NUTRITIONAL MYOPATHY IN DUCKLINGS*

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Plates 7 and 8

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It has been shown in previous papers (1–4) that young chickens on a simplified diet composed of skimmed milk powder, casein, corn-starch, lard, salts, cod liver oil, yeast, and paper pulp, develop a striking disorder of the central nervous system which we have called nutritional encephalomalacia. When this same diet is given to ducklings, no changes are produced in the central nervous system, but they succumb to a remarkable and practically universal degeneration of the skeletal muscles. No other organs or tissues appear to be affected.

EXPERIMENTAL

Day old Pekin ducklings were kept in battery brooders for about 2 weeks, and then transferred to larger cages with adequate water pans. 32 experimental animals received Diet 108,[1] to which water was added to make a paste. 15 normal controls were given the Stock Diet 20.[2]

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[1] Diet 108 is composed of:

	per cent
Skimmed milk powder (Merrell-Soule)	15.0
Casein (Merck's technical)	20.5
Corn-starch	20.0
Lard	21.0
Cod liver oil (Mead Johnson and Co.)	2.0
Yeast (Fleischmann's bakers', dried)	5.0
Salt mixture (McCollum 185)	6.5
Paper pulp (Eastman)	10.0

For the first 2 weeks, the behavior and appearance of the two groups were much alike. The ducks on the simplified diet showed less yellow pigmentation of bill and legs, but they were active and grew fairly well (Chart 1). Quite suddenly in the 2nd or 3rd week, symptoms of extreme weakness made their appearance. The birds were found sprawled flat, or in the earliest stages, walked awkwardly with feet turned in, and sometimes overlapping. When placed on their back, they had difficulty in righting themselves, and lay passively. As the weakness progressed, they could not raise the head from the table. The eyes were somewhat sunken. Sometimes there were coarse tremors and athetoid movements, but, in contrast to the encephalomalacic chicks, the head was not retracted and there were no spasticity, no forced movements, and no stupor. Indeed, aside from the extreme weakness, the ducklings seemed not very ill; they appeared conscious of their surroundings, free from pain, and not at all irritable. There was usually a terminal loss of weight, probably from inability to obtain food.

Most of the ducklings were sacrificed and studied soon after the onset of symptoms; some were found dead, without previous symptoms having been noted. After the symptoms were well established, recovery did not occur.

Pathology.—The subcutaneous fat and intra-abdominal fat was well preserved. Only the skeletal muscles showed pathologic changes. The most obvious feature was their pale color—often creamy

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[2] Diet 20 is composed of:

	per cent
Wheat bran	16.2
Wheat middlings	12.2
Yellow corn-meal	22.2
Ground oats	11.0
Alfalfa leaf meal	6.0
Skimmed milk powder	18.0
Cod liver oil	2.0
Yeast	2.0
Meat scraps	4.0
Bone meal	4.0
$CaCO_3$	1.6
NaCl	0.8

yellow rather than dark red. In the gross, not all the muscles were equally affected, nor was the alteration in the color always extreme. The muscle tissue was watery and translucent, the contractility impaired, but not usually wholly lost.

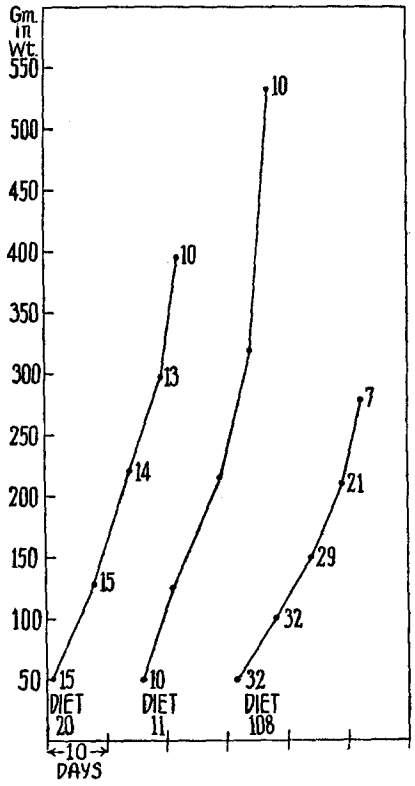

CHART 1. Composite growth curves of ducklings for the first 22 days on Diets 20, 11, and 108. The numerals at the right of each curve indicate the number of survivors.

