The flock should be revaccinated if most birds do not show takes.

The recommended age for vaccinating chickens is 6 to 12 weeks.

Vaccination of baby chicks has been tried but is not generally satisfactory because systemic reactions may slow growth rates. Mortality may be high.

Chickens should not be vaccinated within 4 weeks of the beginning of lay. Turkeys may be vaccinated at any age, except that they should not be vaccinated within 8 weeks of marketing in order to give time for the scabs and vaccination lesions to disappear.

Turkeys should be vaccinated only by the follicle method and on the upper thigh. When the stick method is used, turkeys put their heads under the wing and spread the disease to the head and other parts. Generalized cases of fowlpox are the result. Fowlpox vaccine gives turkeys an immunity that lasts about 6 months. Turkeys that are kept for breeders should be revaccinated before winter.

Chickens do not need to be vaccinated against fowlpox unless pox is prevalent in the neighborhood, the flock is likely to be exposed to the disease, or the flock had the disease the previous year.

After pox vaccine (a live virus) is used on a place, vaccination every year thereafter is necessary.

All susceptible fowls on the premises, including chickens, turkeys, guineas, and pheasants, should be vaccinated at the same time or else segregated.

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Avian Lymphomatosis

B. R. BURMESTER AND NELSON F. WATERS

LYMPHOMATOSIS, an infectious disease, leads to tumorous accumulations of lymphoid cells in various parts of the body. Three forms of the disease complex reflect the location of the pathologic alterations—visceral (internal organs), neural (nerve), and ocular (eye) lymphomatosis.

The disease is widespread in flocks of chickens throughout the United States and the world.

An estimate of its importance was had in results of the New York Random Sample Test, in which many poultry breeders from different sections of the country entered their stock from 1950 to 1954. The yearly average total mortality of the hens, which were hatched and reared from eggs received at the test, was 34 percent. Lymphomatosis was responsible for 13.4 percent; the visceral form caused three-fourths of this mortality. If that death rate were applied to the total number of chickens on farms, it can be estimated that in 1954 more than 59 million mature chickens died of it.

Heavy losses from neural lymphomatosis occurred on many farms in the 1920’s. The visceral form became more prevalent after 1930 and has been responsible for two-thirds to three-fourths of all mortality caused by this disease complex.

At no time has the ocular form caused so high a death rate as the other two forms.

Soon after this disease complex became a definite economic hazard in poultry production, many State ex-
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Experiment stations and other laboratories began investigations for the purpose of developing measures for its prevention and control. Because of the many difficulties encountered in this research, certain agricultural experiment stations sponsored the establishment of a central laboratory. The United States Regional Poultry Research Laboratory, at East Lansing, Mich., was approved on December 23, 1937, by the Secretary of Agriculture. Its purpose was designated as the improvement of viability in poultry. Since its inception, most of the work has been devoted to investigations on lymphomatosis and associated diseases. The work at the laboratory and that done at the State agricultural experiment stations has resulted in much information about many phases of this disease, but a great deal more research will be needed before adequate prevention or controls can be developed.

The three forms of lymphomatosis are related to several other neoplastic (cancerous) conditions, all of which are grouped in "the avian leukemia complex." These associated diseases—of minor or no economic importance—include osteopetrosis (bone disease), the true leukemias, erythroblastosis, granuloblastosis, and myelocytomatosis (blood diseases). The names are based on the pathologic alterations observed without regard to their causes.

Lymphomatosis is limited primarily to chickens. We have indications that its occurrence in turkeys has been increasing. It has been reported in pheasants. Symptoms and lesions typical of neural lymphomatosis have been noted in ducks.

Pathologic manifestations of the disease are varied. The common indication is the uncontrolled accumulation of blood-forming cells and closely related cells.

The visceral lymphomatosis—also known as big liver disease, lymphocytoma, or lymphomatic leukemia—may occur at any age after 4 weeks. Most deaths usually occur soon after the pullets are in heavy production. Mortality then drops gradually but continues as long as the chickens are kept.

Outward signs of the disease are variable. It may be acute or chronic. Fast-growing pullets may become listless, droopy, and succumb within a few days. Laying hens may cease laying abruptly and soon die. Other birds may show similar symptoms over a long period, becoming emaciated and dying after a long illness.

