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.

B. R. Burmester is a biologist at the laboratory at East Lansing. He is in charge of the pathological phases of investigations of avian lymphomatosis. He has undergraduate and graduate degrees from the University of California. Later he obtained the degree of doctor of veterinary medicine from Michigan State University.

Nelson F. Waters is the geneticist at the United States Regional Poultry Research Laboratory at East Lansing, Mich. He has been investigating the inheritance of resistance and susceptibility to avian lymphomatosis since 1939.

**Turkey Erysipelas**

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

Turkey Erysipelas is caused by the same bacterium that causes swine erysipelas, *Erysipelothrix rhusio-pathiae*. 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
Turkey Erysipelas

about the time they are ready for market and the death of even one bird of that age is a sizable financial loss to the owner. Besides, affected birds that might otherwise have commanded top price may be downgraded or rejected.

The occurrence of turkey erysipelas is not necessarily associated with erysipelas in swine, although it can originate from contact with infected pigs. Four States in the Corn Belt, where erysipelas in swine commonly exists, reported that turkey erysipelas was not a statewide economic problem. One other State in this region reported it to be second in economic importance, and two ranked it fifth. It has been also suggested that this disease may be associated with areas where sheep have been ranged, but this may be only coincidental.

Because *E. rhusioptihiae* is so widespread, the soil must have an important part in keeping it alive. Because turkeys are resistant to infection with ordinary laboratory strains of the organism, which in turn can cause sickness in susceptible hogs, some unusual situation must occur to start an outbreak of this disease.

A. S. Rosenwald and E. M. Dickinson observed that in Oregon the disease occurs at the time of increased rainfall and when the turkeys are reaching maturity. Possibly the increased moisture along with the additional nitrogen from droppings in the soil favors the multiplication of the organism.

The toms, which are maturing at that time, fight and cause head injuries, in which infections could start. It has been shown in the laboratory that repeated transfer of the organism from a bird dying of the disease to a living susceptible bird increases the virulence, or killing ability, of the organism. Occurrence of the same situation in the field might account for the sudden severe losses and seasonal occurrence of the disease.

Feed that contains inadequately cooked fish offal has also been suspected as a possible source of the infection.

The death rate following infection may vary from 2 to more than 40 percent.

Outbreaks usually occur in the fourth quarter of the year. The disease rate is much higher in the males than the females, not because of a sex difference in susceptibility but apparently because of infected wounds incurred in fighting among the males.

An outbreak of turkey erysipelas can occur with no warning, and all that will be seen are some dead birds. Dr. Rosenwald and Dr. Dickinson noted that in several outbreaks in Oregon the owners suspected poisoning, stampeding, or some mysterious predators.

In other instances the birds may first appear weak or listless and with wings and tail feathers drooped. The snood, wattles, and bare parts of the head and neck may be swollen and purplish. In birds that live long enough, erysipeloid lesions may appear on the head and resemble somewhat the lesions of diamond skin disease of swine.

An examination of a dead bird reveals evidence of a septicemia. Hemorrhages in the muscles particularly in the breast and thigh, may be seen. The liver and spleen are usually swollen and dark colored, and there may be some degree of enteritis. The blood vessels of the viscera will usually be full. Despite the possible presence of noticeable symptoms and lesions in the bird, a definite diagnosis can only be made in the laboratory. Finding the organism eliminates any doubt as to the diagnosis of the disease.

The only available treatment for many years was specific immune serum, as used in swine. For the most part, however, the results did not appear satisfactory, probably because of the manner in which the serum was used. At any rate, if serum is used it should be administered early in the course of the disease. Sulfonamides have not proved to be worthwhile.
Antibiotics, particularly penicillin, are useful. Used early enough and at adequate levels, they usually give satisfactory results. One should not expect all sick birds to recover, however, and relapses may occur.

Erysipelas adsorbate bacterin has shown promise as a biologic for vaccination, but a final estimate of its value must await further reports from users. For a long time we have needed an immunizing agent that will protect the growing turkey right on through to market time.

Removal of the snood, or dewbill, of day-old turkey poults in areas where the disease is prevalent has been suggested by W. A. Billings, of the University of Minnesota. The basis for such a procedure is that the snood is especially apt to be injured because of its location; removing it would reduce the hazard of wound infection with the erysipelas organism. Debeaking at about 14 weeks also has been suggested in an effort to minimize the effect of fighting among the toms.

Segregation of sick birds, moving the remainder to a new range, and the use of antibiotics will help to control an outbreak. An infected range should not be used for turkeys for a year. Any low points should be landscaped to eliminate areas of standing water, and a cover crop should be put in. As a precaution, particularly in the places where erysipelas is known to exist in hogs or sheep, turkeys should be kept away from any contact with them.

One should remember that the causative organism of turkey erysipelas can infect people (erysipeloid). A person should therefore handle sick or dead birds only with proper care.

**Richard D. Shuman** is a veterinarian in the Bacterial and Mycotic Disease Section of the Animal Disease and Parasite Research Branch. He was graduated from the College of Veterinary Medicine of the State College of Washington in 1938.

**O. L. Osteen** joined the Department of Agriculture in 1932 and has engaged in research in diseases of animals, and poultry.

---

**Chronic Respiratory Disease in Chickens**

**J. F. Sullivan, Clarence H. Thompson, Jr., and O. L. Osteen**

**Chronic** respiratory disease (air-sac infection), a respiratory disease of chickens, has a slow rate of spread and a persistent nature.

The average annual mortality from chronic respiratory disease (CRD) in chickens from 1942 through 1951 was more than 14 million birds. Each year additional losses are due to the decreased gains and lowered feed utilization that accompany the disease, extra time needed to bring broilers to market, a larger number of unmarketable birds, and lower egg production and hatchability.

A pleuropneumonialike organism causes CRD. The organism is extremely small and, like viruses, can pass through certain filters designed to retain bacteria. Like bacteria, it can be seen, following appropriate staining, with an ordinary microscope. It grows readily in the yolk sac of 7-day-old embryonating chicken eggs and can be propagated in suitable cell-free cultural media. The organism has varying degrees of sensitivity to antibiotics, ranging generally from complete inhibition by streptomycin to total resistance to penicillin.