Microscopically, there was a varying proportion of necrotic fibers, showing the usual picture of hyaline or waxy degeneration. In the most acute cases, there was little or no cellular reaction, but the intermuscular tissue was edematous. In cases which survived longer, there was an extreme cellular response in which myoblasts, histiocytes, and leucocytes of different types took part. Occasionally, basophilic

regenerating myocytes were present. The animals did not survive long enough for the necrotic fibers to become replaced by connective tissue or fat. Calcium deposits were not seen. The lesions are illustrated in Figs. 1 and 2. The brain was examined in all the cases, and the spinal cord and sciatic nerves in many. No significant lesions were found. A study of the motor nerve endings by Rogers' method showed, as in the nutritional muscular dystrophy of rabbits and guinea pigs (5), that these structures were not affected (Fig. 3). The only incidental lesions noted in a few animals were erosions of the gizzard mucosa, and in one case a perforating ulcer with peritonitis. The myocardium and smooth muscles were free of change, and nothing abnormal was seen in the other viscera. No detailed histological study has been made of the bones but there were no gross deformities. The epiphyseal cartilages were not widened, and the bones cut with great resistance.

The controls grew well and when killed at corresponding intervals showed normal muscles and viscera.

Cultures (from heart, liver, spleen, and in some animals from the muscle tissue) were made on infusion agar slants, and incubated for 7 days at 37°C. The results are shown in Table I and offer no evidence of an infectious etiology. No bacteria or other microorganisms were demonstrable in the lesions.

Of the 32 ducklings on Diet 108, 3 were found dead. 9 others presented typical myasthenic symptoms, but at autopsy, the muscle changes escaped notice, and the muscles were not sectioned for histological study. The observations upon the remaining 20 ducks are summarized in Table I, from which it can be noted that 17 showed pronounced and obvious symptoms and lesions. There were 3 negative cases. No. D-1895, which was killed on the 30th day because of weakness and prostration, proved to have perforating ulcers of the gizzard, but there were no muscle lesions, and the muscle creatine was within normal limits. No. D-1924 died on the 11th day of a generalized infection, probably due to an organism of the paratyphoid group. No. D-1929 was killed on the 19th day because it walked awkwardly and seemed weak on its legs. Although the muscle creatine was distinctly low, there were no histological changes in the muscle.

In a previous study (6) it has been shown that there is a decrease in the creatine content of the muscles in nutritional muscle dystrophy of rabbits. Similarly in this disease of ducks, the creatine content of the degenerated muscle was reduced.

The individual values for the muscle creatine, as determined by the Rose, Helmer, and Chanutin modification of the Folin method (7) are listed in Table I. In 17 normal ducklings of the same age, the muscle creatine was found to have a mean value of 445 mg. creatine per 100 gm. fresh tissue, with a standard deviation

TABLE I

Effect of Diet 108 upon Ducks

Duck No.	Period on diet	Myasthenic symptoms	Microscopic lesions		Analysis of leg muscle		Remarks
			Central nervous system	Muscles	Moisture	Creatine per 100 gm. fresh tissue	
	days				*per cent*	*mg.*	
1888	K 30	+	−	+++	80.6	310	
1889	K 22	+++	−	+++	77.3	217	
1891	K 24	+++	−	++			
1893	K 19	+++	−	+			
1895	K 30	+++	−	−	77.8	418	Perforating ulcer of gizzard
1898	K 20	+++	−	+++	82.0	301	
1899	K 18	+++	−	+++			
1900	K 20	+++	−	++++	76.9	288	
1902	D 27		−	+++	78.7	236	
1919	K 18	++	−	++++	81.8	243	
1920	K 21	+++	−	++++	82.8	103	
1921	K 14	++	−	++++	—	128	Cultures sterile
1922	K 19	+++	−	++	79.4	187	Liver culture—large Gram-positive diplococcus; spleen sterile
1923	K 33	++	−	++	80.7	302	
1924	D 11		−	−	—	186	*B. paratyphosus* from spleen, liver, and heart blood
1925	D 14	+++	−	++	—	547	
1926	K 16	++	−	++++	82.2	88	
1927	K 19	+++	−	+++	82.9	179	Cultures from leg muscle—sterile; liver Gram-positive bacilli. Erosions of gizzard
1928	K 15	+++	−	++++	82.6	106	Cultures sterile
1929	K 19	+	−	−	77.6	288	Cultures sterile

