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The Fowl Leukosis Complex

I. A review of the current literature on fowl leukosis †

C. D. Lee, D.U.M., M.S.*

A DISEASE complex commonly referred as range paralysis, fowl paralysis, neurolymphomatosis gallinarum, lymphomatosis, leucosis and a variety of other technical names has probably caused more losses to the poultry in this country during the past ten years than any single disease.

The terminology in many instances has been very satisfactory for some expressions of the disease but not satisfactory for a nomenclature of the complex as a whole. In an effort to clarify the nomenclature a group of poultry pathologists meeting at Federal Regional Poultry Disease Laboratory at East Lansing, Michigan adopted the following terminology:

Fowl Leukosis Complex

1. Lymphomatosis
   Ocular (eye type)
   Neural (paralysis)
   Visceral (tumor infiltrations)
   Osteopetrotic (enlarged bones)

Avian leukosis is an infectious disease due to a filterable virus. It is highly infectious and affects chickens, principally between the ages of four and eight months, the majority of cases occurring at about six months of age. Birds of all breeds are affected and no recoveries from this disease have been noted but temporary respite do occur. The disease is not caused by intestinal parasites or by any known nutritional factor.

Review of Literature

A review of the literature on fowl paralysis and the fowl leukosis complex especially that of more recent dates indicates primarily two different schools of thought as to the etiology of these conditions—the one on the etiologic unification of these conditions, and the other that of etiologic dissociation. The disease with its various manifestations presents many complex problems, and the combined work of many investigators over a long period of time may be necessary to settle the many questions now existing.

As aptly expressed by Junghur¹ the most important problem in this field centers around the etiologic unity of fowl paralysis, leucosis and certain neoplasms. And, if the etiologic unity can be established, control measures, either sanitary or genetic will be materially facilitated.

Early Reports

In view of the existant thoughts on etiology one may only review the literature at this time as each school of thought has presented rather convincing evidence in support of their theory. The first report or discovery of this disease was by Marek² in 1907. He, at that time, described a disease with symptoms of paralysis of legs and wings. He also described a definite gross lesion of thickening of the lumbar plexus in one of these birds. He did not

† In the summer issue of The Veterinary Student Dr. Lee will discuss the clinical manifestations of the Fowl Leukosis complex.

—The Editor

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advance an opinion as to the etiological agent.

The first report in this country was by Kaupp\(^3\) in 1921 from his observations made in eastern and southern states in 1914. The pathology described by Kaupp is that generally attributed to this disease. Kaupp studied that disease experimentally but was unable to arrive at any definite results. Repeated inoculation of heart, blood, liver, kidney, spleen, and various affected tissues into guinea pigs, rabbits, and young and old chickens were unsuccessful. In spite of these results Kaupp believed the disease infectious.

Van der Walle and Winkler\(^4\) in 1921 suggested the term neuromyelitis gallinarum for the disease and believed it was infectious and due to a filterable virus. May, Tittsler, and Goorer\(^5\), 1925, reported field observations and laboratory findings in paralysis of the domestic fowl. The pathologic description was incomplete. Aerobic and anaerobic cultures from tissues of affected birds failed to reveal infective organisms. Attempts to transmit the disease by association, contact, feeding, or by inoculation of affected tissues failed. They also concluded intestinal parasites, either worms or coccidia, were not the cause of paralysis.

**Due To Infective Agent**

Beach and Davis\(^6\) in 1925 described a form of paralysis which they attributed to chronic coccidiosis. Coccidia were later incriminated by workers in Kansas, Michigan and Wyoming. Doyle\(^7\) in 1926 completely described the symptoms and pathology of the disease as well as the histo-pathology of the brain, cord and peripheral nerves. Attempts to transmit the disease failed, but the author believed that the disease was due to an infective agent.

Pappenheimer, Dunn, and Cone\(^8\) in 1926 made extensive studies of this disease and termed it neurolymphomatosis gallinarum. Their work established a solid foundation for later studies by many investigators. The visceral lymphomata were associated in a certain percentage of cases. Evidence favoring this association was not accidental and was probably expressions of the same disease. They were able to transmit the disease by subdural or intramuscular injections of tissue suspensions from affected birds. They were also able to transmit the disease by filtrates of some tissues.

