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The Transmission of and Resistance to Fowl Paralysis (Lymphomatosis)

By

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Summary

1. Fowl paralysis is readily transmitted to young chicks by contact.

2. Young chicks from susceptible stock contracted the disease regardless of attempts to isolate them on the premises where paralysis-affected stock existed.

3. Pullets from paralysis-susceptible stock showed a distinct age resistance when not introduced before six weeks of age to premises now or recently occupied by affected stock.

4. Definite evidence is presented that certain families are more resistant than others to the disease.

5. A high degree of resistance to fowl paralysis has been developed through intensive selection.

6. The progeny of hens was shown to be more resistant to the disease than the progeny of pullets in the same affected flock.

7. Confined rearing did not reduce the occurrence of the disease over that attained by range rearing.

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The Transmission of and Resistance to Fowl Paralysis (Lymphomatosis)

By

E. M. GILDOW, J. K. WILLIAMS, and C. E. LAMPMAN*

FOWL paralysis, lymphomatosis, or neurolymphomatosis, as described by Pappenheimer and Dunn (*10*), is a disease of the domestic chicken with symptoms of incoordination in body muscles, notably the legs and wings, partial or complete blindness, and, in some instances, general unthriftiness (*Figs. 1 and 2*).

The disease usually affects birds between 3 and 15 months of age though cases have been seen in birds under 2 months and over 2 years of age by the authors. It is generally agreed that a filterable virus is the causative agent (*4, 10*).

The earliest recorded study of the disease in this country was by Kaupp (*6*), who in 1921 described the disease in the Eastern states. The classical work of Pappenheimer and Dunn in 1926 (*10*) established a solid foundation for later studies. They showed that the disease was caused by a filterable virus which could be transmitted to young chicks by intramuscular or subdural injection of emulsified affected tissue or filtrates from such tissue. They also demonstrated the existence of typical pathological changes primarily in the nervous system but also in the affected iris, ovary, viscera, and other organs or tissues. The predominant lesion is the infiltration of lymphoid cells into the affected tissue and the degeneration of the invaded tissue.

Numerous workers (*4, 9*) have subsequently shown that regardless of the type of tissue used for injection any or all of the manifestations of the disease may be produced. Thus eye, nerve, kidney, or liver lesions may be produced from the injection of macerated eye lesion tissue.

One, therefore, may safely conclude that any form of the disease may be produced from a single type lesion, and that certain forms of enlarged livers are definitely a form of this disease. Many investigators believe that all forms of leucosis are caused by the same virus. Stubbs 1939 (*11*), on the other hand, has shown that some viruses may cause leucosis entirely distinct from fowl paralysis (lymphomatosis). The presence of leucosis in chickens and its associated big liver conditions for many years (7) prior to the advent of extensive paralysis complex outbreaks would indicate that there

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probably is more than one etiological agent involved. Certainly the solution of the etiology of the fowl paralysis complex in chickens has not been definitely reached.

Fowl paralysis has been reported from many countries and from practically every state in the United States and is more or less extensive in practically every major poultry community. The authors have observed its existence over a considerable period of time in three widely separated states in the United States: viz., New Hampshire, Wisconsin, and Idaho. In these areas as well as much of the remainder of the United States, as recorded by others (*5, 8, 9*), the disease has been responsible for extensive losses. In individual flocks these losses range from a few birds up to 75 per cent of the pullet flock.

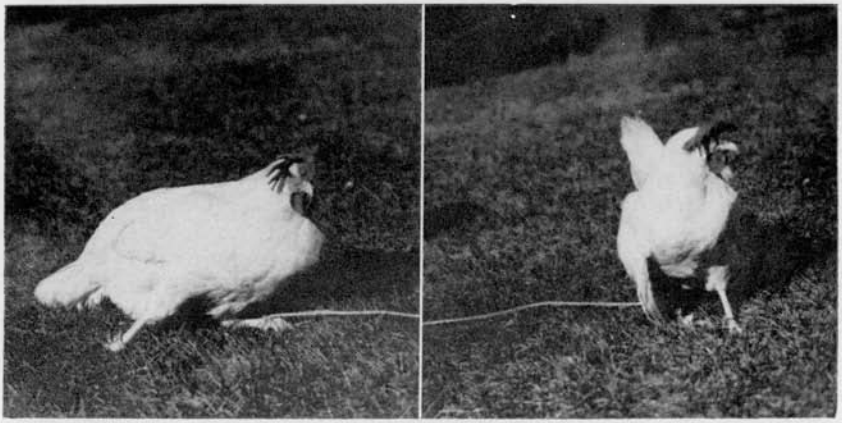

Figure 1.—Two Leghorn pullets showing typical wing and leg type of fowl paralysis.

By 1925 it had already become a major cause of pullet mortality in New Hampshire and had been a serious problem there for four or five years. Indeed, Kaupp (*6*) reported the disease in the New England states as early as 1914 and specifically mentions its presence in New Hampshire in 1917. From 1925 to 1928 enormous losses were experienced in New Hampshire.

The disease first made its appearance in Wisconsin in an occasional flock as early as 1920. However, it did not become an important cause of pullet mortality in Wisconsin and the Middle West in general until about 1925. It gained in extent for the next five or six years and has been prevalent there ever since.

The first cases of the disease in Idaho were recorded in the spring of 1930. At that time the disease was not extensive. During the next five years it spread rapidly. Due to the rapid development of resistance in a few of the key breeding flocks in Idaho, and to

a better knowledge of the disease, its prevalence and economic importance has been decreasing since 1935.

A survey of the literature on fowl paralysis indicates that little variation in susceptibility or resistance to the disease exists between breeds. Great variation does exist in this respect between families or strains within breeds, however.

