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Studies on the Origin and Transmission of Fowl Paralysis (Neurolymphomatosis) by Blood Inoculation

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SUMMARY

1. Forty two chicks were obtained from eggs hatched from fowls showing visible clinical symptoms of fowl paralysis.

2. The blood from 22 of these chicks was inoculated into 527 healthy day old chicks with the exception of two inoculation periods, at ten day periods until 28 inoculations falling into fourteen ten day periods were completed and the donors had reached the age of 136 days. Five hundred and seven chicks were maintained as controls.

3. The blood was transfused immediately without any treatment.

4. A total of 91 birds or 17.1% developed fowl paralysis in the inoculated group, whereas only 18 or 3.55% developed the disease in the control birds.

5. The results also show that there were three periods of age of the donors at which the blood more readily transmitted the disease to the recipients; (a) 20 to 30 days, (b) 50 to 60 days, and (c) 110 to 120 days.

6. These studies indicate that fowl paralysis produced artificially by intravenous inoculation occurred somewhat earlier than natural transmission. Twenty-five cases occurring over a period of 4 months in the inoculated birds before any case appeared in the control birds.

7. The data are also presented which show that the eye form (both inoculated and control) of fowl paralysis develops much later than other forms.

8. In these studies there were no blood changes involved in the fowl paralysis cases.

9. The data also shows that leucosis and tumor formations were not involved to any extent in these investigations. See Figures 10 and 11.

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INTRODUCTION

The Veterinary Department of the Missouri Experiment Station has been making studies on fowl paralysis for a number of years and has from time to time reported in the Missouri Experiment Station annual reports certain observations on this disease. For the past year certain intensive studies have been made on blood transmission of fowl paralysis, and at the present time such portions of the results will be reported as appear complete. Other questions are still under investigation and the results will be reported at a later date.

One of the difficulties in the past concerning progress in the investigation of fowl paralysis has been that of obtaining the virus regularly in a transmissible form in a high percentage of cases. A second difficulty has been the fact that some investigators have maintained that fowl paralysis and leucosis were due to the same virus, while others have held that the two diseases are distinct entities. In any case it is generally agreed that the etiological agent or agents is a virus.

Considerable evidence also has been produced that indicates fowl paralysis is transmitted through the egg from infected fowls to the baby chick when hatched.

It would appear from a study of the literature that no comprehensive control experiments have been carried out on the transmission of fowl paralysis by blood inoculation. In no case have any investigators attempted to transfuse the fresh whole blood from one fowl to another without first subjecting the blood to some form of treatment and a delayed injection. Johnson[5]* gives some data on blood transmission of leucosis and lymphomatosis of fowls in which only a small number of birds were used for the transmission and in which the author had under consideration several different disease manifestations. Furth[4] states that "Neurolymphomatosis is a neoplastic disease allied to leucosis and sarcoma, but it is not produced by the agent that causes erythroleukosis and myeloid leucosis.

*Superscript numerals refer to references, page 18.

Fig. 1.—Battery cages in which experimental birds were confined. The inoculated birds in one side of each battery, and the control birds on the other side.

Neither does the agent of neurolymphomatosis produce erythroleukosis or myeloid leucosis." He further states "The transmitting agent circulates in the blood in high concentration throughout the course of the illness. Blood cells readily transmit the disease but plasma does not." Durant[1] and McDougle attempted to transmit fowl paralysis by blood and tissue inoculation to chickens and guinea pigs with negative results. It should be pointed out in this connection that the donors were older birds.

In the studies here presented the authors have assumed that fowl paralysis is a distinct entity caused by a specific virus and indicated by pathological changes produced in the nervous system, in the eyes, and possibly tumor formations such as observed in the ovary.

The changes occurring in the nervous system are loss of striation, enlargement, discoloration, and edema in the nerve trunks of the affected organs or in localized areas of the nerves.

The basis for this study was the investigation reported by Durant[2] and McDougle in Bulletin 387, Missouri Experiment Station, page 95, 1937. This report showed that 50% of the chicks hatched from visibly affected birds showing the eye form of fowl paralysis developed the disease in from 57 to 171 days.

Further experiments not yet reported showed that 60% of chicks from affected birds developed some form of paralysis in the third

generation. The time, however, for development was somewhat longer, 171 to 337 days.

With this information as a basis it was decided to test the infectivity of the blood of chicks hatched from hens affected with visible clinical symptoms of fowl paralysis. It was reasonable to assume that at least half of the chicks hatched from infected birds would in time develop fowl paralysis and presumably might at some time have the virus in the circulating blood in sufficient quantities for transmission at some time between hatching and the development of visible evidences of fowl paralysis.

EXPERIMENTAL PROCEDURE

Experimental Birds Furnishing Eggs

From an infected flock a group of 14 White Leghorn pullets were selected for the experiment. Most of these birds showed varying degrees of eye lesions of fowl paralysis.

These pullets were mated to healthy White Leghorn males. Thirty-two eggs were obtained for the first incubation and 12 living chicks were hatched. From the second and third hatches 30 chicks were obtained, making the total number of chicks from the 14 hens, 42. Twenty-two chicks were selected from this group to test the infectiousness of the blood. The remainder were kept for observation. All 42 were confined in a room separate from all other fowls.

Technic of Inoculating Baby Chicks

When the 22 chicks were 2 days old, one-half of a c. c. of blood was drawn from the brachial vein or heart of each chick. A short-bevel, 27-gauge needle, or a 23-gauge needle, and 2 c. c. syringe was used for obtaining the blood. Each sample of the blood before it had had time to clot was injected intravenously in equal portions into the two White Leghorn baby chicks.

At the same time an equal number of chicks of the same age and source were placed in opposite cages to those inoculated. Subsequent inoculations were made in exactly the same manner within every 10 day period (with the exception of the 9th period) until 28 inoculations falling into 14 ten day periods were completed (Table 2), and the birds furnishing the blood had reached the age of 136 days. Five hundred and twenty-seven chicks received blood and 507 served as controls for the inoculated birds. The period of time covered for these 28 inoculations was 4½ months. During the course of the 4½ months of inoculations 3 birds of the 22 which furnished the blood died. One of these succumbed just after the first inoculation.

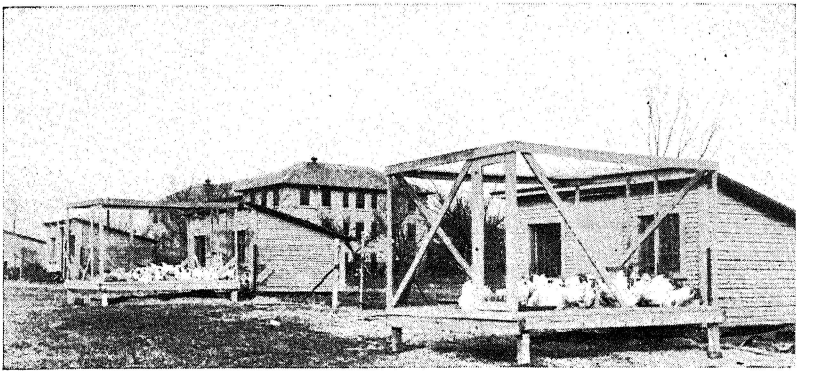

Fig. 2.—Outside brooder houses and runs to which the control birds were moved four months after the beginning of the experiment.

