NUTRITIONAL ENCEPHALOMALACIA IN CHICKS*

Influence of Age, Growth, and Breed upon Susceptibility

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In previous papers (1, 2), attention has been called to a nutritional disorder of young chicks, characterized by severe injury to the central nervous system. During the past 2 years, experimental studies have been continued in the as yet unfulfilled hope of defining the precise factors responsible. The data which have been accumulating enable us to discuss in the present paper, the influence of age, growth, and breed upon the occurrence of this interesting disorder.

The behavior of 172 White Leghorn chicks, belonging to 12 groups that were placed upon the disease-producing Diet 108[1] either at hatching, or after a short preliminary period on a natural foods diet, has been summarized in Table I with respect to the percentage incidence, the occurrence of symptoms, gross and microscopic lesions, and time elapsing until the appearance of the disease. Chicks that died during the 1st week or that had doubtful lesions, are not included in the table.

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[1] Diet 108 has the following composition:

	per cent
Skimmed milk powder (Merrell-Soule)	15.0
Casein (Merck's technical)	20.5
Cornstarch	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) (3)	6.5
Paper pulp (Eastman)	10.0

Table I demonstrates the wide variation between different experimental groups in the characteristics analyzed. For instance, the incidence of the disease as measured by percentage microscopic lesions, is seen to vary from 30 to 100 per cent. Of the total number, about one-third developed neither symptoms nor brain lesions during the arbitrarily chosen experimental period. Of the 99 chicks which showed symptoms, 83 presented lesions which were noted both grossly

TABLE I

Incidence of Nutritional Encephalomalacia in Chicks on Diet 108

Date of hatch	Length of time on stock diet	No. of chicks	No. of chicks with			Per cent with microscopic lesions	Length of time until end of experiment	Mean No. of days until onset of disease
			Symptoms	Gross lesions	Microscopic lesions			
	days						*days*	*days*
June 8, 1930	10	10	6	3	5	50	40	20
July 7, 1930	15	7	7	6	7	100	28	20
Sept. 30, 1930	7	22	8	10	13	54	28	20
Nov. 14, 1930	10	11	5	5	6	54	30	24
Jan. 8, 1931	2	11	7	7	7	64	28	22
Feb. 26, 1931	0	15	10	7	7	47	28	22
Apr. 30, 1931	0	20	9	5	6	30	35	18
Sept. 28, 1931	0	13	9	7	8	62	32	26
Dec. 1, 1931	0	13	7	8	9	69	42	36
Mar. 1, 1932	0	18	8	8	10	56	46	26
Apr. 26, 1932	0	15	12	12	13	87	56	34
June 13, 1932	0	17	11	7	7	41	42	28
Totals.....		172	99	85	98	57		

and microscopically; 12 showed microscopic lesions only, and 2, gross; in 13, no lesions were found. The presence of symptoms without demonstrable lesions may have been due to the fact that it was impracticable to cut serial sections of the entire brain in each case, and that small microscopic lesions may have escaped recognition. Another possible explanation is that the symptoms were due to transient functional disturbance—perhaps vascular spasm—not followed by manifest anatomical changes in the brain tissue.

The variation between groups may be due in part to the fact that

chicks were not allowed to survive for a sufficiently long period. This possibility was not fully appreciated in the earlier experiments, in which all the survivors—*i.e.* those which had not manifested symptoms—were killed after an arbitrary period on the diet, the length of which is listed in Table I. Recently, we have observed a chick in which the disease appeared suddenly after 53 days, and a number of others in which it first appeared after 40 days. In a recent group of 25 chicks, 11 developed the disease before the 28th day and 14 afterwards.