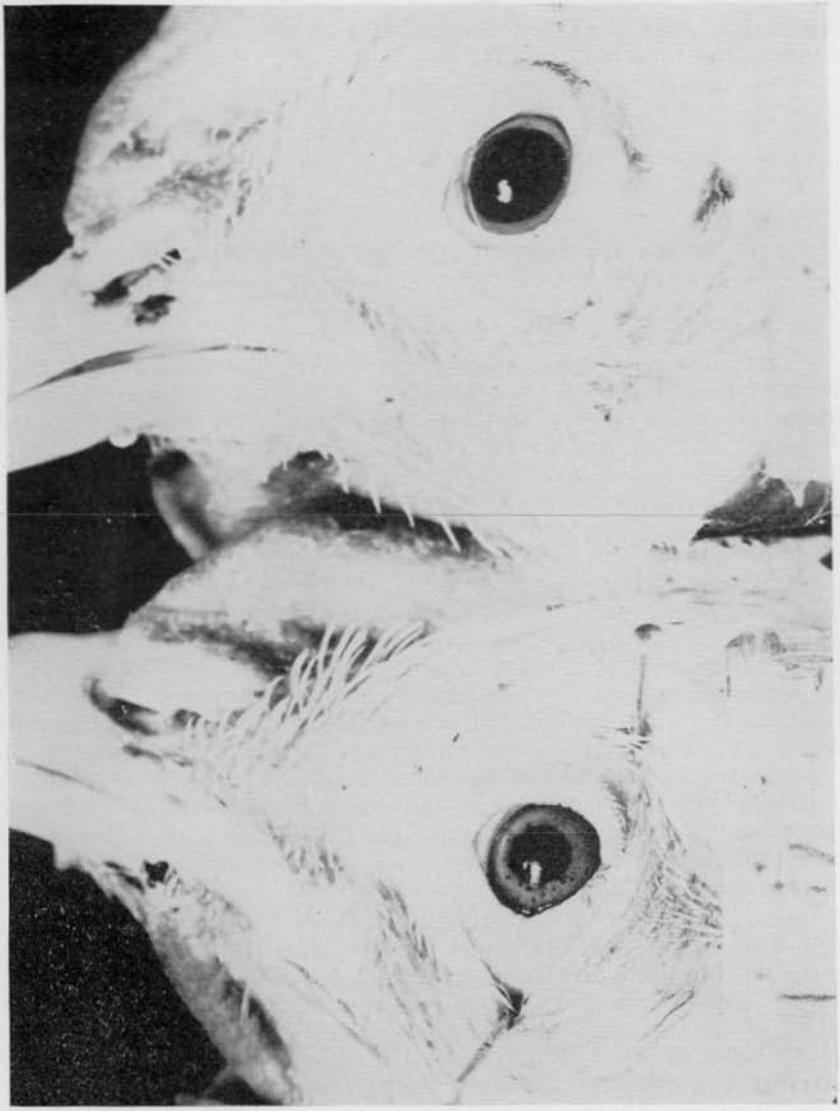

Figure 2.—Comparison of a normal eye above and a so-called fish eye below commonly seen in fowl paralysis.

Object of Experiment

Paralysis made its appearance in the Idaho Experiment Station Leghorn flock in the summer of 1930. It increased in prevalence until in 1932 and 1933 about 40 per cent of the pullets were affected during their pullet year. By this time considerable evidence of an expansion of the disease in Idaho was apparent. In order to learn more about the disease as well as to curb its extent in the station flock and other flocks in the State a project was set up to determine the following points:

1. Is the disease readily transmitted through contact?
2. Is there an age at which birds show resistance to the disease?
3. Is there a distinct inheritance of resistance to the disease?
4. Is there a difference in resistance in offspring of hens as compared with pullets?
5. Is there a difference in incident of the disease in confined—as compared with range-reared birds?

Methods of Procedure

Series I was started in the spring of 1933 when 452 day-old S. C. White Leghorn chicks were purchased from a breeder in southern Idaho, in whose flock paralysis had never occurred. These were divided into four lots of 113 chicks each with Lot I being placed in a brooder house and Lots II, III, and IV in a battery brooder. On the same date 228 pullet-hatched chicks (chicks from birds in their first year of production) and 226 hen-hatched chicks (from birds in their second year of production or older) from the flock of the Idaho Agricultural Experiment Station were also divided into four lots of 57 pullet-hatched chicks and 56 or 57 hen-hatched chicks each. Lot I of the station stock was placed in the brooder house with Lot I of the purchased group, and Lots II, III, and IV were placed in a battery brooder in a separate room from Lots II, III, and IV of the purchased chicks. This procedure was carried out in an attempt to prevent the transmission of the disease from the infected station stock to the introduced stock. The two groups in batteries were managed alike and fed the same ration and were cared for by the same caretaker. Hereafter the station birds will be called "affected stock" while the introduced birds will be called "clean stock."

Lots II, III, and IV of both groups were kept in batteries for 2, 4, and 6 weeks respectively, at which times they were placed together in the brooder house. As each lot was placed in the brooder house it was separated from the other lots for a week or two by wire panels. Cockerels were removed at 8 weeks of age and data kept on the pullets only. The per cent mortality was figured upon the number of pullets present at the end of the brooding period.