The veins of day-old chicks are exceedingly small and it is only with careful and precise technic that it is possible not only to draw the blood from such young chicks but to be able to transfuse this blood, before it is clotted, into the veins of healthy day-old chicks.

Down from the inner humeral surface of both wings and the distal tibial region of both legs was removed. This precaution is necessary, as occasionally more than one vein is used for a successful transfusion. In case of failure to get the blood intravenously, a very few birds received the blood subcutaneously (4 birds). No anti-coagulant or any chemical was used to prevent clotting of the blood as it might have some detrimental effect on the virus which may be present in the circulating blood of the birds under the test for infectiousness. In making the transfusions it is necessary to work rapidly, otherwise the injection is invariably fatal if the blood has started to clot.

Source of Chicks for Inoculations and Controls

The chicks that received the blood and controls were secured from a selected flock of purebred White Leghorn chickens. No fowl paralysis had been reported in this flock, though later indications were that there may have been a slight amount of fowl paralysis, since months later in the flock from which these chicks originated one case of the eye form of fowl paralysis was suspected in a male bird. Subsequent observations of the chicks hatched from this flock and used in these experiments indicated that fowl paralysis was present to a slight extent.

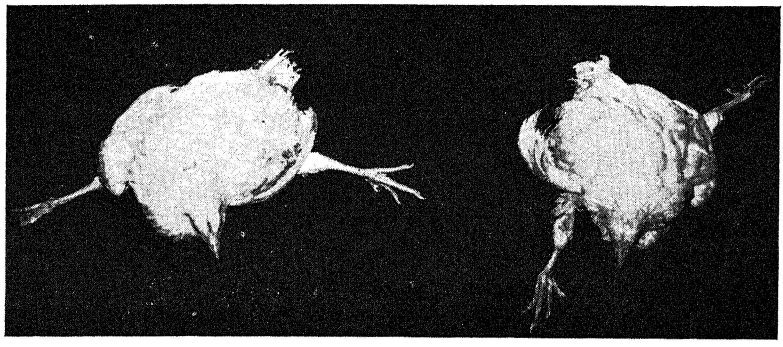

Fig. 3.—Inoculated birds, Nos. 1477 and 1499, showing clinical symptoms of fowl paralysis. Both birds on autopsy showed an extensive involvement of the sciatic nerves 87 days after inoculation.

Confinement of Experimental Birds

The birds receiving the blood and their controls were confined in small cages in a large laboratory. These cages were built in groups of 16 with four solid walls for each cage, each was covered with chicken wire, and the floor with hardware cloth (Fig. 1). The control birds were confined for 4 months or more adjacent to those receiving the blood, after which they were removed to brooder houses with raised wire runs (Fig. 2).

During the time the two groups of birds were confined in the batteries no special effort was made to prevent the transmission of disease from the inoculated birds to the control birds other than the arrangement of separate compartments. All the birds were fed and cared for exactly alike, receiving the same food by the same care-taker.

Examination of Experimental Birds

The inoculated and control birds were carefully examined twice a day or more often for clinical symptoms of fowl paralysis, or any other serious condition. All birds were removed as soon as any disability was observed and when the proper stage for post-mortem examination was reached they were autopsied and carefully examined for changes indicative of fowl paralysis. A systematic post-mortem examination was made, particular attention being given to the nervous system, especially those nerves which supplied parts which were causing altered function. All birds that developed typical eye changes were included in the fowl paralysis group. No bird died or was killed, during the course of the experiment,

that was not carefully autopsied and examined for gross evidences of fowl paralysis.

The following nerves were all carefully examined: brachial plexus, the brachial or median, ulnar, and other nerves of the wing; sacro-sciatic plexus, sciatic, tibial and other nerves supplying the legs; the vagus trunks and branches of this nerve supplying the crop; coeliac plexus and branches, and the cervical nerves. Specimens of nerve tissues showing evidences of fowl paralysis were preserved for histological study. Blood smears stained with Wilson's stain were preserved and studied from many recipients showing signs of paralysis.

Early in these investigations blood smears were studied and counted from *all* affected recipients but no pathological changes were observed. As time progressed with more birds developing symptoms a complete blood study could not be made of each suspect (either recipient or control); the blood studies therefore were discontinued. However, the blood of all anemic birds was studied to determine whether the birds were suffering from leucosis or some other dyscrasia.

EXPERIMENTAL RESULTS

Results of these experiments are shown in the following tables, charts and discussions.

Because of the somewhat uncertain status as far as the etiology of fowl paralysis and leucosis is concerned, complete and carefully compiled figures on tumors and leucosis have been included so that anyone who wishes may include or compute these changes along with the fowl paralysis records, thereby completing the picture as far as those are concerned who are of the opinion that fowl paralysis and leucosis are due to the same virus.

When interpreting the results of the blood inoculations the authors have made no attempt to demonstrate histologically by destruction of the fowl and examination of the nerve tissue microscopically the presence or absence of fowl paralysis. They have depended entirely on clinical symptoms and manifestations of the disease followed by post-mortem examination of the visibly affected birds either at death or when killed because of advanced stages of disease.

This is mentioned especially because of the fact that the records of the inoculated birds show that 8 of the inoculated birds developed partial paralysis of the wings or neck and were removed from the experimental group as suspects of fowl paralysis. However, this partial clinical paralysis disappeared in a short time and an ex-

amination of the nerves of these birds was not made at the time. The records also show that none of the control birds of the same age developed this partial paralysis. It is possible that blood containing the virus of fowl paralysis may cause a light form of the disease, producing temporary symptoms that disappear (Fig. 4). If this be true the number of birds actually affected with fowl paralysis from the inoculations in these experiments might be increased from 91 to a total of 99 birds. It is true that none of the 8 birds mentioned, however, showed any evidence of fowl paralysis at autopsy, which was carried out several months after they had shown the transient symptoms of partial paralysis.

Fig. 4.—Two inoculated birds, Nos. 2729 and 1625, showing clinical symptoms of paralysis of a transient form affecting the wings and neck.

BLOOD STUDIES

Blood preparations and studies have been made throughout the course of these investigations.

The method of blood count used was reported by Durant[3] and McDougle in which a system of staining and classification, that appears to be quite accurate, was developed. The blood smears were stained with Wilson's blood stain, after which 1000 cells were counted on each slide and were classified into one of four cell types. Although quite a laborious procedure, this type of counting appears to give a very accurate picture of the blood of fowls in health or disease.

