in normal treated rats. Similarly, the serum of these animals also contained low titres of gonad-stimulating material, comparable to treated normal rats, with estrin producing ovaries. Nine hypophysectomized spayed animals, identically treated, gave high serum titres in spite of estrin injections (Table).

5. *Conclusion.* It appears that the hypophysis under the influence of estrin, whether supplied by injections or by overstimulated ovaries, actually inhibits possibly through the intermediary of some other endocrine gland the action of gonadotropic hormone.

8684 C

Transmission of Nutritional Muscular Dystrophy to Rabbits *in Utero.*

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When young rabbits are placed upon muscle-dystrophy producing Diet 11, the majority die within a period of several months.[1] Rarely the disease runs a protracted course; the animals may survive for a year, only to succumb eventually with characteristic degeneration of the skeletal muscles. One such adult dystrophic rabbit successfully completed gestation and gave birth to living young. This exceptional event, has permitted us to observe that nutritional muscular dystrophy may be passed on from mother to offspring.

Rabbit 241 was born in the laboratory on Jan. 15, 1935. On Feb. 19th, it was weaned and given Diet 11.* A biopsy from the thigh muscles was taken on Jan. 6th, 1936, when the animal had attained a weight of 2.4 kilos. A few necrotic fibers were found in each low power field. The muscle creatin was 540 mg.—within the normal range. Two subsequent biopsies on Nov. 19th and Dec. 11th showed little progression of the lesions.

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[1] Goettsch, M., and Pappenheimer, A. M., *J. Exp. Med.*, 1931, **54**, 167.

* Diet 11.

Rolled oats (Quaker)	355 parts
Wheat bran (Pillsbury)	180 "
Casein (Merck technical)	75 "
Lard	80 "
Cod liver oil (Mead-Johnson)	10 "
NaCl	10 "
$CaCO_3$	15 "

On Jan. 7th, 1936, she was mated with a stock breeding male, which was known to have sired several normal litters, and which had been maintained on a stock diet of grains and alfalfa. On Feb. 8th, Rabbit 241 gave birth to a litter of 2. The young seemed somewhat scrawny, but they were not carefully examined for fear of disturbing the mother. The following morning, the newborn rabbits were found dead.

Their skeletal muscles at autopsy were strikingly pale. No other pathological change was recorded.

Histologically, sections taken from various muscles all showed extreme lesions (Fig. 1). Many of the fibers—in some field al-

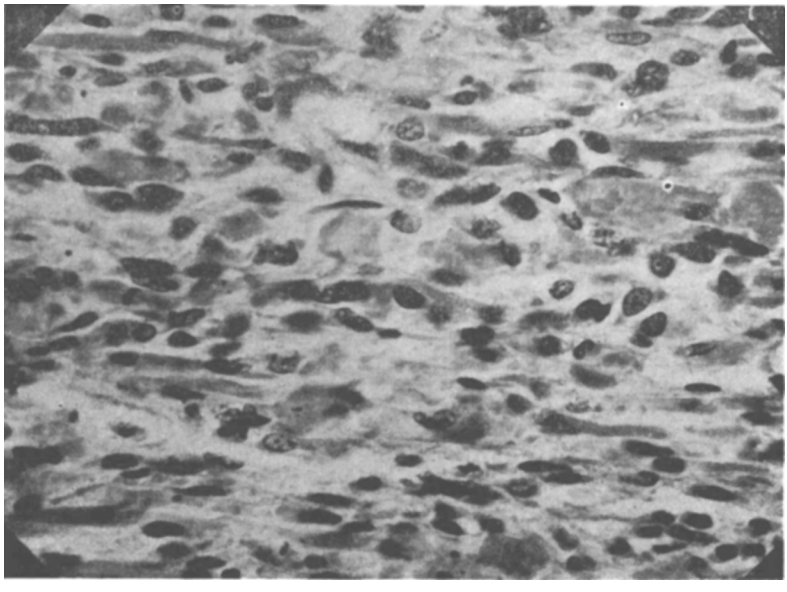

FIG. 1.

Rabbit 241 A. Section of thigh muscle showing necrotic fibers, myocytes and histiocytes. H-E. x 460.

most 50%—were undergoing hyaline necrosis, with segmentation and rupture. They were widely separated by an œdematous stroma in which were great numbers of elongate fusiform cells with vesicular nuclei and basophilic cytoplasm. These were identified as myocytes. There were also many rounded histiocytes, and a few mature lymphocytes. The lesions were thus identical with those found in certain stages of adult nutritional muscular dystrophy.[1] As in the post-natal disease, the cardiac muscle was not affected.

Since these young rabbits lived but a day, these extreme muscular

lesions must have developed *in utero*. The further history of the mother is interesting. Following the birth of the young, she lost weight rapidly—about one kilo in 17 days. Towards the end, she ate but little, and there was great prostration with rapid wasting of the muscles. She was killed on Feb. 26th. At autopsy, the subcutaneous and intraäbdominal fat was still preserved; the muscles were pale, streaky and devoid of contractility. The uterus was involuted. There were no incidental lesions of significance.

