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ARTICLES

EASTERN EQUINE ENCEPHALITIS IN THE UNITED STATES*

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Although Eastern equine encephalitis (EEE) was not recognized as a distinct disease entity until 1933, there is good evidence that this malady has caused severe epizootics among horses and mules in the United States for at least 100 years. Many of the outbreaks formerly designated as cerebrospinal meningitis, blind staggers, and forage poisoning were no doubt caused by the virus of Eastern equine encephalitis. These included the outbreaks of 1847 in Long Island, New York (1), of 1902 in North Carolina (2), and of 1912 in Maryland, New Jersey, and Virginia (2, 3).

During the past 20 years, the Eastern type virus has caused outbreaks among horses, humans, and birds in various sections of eastern United States. At present this virus has been demonstrated in 17 states—all except Michigan, Missouri, Arkansas, and Tennessee located along the Atlantic seaboard or the Gulf of Mexico (4, 5, 6).

Outbreaks of Eastern Equine Encephalitis in the United States. Notable outbreaks (principally epizootics) of EEE during recent years include the following: (1) 1933 along the North Atlantic coast; (2) 1938 in New England; (3) 1939 along the South Atlantic coast; (4) 1941 in Texas; (5) 1942–43 in Michigan; (6) 1947 in Louisiana and Texas; and (7) 1949 in Louisiana and Arkansas.

The 1933 epizootic, centered along coastal and tidewater areas of Virginia,

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New Jersey, Delaware, and Maryland, was noteworthy not only because of its magnitude and severity—at least 1,000 horses were affected, with a mortality approximating 90 percent (7)—but also because isolations of virus from the brains of dying animals proved it to be a distinct disease (some of these first virus isolations were from Virginia cases) (3, 8, 9).

The 1938 outbreak of encephalitis in the New England states proved to be an epochal one, for it was learned for the first time that humans, especially children, are susceptible to the virus of EEE (10, 11, 12). An epizootic in horses preceded the human epidemic. The epizootic started in July in southeastern Massachusetts and later extended into Rhode Island and Connecticut. From July to late October, 381 cases were reported from these 3 states with a mortality approaching 93 percent (13).

A highly fatal encephalitis outbreak among children occurred in southeastern Massachusetts in August and September. Thirty-four cases were definitely identified, of which 74 percent terminated fatally (14). Of the nine survivors, at least six showed evidence of permanent brain damage (15). Seventy percent of the 34 cases were under 10 years of age.

An interesting epidemiological finding in connection with this outbreak was the occurrence of encephalitis among birds in the area. Unusual losses among pigeons and ring-necked pheasants were reported in the affected area, and EEE virus was isolated from a pigeon in Massachusetts (16), and from four ring-necked pheasants in Connecticut (17). Virus was also isolated from two pheasants in nearby New Jersey (18).

A mosquito survey was started in the affected area on September 19, 1938, but unfortunately a severe hurricane swept through New England 2 days later and destroyed many mosquitoes. When the survey was resumed in October, *Culex pipiens* was found to be the predominant species (14).

An extensive mosquito survey was conducted in Massachusetts the following year, and from the results of this study it was concluded that *Aedes vexans,* because of its distribution and abundance, was probably the chief vector of EEE in the area, but that salt-marsh mosquitoes—particularly *A. sollicitans* and *A. cantator* —might have been involved along the coast (19).

In 1939, the EEE virus presumably spread south along the Atlantic coast. In Florida, Georgia, and North Carolina, there were 524 reported cases among equines, with a mortality of 92 percent (20).

A relatively small localized epizootic of EEE in the vicinity of Brownsville, Texas, in 1941 was of particular interest because it constituted the first knowledge of the occurrence of the virus west of the Appalachian line, and the disease appeared in this area during April and May (4, 21). Usually EEE is a disease of summer and early fall.

When epizootics of EEE occurred in southwestern Michigan in 1942 and 1943, it became evident that this disease is not restricted to seaboard areas. Reported equine cases from Michigan for the 2 years totaled 102 and 367, respectively, with a mortality of 91 and 95 percent (22, 23). This area of Michigan is characterized by numerous lakes, ponds, swamps, and marshes (24, 25). It may be significant to note that a 1,000-acre bird sanctuary lies in the center of the epizootic area.