TABLE II

Incidence of Nutritional Encephalomalacia in Chicks That Were Placed on Diet 108 at Different Ages

Chicks Hatched April 26, 1932

Group No.	Length of time on stock diet	No. of chicks	No. of chicks with			Per cent with microscopic lesions	Mean No. of days until onset of disease
			Symptoms	Gross lesions	Microscopic lesions		
	days						*days*
1	0	15	13	13	13	87	34
2	12	15	13	11	14	93	31
3	19	14	9	8	9	62	23
4	26	15	5	4	6	40	19
5	40	16	3	3	4	25	21
6	54	8	2	1	1	14	10
7	68	7	0	0	0	0	

The period elapsing until the onset of the disease has been roughly determined by killing the chick upon the first appearance of symptoms and establishing the diagnosis of encephalomalacia from the presence of characteristic lesions. In a certain number of chicks which were found dead or which were killed at the end of the experiment, no clinical abnormalities had been noted, but since these chicks presented fresh lesions at autopsy, they are included in Table I. Not included, however, are chicks showing symptoms but no lesions, or only old healed or healing lesions.

Table I shows that the variation is not dependent upon seasonal influences. Thus in April, 1931, only 30 per cent showed the disease, as against 87 per cent in the same month of 1932.

The Influence of Age upon Susceptibility

The experiments summarized in Table I were carried out upon day old or very young chicks; our few negative results with older birds had given the impression that this disorder could be induced only during the early growth period. In order to obtain more definite information upon this point, groups of chicks of the same hatch were placed upon the disease-producing Diet 108 after varying intervals upon the natural foods Diet 634 of Hogan, Hunter, and Kempster (4).[2] Those that showed no symptoms were kept under observation for at least 40 days.

The incidence of the disease in these successive groups and the time elapsing until the onset are shown in Table II.

From the results of this experiment it would appear that the susceptibility to the disease diminishes as the preliminary period upon the natural foods diet is extended. It might be supposed that the older chicks would exhibit the disease in a milder form and that it would take longer to develop. This is definitely not the case. Thus in Chick 1294, which had been on the natural foods diet for 54 days, and which was the only one of 8 in this group to show the disease, severe symptoms appeared suddenly after 10 days on Diet 108. It was immediately killed and extensive fresh lesions were found in the cerebellum.

Table II demonstrates the unexpected fact that the disease tends to develop after a shorter period in the older chicks, in spite of the fact that fewer of them become affected.

Growth and Susceptibility

For purposes of comparison with the growth of chicks on Diet 108, a

[2] Diet 634 of Hogan, Hunter, and Kempster:

	per cent
Whole wheat	55.6
Whole milk powder	8.2
Casein	12.3
Alfalfa meal	2.5
Butter fat	4.2
NaCl	0.9
$CaCO_3$	1.3
Cod liver oil	3.0
Yeast	12.0

composite growth curve A in Chart 2, was constructed of 78 chicks on the Hogan, Hunter, and Kempster natural foods Diet 634 (4).

The standard deviations for the initial weight and the weights at 21 and 28 days respectively were calculated in addition to the mean weights and plotted in Chart 1 in order to illustrate the extent of