Ellerman\(^9\), 1913, differentiated intravascular erythroid and myeloid leukosis and an extravascular lymphoid type all of which were considered to be transmitted by a common etiological agent, a filterable virus.

**Transmissability**

Anderson and Bang\(^10\), 1929, expressed doubt as to the transmissability of lymphoid leukosis. Furth\(^11\), 1931, made comprehensive studies of erythroleukosis and anemia in fowls. He was able to transmit erythroleukosis and myeloid leukosis and showed that they may be transmitted freely by injection. The two types developed irrespective of the type used for infection.

Furth\(^12\) in 1931 showed that myeloid leukosis or erythroleukosis can be transmitted from one bird to the other by emulsions of infiltrated organs, whole blood cells, and plasma. In many instances mixed forms occurred. Failure to transmit myeloid leukosis or erythroleukosis by filtrates was blamed to technical difficulties of filtration.

Furth and Miller\(^13\), 1932, found the transmitting agent of leukosis passed all types of silicious filters. Filtration is particularly successful when material is first passed through larger filters. Filtration apparently reduced the concentration of material. Filtration through collodion membranes indicates the virus approximates the size of bacteriophage. The filterable agent also resists drying. The agent may be preserved in glycerin.

**Filterable Agents Described**

Furth\(^14\) in 1933 described a strain of filterable agents that successfully transmitted three types characteristically ascribed to three different types of filterable agents: namely, (a) the agent of Eller-
man and Bang that causes erythroleukosis and myeloid leukosis in chickens; (b) the agents of Rous that stimulate connective tissues of chickens to neoplastic growth and may cause endothelioma; (c) the agent of Pappenheimer, Dunn, and Core that produces fowl paralysis. The available evidence is in favor of the assumption that a single agent can produce lymphomatosis, with or without paralysis, myelomatosis and endothelioma. All these conditions were transmitted by a single filterable agent by material free of viable cells.

Furth[16] in 1936 reports experiments with the strain causing osteochondrosarcoma in a bird previously inoculated with strain 2, as mentioned above.

In successive passages made by intramuscular implantation of tumor tissue deriving from their strain there were produced osteochondrosarcoma free from leukosis, leukosis free from osteochondrosarcoma, or osteochondrosarcoma with leukosis. He concludes the possibility of a mixed strain.

Interactions Occur

It is known that four interactions of two strains of virus may occur: (1.) A virus may inhibit the development of another virus in the host tissues. (2.) The second virus may multiply in the tissue without producing typical disease symptoms. (3.) The two virus may multiply each producing typical disease symptoms. (4.) The effect of a second virus may cause a more severe and sometime atypical disease than either virus might produce alone.

Furth[16] in 1937 states that the blood of paralyzed chickens contains the etiological agent at all periods of their illness and when introduced intravenously produces neurolymphomatosis not distinguishable from natural infections.

Ratcliffe and Stubbs[18] in 1935 were unable to transmit fowl leukosis by mosquitoes or mites. They found the virus became inactivated in twenty-four hours in mosquitoes and mites.

Junghur[19] in 1937 made comprehensive studies on transmission and the pathology of fowl paralysis and associated conditions. He concluded that lymphomatosis is transmissible by fresh or dessicated bacteriologically sterile tissue material and occasionally by feces, that transmissability cannot be predicted by morphological figures, that iritis may be a manifestation of lymphomatosis, that small round cell neurolymphomatosis and large round cell lymphomatosis are indistinguishable. Lymphomatosis and Rous sarcoma are etiologically different. Typical erythro- and myeloid leukosis are not related to lymphomatosis.

Common Etiology

Johnson[20], 1934, concluded that neurolymphomatosis of Pappenheimer and erythro-leukosis or myeloid leukosis of Furth could be produced by a common filterable agent. He shows that bacteria free, cell free filtrates, or the blood and other tissue affected with any of the forms may produce the same form as the donor, or they may produce any of the several expressions of the disease, and that one form may change into another during the course of disease, or two or more forms may exist in combination.

Olson[21], 1937, was unable to transmit neurolymphomatosis by either nerve implantation or by blood.

Junghur[22] in 1938 after investigating the so-called marble bone (osteopetrosis) concluded that the causitive agent of this condition was non-dissociable with the agent of lymphomata and that it appeared certain strains of lymphomatosis are endowed with active or latent potentiality for neoplastic osteogenesis.