The largest proved EEE epizootic of record occurred in southern Louisiana and adjoining Texas in 1947. The official report of the Bureau of Animal Industry of the U. S. Department of Agriculture (26), gives 3,813 and 715 cases from Louisiana and Texas respectively; but a later report by the Department of Veterinary Science, Louisiana State University (27), lists 14,334 cases with 11,722 deaths for the State of Louisiana. The disease, which began in May in a parish on the Gulf coast line in southwest

Louisiana, spread north and west and finally involved southeastern Texas. The last cases were reported in October.

Accompanying this epizootic was a small outbreak in humans. From September 8 to October 23, 10 cases were definitely diagnosed as due to the Eastern type virus; 7 of the 10 died. All cases except 1 were in children, and 7 were under age 10 (28, 29, 30).

In 1949, Louisiana and Arkansas experienced an acute fulminating type of encephalitis which involved at least 1,700 equines (6). The mortality was reported as 98 percent. This epizootic, which started on the coast near the initial site of the 1947 outbreak, progressed northward to the middle of Arkansas.

The 1947 and 1949 outbreaks in Louisiana had certain characteristics in common, namely: (1) they started in an area where there are several bird refuges; (2) they occurred in districts with large areas of swamps, bayous, and rice fields; (3) the equine population was dense; (4) wading birds, such as egrets, and blackbirds were common; and (5) the mosquito population was high (31).

Reported Cases of Equine Encephalitis in Virginia. A total of 294 cases among horses and mules in Virginia was reported by the Bureau of Animal Industry during the 16-year period, 1935–1950. The number varied from 0 in 1941 to 50 in 1945. Encephalitis in equines has been reported from 24 counties in the state—all in the Coastal Plain physiographic province except Stafford and Spotsylvania in the Piedmont province. As far as I have been able to determine, no confirmed human cases of EEE have been reported in Virginia.

Epizootics in Pheasants. In addition to the previously mentioned episode of encephalitis in pigeons and pheasants that accompanied the 1938 outbreak in the northeastern part of the country, several EEE epizootics among pheasants have been reported in New Jersey, and one in Connecticut in 1951. Beaudette has reported 13 definite outbreaks of EEE in New Jersey pheasants from 1938 through 1946 (32). Examples of reported losses may be cited for two counties. In Burlington County, losses were 1,120 of 3,350 birds in 1943, 1944, and 1945. In Ocean County, deaths were 4,131 of 5,580 birds for the years 1945 and 1946.

The recent report of encephalitis in Connecticut (33) is of interest since it represents the first time that EEE has been recognized in the New England states since the 1938 outbreak. A study of the 1951 episode revealed that at least three game-breeding pheasant farms were involved and reported losses were as follows: On the first farm, 200 of 800 pheasants; on the second farm, 173 of 200 (the final 27 were destroyed by the owner); and on the third farm, 100 of 1,800 pheasants. EEE was identified by virus isolations from the brains of birds on the second and third farms and by virus neutralization tests from recovered birds on the first farm. The disease was first recognized on August 24, September 21, and the first part of October for the three farms respectively. During 1951, EEE infection was not recognized in horses or humans in Connecticut.

Mode of Transmission. It was suspected that equine encephalitis was insect-borne because of the prevalence of the disease during the insect season. Since EEE outbreaks were frequently associated with coastal and tidewater areas, some of the first experimental work on transmission included salt-marsh mosquitoes. In 1934 (34), Merrill and his co-workers found that EEE virus could be transmitted in the laboratory by *Aedes sollicitans* and *A. cantator;* also by the domestic mosquito, *A. aegypti.* In 1935, *A. taeniorhynchus* and *A. vexans* were added to the list of laboratory transmitters (35). When Davis (36), in 1939, demonstrated transmission of EEE virus by six species of *Aedes,* including *A. triseriatus* and *A. atropalpus,* but could not demonstrate conveyance by *Culex pipiens, C. salinarius, Anopheles punctipennis,* and *Mansonia perturbans,* many epidemiologists were convinced that *Aedes* mosquitoes were the principal vectors of the Eastern virus.