As a basis for comparison, stained blood smears were studied from 16 healthy White Leghorn birds, using the method of cell classification as explained. The studies were started when the chicks were three days of age and repeated at approximately 10 day intervals until the birds were mature or in production.

NO SIGNIFICANT BLOOD CHANGES IN AFFECTED BIRDS

Because of the large number of fowls involved it was not possible to make a detailed study or record but sufficient evidence has been accumulated which indicates that the form of fowl paralysis with which this investigation is dealing is not indicated by any specific blood changes which are detectable by ordinary methods of blood examination (Table 1).

TABLE 1.—A COMPARISON OF THE BLOOD COUNT OF 20 BIRDS AFFECTED WITH FOWL PARALYSIS, AND 20 CONTROL (HEALTHY) BIRDS OF IDENTICAL AGES.

	From Paralysis Birds	From Normal Birds
Normal R.B.C.	977.25	973.95
Microcytes	.05	.05
Primitive R.B.C.	.05	.05
Poikeliocytes	.15	.15
Non-nucleated R.B.C.	.05	.00
Lymphocytes	9.15	10.50
P.M.N.E. Rods	2.10	1.60
P.M.N.E. Granules	1.25	.35
Basophils	.05	.30
Transitionals	.00	.05
Platelets	9.70	13.00
Baskets	.20	.00
Total	1000.00	1000.00

Table 1.—Would seem to indicate that there were no significant changes in the blood of birds affected with fowl paralysis in these investigations.

SUMMARY HISTORY OF THE 22 BIRDS FURNISHING THE BLOOD AND THEIR 20 CONTROLS IN THESE EXPERIMENTS

Of the 42 birds (see page 5) comprising the 22 donors or those furnishing the blood for the experiment and 20 control birds, all of which were hatched from visibly affected hens, 12 of the 22 donors are still living. Eight or 36.36 per cent have shown visible symptoms or lesions of fowl paralysis and yet 19 of the 22 birds apparently carried the virus, as chicks developed fowl paralysis after blood from these donors was injected intravenously into them. The remaining birds of this group are now 312 and 304 days of age. The possibilities are that some of those which apparently have furnished infected blood may later develop typical symptoms and manifestations of fowl paralysis. If this does not occur it would seem to indicate that many birds carry the virus of fowl paralysis in the circulating blood without ever showing any symptoms of the disease and may serve as a potential reservoir of the infection.

Of the 20 control birds only 3 or 15 per cent have developed symptoms or lesions of fowl paralysis. These 20 control chicks, however, were hatched from eggs which were laid later than those eggs from which the chicks were hatched that furnished the blood, and the possibility exists that the first eggs laid by infected hens may carry more of the infectious agent than later eggs.

In a study of Table 2, which is a summary of the results of the blood inoculation, several interesting things may be observed.

The table is arranged so that the age of the donor is given at the time of inoculation, and showing the number of birds inoculated and the results of the inoculation of not only fowl paralysis but of leucosis and tumors as well. The two columns showing the development of fowl paralysis in the controls and in the inoculated birds have been brought together for direct comparison. This table shows that a total of 91 birds (17.1%) developed fowl paralysis in the inoculated group, whereas only 18 (3.55%) developed the disease in the control birds. Five cases of paralysis developed in the in-

TABLE 2.—THE NUMBER OF RECIPIENTS AND CONTROLS DEVELOPING FOWL PARALYSIS, LEUCOSIS, OR TUMORS WITHIN 10 MONTHS FROM TIME OF INOCULATION.

Age of Donor at time of inoculation	Total	Recipients Developing			Controls Developing			Total
Days	Recipients	Tumors	Leucosis	Paralysis	Paralysis	Leucosis	Tumors	Controls
2	4	0	0	0	0	0	0	3
7	18	0	1	3	0	0	0	18
10	16	1	0	2	0	0	0	16
18	25	1	0	0	1	1	0	25
22	15	1	0	4	2	0	1	15
25	21	1	0	4	3	0	1	21
29	16	0	0	5	1	0	0	16
32	20	0	0	3	0	0	0	20
39	24	0	1	0	1	0	0	24
40	18	0	1	5	1	0	0	18
46	19	1	0	2	0	0	1	13
47	18	0	0	3	1	0	0	18
54	38	1	1	10	0	0	0	29
61	31	0	0	9	0	0	0	31
69	18	1	0	1	0	1	0	18
76	18	1	1	2	1	0	0	18
77	*12	0	0	1	0	0	1	10
92	*18	0	0	2	1	0	0	18
94	18	0	0	1	1	1	0	18
100	17	0	0	2	0	0	0	17
108	18	0	0	3	1	0	0	18
109	18	0	0	6	1	0	0	18
114	18	0	0	6	1	0	0	18
115	18	0	0	4	0	1	1	18
121	15	0	0	3	1	0	0	15
123	18	0	0	3	0	0	0	18
129	20	0	0	4	1	0	0	18
136	18	0	0	3	0	0	0	18
Total	527	8	5	91	18	4	5	507

*14 to 70 days of age when inoculated.

