One thousand four hundred eighty-nine cases had been reported in the horses and mules of Louisiana as of the first of September. DDT spraying operations for reduction of insect populations in farm buildings, which were proceeding during the outbreak of the disease in Avoyelles Parish, did not appear to reduce the number of cases reported in this area, which rose from 40-42 on July 25 to 650 on September 1. Too few horses were vaccinated with Eastern equine encephalomyelitis vaccine to affect the course of the epidemic in this parish. Whether or not spraying may have had some effect on the incidence of human cases is a matter of speculation. It is interesting that in both Louisiana and Arkansas human cases remained relatively few or unrecognized. Certainly, there were no reports to indicate significant human involvement.

Various species of mosquitoes were collected for virus studies which have not yet been completed. The outstanding feature of the 1949 epidemic was its apparent tendency to follow water courses northward.

Surveys of serum antibodies have led toward the incrimination of wild and domestic birds as a possible reservoir for the virus of Western equine encephalomyelitis. Information in this regard has been lacking for Eastern equine encephalomyelitis. One hundred sixty domestic fowl were sampled in the area involved in the 1947 epizootic in Louisiana and tested for neutralizing antibodies at the Montgomery, Ala., virus laboratory. Only 2 chickens of 109 gallinaceous birds (chickens and turkeys) gave positive titers for neutralizing antibodies against Eastern equine encephalomyelitis. No positive sera were observed in 13 anserines (ducks and geese) or 38 Columbiformes (pigeons). Two cormorants (diving sea birds) showed neutralizing antibodies.

Six of eighteen unvaccinated horses with no history of encephalitic symptoms gave positive neutralization indices. Ten of eleven recovered equines were positive and all of four horses bled in the terminal stages gave positive titers.

**SUMMARY**

CDC investigated epizootics of Eastern equine encephalomyelitis in Louisiana and Arkansas during the summers of 1947 and 1949. These two epizootics possessed certain things in common. Both started on the Gulf coast within a few miles of each other in the late spring and spread at a rather regular rate covering approximately 25 miles every 2 weeks. Both occurred in districts which have large areas covered by still water such as swamps, bayous, and rice fields. The equine population was dense in each case, the morbidity high, and the mortality about 90 percent. Under apparently equal conditions for exposure, mules were more resistant to the disease than horses. Serum surveys revealed very few domestic fowl with significant antibody titers. Wild fowl, such as wading birds and blackbirds, were common in the epizootic areas and the mosquito population was high. Further investigation, with emphasis on the role of birds and associated arthropod vectors, has been planned by the Virus and Rickettsia Section of the Communicable Disease Center. This will be carried out in the near future in cooperation with Epidemiologic and Veterinary Public Health Services.

# *Western Equine Encephalomyelitis*

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History bears out the fact that Western equine encephalomyelitis has been with us for a long time. As early as the middle of the nineteenth century, western veterinary practitioners recognized and reported outbreaks of a febrile disease of horses, with symptoms comparable to those observed in equine encephalomyelitis today. The literature contains information on about 10 major epizootics

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occurring between 1847 and 1930; undoubtedly there were many other outbreaks that were not recorded.

It is of historical interest to note that reports of the U. S. Bureau of Animal Industry for 1912 show that 16 States had a high incidence of the disease. Kansas and Nebraska had the greatest incidence. Estimates indicate that in these two States about 35,000 equines died of this disease. Early reports of these epizootics referred to the infection under several names such as "Kansas-Nebraska horse plague," "forage poisoning," "cerebral spinal meningitis," "Borna disease," and "sleeping sickness." Some of these terms are still being used by farm people.

Specific knowledge of this disease dates from the work of Meyer, Herring, and Howitt, who in 1931 isolated the causal agent of Western equine encephalomyelitis from the central nervous system of horses.

The importance of this virus in relation to human disease became established in 1938, when Howitt isolated the Western equine strain from brain tissue of a child.

The incidence of this disease in horses is reported annually to the U. S. Bureau of Animal Industry by nearly every State west of the Appalachian Mountains. The highest incidence is found in the agricultural areas of the Midwest and the West Coast where it is endemic over wide areas and where it produces large epizootics periodically. For instance, in 1936, the 10 States in the Missouri River Basin reported a total of only 2,926 cases, whereas in 1937, some 146,587 cases were reported in the same States. The first cases usually appear with the advent of warm weather, continuing until the first freezing weather in the fall. Western equine encephalomyelitis is so closely related to the other encephalitides, that it is impossible to differentiate between them clinically or pathologically. To make a specific diagnosis, it is necessary to identify the virus by serological determinations or by isolation of the virus itself.

During 1941, 10 Midwestern States reported 28,899 equine infections. This epizootic was almost concurrent with one of the largest outbreaks of human encephalitis which occurred in the same general area. There were approximately 4,000 human cases in this epidemic with mortality of 8 to 15 percent as compared with the equine rate of 25 to 30 percent.

Animal care consists chiefly of good nursing in quiet, cool, comfortable, well-bedded quarters, protected from sun and rain. The use of anti-encephalomyelitis serum has not proved to be very satisfactory. However, there is available a very satisfactory vaccine which will produce, in 10 days to 2 weeks, nearly 100 percent immunity lasting upwards of 6 months.

The epidemiology of this virus infection is not clearly defined and, although considerable work has been done, the relationship of known facts cannot be explained at this time. Evidence of the virus has been found in many species of animals. Of the mammals, the horse seems to be the most susceptible; however, antibodies have been found in other domestic animals as well as in wild animals. Sufficient work has not been done in the Midwest to ascertain the percentage of mammals with antibodies present.

Very little has been published relative to the frequency of the disease in midwestern birds. From work done by Hammon in Oklahoma, only about 8 percent of the domestic birds and 5 percent of the wild birds showed Western equine encephalomyelitis antibodies. This is considerably lower than a comparable study made in California where 50 percent of the domestic birds and 17 percent of wild birds were found positive.

Several of the arthropods apparently play a very important part in the transmission of the disease. However, much additional work in this field is necessary before the complete cycle of the disease can be described. Several varieties of mosquitoes, mites, lice, as well as *Triatoma*, have been found harboring the virus in nature.

Realizing the importance of encephalomyelitis, the CDC is supporting an expanded investigation of this disease. An epidemiological study of encephalomyelitis in the Midwest was started in 1949. This work involves studies of the ecology of the various strains present, during both epidemic and interepidemic periods. A considerable amount of data has been obtained during the past season. Reports delineating the problem in the Midwest and presenting results of investigation to date are being prepared for publication.