lated with measles virus, either from tissue culture fluids or clinical materials, but were not observed in tubes inoculated with mixtures of measles virus and measles-immune sera (Fig. 2). Within 7-14 days, necrosis was virtually complete even when dilute inocula were employed. Syncytial formation was almost completely suppressed by the addition of freshly prepared glutamine to the medium. The latter effect is similar to that reported in Hep-2 cultures infected with measles virus to which glutamine had been added(7).

Eosinophilic intranuclear and intracytoplasmic inclusions have been observed as early as 3 days following inoculation of large quantities of virus. Complement fixing antigen (1:8) was detected on the 4th-5th day in tissue culture fluids harvested from roller tube cultures containing 2.0 ml of medium. The average infectious titer per 0.1 ml of tissue culture fluid was 10^{5} TCD_{50}. Twenty-one serial passages of measles virus have been made in stable amnion cultures.

The relative ease not only in the preparation of stable human amnion cultures but also in the early recognition of specific cytopathogenic effects suggests that these cells be used in preference to primary trypsinized human amnion cultures for measles virus studies. In our hands, these cells are preferable to either monkey kidney or Hep-2 cells in performing serum neutralization tests. They have been used also in the preparation of standard antigens for the complement-fixation test. Further work would be required to determine their general usefulness in the isolation of measles from clinical materials.

Summary. Cytopathogenic changes induced by measles virus propagated in a stable line of human amnion cells is described. The prominent change is necrosis in contrast to formation of "giant multinuclear cells" occurring in primary human amnion cultures infected with measles virus. Preliminary studies indicate the general utility of this line of cells for investigations on measles virus.

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Effect of Reserpine on Prolactin Content of Rabbit Pituitary.* (23866)

JOSEPH MEITES†
Department of Physiology and Pharmacology, Michigan State University, East Lansing

A single intravenous injection of reserpine into mature female rabbits induced a limited degree of lactation after prior treatment with estrogens or at the end of pseudopregnancy (1-3). Administration of reserpine(3) and chlorpromazine(4-7) have also been reported to occasionally initiate lactation in women patients. These results, as well as the finding that reserpine may prolong luteal function in the rat(8), suggests that this drug may stimulate production and release of prolactin from the pituitary. The present experiment demonstrates that reserpine can increase the prolactin content of the pituitary of rabbits.

Methods and results. Twenty-two mature female rabbits, predominantly New Zealand White in origin, were divided into 4 groups of 5 to 6 each. The rabbits averaged 6-9 pounds in body weight and were fed a standard rabbit diet. Treatment of the 4 groups was as follows: 1, controls; 2, a single intravenous

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TABLE I. Effects of Reserpine and/or Estradiol on Prolactin Content of Pituitary.

Group & total No. of rabbits	Treatment	Avg pit. wt, mg	Avg pigeon units/pit.	Avg pigeon units/mg pit.
(1) 5	Controls	30.4	0.37 (.016)§	.012 (.0005)§
(2) 5	Reserpine*	31.7	2.00 (.090)	.060 (.0027)
(3) 6	Estradiol†	38.4	4.75 (.214)	.123 (.0055)
(4) 6	Estradiol & reserpine‡	38.9	6.75 (.304)	.170 (.0076)

* Reserpine inj. once and rabbits killed 1 wk later.
† Estradiol inj. for 10 days and rabbits killed 1 wk after last inj.
‡ Estradiol inj. for 10 days followed by 1 inj. of reserpine, and rabbits killed 1 wk later.
§ International Units [1 I.U. = 22.2 pigeon units(9)].

injection of 1 mg reserpine/kilo body weight and killed 1 week later; 3, 0.2 mg estradiol daily for 10 days and killed 1 week later; 4, 0.2 mg estradiol daily for 10 days, followed by a single intravenous injection of 1 mg reserpine/kilo body weight on the 10th day, and killed 1 week later. The pituitary from each rabbit was removed, weighed to the nearest 0.1 mg and combined from each group for assay. They were macerated into a paste with a small agate mortar and pestle, taken up as a fine suspension in distilled water, and assayed in mature White Carneau pigeons by the sensitive intradermal technic(9). The 5 pituitaries from the control rabbits were directly compared in the same 10 pigeons against the 5 pituitaries from the reserpine-injected rabbits by injecting over both sides of each crop sac. Since estrogens increase pituitary prolactin content(10), the equivalent of only 2 pituitaries from the estradiol-injected rabbits was directly compared in 10 pigeons against the equivalent of 2 pituitaries from the rabbits given both estradiol and reserpine. Representative mammary glands from each of the rabbits were examined for gross and microscopic evidence of growth and lactation.