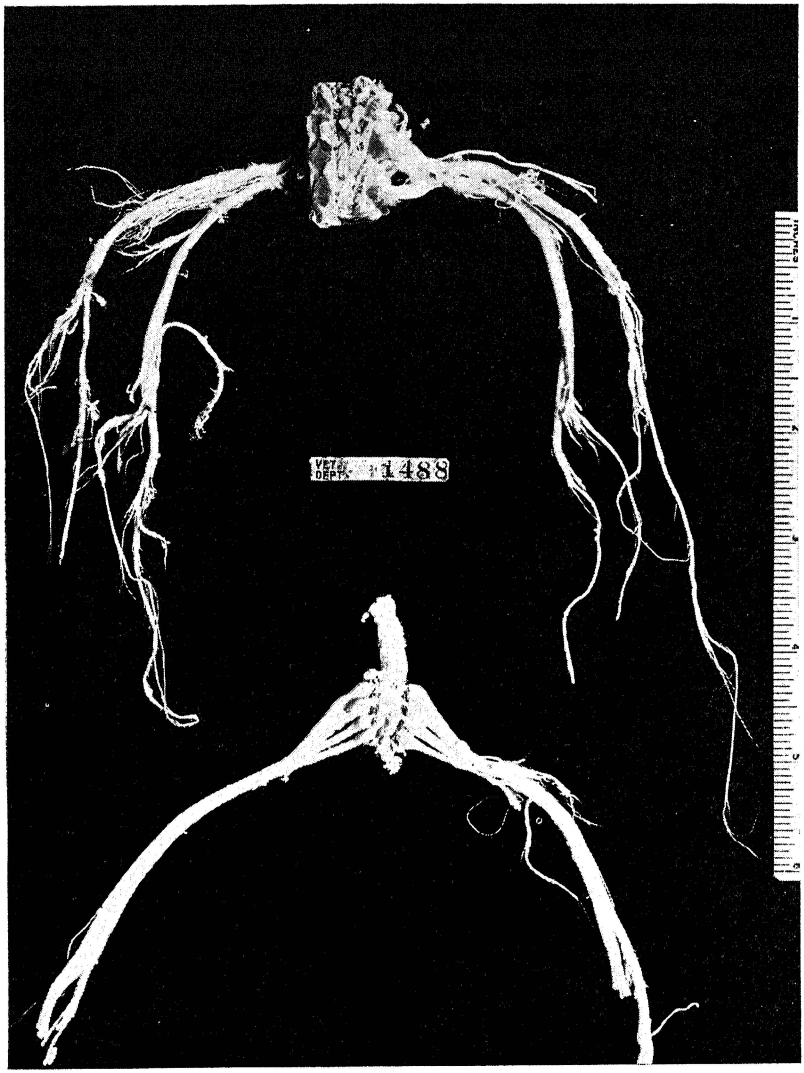

Fig. 5.—Brachial (above) plexus, and sciatic (below) plexus with their branches of bird No. 1488 affected with fowl paralysis 80 days after inoculation as a day-old chick. All nerves involved were greatly enlarged as indicated by comparison with scale of inches at right.

oculated birds in the first ten days of age of the donors, and none developed the disease in the control group of the same age. Furthermore, when the donors were 54 days old, 10 (26.31%) of the 38 inoculated birds developed fowl paralysis, whereas of the 29 controls of the same age none developed the disease. Likewise when the donors were 61 days old, 9 (29.03%) out of the 31 inoculated birds developed fowl paralysis and none of an equal number of controls of the same age developed the disease.

Filtrate of Affected Nerves Fails to Transmit The Disease

One of the experimental birds (No. 1487-88) which developed fowl paralysis from the blood inoculations should be mentioned especially because of the nature of the pathological changes observed in the nerves. The brachial, and sacro-sciatic plexuses with their branches were enormously enlarged, oedematous and easily broken.

Because of the unusual nature of the changes it was thought advisable to test the infectivity of a filtrate from these affected nerves. The nerves were ground in sterile saline and filtered through a Seitz filter. Four three weeks old White Leghorn chicks were given one c.c. intravenously; and four 3 day old White Leghorn chicks were given .5 c.c. intravenously. Eight controls were maintained at the same time with the 8 inoculated birds. Ten months has elapsed since these inoculations. Two of the inoculated birds have died from other causes, (1 Pullorum disease, and 1 crop bound and air sac infection). One control has died (Peritonitis) but none has developed fowl paralysis, leucosis or tumors during that period in either group.

Leucosis in Inoculated and Control Birds

A further study of Table 2 shows that five birds of the 527 that were inoculated developed leucosis, which is only one more than occurred in the 507 controls—indicating that the incidence of the disease was not influenced by the inoculation.

Pendulous or Paralyzed Crop Due to Fowl Paralysis

During the course of these investigations several cases of pendulous crops were encountered. Fowl paralysis was not at first suspected, but due to the careful routine autopsies of each case involved it was discovered that a branch of the left vagus nerve which supplies the crop was greatly enlarged and typical of nerves affected with fowl paralysis.

It should be pointed out that all cases of pendulous crop are not due to fowl paralysis, but a diligent, systematic examination of the nervous system should be made as the pendulous crop may be a clinical symptom of fowl paralysis.

Possible Relation Between Fowl Paralysis and Tumor Formation in Fowls

It would appear possible from observed results that the true fowl paralysis virus might cause tumor formation in the ovary, adrenals and other organs. It has been observed in a few cases of tumors (3 out of 8) which have occurred in the course of these studies of fowl paralysis that enlargement of the nerves adjacent to the ovary and the ovary are simultaneously affected. This is merely an observation and there is not sufficient experimental data to draw a definite conclusion. Attention is merely called to such a possibility because of certain data which are presented on tumor formation in Table 2. Of the eight inoculated birds developing tumors, three or 37.50% of the birds had tumors of the ovary.

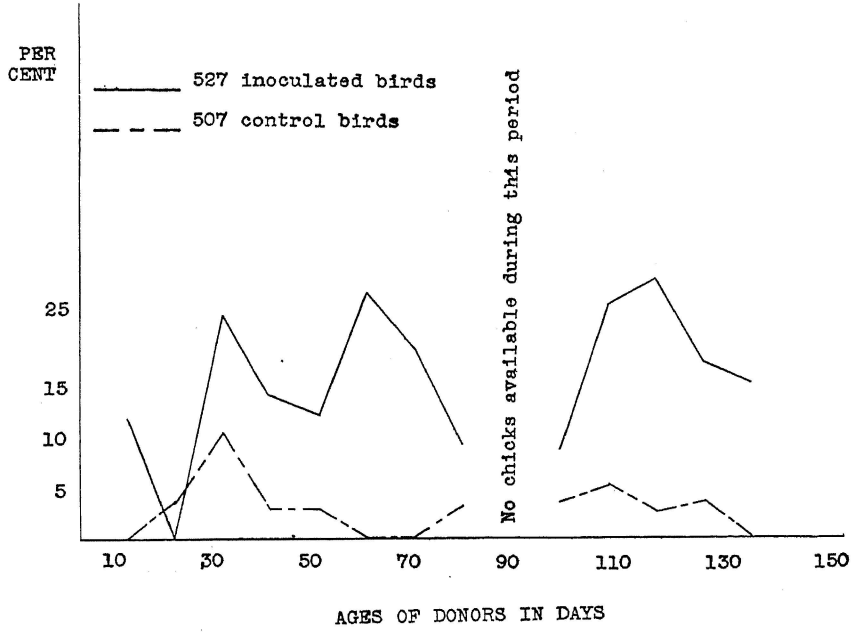

Fig. 6.—This chart shows the ages at which the birds furnishing the blood transmitted the disease. During the 140-day period of inoculation there were three definite ages at which the disease was most readily transmitted. The first period was 20 to 30 days, the second 50 to 60, and the third 110 to 120 days.

Fig. 6 is a graphic illustration to show the ages at which birds furnishing blood transmitted the disease. During the period of 140 days there were three distinct ages at which the blood appeared to be more infectious. The explanation for this phenomenon cannot be ascertained from the data presented, but may have importance for the future investigation of fowl paralysis, if these results would prove in the future to be constant. Plans are now going forward to check these periods to see if the high infectivity can be duplicated.