At the end of the brooding period 89 pullets from the clean stock and 96 pullets from the infected stock, making a total of 185 pullets,

were transferred to a colony house on the regular growing range. A total of 186 pullets including 89 from the clean flock and 97 from the infected flock, was continued in the brooder house and reared in confinement during the entire growing period (*Fig. 3*). During the last week in September 240 of the better looking birds were transferred to four pens in the experimental laying house. Each pen contained 60 birds—30 from the clean stock and 30 from the infected stock. In the infected group half the birds were from hen stock and half from pullets. The remainder of the birds were

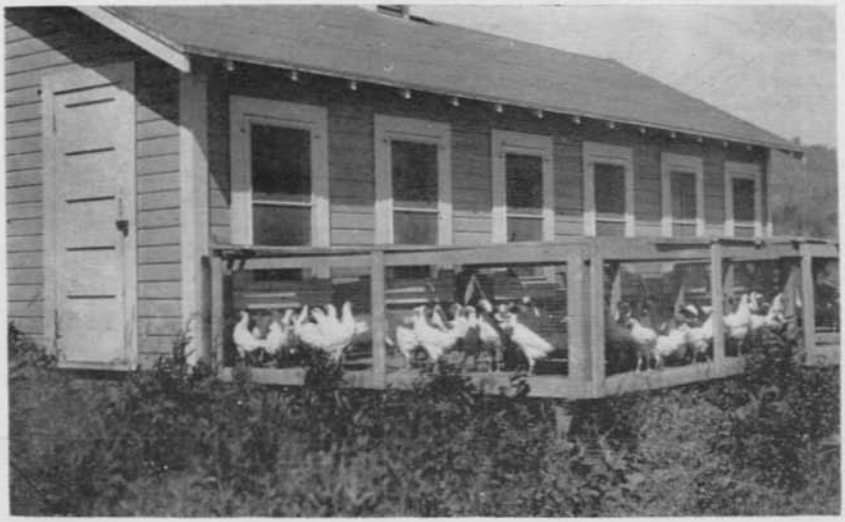

Figure 3.—Series I pullets in confinement on wire-floored yards in front of brooder house in confined rearing as compared with range rearing phase of fowl paralysis project.

housed in other pens on the poultry plant. Records of mortality and post-mortem were taken from the entire number of pullets raised on the project, regardless of where they were housed. The birds were kept in the laying pens for 11 months or until September 1 of the following year.

Series II was started in the spring of 1934 and was largely a duplicate of the previous year's work. The chicks were secured from a different breeder than the year before, and, like that of the preceding year, this breeder had never had an outbreak of paralysis in his flock. Three hundred fifty-one day-old chicks were purchased, divided into three lots, and placed with an equal number of chicks from the station stock at hatching, and at 4 and 8 weeks of age. Lots II and III were again kept in separate rooms but in the same building at the poultry farm until they were placed together in the brooder house at 4 and 8 weeks of age. One-half

of the chicks from the station flock were again from pullets in their first year of production and one-half were from hens 2 years old or older. At the end of the brooding period half the pullets from each group and each lot were again taken to a clean alfalfa range while the other half were grown in confinement. At the end of the growing period all groups and lots were again divided equally into four pens in the laying house and kept through one laying year or until approximately 17 months of age.

In Series III, which was started in the spring of 1935, one big change was made. Instead of getting chicks from a breeding flock in which paralysis had never occurred, hatching eggs were purchased from an R. O. P. breeder who had had a severe outbreak of paralysis in his flock several years previously but had apparently eliminated the disease by selective breeding. These birds will be called "resistant stock." These chicks were divided into three lots and placed with chicks from the station flock at hatching and at 4 and 8 weeks of age. These birds were managed during the brooding, growing, and laying periods in the same manner as the preceding years except that all pullets were placed on range during the growing period instead of leaving some in confinement.

Each succeeding year of the experiment the occurrence of paralysis among the pullets from the affected station flock had become less, and it was thought that this was possibly one reason why the occurrence of the disease was so low in the imported stock in Series III. Therefore, in Series IV chicks were again secured from the same non-affected flock as those secured in Series I, together with chicks from the same resistant flock as in Series III. In addition to securing day-old chicks from the non-affected flock, pullets from the same hatch as the day-old chicks were kept on the breeder's farm and shipped to the Experiment Station at 6 and 12 weeks of age, respectively. They were placed with pullets of the same age from the station flock immediately upon their arrival. All pullets were reared on range and were managed the same way as the previous years. All birds that developed typical symptoms of paralysis and all birds that died after 8 weeks of age were autopsied and records taken concerning the symptoms, lesions, and cause of death.

During the study a few over 2,500 birds were used.

Diagnosis

The diagnosis of fowl paralysis in an affected flock is not difficult since some individuals will show the common symptoms and typical lesions of the disease (*Figs. 1 and 2*). A definite diagnosis of the disease in an individual bird is, however, more difficult. The differences of opinion that now exist in regard to whether or not all types of leucosis are caused by a single etiological agent has already been mentioned. Jungherr in 1934 (5) came to the con-

clusion that spontaneous cases of fowl paralysis could be reasonably diagnosed on the basis of macroscopic and characteristic microscopic changes in the peripheral nerves.

Pappenheimer and Dunn (*10*) considered that lymphomatosis tumors of the ovary and other viscera are composed of the same type of lymphoid cells found in the nerve lesions of this disease and can, therefore, be considered as typical lesions of the disease. They further state that enlarged livers and spleens are rarely associated with fowl paralysis.