Results. A single injection of reserpine (group 1) increased pituitary prolactin content at least 5-fold as compared to the controls, but did not initiate lactation (Table I). There was no evidence that reserpine induced a significant degree of mammary growth. Injections of estradiol (group 2) increased the prolactin content of the pituitary at least 10-fold as compared to the controls. Although some growth of the mammary duct system occurred in these animals, milk secretion was not initiated. The greatest increase in pituitary prolactin content was elicited when estradiol was followed by reserpine (group 4). The mammary glands of all these rabbits showed considerable lobule-alveolar development and a limited but definite degree of lactation. This was described previously(1). In most of the rabbits given reserpine, drowsiness or sleep was induced for several hours after injection of the drug.

Discussion. These results show that a single intravenous injection of a relatively large dose of reserpine elicits a pronounced increase in the prolactin content of the pituitary of mature female rabbits. A single injection of reserpine alone, however, failed to initiate lactation, presumably because the mammary glands remained rudimentary in structure. Treatment with estradiol alone for 10 days produced a greater increase in pituitary prolactin than reserpine, but mammary growth was only slightly stimulated and lactation was not initiated. Long-term treatment of rabbits with estrogens has induced extensive mammary growth, increases in pituitary prolactin content and lactation(11,12). When reserpine was given following injections of estradiol for 10 days, there was considerable lobule-alveolar development, an increase in pituitary prolactin and milk secretion. This suggests that the presence of adequate mammary tissue is essential for the prolactational action of reserpine in the rabbit.

In this laboratory, injections of reserpine or chlorpromazine failed to induce lactation when injected into mature female rats for 20 days, and did not increase milk production when injected into parturient lactating rats or into goats during the declining phase of lactation (unpublished results). Other investigators have independently observed that these drugs do not induce lactation in rats or guinea pigs, nor increase milk yields in goats(3,13). We have injected both drugs into mature

White Carneau pigeons for 20 days without eliciting crop milk secretion. It appears therefore, that a species difference exists in the ability of these drugs to influence prolactin secretion and perhaps other hormones which regulate mammary growth and lactation.

Summary. 1. A single intravenous injection of 1 mg reserpine kilo body weight into 5 mature female rabbits, followed by sacrifice 1 week later, resulted in at least a 5-fold increase in pituitary prolactin content over 5 controls. There was no evidence that reserpine induced mammary growth or lactation. 2. Subcutaneous injections of 0.2 mg estradiol daily for 10 days into 6 rabbits elicited 1 week later at least a 10-fold increase in pituitary prolactin content, a slight growth of the mammary duct system and no milk secretion. 3. When estradiol treatment was followed by a single intravenous injection of reserpine (above dose) and the rabbits killed 1 week later, pituitary prolactin content was at least 16 times greater than in controls. This was accompanied by a considerable degree of lobule-alveolar development and a limited amount of milk secretion in all 6 animals.

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Potentiation of Barbiturate Hypnosis by Certain Uncoupling Agents.*†‡ (23867)

K. F. KILLAM, T. M. BRODY AND J. A. BAIN
Departments of Pharmacology, University of California at Los Angeles, University of Michigan, Ann Arbor, Emory University, Ga.

The hypothesis that barbiturates produce their depressant effects by uncoupling oxidative phosphorylation has been proposed(1-4). An objection to this premise is the observation that other uncoupling agents are not hypnotics or anesthetics in the intact animal. The possibility that many uncoupling agents fail to depress *in vivo* because of limited penetrability into the central nervous system has been discussed(4). While most uncoupling agents possess little or no hypnotic properties *per se,* the present study will demonstrate that they potentiate hypnosis induced by the barbiturates. The uncoupling agents studied were: 2,4-dinitrophenol, (DNP) 2,4-dichlorophenoxyacetic acid (2,4-D) and chlorotetracycline (Aureomycin).

Methods. Sleeping time was measured in white mice of the Harlan strain of both sexes weighing 18 to 25 g. The test drugs or saline solutions were made up in such a manner as

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