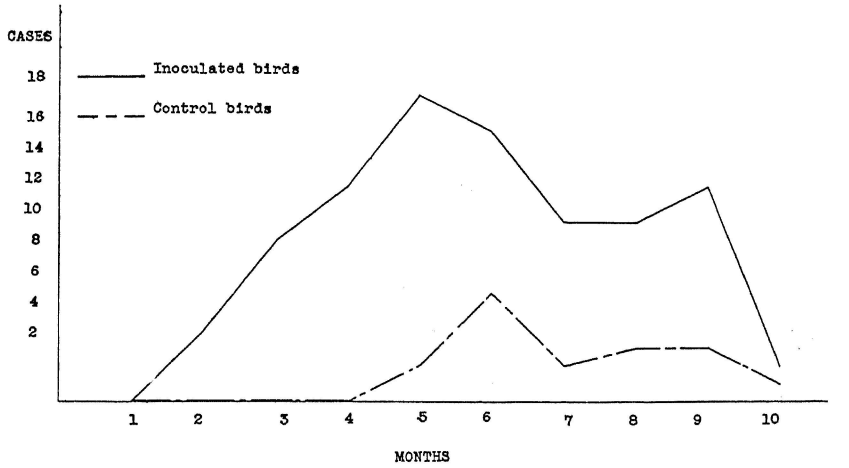

Fig. 7.—Number of days from inoculation until definite symptoms or autopsy showed the birds to be affected with fowl paralysis.

In Fig. 7 two curves are shown representing the number of days from inoculation until definite symptoms or autopsy showed birds to be affected with fowl paralysis. These curves, indicate that fowl paralysis produced artificially by intravenous inoculation occurred somewhat earlier than natural transmission. Over a period of four months, 25 cases of fowl paralysis occurred in the inoculated birds before any cases appeared in the control group. The peak of the disease for the inoculated birds also occurred about one month earlier than the peak for the control birds.

In Figs. 8 and 9 are shown by means of curves the length of time for the different forms of fowl paralysis to develop in birds—particular attention being given to the differentiation of the eye form as compared to other forms of fowl paralysis.

In Fig. 8 in the inoculated birds, as well as in Fig. 9 of the control birds, it will be observed that the eye form developed much later than the other common forms of fowl paralysis, and that there was no difference in the time of appearance of the eye form in the

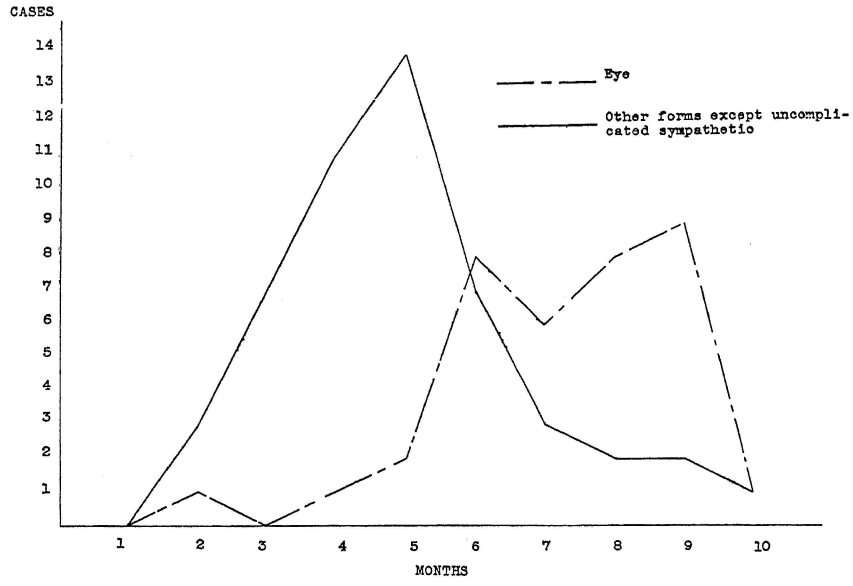

Fig. 8.—Length of time to develop different forms of fowl paralysis in artificially infected birds.

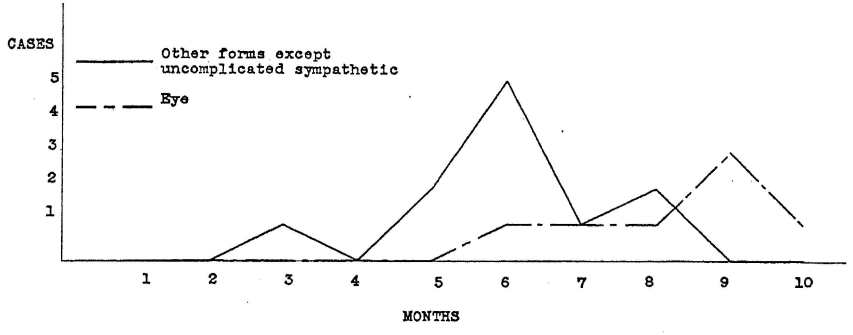

Fig. 9.—Length of time to develop different forms of fowl paralysis in control birds.

inoculated and control birds, whereas the other forms of fowl paralysis occurred much earlier in the inoculated birds than in the control birds, and that the peak of the other forms occurred one month earlier in the inoculated birds than in the control group.

Fig. 10 is a graphic illustration of the incident of leucosis in the groups of birds (both inoculated and controls) on this experiment. These curves show no difference in the incident of leucosis since Table 2 shows that there were only four cases in the control group, and five cases in the inoculated birds.

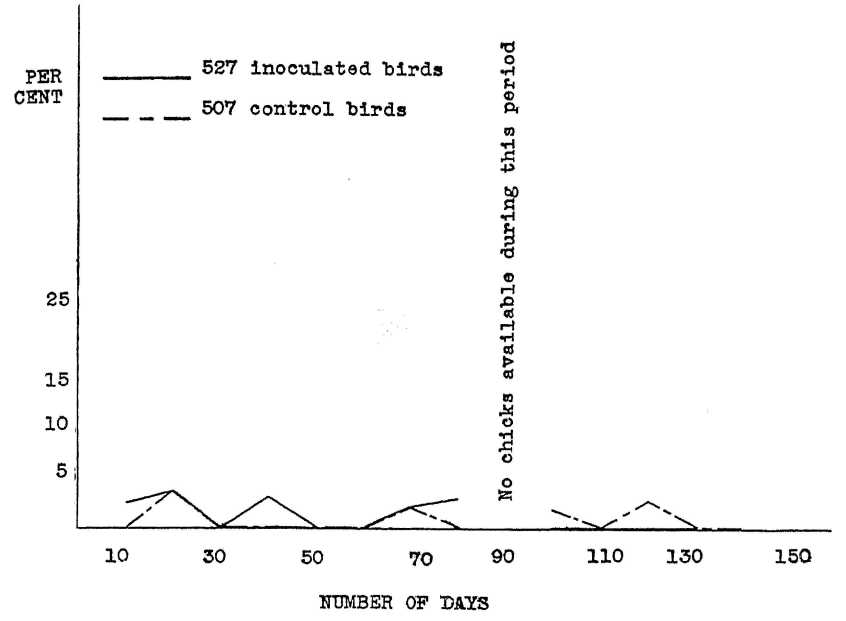

Fig. 10.—This chart shows the incident of leucosis in the group of birds (both inoculated and controls) on this experiment. There was no significant difference in the incident of leucosis.