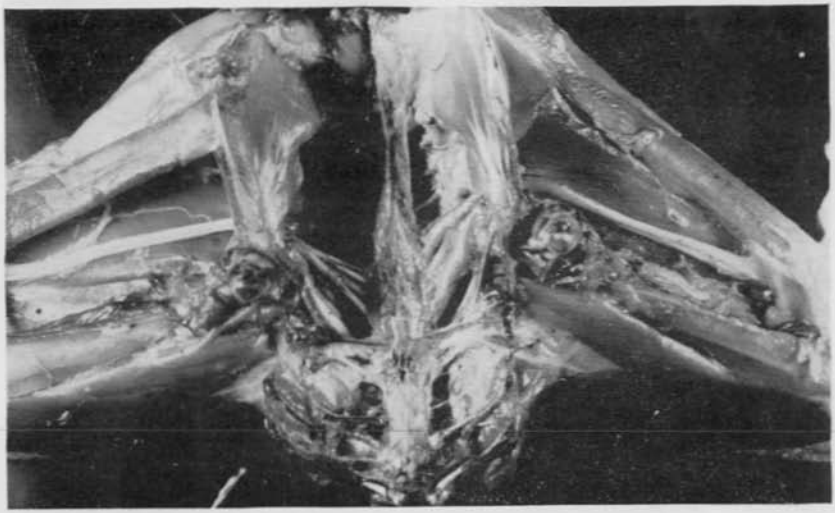

Figure 4.—Ventral view of the enlarged sciatic plexisis and femoral nerve on the right side in contrast to the normal nerves of the left side. Notice the normal cross striations on the left femoral nerve.

In the study of fowl paralysis at the Idaho Agricultural Experiment Station, where a large number of birds were involved, the diagnosis of the disease was based largely on clinical symptoms and macroscopic detection of lesions. The symptoms used in diagnosis were unilateral or bilateral lameness; a weakness or drooping of one or both wings; iritis as manifested by an irregular or contracted pupil or a slate-gray discoloration of the iris; and occasionally a distension of the eyeball. Wry necks and tumors of the skin or muscles that were detectable before autopsy were also considered.

Enlargement of peripheral nerves was considered as a diagnosis lesion (*Figs. 4 and 5*). Usually these enlarged nerves showed a tendency to become more or less yellow in color and the normally distinct cross striations of the nerves became less distinct or were absent. A definite extension of infiltration to surrounding tissues was sometimes detected. Typical appearing macroscopic tumors of

the ovary and other viscera as well as of the muscles and skin were considered diagnostic of the disease (*Fig. 6*). Tumors of the mesentary that were frequently associated with peritonitis caused by ruptured egg yolks, etc., were not considered as lesions of the disease unless they were associated with tumors of the ovary. Enlarged friable or distinctly fibrous livers with or without en-

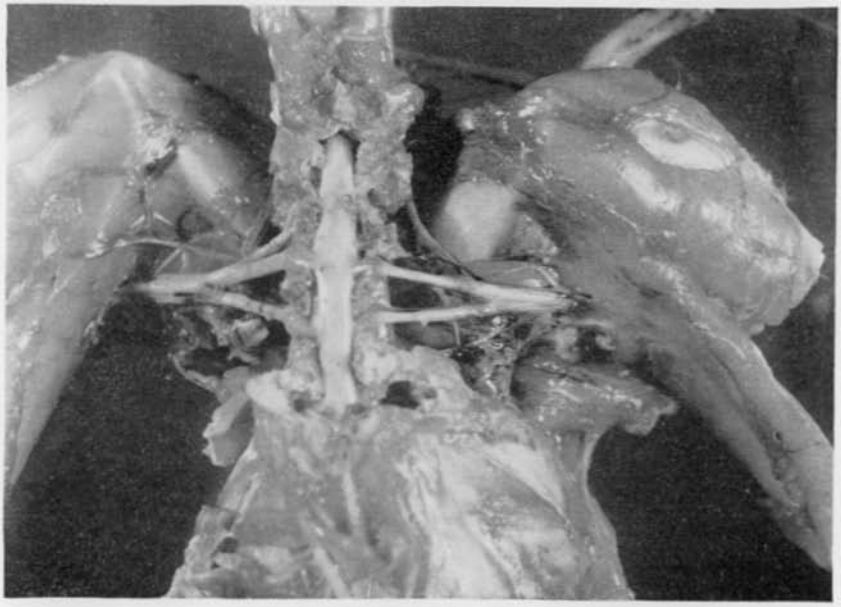

Figure 5.—Dorsal view of the brachial plexisis showing enlargement of the dorsal ganglia on the left side, also slight enlargement of the No. 2 branch of this plexisis on the left side.

largement of the spleen and distinct circumscribed tumors in the liver or spleen were not considered as lesions of fowl paralysis. Admittedly some error in accurate diagnosis may be present in properly classifying these tumors but the large number of birds

Table 1.—Prevalence of symptoms and lesions in pullets from affected and non-affected stock

Year	Source	Symptoms or lesion					
		Leg paralysis	Wing paralysis	General paralysis	Nerve lesions	Tumor growth	Eye lesions
1933	Affected	8	3	11	25	16	25
	Non-affected	13	9	12	23	28	36
	Total	21	12	23	48	44	61
1934	Affected	11	2	1	13	10	9
	Non-affected	23	5	4	33	17	27
	Total	34	7	5	46	27	36

involved and the relative small number of diagnoses made on this basis reduces the chances of great error in final conclusions.