Two Diseases

Fenstermacher[23] in 1936 concluded from his experiments that lymphocytoma and neurolymphomatosis were two different diseases, the former non-transmissable and the latter transmissible.

Emmell[24-25], 1935-36, regarded hemocytoblastic changes of the blood picture as the basic disease which might later manifest itself as various forms of leukosis or

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the brilliance of his coat. This is followed by a shower and a special shampoo for the mane and tail. On special parade days, the manes and tails are waved.

The Palomino breed is not well established, and the color breeds true only 50 percent of the time. Arabians and Thoroughbreds are being used in the developments of the breed, as are the Morgans and the American Saddlehorse. The mating of two Palominos often results in a bleached color or albinism. This makes it necessary to mate Palomino stallions to self-colored chestnut or sorrel mares, which results in a more consistent progeny of the desired color. The Palomino color develops from a cream color at birth to a fixed shade of the desired golden color at two years of age. Sometimes the color is not fixed at this age and so may change into a deeper copper color.

It is generally agreed that the Palomino horse is heterozygous for at least one pair of color genes. Salisbury and Butler state that through study they have concluded that the Palomino color is "produced by an incompletely dominant dilution gene super-imposed over basic chestnut or sorrel color." The dilution gene varies the degree of sorrel pigment which results in the desired Palomino color. Salisbury and Butler also state that unidentified genes and gene interactions play a responsible part in the color production.

The Palomino horse is today becoming a favorite American pleasure horse. Especially is this so in the southwest, where almost every public exposition features Palominos. The annual Rose Bowl parade is headed by smart stepping Palominos. At the colorful Old Spanish Fiesta Days which are held every year at Santa Barbara, California, the best Palominos in the southwest are present to lend their color to the festival. Abilene, Texas, holds an All-Palomino show every October. But more important than just leading a parade or adding color to a festival, the Palomino with his shining golden coat and his flowing silver mane and tail is making it possible for many proud owners to fulfill their wish of having "the most beautiful horse in the land."

**FOWL LEUKOSIS**

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neurolymphomatosis. It was etiologically interpreted by him as a sequence to a primary non-specific infection with bacterial organisms of the genus *saomonella*. Butler, Warren, and Hammersland reported that much fowl paralysis was due to an enteritis reduced by dietary changes of Vitamin E deficiency. They reported recovery of many affected birds by feeding or injection of wheat germ oil. This etiological theory was disproven by Jung-hur and others.

Patterson and his co-workers, 1931, concluded from their experimental work that erythroleukosis, myeloid leukosis, lymphomatosis, iritis, and neurolymphomatosis gallinarum were one disease with a common etiological agent which was a filterable virus. Lee, Wilcke, and Murray, 1937-39, have held the same unitarian idea.

Johnson and Bell, 1936, in filtration experiments proved quite conclusively that bacteria were not etiological factors and that the disease was due to a filterable virus. They were unable to separate two or more filterable agents on the basis of size particles as factors in the etiology of the various forms of leukosis. They conclude that the various manifestations are due to a single agent whose size lies between limits 400 to 100 millimicrons or less.

Lee and Wilcke in 1939 subjected the etiologic agent to electrophoresis in an attempt to dissociate the virus. Their observations show that migration takes place toward the negative pole from pH values 4.01 to 6.01. The shift occurs to the positive pole at pH 7.01, with the isoelectropoint being between 6.01 and 7.01. Later unpublished work shows it to be between pH 6.5 and 7. The behavior of the material to electrophoresis is so like that of a virus that there seems but little doubt that this disease is due to filterable virus.

Durant and his co-workers have been able to transmit fowl paralysis to susceptible chicks from blood of 2 day old and
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older chicks hatched from adults affected with paralysis.

This review of literature is not all inclusive but is believed sufficient to show the wide difference of opinion as to the etiology of fowl paralysis and associated conditions.

It appears quite generally believed that the etiological agent in all transmissible forms is a filterable virus and quite generally agreed that myeloid and erythroid leukemia are due to a common etiological agent. It is not agreed in general as to the classification of lymphoid leukosis nor its transmissability in all forms.

It would seem many failures to transmit the various forms of the disease has been the use of birds of an age which are less susceptible. It is also true that many birds have been discarded from experiments as negative much too early to obtain definite results.

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