of ±56; the probable error of the mean was ±9.2, and that of the standard deviation, ±6.9. Although the creatine content of normal muscle varies greatly, the values given in the table for degenerated muscle are seen to be significantly low, ranging from 88 to 310 mg.

The only exception is No. D-1925, in which fairly marked lesions were present with a creatine of 547 mg. However, different blocks of muscles in this animal showed great variation in the intensity of the histological changes, one being normal, and the sample taken for creatine determination may not have been representative.

Moisture determinations were also made. The mean value obtained in 17 normal ducklings was 78.3 per cent with a standard deviation of ± 1.8; the probable error of the mean was ± 0.3 and that of the standard deviation was 0.2. It is apparent from the table that in many instances, the moisture content of the degenerated muscle was increased, a fact which is in accord with the observed edema.

Studies of the oxygen consumption and irritability were made by Dr. Joseph Victor and will be reported in a separate communication.

The resemblance of these muscle lesions to those which are produced in rabbits or guinea pigs by nutritional means (8) led to an experiment in which 10 ducklings were given Diet 11.[3] Although some of the animals showed a certain awkwardness in gait, in a few instances, so severe as to incapacitate them, this seemed to be associated with deformity of the bones instead of muscle weakness or disease of the central nervous system. Most of the birds showed excellent growth, and only in the 4 so crippled that they had difficulty in obtaining nourishment did death result.

The muscular system, both grossly and microscopically, was not affected. The creatine content of the muscle was within normal limits. The brain, cord, and peripheral nerves were also unaffected. The bones were not subjected to careful study. In one instance, however, rachitis-like changes—widening and irregularity of the cartilage, slight excess of osteoid, and fibrosis of the marrow, were present in the upper extremity of the tibiotarsus.

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[3] Diet 11 is composed of:

	per cent
Rolled oats	35.5
Wheat bran	12.0
Casein (Merck's technical)	7.5
Lard	8.0
Cod liver oil (Mead Johnson and Co)	1.0
NaCl	1.0
$CaCO_3$	1.5
Skimmed milk powder	27.5

DISCUSSION

The most interesting aspect of these observations is that the same diet produces in two different species of birds widely different pathological effects. In chickens, the injurious action is limited to the brain —in ducks, to the voluntary muscles. In both cases, the lesions are associated with characteristic clinical symptoms.

It would be futile at this time to explain either condition in terms of known dietary factors, or to speculate as to why such dramatically diverse changes should occur on the same diet in the two species.

In spite of the close resemblance between the pathological lesions in the muscles of ducks on Diet 108 and those produced by Madsen, McKay, and Maynard (9) in goats, rabbits, and guinea pigs on synthetic diets, it would be unwise to assume that the same nutritional fault is responsible. The fact that Diet 11, which leads to severe muscle degeneration in rabbits and guinea pigs, is without effect upon the muscles of ducks makes the identity of the avian and mammalian disease very doubtful.

CONCLUSIONS

Ducklings fed on a diet of skimmed milk powder, casein, corn-starch, lard, cod liver oil, yeast, salts and paper pulp rapidly develop a disease characterized by extreme and progressive myasthenia, ending in death within a few days.

Pathological changes are found in the skeletal muscles. These show widespread hyaline necrosis of fibers, with edema and cellular reaction. The brain and other parts of the central nervous system are not affected, and no significant alterations are found in other viscera or tissues. The creatine content of the muscles is reduced in proportion to the muscle injury. Controls on a natural food diet remain free from the disease.