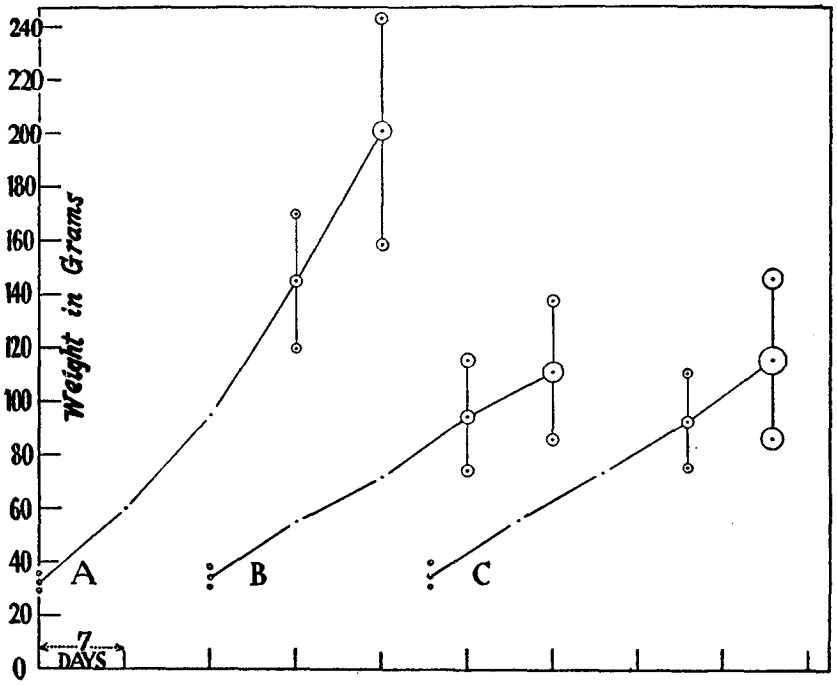

CHART 1. Composite growth curves including males and females. A, 78 normal chicks on Diet 634. B, 21 chicks with lesions on Diet 108. C, 30 chicks without lesions on Diet 108. Vertical lines represent standard deviations. Circles represent probable errors.

biological variation. The probable errors of the mean and of the standard deviation have been depicted by circles. The rate of growth of these chicks on Diet 634 compares favorably with that cited by Hogan, Hunter, and Kempster in their description of the diet, and with that obtained by Mitchell, Card, and Hamilton (5) in a recent study of growth in the White Leghorn chick.

The growth curves of chicks on Diet 108 were plotted in a similar fashion. Curve B was constructed from 21 chicks with encephaloma-

lacia that survived the diet for more than 28 days before showing symptoms. Curve C refers to 30 chicks in which the brain was normal at autopsy. Curves B and C are seen to be similar. The growth is not nearly so good as that for chicks on the natural foods Diet 634.

The records of many chicks that did not survive even 21 days were available. The average daily increase in weight of these is included in the data given in Table III.

TABLE III

Mean Daily Increase in Weight of Chicks on Diets 108 and 634

Diet	No. of chicks	Mean daily increase in weight	P. E. of mean	Standard deviation	P. E.	Coefficient of variation
		gm.	*gm.*	*gm.*	*gm.*	*gm.*
108 (with lesions)	67	2.9	0.08	0.9	0.05	31.6
108 (without lesions)	56	2.8	0.09	1.0	0.06	36.9
634 (normal chicks)	85	6.2	0.11	1.5	0.08	24.9

TABLE IV

Incidence of Nutritional Encephalomalacia in Different Breeds of Chicks on Diet 108

Chicks Hatched March 1, 1932

Breed	No. of chicks	No. of chicks with			Per cent with microscopic lesions	Mean No. of days until onset of disease
		Symptoms	Gross lesions	Microscopic lesions		
						days
White Leghorn	18	9	11	10	56	26.1
Barred Plymouth Rock	16	11	11	10	62	28.7
White Wyndotte	20	15	12	12	60	28.5
Rhode Island Red	18	11	14	15	83	29.2

From Table III it is apparent that chicks on Diet 108 had the same mean daily increase in weight irrespective of whether or not they developed the disorder.

The Susceptibility of Different Breeds to Nutritional Encephalomalacia

In the following experiment, the susceptibility of White Leghorns, Barred Plymouth Rocks, Rhode Island Reds, and White Wyandottes

was compared. Twenty chicks of each breed were placed on Diet 108 from the day of hatching; 5 additional chicks of each lot were given the natural foods Diet 634. It was found, as shown in Table IV, that the disease is readily produced in each of these breeds. The differences in percentage incidence are probably without significance, in view of the variations noted in the White Leghorn series (Table I).

Not only was there a high incidence in all groups, but the average time on the diet before the appearance of symptoms was virtually the same. The character and distribution of the lesions showed the customary variations in all the groups. One may conclude, therefore, that none of the breeds tested showed a peculiar susceptibility or resistance to the disease, and all are equally suitable for experimental purposes.

CONCLUSIONS

1. Nutritional encephalomalacia may be induced in chicks up to the age of approximately 2 months. As the preliminary feeding period on a natural foods diet is increased, the percentage incidence of the disease becomes progressively less. The average time between institution of diet and appearance of the disease tends to diminish.

2. There is no correlation between growth and incidence of the disease.

3. White Leghorns, Barred Plymouth Rocks, Rhode Island Reds, and White Wyandottes are equally susceptible.

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