline(4). Following an injection of a bacterial antigen in water-in-oil emulsion, the injection of antigen in saline was as effective a "booster" as antigen emulsified in paraffin oil(5).

Conclusions. 1. The formation of neutralizing antibodies in rhesus monkeys is enhanced when the antigen, spinal cord containing poliomyelitis virus, is incorporated into a water-in-oil emulsion. 2. The addition of a small amount of killed *Myco. butyricum* to the water-in-oil emulsion further augments antibody formation. Some of the monkeys, however, develop fatal allergic encephalitis. 3. By repeating the injections of antigen without these adjuvants, antibody titers can be produced which are equal to those found after the injection of antigen in water-in-oil emulsion, but not as high as in monkeys receiving the combination of antigen, paraffin oil and killed *Myco. butyricum.*

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Lack of Effect of Lactogenic Hormone on Mammary Adenocarcinoma in Mice.* (17965)

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Many clinical observations have indicated that the growth of mammary cancer in women is accelerated by pregnancy and lactation (1,2). The effect of lactation alone is not known, although Geschickter(3) believes that it does not influence the growth rate of infiltrating cancer of the breast. Pregnancy stimulation is usually ascribed to the high estrogen titre, a situation which does not apply during lactation(3).

In mice, estrogens exert a carcinogenic effect on the mammary gland(4), but the estrogen preponderance of pregnancy ap-

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TABLE I.
Effect of Pure Pituitary Lactogenic Hormone and Castration on Mammary Adenocarcinoma. Dissected tumor wt in g.

Saline-injected		Hormone-treated	
Intact	Castrate	Intact	Castrate
.16	1.45	2.47	.27
1.52	.15	1.10	.48
.66	.24	2.45	1.22
1.19	2.11	.41	.05
1.26	.03	.40	1.71
1.82	2.86	.03	1.25
1.82		.16	.49
.02		.31	
1.06 ± 0.24*	1.14 ± 0.48	0.92 ± 0.35	0.78 ± 0.22

*Mean value ± standard error.

parently does not accelerate the tumor growth(5). The influence of lactation, with its high lactogenic titre, has not been established, although it has been shown that a crude prolactin preparation did not modify the growth rate of these tumors(6). With the recent availability of homogeneous pituitary lactogenic hormone, it was thought worth while to test this substance for its possible effect upon the growth of mammary adenocarcinoma in mice.

Procedure. Virgin female mice of the C3H strain, 5 to 6 months old, were used. To obviate any ovarian influence half the group was oophorectomized. After a 7 week recovery period, uniform fragments of a transplantable C3H strain mammary adenocarcinoma were implanted by trocar in the dorsal subcutaneous tissues. Eight days later subcutaneous injections were begun. Half of each group, castrated and intact, received two 0.1 cc injections daily, each containing 1 mg of purified lactogenic hormone assaying 30 I.U. per mg(7). The control groups received equal volumes of normal saline. Periodic recordings were made of body weights and tumor diameters. Daily vaginal smears of the hormone-treated mice showed anestrous periods of 12 to 15 days occurred regularly throughout the experiment, thus demonstrating the continued potency of the hormone. After 41 days of injection the experiment was terminated, and the tumors were dissected free and weighed. Necropsy revealed no gross visceral metastases. Of the original 40 mice in the experiment, eight were disqualified for developing spontaneous tumors, and three died of intercurrent disease.

Results. The weights of the dissected tumors are shown in Table I. All tumors had grown considerably since transplantation, although there was great variation between those of individual animals. The tumors of the hormone-treated animals were slightly smaller than those of the controls, but the difference is not statistically significant.

Since neither the hormone nor castration significantly altered the final tumor weight it is interesting to combine the various groups into the categories shown in Table II and note the similarity of results with these larger groups. The hormone did not affect body weight gain.

Conclusion. Homogeneous pituitary lactogenic hormone (2 mg injected daily for 41 days) did not significantly modify the growth rate of a transplanted mammary adenocarcinoma in C3H mice. Castration after sexual maturity did not alter the growth rate of these tumors.

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TABLE II.
Combinations of Original Groups to Show Effect of Lactogenic Hormone and Castration on Tumor Growth and Body Weight Gain. Mean values in g.

Group	No. in groups	Body-wt increment	Tumor wt
All saline-treated (castrated and intact)	14	2.6	1.09
All hormone-treated (castrated and intact)	15	2.5	.85
All intacts (control and hormone-treated)	16	3.4	.98
All castrates (control and hormone-treated)	15	1.3	.95