The prevalence of symptoms and lesions during the first two years of this study for both the affected station stock and the introduced birds from non-affected stock is shown in Table 1. Eye

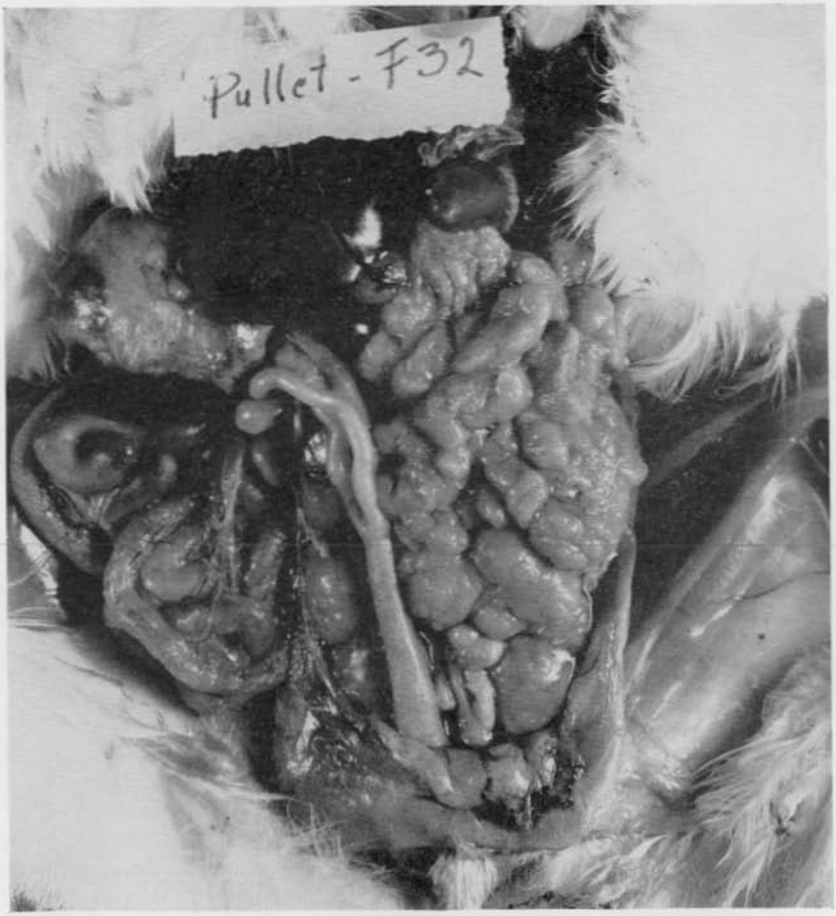

Figure 6.—Ventral view of the opened abdominal cavity showing one of the larger tumors of the ovary in fowl paralysis.

and nerve lesions were most prevalent, tumors and leg paralysis next in importance, and general paralysis and wing paralysis least prevalent.

In Table 2 is shown the monthly occurrence of paralysis for the first two years of this study. The high occurrence recorded for the 17th month (Series I) is misleading since eye lesions were not

Table 2.—Monthly occurrence of paralysis

Year	Source	Age in months																
		2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	Total
1933	Non-affected	1	2	7	5	7	4	8	5	5	4	7	1	2	7	1	11*	77
	Affected	0	1	3	1	2	4	4	5	7	3	3	2	4	3	4	14*	60
	Total	1	3	10	6	9	8	12	10	12	7	10	3	6	10	5	25	137
	Per cent of cases monthly	.73	2.19	7.03	4.38	6.57	5.84	8.76	7.30	8.76	5.10	7.30	2.19	4.38	7.30	3.65	18.25	100
1934	Non-affected	6	2	6	3	11**	5	12	1	6	0	1	0	7	3	3	1	67
	Affected	2	3	1	6	5	2	1	4	1	0	0	1	1	0	1	2	30
	Total	8	5	7	9	16	7	13	5	7	0	1	1	8	3	4	3	97
	Per cent of cases monthly	8.3	5.1	7.2	9.3	16.5	7.2	13.4	5.1	7.2	0	1	1	8.3	3.1	4.1	3.1	99.9

*Includes 9 birds from the non-affected flock and 10 birds from the affected flock which were blind at the end of the experiment and not previously recorded. In the 1933 series no accurate monthly record of eye lesions was kept. Eye lesions were recorded only as birds died, became totally blind, or were removed for other causes. This tends to decrease the monthly incident in the earlier months and to increase it in the later months. In the 1934 series monthly observations on eye lesions were made after the pullets were housed.

**Seven pullets showed eye lesions when placed in laying house at 6 month of age.

recorded monthly but were checked for in living birds at the termination of the trial only. First cases showed up before the birds were 3 months old and continued through the 17th month which terminated the collection of data. The prevalance was highest up to 10 months of age. However, occasional high points were reached after the 10th month. The high point in incident of the disease could probably be placed at about the 10th month.

Experimental Results

Transmission

Some evidence had been accumulated prior to the time this project was started (1933), indicating that fowl paralysis was transmissable. Pappenheimer and Dunn 1926 (*10*) showed that it was transmitted to 25 per cent of chicks injected subdurally or intramuscularly with suspensions of nerve lesions of affected birds. Since 1933 considerable additional evidence concerning its transmissability has been accumulated. Kennard and Chamberlin in 1936 (*8*) gave the most comprehensive practical demonstrations of transmission by contact. They found that the disease was readily transmitted by contact to susceptible chicks from flocks that had never been affected with fowl paralysis when placed on the Ohio Station poultry farm where the disease was then prevalent. In fact they found that the disease was transmitted to such chicks even though they were placed on a clean piece of ground ½ mile from the station grounds when they were cared for by the same individual that cared for the affected station stock.

Wilcke et al, 1938 (*12*) show that 7 out of 8 families of chicks contracted fowl paralysis from pen contact to the same extent as when actually injected with virus.

In Series I, II, and IV (*Table 3*) it is shown that young, susceptible chicks from a flock that had never been known to have the

Table 3.—Paralysis mortality in progeny from various types of breeding flocks (in per cent)

Trial	Affected station stock	Introduced progeny			Station stock		All stock	
		Paralysis-free stock*		Resistant stock	Hen chicks	Pullet chicks	Range	Confined
Series I 1933-34	31.1	43.3		.....	27.4	34.7	37.3	37.1
Series II 1934-35	19.9	41.6		.....	17.0	24.6	25.0	26.3
Series III 1935-36	6.9	.....		3.1	.....	.....	.....	.....
Series		Introduced at						
IV 1936-37	7.6	1 dy. 41.8	6 wk. 4.0	12 wk. 8.0	5.5	.....	.....	.....