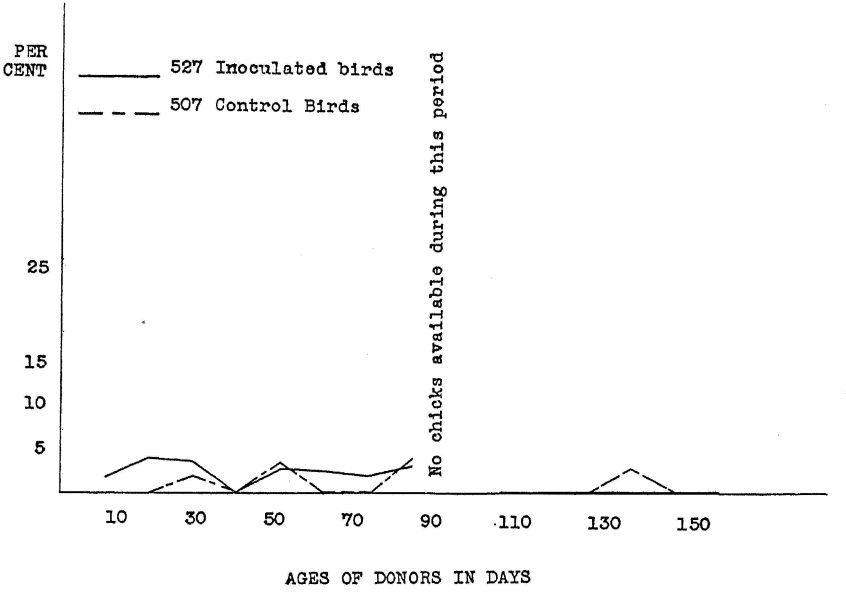

Fig. 11.—This chart shows the incident of tumor formation in the group of birds (both inoculated and controls) on this experiment. There was a slight difference in the incident of tumor formation between the controls and the inoculated birds.

Fig. 11 shows the incident of tumor formation in the group of 527 inoculated birds, and 507 controls. Eight birds of the in-

oculated group developed tumors whereas five of the controls were affected. This difference in tumor formation is apparently not significant when the total number of birds is considered.

REFERENCES DEALING WITH SPECIFIC SUBJECT OF BLOOD TRANSMISSION OF FOWL PARALYSIS

1. Durant, A. J., and McDougle, H. C., 1936. *Fowl Paralysis or Neuritis of Fowls.* Work of the Missouri Agricultural Experiment Station. Bulletin 370, page 84.

2. Durant, A. J., and McDougle, H. C., 1937. *Fowl Paralysis.* Work of the Missouri Agricultural Experiment Station. Bulletin 387, page 95.

3. Durant, A. J., and McDougle, H. C., 1937. *Fowl Leucosis.* Work of the Missouri Agricultural Experiment Station. Bulletin 387, page 96.

4. Furth, J., and Breedis, Chas., 1935. *Lymphomatosis in Relation to Fowl Paralysis.* Arch. of Path. 20: 379-428.

5. Johnson, E. P., May, 1934. *The Etiology and Histogenesis of Leucosis and Lymphomatosis of Fowls.* Virginia Polytechnic Institute, Virginia Agricultural Experiment Station. Technical Bulletin 56.

BIBLIOGRAPHY OF GENERAL FIELD OF FOWL PARALYSIS

Bayon, H. P.
Acute Neuro-lymphomatosis gallinarium in a strain of Rhode Island Red fowls. Sept. 5, 1931. Vet. Rec. 11. 907-911.

Bayon, H. P.
The pathogenesis of Neuro-lymphomatosis gallinarium and similar forms of fowl paralysis. (1932) Vet. Rec. 12, No. 17, 457-467.

Beach, J. R.
Paralysis of fowls (Neuro-lymphomatosis gallinarium). North Amer. Vet., 11 (1930) No. 10, 49-54.

Beach, J. R.
Experiments in the transmission of fowl lymphomatosis by inoculation. Poultry Sci. 17: (January, 1938), 67-71.

Beaudette, F. R.
Fowl Paralysis. N. J. Agr., 15 (1933) No. 3, 2.

Beaudette, F. R., and Hudson, C. B.
Fowl Paralysis. N. J. Sta. Hints to Poultrymen 19 (1931) No. 11, 4.

Biely, J., Palmer, V. E. and Lerner, I. M.
Fowl paralysis (Neurolymphomatosis gallinarium) in chicks under three months of age. Canad. Jour. Research, 8, (1933) No. 4, 305-311.

Biely, J., and Palmer, V. E.
Observations on the gonads of male birds affected with fowl paralysis (neurolymphomatosis gallinarium). Canad. Jour. Research. 7 (1932), 293-299.

Biely, J., Palmer, E., and Asmundson, V. S.
Inheritance of resistance to fowl paralysis (Neurolymphomatosis gallinarium) II and a significant difference in the incidence of fowl paralysis in two groups of chicks. Canad. Jour. Research 6 (1932) No. 4, 381-386.

Biely, J., Asmundson, V. S.
Inheritance of resistance to fowl paralysis (neurolymphomatosis gallinarium). I Difference in susceptibility. Canad. Jour. Research 6 (1932) No. 2, 171-176.

Biely, J., Palmer, V. E., and Lerner, I. M., and Asmundson, V. S.
Inheritance of resistance to fowl paralysis (neurolymphomatosis gallinarium). Science 78 (1933) No. 2011, 42.

Biely, J., and Palmer, V. E.
The etiology of fowl paralysis. (A review of literature) Vet. Rec., 12 (1932) No. 44, 1302-1309.

Blakemore, F.
Leucocytes of fowl blood with special reference to fowl paralysis. Vet. Rec., 14 (1934) No. 16, 417-422.

Blakemore, F., and Dalling, T.
Some recent observations on Fowl Paralysis (neurolymphomatosis). Proceedings Seventh World's Poultry Congress and Exposition (1939). 282-286.

Blount, W. P.
Vitamin B and fowl paralysis. Vet. Jour., 88 (1932) No. 7, 289-299.

Blount, W. P.
Studies on fowl paralysis. III. Gastronomic enteritis. Vet. Jour., 88 (1932) No. 6, 236-240.

Bthke, R. M., Record, P. R., and Kennard, D. C.
A type of nutritional paralysis affecting chicks. Poultry Science. 10 (1931) No. 7, 355-368.

Calif. Dept. of Agr., Mo. Bul., 22 (1933) 243.

Cole, R. K.
Vitamin E and Avian neurolymphomatosis. Science n s 88: 286-7, Sept. 23, 1938.

Conn., Storrs, Sta. Bul., 136 (1925) 439.