As the disease advances, the comb becomes shrivelled and pale or bluish. A white, pasty deposit of uric acid alone or in combination with greenish bile pigments often is found on the abdominal feathers.

Certain internal organs of birds that are not excessively fat may be felt; enlargement of the liver may be recognized in that way by its projection beyond the keel and the margin of the ribs.

The lesions characteristic of visceral lymphomatosis are found oftenest in the liver, spleen, and kidneys, but all organs, including the skin, at times are affected. The gross alterations may be a general enlargement, with a variation in color from dark red to light gray in organs such as the liver, spleen, and kidneys. These represent the diffuse type of involvement and often require microscopic examination for diagnosis. More frequently discrete tumor nodules, one-sixteenth to one-half inch or more in diameter, are located in the organs. These nodules are spherical. When they are on a surface, they are flattened or merge with other nodules. They are grayish, and the cut surface is uniform in color; similar nodules seen in tuberculosis are white and usually contain yellowish centers.

The microscopic picture is comparatively uniform and is characterized by massive accumulations of lymphoid cells, which are of two types. The small round cells, resembling lymphocytes, usually make up the tumors of chronic cases. The large cells, like lymphoblasts, occur in the acute type.
Intermediates and mixtures of the two types are often found.

In the absence of large accumulations, difficulties often arise in the interpretation of microscopic changes. Birds, with few exceptions, are without organized lymph nodes, but microscopic lymphoid foci are present in the glandular organs and the digestive tract. Because these foci respond to the presence of several disease agents, it often is difficult to state whether a given lymphoid focus represents normal, reactive, or neoplastic tissue. All such foci should be considered abnormal; they may represent a predisposition toward lymphomatosis.

**The Neural Lymphomatosis**—also known as neurolymphomatosis, fowl paralysis, or range paralysis—occurs oftenest in birds 2 to 6 months old, although the rate of its appearance reduces only slowly after 6 months and it has been seen in birds as early as 3 weeks of age.

The sign of this disease most familiar to the poultryman is a sprawling position, in which the chicken may lie on its side with one leg extended forward and the other backward. Less advanced cases may show a weakness or lack of coordination of the legs, wings, or neck, so that the bird has trouble in standing or walking, and a drooping of wings and head.

Nerves supplying the digestive tract may be affected, so that there are loss of weight and appetite, dilation of the crop, and diarrhea.

Respiratory distress, such as gasping, may result when one or both vagus nerves are involved. Only one of several nerves of the same bird may be affected. Survival depends largely on the extent, location, and function of the nerve or nerves affected.

Partly paralyzed birds may live many months and sometimes show partial recovery when placed in individual cages with access to feed and water.

Similar symptoms may occur in chickens with other diseases, such as those due to riboflavin deficiency or avian encephalomyelitis. A microscopic study of the affected nerves is the basis of a differential diagnosis.

The gross pathologic alterations of the affected nerves are characterized by localized or extensive soft swellings of the peripheral nerve trunks, which are yellowish or slightly grayish. In less obvious cases, the affected nerves may show only a loss of cross-striations, which are seen in normal nerves. Microscopic examination is often necessary for an accurate diagnosis. The nerves most commonly affected are the sciatic (large nerve in thigh) and its roots, the vagus (easily seen in the neck), and the brachial (shoulder and arm), celiac, and lumbar (viscera) plexuses.

Microscopic examination of affected nerves shows an infiltration with the small, round lymphocyte with a few large cells not unlike the lymphoblast. Occasional infiltrations are made up predominaately of cells of the latter type.

**Ocular Lymphomatosis**, formerly known as gray eye, pearly eye, or iritis, causes impairment of vision or complete blindness in one or both eyes. That is because lymphoid cells enter the iris and other vascular parts of the eye. This infiltration occurs over a period of several weeks and finally stops the normal dilating and constricting movements of the iris. The iris becomes fixed, usually in a restricted state, and the pupil becomes small. The pupillary border may become irregular, show indentations, and lose its regular circular shape.

These changes in form and loss of movement are perhaps more diagnostic than the change in iris color, which is a more commonly observed symptom.

The infiltrating cells also mask the pigments of the iris and cause a change from an orange or reddish brown to a pearly gray. The amount of pigment in the iris is influenced by the diet, the rate of egg production, and the age of
Avian Lymphomatosis

The chicken; furthermore, some breeds and crosses naturally have a gray iris. It is important to consider these normal variations in the color of the iris in arriving at a diagnosis of ocular lymphomatosis.