*Paralysis-free stock for Series I and IV from same source. Period of mortality included growing period and first laying year (8 weeks through 17 months).

disease contracted it readily when placed with chicks from the affected flock at the Idaho Agricultural Experiment Station. In each of these three series susceptible chicks from a paralysis-free source contracted the disease to the extent of over 40 per cent during the growing period and first-laying year as compared with 31.1, 19.9, and 7.6 per cent, respectively, in the affected station flock. It was found impossible to prevent young, susceptible chicks from contracting the disease even though they were isolated in a separate battery room and fed and watered in separately, carefully cleaned containers when they were fed by the same caretaker who also fed other chicks on the station and the source of feed was the same for both groups of chicks. This is in close agreement with the results obtained by Kennard (*8*) and by Wilcke (*12*).

Several transmission experiments have been reported where difficulty was experienced in transmitting the disease. One important factor to be taken into consideration in this problem is that of age resistance of otherwise susceptible chicks. This phase of the fowl paralysis problem is discussed under "Age Resistance to Paralysis."

Age Resistance to Paralysis

Pappenheimer and Dunn, 1926, (*10*), first noted that pullets of laying age from a fowl paralysis-free source were highly refractory to fowl paralysis. They also noted that birds 8 weeks old moved from a farm on which the disease was prevalent to a clean farm came down with the disease when they were 12 weeks old, indicating that they had probably contracted the disease before 8 weeks of age.

Kennard and Chamberlin, 1936, (*8*), also found that ready-to-lay pullets from a flock known to be free from fowl paralysis did not suffer from the disease when placed with their own affected stock, whereas when day-old chicks from the same source were reared on the station poultry farm a high incident (50 to 60 per cent) of the disease occurred. Other evidence is available, indicating that chicks rapidly become more refractory to the disease as they become older.

In an attempt to determine the relation of age to susceptibility the chicks from the paralysis-free source in Series I and II (*Table 4*) were divided into several lots (see *Methods of Procedure*) some of which were isolated in clean batteries separate from other stock and were placed with the chicks from the affected station stock at ages between 2 and 8 weeks. Losses from fowl paralysis were about comparable in each of the age lots. Data in Table 4 show that a high percentage of the birds in all of these lots readily contracted the disease. Evidence that transmission took place before the affected and non-affected birds were mixed at 8 weeks of age in the 1934-35 trial is apparent since one of the chicks from

the paralysis-free progeny came down with the disease prior to the time the birds were mixed. This transmission probably took place through contact with the main supply of feed and caretaker, both of which were common to both the isolated birds and to the affected station stock.

In Series IV (*Table 4*) are shown the results of a further attempt to determine the age resistance of chicks to fowl paralysis. One lot of chicks from the paralysis-free stock was placed with chicks from the affected station stock as day-old chicks. Pullets

Table 4.—Relationship of age at exposure to the occurrence of paralysis when chicks from a paralysis-free source are placed with chicks from an affected flock at various ages*

Series	Chicks mixed at:	Lot No.	No. cases in clean stock	No. cases in affected stock	Per cent cases in clean stock	Per cent cases in affected stock
Series I 1933–34	Hatching	1	20	14	46.5	31.1
	2 wk.	2	20	9	40.0	18.4
	4 wk.	3	17	17	37.8	31.5
	6 wk.	4	20	20	50.0	44.4
Series II 1934–35	Hatching	1	18	8	29.1	25.5
	4 wk.	2	18	13	30.0	21.7
	8 wk.*	3	22	6	41.5	11.5
Series IV** 1936–37	Hatching	1	23	5	41.8	7.6
	6 wk.	2	1	....	4.0	....
	12 wk.	3	2	....	8.0	....

*The possibility of transmission of the disease before the birds were mixed is discussed under "Age Resistance to Paralysis."

**The pullets (lots 2 and 3) in Series IV were retained on the farm from which they were secured until 6 and 12 weeks of age when they were shipped to the station and mixed with the affected station flock.

from the same hatch from the paralysis-free stock were secured at 6 and again at 12 weeks of age and were placed with the chicks of the same age from the affected station flock. The incident of the disease was 41.8 per cent in the chicks introduced as day-old chicks, 4.0 per cent for those introduced at 6 weeks of age, and 8.0 per cent for those introduced at 12 weeks of age. This indicates considerable resistance for 6- and 12-week-old chicks and further indicates that 6-week-old pullets are as resistant as those 12 weeks of age from the same source. This factor of age resistance may account for some of the variations secured in transmission experiments with this disease.

Susceptibility of the Progeny of Hens as Compared with Pullets

Kennard and Chamberlin, 1936, (*8*), showed that the progeny of hens were more resistant to fowl paralysis than the progeny of pullets. In two consecutive years the occurrence of fowl paralysis

in hen progeny as compared with pullet progeny was 20 to 32 and 14 to 24 per cent, respectively.

In the first two years of study at the Idaho Agricultural Experiment Station, as shown in Series I and II (*Table 2*), the incident of fowl paralysis in pullet progeny was approximately 35 per cent greater than that in hen progeny. The occurrence for hen progeny as compared with pullet progeny in the two series was 27.4 to 34.7 and 17 to 24.6 per cent, respectively. This is very similar to the situation as recorded by Kennard and Chamberlin (*8*). It is readily appreciated that many susceptible birds die during the pullet year, leaving the more resistant hens as breeders the following year. This situation exists, however, only when the hens as young chicks have passed through an active outbreak of the disease as demonstrated at the Idaho Station.