We are greatly indebted to Dr. Abner Wolf for preparations of the neurites and motor end plates; and to Dr. C. A. Slanetz for the bacteriological examinations.

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EXPLANATION OF PLATES

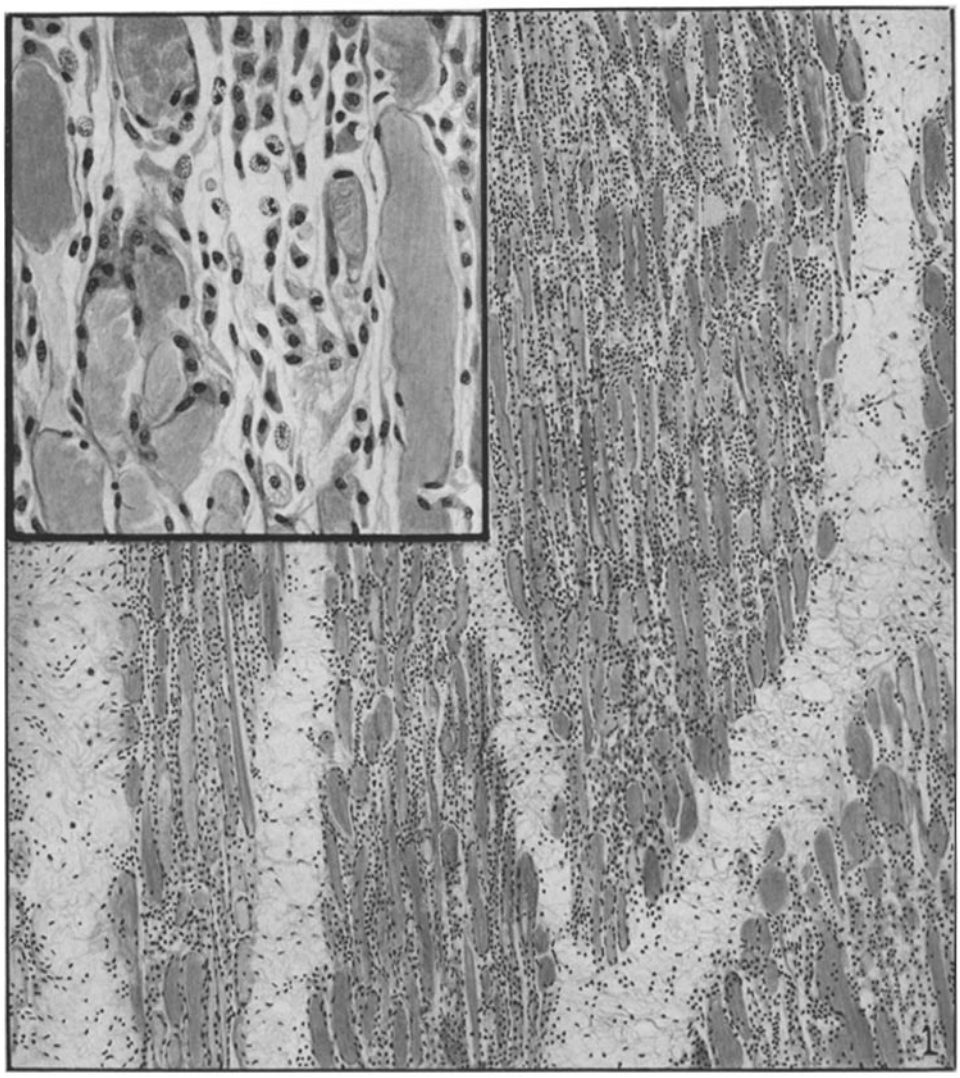
PLATE 7

FIG. 1. Duck 1898. Killed after 20 days on Diet 108; complete prostration. Gluteus muscle. Extreme waxy necrosis. The degenerated fibers are surrounded by myoblasts and histiocytes; the stroma infiltrated with great numbers of mononuclear and polymorphonuclear leucocytes.