OSTEOPETROSIS, commonly known as marble bone or thick leg disease, afflicts an occasional bird in some flocks or a sizable number in other flocks, but it is not so widespread as to be of major economic importance. An enlargement of the shanks, giving a convex appearance, is usually the first symptom in chickens.

As the disease progresses, other bones, especially the long bones of the wing and of the leg, become enlarged. Articulation becomes restricted, and the chicken walks in a stiff, stilted manner. The histopathologic picture varies with the stage of the disease in the bone examined. The excessive deposition of hard bone appears to be due to a greatly increased proliferation and activity of the bone-forming cells of the inner and outer surfaces of the shaft and a reduced activity of the bone-dissolving cells.

THE TRUE LEUKEMIAS in chickens are of two types.

In erythroblastosis, the immature forms of the erythrocyte (the red blood cell) are dominant in the blood. When the leukemia is due to an overabundance of the immature forms of the granulated white cells, it is known as myeloblastosis or granuloblastosis. Often both cell lineages are involved. This condition is referred to as erythromyeloblastosis.

The occurrence of erythro- and myeloblastosis is similar to osteopetrosis and is in contrast to the various forms of lymphomatosis in that the occurrence is comparatively rare and sporadic. Symptoms are similar for the two types of leukemia. The comb and other unfeathered parts of the body become pale and often yellowish. The bird becomes emaciated, listless, droopy, and shows evidence of diarrhea.

The blood appears watery and grayish red, instead of the bright red or dark red of normal blood. Microscopic examination reveals a high proportion of immature blood cells. The liver and spleen are usually enlarged and are gray or bright red, depending on the cell type involved. The red bone marrow increases and takes over portions that are normally a fatty yellow.

Myelocytomatosis, an aleukemic neoplasia of the granular white blood cells, occurs only occasionally and is of no economic consequence. The cells are massed into distinct white nodules, which are located in the muscle and visceral organs. The cell type is the myelocyte, which contains red staining granules.

THE CAUSATIVE AGENT or agents of the various forms of lymphomatosis and associated diseases are submicroscopic and are believed to be viruses. Conclusive evidence that such is the case has been obtained only for visceral lymphomatosis, osteopetrosis, and erythro-myeloblastosis. The rapid spread of neurolymphomatosis from one farm to another and from one section of the country to another suggested that the disease is contagious and is caused by an infectious agent. Attempts to reproduce neural or ocular lymphomatosis experimentally, however, have not been sufficiently successful to warrant any conclusions regarding their cause.

Early investigators considered the different forms of lymphomatosis as separate entities, caused by different agents. Later many research workers obtained the different forms, including the leukemias, in chickens of the same experimental lots and even the same chicken. In fact, under natural conditions two or three forms of lymphomatosis are usually present in any flock, with one form predominating. It is not unusual to find individual chickens with visceral-neural, visceral-ocular, or ocular-neural involvement. A combination of the three forms also occurs. These observations led to the theory that one agent causes all forms
of lymphomatosis, osteopetrosis, and erythromyeloblastosis.

Recent investigations have led some research workers to return to the earlier view that most, if not all, of the neoplastic conditions described here are caused by similar viral agents that are different but have some similarities. Thus the virus causing visceral lymphomatosis does not result in the neural or ocular form or in erythro- or myeloblastosis. The virus causing the latter does not produce lymphomatosis in any of its forms.

The appearance of several forms in one flock or in one chicken may be explained by a tendency of the causative agent to remain latent and create inapparent infections in many chickens. Thus, a bird that has died of one type of neoplasm also may have been infected with viruses that cause other types. An inoculum obtained from such a bird is likely to contain a mixture of viruses and result in different types of tumors in the chickens.

Experimental studies at the Regional Poultry Research Laboratory disclosed that only a part of the naturally occurring cases of visceral lymphomatosis could be transmitted readily to susceptible chicks. Why this is so has not been determined.