Although no definite project has been set up since Series II to further test the susceptibility of hen as compared with pullet progeny, an analysis of general flock data on pedigree stock shows that in some instances where special selections and planned matings are made, the progeny of pullets may be as resistant to fowl paralysis as the general hen flock used for chick production the same year. One may conclude from this study that hen progeny in a flock affected with paralysis will be more resistant to the disease than pullet progeny, but if careful selection of pullet breeders is made when pedigree breeding is practiced, the pullet progeny may be as resistant as the general hen progeny of the same year.

Inheritance of Resistance

Pappenheimer and Dunn, 1926, (*10*), first concluded that resistance and susceptibility to fowl paralysis seemed to vary considerably between different families or strains in a breed of poultry though no individual breed seemed to be more resistant than another breed. Asmundson and Biely, 1932, (*1*) came to the same conclusion as have many other workers; Wilcke, Lee, and Murry (*12*), Kennard and Chamberlin (*8*), Gildow, Williams, and Lampman (*3*).

Early in the work on fowl paralysis at the Idaho Agricultural Experiment Station one family of S. C. White Leghorn chickens, descendants of a single hen (A375), showed a very low occurrence of the disease.

She was hatched in 1928 before fowl paralysis had established itself in the station flock. By comparing her daughters and granddaughters with 22 other hens of the same age, definite evidence of a distinct variation in resistance was apparent. Of 24 daughters and granddaughters of A375, only one or 4.2 per cent died of paralysis. In contrast to this, of 124 daughters and granddaughters of the other 22 hens of the same age, 47 birds or 37.9 per cent died of paralysis. To show more direct contrast, 4 hens of the same age as A375, selected because they had the largest number of progeny,

showed 7 cases of paralysis out of 18 pullets, 5 out of 13, 5 out of 11, and 3 out of 11.

The progeny of different male birds varies extensively in their resistance to the disease. Reference to Table 5 will show this variation by years and the deviations from the average incident of paralysis for the progeny of all males and that shown by the high and low males. For instance, in 1933-34 the high male had a mortality of 33.3 per cent from paralysis while the low male showed only 18.4 per cent. In 1937-38 the high male showed 11.1 per cent

Table 5.—Average per cent laying-house mortality of the daughters of all breeding males and the males showing the highest and lowest total mortality for each of the six years from 1933 to 1939

Year	Pen	Total mortality	Paralysis mortality	Leukemia mortality	Other mortality
1933–34	Av. all males	44.6	21.4	2.7	20.5
	High male	60.0	33.3	11.1	15.6
	Low male	34.2	18.4	0	15.8
1934–35	Av. all males	37.0	11.1	9.4	16.5
	High male	46.9	18.8	9.4	18.7
	Low male	19.8	4.7	7.0	8.1
1935–36	Av. all males	23.7	4.9	4.9	13.9
	High male	40.9	6.8	8.0	26.1
	Low male	8.3	2.1	2.1	4.1
1936–37	Av. all males	27.9	6.0	7.2	14.7
	High male	45.9	13.5	8.1	24.3
	Low male	17.6	5.9	2.9	8.8
1937–38	Av. all males	17.6	2.5	4.4	10.7
	High male	31.5	11.1	5.6	14.8
	Low male	6.6	0	0	6.6
1938–39	Av. all males	7.14	0	2.52	4.61
	High male	13.68	0	5.26	8.42
	Low male	2.04	0	2.04	0

Note—The figures under the headings "Paralysis mortality," "Leukemia mortality," and "Other mortality" are not comparative figures but show only the proportion of the total mortality due to each of those causes.

while the low male had no evidence of the disease in his progeny. The genetic make-up of the hens mated to these males undoubtedly influenced these results although no attempt was made to favor any specific male. All hens were selected on the basis of resistance to paralysis as well as other factors.

The column labeled "Paralysis mortality" in Table 5 shows the satisfactory progress made in the control of the disease by breeding for resistance. In six consecutive years the average percentage occurrence of the disease in the entire Leghorn flock decreased as follows: 21.4, 11.1, 4.9, 6.0, 2.5, 0. Evidence that progress in the control of the disease up to 1936 was due to inherited resis-

tance rather than a decreased virulence of the causative organism is presented in Table 3 in the column headed "Paralysis-free stock." The introduced chicks in Series I and IV were both from the same susceptible flock. Mortality from fowl paralysis in those introduced as day-old chicks was 43.3 per cent for Series I and 41.8 per cent for Series IV as compared with fowl paralysis mortality of 31.1 and 7.6 per cent in Series I and IV of the affected station flock. This continued high incident in introduced susceptible stock through 1937 indicates that the virulence of the causative organism had not decreased to that time and that the reduction of fowl paralysis in the station Leghorns was due to a definite resistance to the disease. That this resistance is inherited seems evident though definite genotypic studies of the mode of this inheritance have not been made.

Evidence of a high incident of fowl paralysis among the progeny of affected birds was established early in this study. In the spring of 1934, a few hens from the affected station flock which showed contraction or irregularity of one or both pupils were mated to a male which showed contraction of both pupils. From this mating 31 pullets were secured, of which 14, or 45.2 per cent, developed some form of fowl paralysis by the end of the first laying year. Inasmuch as only 19.9 per cent of the affected paralysis project stock and only 11.1 per cent of the general station flock developed the disease the same year, this data would indicate that the offspring of birds affected with paralysis are more highly susceptible to the disease than those not affected.