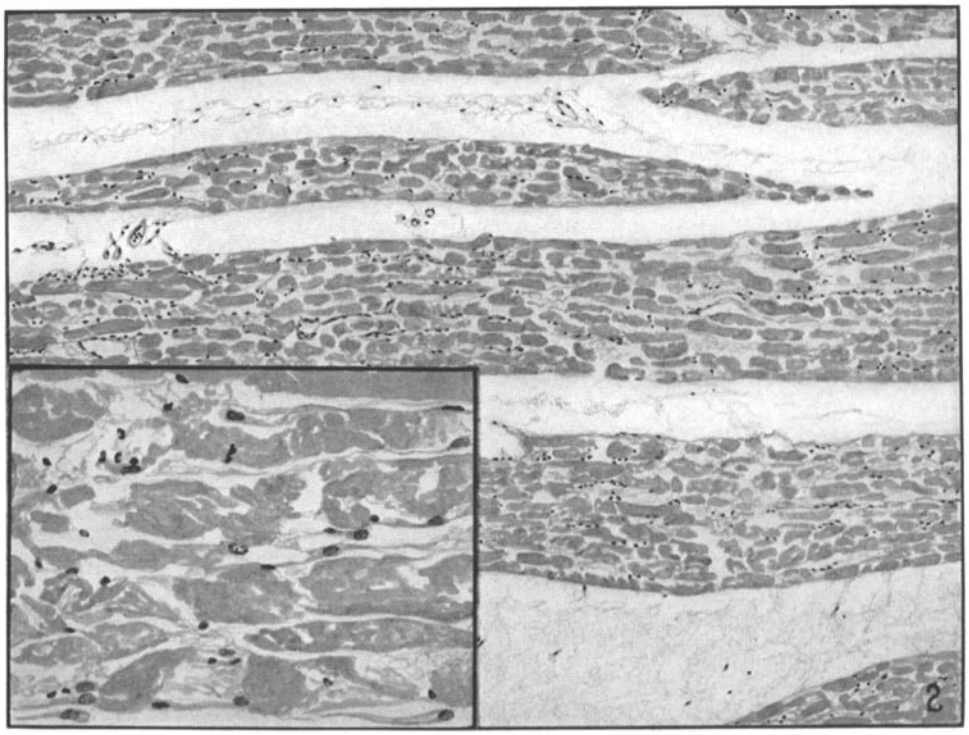
PLATE 8

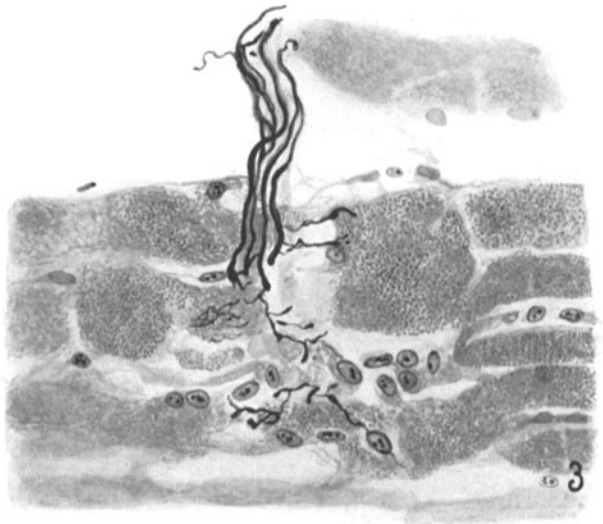
FIG. 2. Duck 1921. Killed after 14 days on Diet 108. Extensor muscles of leg. Extreme necrosis and fragmentation of fibers with interstitial edema. Little cellular reaction. Insert shows higher magnification.

FIG. 3. Duck 1926. Killed after 16 days on Diet 108, when unable to stand erect. Gluteus muscle—Rogers stain. In spite of extreme necrosis of fibers, the neurites and motor end plates are normal in appearance.



(Pappenheimer and Goettsch: Nutritional myopathy in ducklings)





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