Microscopically, blocks taken from numerous muscles showed an advanced stage of dystrophy, with many recently necrotic fibers, as well as much interstitial fibrosis and fat replacement (Fig. 2).

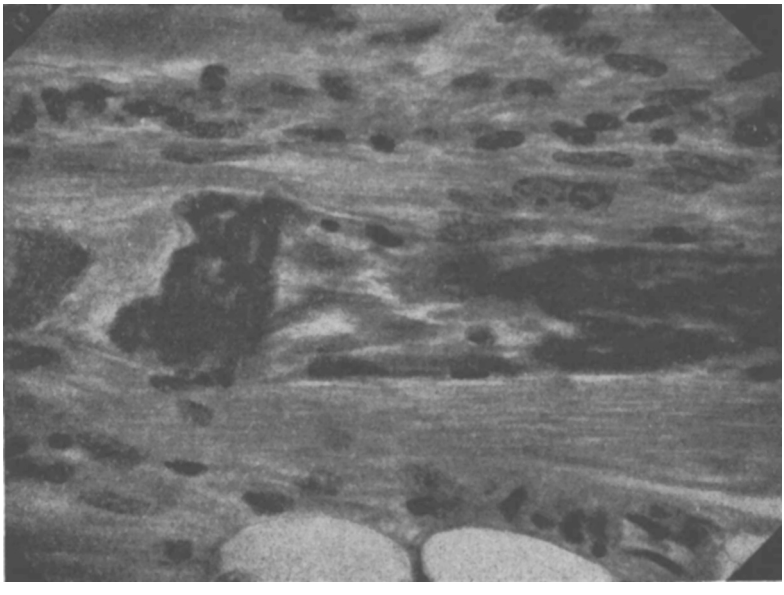

FIG. 2.
Rabbit 241. Mother. Section of adductor muscle, showing necrosis of fibers, multiplication of myocytes and histiocytes. H-E. x 460.

The precise factors concerned in the production of nutritional muscular dystrophy of herbivora have not yet been clearly defined. It is therefore not justifiable at the present time to speak of this condition as a deficiency disease. But evidence is accumulating that the addition of vegetable oils (soybean oil,[2] cotton seed oil[3]) to the experimental diet exercises a protective effect. In our lab-

[2] Goettsch, M., and Pappenheimer, A. M., *Proc. Meetings Am. Soc. Exp. Biol.*, Washington, 1936, March 28 (In press).

[3] Madsen, L., *J. Nutrition*. Personal communication.

oratory we have succeeded in raising animals to maturity and in keeping their offspring in apparent health by replacing the lard in Diet 11 by soybean oil. The protective factor must therefore pass to the fetus, because in its absence, the offspring are born with advanced muscular lesions.

8685 C

Effects of Synthetic Androsterone on Accessory Reproductive Organs of the Male Ground Squirrel.*

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Accessory organs of immature, non-rutting adult and castrated male ground squirrels (Citellus tridecemlineatus) have been shown to respond greatly to injections of male hormone prepared from bull testes and human male urine.[1] Synthetic androsterone,‡ reported to stimulate male accessories in castrated birds and mammals,[2-6] was administered to annual-breeding male ground squirrels in order to determine its effects on accessory reproductive organs during the season when testis hormone is released in minimal quantities, if at all, by gonads in normal males of this species.

The low state of reproductive organs in 25 normal males was evaluated by the following methods without the actual removal of tissues, in order to evade possible compensatory hypertrophy: (1) inspection of scrotal skin for pigmentation, (2) manual palpation of bulbar gland, (3) examination of epididymis and ductus deferens at exploratory laparotomy, and (4) measurement of the testis

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† Much of the investigation reported in this paper was done in the Department of Zoology of the University of Chicago. The writer wishes to thank Dr. Carl R. Moore, who provided research facilities in his laboratory during the summer of 1935.

1 Wells, L. J., *Anat. Rec.*, 1934, **60** (Suppl.), 53; 1935, **62**, 409.

‡ Prepared from cholesterol. Kindly furnished by Drs. Ruzicka and C. C. Haskell and the CIBA Company.

2 Ruzicka, L., and Tschopp, E., *Schweiz. Med. Woch. Schr.*, 1934, **64**, 1118.

3 Callow, R. K., and Parkes, A. S., *Bioch. J.*, 1935, **29**, 1414.

4 Callow, R. K., and Deansely, R., *Bioch. J.*, 1935, **29**, 1424.

5 Korenchevsky, V., and Dennison, M., *Bioch. J.*, 1935, **29**, 1720.

6 Tschopp, E., *Klin. Woch.*, 1935, **14**, 1064.