The readily transmitted tumors may be reproduced in two different ways. Living tumor cells in suspension, when injected into almost any part of chickens of any age, will grow into a tumor in 5 to 20 days. Some of the tumor cells escape into the blood vessels during that time and are carried to the liver and other organs, where they multiply rapidly and cause the death of the bird. Some birds survive such transplants of tumor cells and are then immune to further transplants of the same type of tumor, but they are not immune to the action of the viral agent, which requires a much longer time for its action.

The second method of propagating such tumors is by the use of cell-free viral preparations. The virus may be obtained from such sources as feces, saliva, or embryos of eggs from infected chickens. A more concentrated source is blood plasma of lymphomatous birds and extracts of the tumors. Such materials may be centrifuged and passed through fine filters capable of removing all bacteria and particles of tissue. In order to produce a high incidence of disease with this type of preparation, baby chicks of a susceptible line must be exposed. The exposure may be by one of several routes, such as intravenous or intraperitoneal injection or application to the eye, nose, or trachea.

The development of the disease is similar to that which takes place after natural means of exposure. Tumors develop in the viscera irrespective of the route of infection, and the birds die of lymphomatous involvement of the visceral organs over a wide range in age. When large doses are used and when the chickens are very susceptible, most of the deaths occur in from 4 to 16 weeks, but when small doses or more resistant chickens are employed, the birds die at ages more nearly approaching those occurring after natural exposure.

The dose and susceptibility, which together determine largely the range of the age at death, also determine the type of microscopic pathology, which is easily recognized in sections of the liver. Thus, in several inoculation experiments, about 90 percent of the chickens that died within 4 months had the intravascular type of tumor involvement (the lymphoid cells were located within the walls of the blood vessels), whereas about 90 percent of those that died after that age had the extravascular type—the lymphoid cells were outside the walls of the blood vessels. The latter is the type found in more than 90 percent of the cases that occur after a natural exposure.

Potent inoculums which have passed through bacteria-retaining filters have been obtained from lymphomatous liver. In one test, 73.5 percent of the chickens died with visceral lymphomatosis before they were 9 months old,
after being inoculated at the age of 1 day by the intraperitoneal route. The dose was at the rate of filtrate equivalent to that obtained from one hundred millionth of a gram of tumor. The virus causing visceral lymphomatosis contained in blood plasma or tumor filtrate has been kept viable for several hundred days when stored at the temperature of dry ice and has been inactivated at a temperature of 131° F. by treatment with low concentrations of formaldehyde or by ultraviolet irradiation.

VISCERAL LYMPHOMATOSIS is transmitted by contact and through the hatching egg. The virus is spread widely by various means, by chickens of various ages, and in various stages of the disease.

Research workers at the Regional Poultry Research Laboratory have made extensive infectivity tests of oral washings, of extracts of feces, and of extracts of embryonating eggs by injecting such materials into susceptible chicks and noting the incidence of visceral lymphomatosis. The studies have revealed that the virus is shed in the feces and in the saliva of chickens having gross lymphoid tumors, but—it is also shed in this manner by chickens apparently normal in every way except for a latent or an inapparent infection.

Chicks inoculated at 1 day of age have, in turn, shed the virus in their saliva at 10 days of age. Noninoculated chicks of the same hatch in direct contact with the inoculated ones became infected and started to shed significant amounts of virus at 90 days of age. The average amount shed gradually increased for the first 6 months.

It was also found that day-old chicks, hatched from hens which have a latent infection, shed virus in their saliva and feces. Thus it is quite evident that many apparently normal chickens may shed the virus of visceral lymphomatosis in their saliva at any age from the baby chick to the mature hen. It is evident that virus from either the saliva or the feces may serve to infect the environment including the drinking water, the feed, and the air.

Studies on possible natural modes of entry of the virus showed that it is possible to induce a high incidence of visceral tumors by applying the inoculum to such natural body openings as nasal passages, conjunctiva, mouth, trachea, and cloaca. Transmission was also obtained when the virus was sprayed into the air above the baby chicks. Thus any mucous membrane normally exposed to the external environment may serve as the avenue of entry.

The infected egg is another major means by which the disease is spread. Workers at the Regional Poultry Research Laboratory have provided direct proof of transmission by eggs. They selected hens of an infected flock at random. They marked the eggs as to source, incubated them, and prepared inoculums from embryos of eggs of individual hens. The presence of the virus in such inoculums was then tested by intraperitoneal inoculation of susceptible baby chicks.