Conn. Sta. Bul., 142 (1926) 168, 180.

Dalling, T.
Fowl paralysis. Vet. Rec., 11, No. 36, 638 (1931).
Doyle, L. P.
The differential diagnosis of leg weakness in chicks. Poultry Science, 1931. X, 393.
Doyle, L. P.
Neuritis or paralysis of fowls. Poultry Science. 8 (1929) No. 3, 159-160.
Doyle, L. P.
Jour. A. V. M. A. LXVIII, p. 622.
Doyle, L. P.
Neuritis and paralysis in chickens. Jour. A. V. M. A., 72 (1928) No. 5, 585-587.
Emmel, M. W.
Etiology of fowl paralysis, leucosis, and allied conditions. Vet. Med., 30 (1935) No. 2, 68-70.
Emmel, M. W.
Importance of endotoxin of salmonella aertrycke in the development of fowl paralysis. A. V. M. A. 90: 749-54, June, 1937.
Emmel, M. W.
Hemocytoblastosis and its relation to the development of fowl paralysis. Jour. A. V. M. A., 88 (1936) No. 1, 45-50.
Emmel, M. W.
Fowl leukemia and paralysis. V. S. Egg and Poultry Mag., 42 (1936). No. 2, pp. 82, 126-127.
Emmel, M. W.
Etiology of fowl paralysis, leukemia, and allied conditions in animals; pathologic manifestations of the causal micro-organisms in the fowl. Fla. Agr. Exp. Bul., 293: 18-23, 1936.
Emmel, M. W.
The etiology of fowl paralysis, leukemia, and allied conditions in animals. Fla. Agr. Exp. B. 305: 1-66, 306: 1-42, 1936.
Emmel, M. W.
Etiology of fowl paralysis, leukemia, and allied conditions in animals; history and a bacterial theory of the etiology of these diseases. Fla. Agr. Exp. Bul. 284: 1-18, 1935.
Emmel, M. W.
The etiology of fowl paralysis (neurolymphomatosis gallinarium) Pappenheim, leucosis, and allied conditions in the domestic fowl. Jour. A. V. M. A., 85 (1934) No. 1, 96-97.
Emmel, M. W.
Etiology of fowl paralysis, leukemia, and allied conditions in animals; intestinal flora of chickens infected with enteritis associated with intestinal parasitism. Fla. Agr. Exp. Bul. 293: 5-17, 1936.
Feldman, W. H., Olson, C.
Pathology of Spontaneous leucosis in chickens. Jour. A. V. M. A., lxxxll (1933), N. S. 35 (6), pp. 875-900.
Feldman, W. H.
The so-called lymphoid hyperplasias of animals. Jour. A. V. M. A. 30: 3, 294-312.
Fenstermacher, R.
Jour. A. V. M. A. N. S. 33, 791.
Fenstermacher, R.
Lymphomacytoma and fowl paralysis. A. V. M. A. 88 (1936) No. 5, 600-613.
Fenstermacher, R.
Familial incidence of lymphocytoma in three generations of the domestic fowl. Jour. A. V. M. A. N. S. 37, No. 6, pp. 863-876.
Florida Sta. Rept. (1934) pp. 37, 41, 43, 45, 46.
" " " (1932) pp. 51, 54, 55.
" " " (1931) pp. 55-56.
" " " (1929) pp. 40-42.
" " " (1928) pp. 79-85.

Furth, J.
Observations on fowl paralysis (neurolymphomatosis). Soc. Exp. Biol. and Med. Proc., 31 (1934) No. 8, 921-923.
Furth, J., and Breedis, Chas.
Lymphomatosis in relation to fowl paralysis. Arch. of Path. 20: 379-428 (1935).
Galloway, I. A.
Proc. Roy. Soc. Med., 22, 1167 (1929).
Gibbs, C. C.
Observations and experiments with neurolymphomatosis and the leukotic diseases. Mass. Agr. Bul., 337: 1-31, 1936.
Gibbs, C. S., and Johnson, C. G.
Leucosis and avian paralysis. Mass. Agr. Exp. Sta. Bul. No. 315, p. 78.
Gibbs, C. S., and Johnson, C. G.
Differentiation of the pathological cell in neurolymphomatosis from lymphocytes of the blood. The differentiation of neurolymphomatosis from lympholeukosis. Mass. Agr. Exp. Sta. Bul., 327, 15: 244-251.
Gildow, E. M., Williams, J. K., and Langsman, C. E.
Transmission of fowl paralysis (neurolymphomatosis gallinarium) Poultry Science. 14 (1935) No. 5, 317.
Gildow, E. M., and Others.
Transmission of fowl paralysis (lymphomatosis). Poultry Science, 15 (1936) No. 5, 244-248.
Gildow, E. M.
New Hamp. Agr. Exp. Sta. Bul. No. 227.
Hall, G. E., and King, E. J.
Calcium and phosphorus metabolism in the chicken. II. Range paralysis. Poultry Science. 10 (1931) No. 5, 259-268 or No. 6.
Hall, Norman
Polyneuritis in fowls. Vet. Jour., 88, 337-340.
Henderson, E. W.
Influence of various nutritional factors on blindness and range paralysis in chickens. 1931. Iowa Agr. Exp. Sta., Annual Report 37.
Idaho Sta. Bul., 205 (1934) 28-34.
Idaho Sta. Bul., 430 (1935). *Factors in the spread of fowl paralysis.* pp. 18-21 (36-38).
Indiana Sta. Rept. (1928) 61-64.
" " " (1927) 10, 54, 57.
Iowa Sta. Rept. (1934) pp. 60, 61, 142.
" " " (1933) pp. 41, 42, 114.
" " " (1932) pp. 30, 31, 90.
" " " (1931) (22-31) (33-35-37).
" " " (1931) 36, 37, 88, 89.
Johnson, E. P., and Conner, B. V.
Blood studies on fowls with various forms of lymphomatosis (fowl paralysis). Jour. A. V. M. A. 83 (1933) No. 3, 325-343.
Johnson, E. P.
The etiology and histogenesis of leucosis and lymphomatosis of fowls. Va. Sta. Tech. Bul., 56 (1934) p. 32.
Johnson, E. P.
A study of lymphomatosis of fowls (fowl paralysis). Virginia Sta. Tech. Bul. 44 (1932) 22.
Johnson, E. P.
Present Status of Fowl Leucosis (Fowl Paralysis). Proceedings Seventh World's Poultry Congress and Exposition (1939) 286-288.
Jungherr, E.
Observations on the macroscopic diagnosis of fowl paralysis. Poultry Science 12 (1933) No. 3, 184-188.
Jungherr, E. (1939).
Wheat germ oil in control of fowl paralysis and kindred diseases. Vet. Med., 34, No. 1, p. 8.