Extension poultrymen, and others closely associated with poultry in the field, observe that fowl paralysis runs a general course in most breeding flocks that become affected. The occurrence of the disease reaches a peak the second or third year and gradually becomes less thereafter. The rate of decrease depends upon the practices followed by the breeder. The incident is reduced least rapidly in non-pedigreed breeding flocks that use a predominance of pullets as breeders. The reduction is more rapid where old hens are used extensively as breeders, where males from highly resistant flocks are introduced as breeders, and where pedigree breeding is followed and selective matings are made. However, a regular reduction in the disease is found to occur even though an attempt to select for susceptibility is practiced, as recorded by Lee et al, 1937, (9) and as experienced by other workers.

The one outstanding factor responsible for the rapid increase in resistance in the Idaho Station flock has been the extreme care taken in making selection of males with high resistance and in the use of hens showing high resistance. This selection has been based most extensively on the following criteria: first, a breeding male to qualify must be from a male that had a low average occurrence of the disease in all of his progeny; and, second, he must be from a medium- to large-sized family in which as little evidence as

possible of the fowl paralysis had appeared prior to the time he was used as a cockerel. If a high percentage of the pullets in his immediate family remained free from the disease through the first laying year and his daughters had a low comparative incident of the disease up to the next breeding season he was again used in the breeding flock if he was available, and providing the size, type, and production characters of his sisters and daughters were also satisfactory.

Hens whose progeny were least affected by fowl paralysis were selected. Factors other than their resistance to paralysis were considered in the selection of all birds. In this program of developing a high resistance to fowl paralysis great emphasis has been placed upon the family records of those birds used as breeders. Certainly, the family record is of major importance in the selection of resistant individuals.

Incident in Range-reared as Compared with Confined Chicks

The relationship of sanitation to the occurrence of fowl paralysis has been reported by Kennard and Chamberlin (*8*). They found that the use of batteries for brooding and rearing chicks, the use of wire-floored yards for developing pullets, or the use of a clean isolated range for pullets that were started in clean brooders did not reduce the paralysis mortality when compared with birds of the same general breeding that were brooded and ranged on the regular poultry yards.

Data accumulated at the Idaho Agricultural Experiment Station (*3*) show that confining pullets to a brooder house with wire-floored yards did not reduce the incident of the disease when compared with an equal number of birds that were placed on clean range at 8 weeks of age. In Series I and II (*Table 3*) are shown the results of two years' comparisons of these methods of development. The occurrence of fowl paralysis up to 17 months of age was 37.3 and 37.1 per cent the first year and 25.0 and 26.3 per cent for the second year for the confined vs. range-developed pullets, respectively.

It is possible that internal or external parasites or other diseases of poultry may have some effect upon the occurrence of the disease in an affected flock (*2*). However, little evidence is at hand indicating that in the absence of the virus of the disease any of these could actually cause fowl paralysis.

The general sanitary practices commonly employed by poultrymen seem to have little effect upon the development of fowl paralysis in an affected flock. Unpublished data from the Western Washington Experiment Station, Puyallup, Washington (conversation) indicated that it is possible to reduce the incident of the disease if chicks are brooded and grown in clean buildings and on clean ground entirely isolated from the affected flock. Kennard and Chamberlin (*8*) report that day-old chicks from their affected

station flock, sent to substations in Ohio where fowl paralysis did not exist, remained free from the disease.

Three lots each of day-old chicks from the affected Idaho Agricultural Experiment Station flock that were reared by flock owners where fowl paralysis did not exist were reported to have remained free from the disease during the first laying year.

Discussion

This report deals primarily with three major factors: that is, transmission of, age resistance to, and inherited resistance to fowl paralysis in White Leghorn chickens. This report, in confirmation of some other reports, shows that the disease is usually naturally contracted early in the life of the chick (before 6 weeks of age); however, a small percentage (4 to 8) of highly susceptible stock will contract the disease naturally through contact after 6 or 12 weeks of age. On the basis of this information it would seem that if maximum occurrence of the disease is expected, transmission experiments on chicks should be conducted during the first few days of their life.

Very little evidence is available concerning the comparative resistance of chicks of different ages to fowl paralysis. Work at Idaho indicates that 6-week-old chicks from highly susceptible stock are highly resistant to paralysis when not brought into contact with the disease prior to that age. Six- and 12-weeks-old chicks are slightly susceptible.

Information obtained in this study as well as from many other sources shows that fowl paralysis is so readily transmissable that it is very difficult to prevent such transmission when affected birds are, or have been recently, present on the premises. This may account for the difficulty generally experienced in preventing the control chicks in transmission experiments from developing the disease.

Many reports are available which show unquestionable evidence that there are varying degrees of resistance to fowl paralysis in domestic poultry. This variation is not restricted to any breed or strain of birds. That this resistance or susceptibility is definitely inherited is proved though the genotypic manner of this inheritance has not been established.

It is not difficult to increase this resistance in a flock of poultry; in fact, natural selection through the death of the most susceptible pullets before the hatching season each year (thus permitting the more resistant survivors to become the parents of the next generation) is sufficient to concentrate to a certain extent the factors for resistance. Some pullets from which eggs are saved for hatching subsequently die of the disease. Therefore, even greater natural selection takes place if only old hens are used as breeders. Where pedigree breeding is being carried out and records are kept of the

extent and cause of mortality, very rapid progress can be made in the increase of resistance to fowl paralysis by selecting only those birds for breeders whose brothers and sisters have shown the greatest resistance to the disease. It is hoped that eventually a genetic analysis of the factors responsible for resistance to fowl paralysis will be made. This should increase the rate of establishing highly or completely resistant stock.

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