Results from many tests show that the embryos of most of the hens tested harbored significant amounts of the virus of visceral lymphomatosis—even though all the hens appeared healthy at the time the eggs were laid and none of the embryos showed any evidence of gross or microscopic pathology.

Thus it would appear that large numbers of chickens, apparently normal in all respects, can transmit the virus of visceral lymphomatosis to their eggs. The virus in turn is taken up by the embryo. No test which could be used to identify such carrier hens in a breeding flock is available. Hatcherymen, therefore, should not be held responsible for the dissemination of the disease by egg transmission.

The progeny of carrier hens may not develop a high incidence of lymphomatosis. Experiments conducted at the Regional Poultry Research Laboratory show that the visceral lymphomatosis
among progeny of infected hens was not significantly greater on the average than that found in progeny of hens classified as noninfected. The chicks of carrier hens are an important source of infection for chicks of noninfected stock, however.

It has been demonstrated that an extract of dust, down, and other debris collected from the hatching unit of an incubator containing chicks of an infected flock, when injected into other susceptible chicks, caused a high incidence of visceral lymphomatosis. Furthermore, when chicks of an infected flock were hatched and brooded with chicks of a noninfected flock, the latter developed a high incidence of tumors, but the former did not. In this experiment the brooding period appeared to be more important than the hatching period in the transmission of the disease.

Thus it is quite possible that the importance of egg transmission lies not in the disease, which may or may not occur among chicks hatched from infected eggs, but in the disease that is transmitted by direct or indirect contact from chicks hatched from infected eggs to chicks hatched from eggs of hens that have had no contact with the virus.

Neural lymphomatosis appears to be transmitted primarily by contact with an infected environment during the early brooding period.

In experiments at the New York Agricultural Experiment Station, the distance between chicks and adult chickens has influenced greatly the incidence of this form of the disease.

When chickens were reared in an isolated area, only a few cases of neural lymphomatosis developed. There is no direct evidence and little circumstantial evidence for the transmission of the causative agent of neural lymphomatosis from the hen to the chick through the hatching egg. It therefore would appear that the most important source of infection must be the growing or the adult chicken.

Nothing definite is known concerning the transmission of ocular lymphomatosis.

Many factors affect the resistance of chickens to lymphomatosis. Among them are genetic resistance, virus-stimulated antibodies, and the age of the chicken at the time of exposure.

A partial control of naturally occurring visceral or neural lymphomatosis can be accomplished through selective breeding. Workers at the Regional Poultry Research Laboratory and the New York Agricultural Experiment Station demonstrated that breeding for resistance is practical and will reduce losses materially.

Only breeders whose practices permit the identification of all chickens hatched are in a position to select toward resistance to either form of lymphomatosis. Pedigree breeding will permit the identification of the most viable families, together with their sires and dams, which show the greatest resistance to the disease. Continued selective breeding from the most viable families will show, within three or four generations, a marked improvement of resistance to either the neural or the visceral form of the disease.

A program of breeding for increased resistance to either of the two forms of lymphomatosis often is difficult because of a lack of adequate infection among the breeding chickens.

The history of many flocks indicates that continued elimination of families of chickens that show high mortality from lymphomatosis will reduce losses to a low figure. This reduction encourages the breeder to believe his chickens have become more resistant.

Actually the chickens may have stopped dying because the disease complex and the causative agents disappeared, or nearly so, from the flock. Thus, when this breeder's chickens are placed on many different farms, certain of these farm flocks encounter substantial losses. So it would appear that the chickens going to some farms are subjected to a greater exposure of
lymphomatosis than those on other farms—an indication that the stock was not so resistant as indicated by the performance on the farm.

In any attempt, then, to use selective breeding as a means of increasing family or strain resistance, one must first subject the chickens to a reasonably constant level of infectious agent. Since a proved method of artificially infecting chickens is not available, any exposure must come from naturally infected stock. If resistance to both neural and visceral lymphomatosis is desired, then the infectious agent of both of these forms must be present.