Jungherr, E.
Studies on fowl paralysis; transmission experiments. Conn., Storrs Agr. Exp. Bul. 218: 1-47, 1937.
Jungherr, E.
Studies on fowl paralysis. I. Diagnosis. Storrs Sta., Bul. 200 (1934) 28.
Jungherr, E.
Etiological and diagnostic aspects of the fowl paralysis problem. Jour. A. V. M. A. (1935) No. 3, 424-432.
Jungherr, E., and Landauer, W.
Studies on fowl paralysis: Condition resembling osteopetrasis (marble bone) in the common fowl. Conn. Storrs Agr. Exp. Bul. 222: 1-34, 1938.
Jungherr, E.
Neurolymphomatosis Phasianorum. J. A. V. M. A., Jan. 1939. p. 49.
Kaupp, B. F.
A contribution to the study of Tumors of the Domestic Fowl. Vet. J. London, 1921, n. s.; 827-830.
Kaupp, B. F. (1921).
Journal Am. Assoc. Inst. and ? Poultry Husb., VII, p. 25.
Lee, A. M.
Some lesions associated with fowl paralysis. Jour. A. V. M. A., 78 (1931) No. 2, 203-10.
Lee, A. M., and Elder, C.
Fowl Paralysis. Wyo. Ext. Cir. No. 31, 1930.
Madsen, D. E.
Effect of iritis of breeding hens on their progeny. Poultry Sci. 16: 393-397, Nov., 1937.
Marginson, G. C., and McGaughey, C. A. (1931).
Field observations on the spread of fowl paralysis by eggs and young chicks. Vet. Rec., XI, p. 573.
Mass. Sta. Bul., 305 (1934) 56-59.
" " " 293 (1933) 56-58.
" " " (1932) 239-242.
Mathews, F. P., and Walkey, F. L. *Lymphodenomas of the common fowl.* Jour. Cancer Res. XIII (1929), 4, p. 383.
May, H. C., Fittsler, R. P., and Goodner, K. (1925).
Field observations and laboratory findings in Paralysis of the domestic fowl. Bul. 202, Agr. Exp. Sta. of R. I. State College, Dec. 1925.
Mayhew, R. L.
Studies on coccidiosis: Observations on paralysis with special reference to coccidial infections. Poultry Sci. 11:289-92, Sept. 1932.
Murray, Chas., Lee, C. D., Patterson, F. D., Wilke, H. L., and Henderson, E. H.
Studies on the so-called range paralysis in chickens. Ames, Iowa Rept. on Agr. Res. p. 60.
McGaughey, C. A.
The prevalence of fowl paralysis in England. Vet. Rec. (1930) X, 1143.
McGaughey, C. A., and Downie, A. W.
Preliminary report on an outbreak of fowl paralysis in England. Jour. Comp. Path. and Ther., 43 (1930) No. 1, 63-76.
McLennan, G. C.
Fowl paralysis: "Range paralysis"—Neurolymphomatosis gallinarium. Aust. Vet. Jour., 11 (1935) No. 2, pp. 42-62.
N. H. Sta. Bul., 270 (1933) 21-23.
" " " 262 (1932) 25-27.
" " " 250 (1930) 13, 14, 27.
New Jersey Sta. Rept. (1934) pp. 34, 80, 84.
Niemann, K. W.
Autopsy findings in field cases of fowl paralysis. Jour. A. V. M. A., 75 (1929) No. 1, 38-50.
Oakley, C. L.
Lymphomatosis. Proc. Roy Soc. Med., XXVIII (1935) 999-1002.

Ohio Sta. Bul., 284 (1935).
" " " 516 (1935) 78, 81, 83, 88, 89.
Olson, C.
Attempts to transmit fowl paralysis. (1937) Jour. Infect. Dis., 61, No. 3, 325-330.
Pappenheim, A. M., Dunn, L. C., and Cone, V. A.
Jour. Exp. Med. XLIX, pp. 63-86, and 87-102. (1929).
Pappenheimer, A. M., Dunn, L. C., and Cone, V.
A study of fowl paralysis (neurolymphomatosis gallinarium). Conn. Storrs Sta. Bul., 143 (1926), 185-290.
Patterson, F. D., Wilke, W. H., Murray, C., and Henderson, E. W.
So-called range paralysis of the chicken. Jour. A. V. M. A. 81 (1932) No. 6, 746-767.
Roberts, R. S.
The epidemiology of fowl paralysis as an indication of its mode of transmission. Vet. Jour., 91, 127-132 (1935).
Seagar, E. A.
The pathology of fowl paralysis, with some aspects of its cause and control. Vet. Jour., 89 (1933) No. 10, 454-473.
Seagar, E. A.
Cellular reactions in the blood in Neurolymphomatosis gallinarium (fowl paralysis). Wt. Jour. 89, 557.
Stafseth, H. J., and Mallman, W. L.
(1928) Poultry Science 8, 19.
Stafseth, H. J., and Johnson, E. P.
Mich. Agr. Exp. Sta. (1927). 40th Annual Rep., pp. 299.
Stafseth, H. J.
Fowl paralysis and the roup complex. Jour. A. V. M. A. 78 (1931) No. 3, 423-429.
Stafseth, H. J.
Jour. A. V. M. A., 1931, LXXVII, pp. 793-816.
Thomas, E. F.
Paralysis of the domestic fowl. Florida Sta. Report (1930) 54.
Thomas, J. C., and Hamilton, H. P.
Fowl Paralysis: An analysis of one-thousand cases. Vet. Jour., 91 (1935) No. 12, 526-536.
Tomhane, A. E., and Mumford, C. W.
Fowl paralysis. Del. Sta. Bul., 172 (1931) 25.
Twisselmann, N. M.
A study of the cell content of the blood of normal chickens, with special reference to comparative differential leucocytes counts made with supravital and Wright's staining technics.
Upp, Chas., and Tower, B. A.
The incidence of blindness and paralysis according to family. Poultry Science, 15: 421 (Aug. abstract).
Van Derwall, N., and Winkler, E.
The Neuritis outbreak in fowls at Barnesveld in 1921. 1924, Tijdscher. v. Vergelijk, Genesk, Leydon, 10, 34.
Warrack, G. H., and Dalling, F.
So-called fowl paralysis, also neuritis in chickens, range paralysis, neurolymphomatosis gallinarium. Vet. Jour., 88 (1932) No. 1, 28-43.
Welch, H.
Fowl Paralysis. Mont. Agr. Ext. Cir. 46 (1937).
Wilcke, H. L., Patterson, F. D., Henderson, E. W., and Murray, C.
The effect of the ration upon the incidence of so-called range paralysis. Poultry Science, 12 (1933) 226-232.
Williams, J. K., and others.
Some factors affecting the transmission of fowl paralysis. V. S. Egg and Poultry Mag. 44:24-27. Jan., 1938.
Wisconsin Sta. Bul. 430 (1935), pp. (116-121) (137-142) (144-149).
Cause of one type of paralysis.