Experiments indicate that an immunity to visceral lymphomatosis may be induced. When certain adult hens were given a series of intraperitoneal injections with preparations of the virus, the chicks from such hens showed a high degree of resistance. In contrast, chicks from the same dams were highly susceptible when hatched from eggs laid before the hens received the injections. Neutralization tests demonstrated that the injections stimulated the formation of antibodies in the dams. Presumably sufficient antibodies were transferred to the hatching egg to give the chick a passive immunity, which protected them from the inoculated virus. This type of immunity may be the basis for the low incidence of disease encountered in the progeny of known carrier hens. It would appear that such hens shed the virus into their eggs and also shed antibodies, which give some protection to the progeny from the egg-transmitted infection.

The younger the chicks are at the time of exposure to the infectious agent—under natural conditions, by inoculation, or by contact with inoculated chickens—the greater will be the incidence of the disease. Thus many experiments have shown that age increases the resistance of chickens to the disease. This resistance is acquired rather rapidly, but it is still not known at what age the maximum resistance is first attained.

In one experiment fewer than half as many chicks exposed to natural infection at 30 days of age developed lymphomatosis as those exposed at 1 day of age. In another experiment, chicks 1, 30, and 60 days old were exposed to others of the same hatch that were inoculated at 1 day of age with the virus of visceral lymphomatosis. The incidence of tumors resulting from this contact exposure was 70, 56, and 21 percent, respectively. Similar results were obtained in chickens inoculated at those ages.

Other factors, some of which are unidentified, influence significantly the expression of the disease.

Females and capons generally are more susceptible than males. We have some indication that the female hormone increases and the male hormone decreases the susceptibility of chickens to lymphomatosis.

Some poultrymen believe that coccidiosis or other intestinal parasites may act as predisposing or precipitating factors. Nutritional factors have not been ruled out. Various stress factors may also determine the incidence of the tumors.

None of the various forms of lymphomatosis can be prevented by vaccines available in 1956 or by the use of other prophylactic measures. No chemotherapeutics, antibiotics, or other therapeutic treatments are known to be effective in the cure of chickens with this disease.

Chickens affected with lymphomatosis sometimes spontaneously show a partial recovery. Even though such cases are rare and never complete, they may be the basis of an erroneous assessment of a therapeutic treatment unless adequate controls are maintained during the same treatment period.

Isolation and quarantine procedures during the hatching and brooding period are helpful in restricting the spread of lymphomatosis.

Neural lymphomatosis is transmitted primarily by direct or indirect contact with older growing or adult poultry.
with no indication of significant egg transmission. The primary consideration therefore is to rear the chicks at as great a distance as possible from such stock and to restrict the interchange of personnel and equipment between chicks and older stock.

The primary source of infection for visceral lymphomatosis appears to be the maternally infected baby chick—there is no indication that the adult stock is an important source. Therefore the procedures that were recommended for checking the spread of neurolymphomatosis are not effective for the visceral form. A practical test for the identification of birds having a latent or inapparent infection is not available. Elimination of such carrier birds from the breeding flock thus is not possible at present. The only recourse available is to hatch and brood chicks from different flocks in separate compartments, pens, or houses insofar as possible. This recommendation is based on the premise that the extent of this disease in a breeding flock, and hence the proportion of chicks with a latent infection and protective antibodies, will vary from flock to flock. This procedure would prevent the mixing of chicks, most of which have a latent infection and shed virus, yet are protected by maternal antibodies, with chicks from other flocks, most of which are from disease-free parents and are highly susceptible to the virus shed by chicks of infected parents. Because most flocks are infected with both neural and visceral lymphomatosis, it is desirable to follow the isolation procedures suggested for both forms of the disease.

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**Turkey Erysipelas**

**Richard D. Shuman and O. L. Osteen**

**Turkey Erysipelas** is caused by the same bacterium that causes swine erysipelas, *Erysipelothrix rhusiopathiae*. The organism also attacks ducks, chickens, pheasants, pigeons, parrots, peacocks, quails, and a variety of small wild birds.

Since 1934, when the disease was first recognized in the United States in New Jersey, by F. R. Beaudette and C. B. Hudson, turkey erysipelas has been reported in turkey-raising areas throughout the country. Officials in 17 rather widely separated States indicated in 1953 that turkey erysipelas ranked first to fifth in economic importance among the bacterial diseases. But of 16 States that produced more than 1 million birds each in 1952, 10 indicated that turkey erysipelas was not a problem. The others ranked the disease from first to fifth in importance.

Estimated annual losses in 1942–1951 approached 2 million dollars. Death losses and the setback turkeys have from the infection usually come