It should be stressed, however, that no species of *Aedes* has been found naturally infected with this virus. The ability of a species to transmit encephalitis under laboratory conditions and under field conditions may not be the same. Although salt-marsh mosquitoes have been incriminated as vectors, such mosquitoes are not known to occur in Michigan, where at least two epizootics of EEE have occurred.

Recently, Chamberlain of the CDC Virus Laboratory at Montgomery, Alabama, has found that the Eastern type virus can be transmitted experimentally by *Psorophora* mosquitoes: *P. ferox, P. confinnis, P. ciliata,* and *P. howardii.* Workers at the Virus Laboratory have also carried on experiments with *Culex quinquefasciatus.* These experiments indicate that few specimens retain virus for more than a few days after an EEE-infected blood meal. Although one transmission to a chick was accomplished after a mass feeding of "infected" specimens, it is not believed that *C. quinquefasciatus* could, under ordinary circumstances, be an important vector (37).

To date, only two species of mosquitoes have been reported as naturally infected with the virus of EEE. These are *Mansonia perturbans,* collected in Georgia in August 1948 (38), and *Culiseta melanura,* trapped in southeastern Louisiana in August 1950 (39). The epidemiological significance of these two species cannot be evaluated at present.

The Eastern type virus has also been isolated from chicken mites and chicken lice collected in Tennessee in August 1947 (5). In the laboratory, attempts to transmit EEE virus by chicken mites have yielded mostly negative results. Thus the role of their importance as potential vectors needs clarification.

Out of this somewhat spotty and confused picture, it is generally considered that mosquitoes are the usual vectors of the Eastern equine virus, but precise information as to genus or species is lacking.

In connection with the transmission of the EEE virus, the question arises as to what is the principal source of mosquito infection. Although both mammals and birds have been suggested as potential "reservoirs" of the viral agent, current evidence points to the theory that birds are the more important of these two groups. Studies show widespread infection in birds—mostly unapparent—with a high titer of virus in their blood stream for a short period of time. It is of interest to note that a wild bird—a purple grackle—which was later shown to have EEE virus in its blood was shot in June 1950 in a swamp in Louisiana (40), only a few hundred yards from the location where the naturally infected *Culiseta melanura* was collected in August 1950. Present knowledge of the epidemiology of EEE indicates that the basic cycle is mosquito-bird-mosquito, with man and horse as accidental entries into the infection chain. But the crux of the problem still remains: How is the virus carried over from one season to another? Birds are not considered long-term reservoirs of the virus since they contain circulating virus for only a few days, and they are not known to have recurrent viremias. It is hoped that the intensive investigations now under way will provide the answer to this unsolved problem.

PUBLIC HEALTH PROBLEM

One may well ask: Is EEE a public health problem? It must be recognized that this disease has been quite unimportant as far as numbers of humans affected are concerned, but it should be emphasized that the importance of this disease is not that of numbers. The importance lies in hysteria or panic which accompanies the unpredictable outbreaks. People are stricken with fear, and consequently publicity is tremendous. Health departments are bombarded with all kinds of questions, most of which they are unable to answer. In the wake of an outbreak, the public demands action. Thus it behooves all public health workers to be as well informed as possible on the epidemiology of the disease so that when an

outbreak does occur, the public can be properly informed.

CONTROL

On the basis of our present knowledge concerning the disease, the only feasible approach to the control of encephalitis is a program of prevention. Prevention of encephalitis consists of two principal avenues: (1) immunization; and (2) mosquito control. Vaccination is recommended as a prophylaxis for equines, but it is neither practical nor indicated for the general human population.

Mosquito control can hardly be justified solely on the basis of encephalitis prevention. However, it is frequently justified from the pest mosquito standpoint. Therefore, an encephalitis prevention program should be combined with nuisance mosquito control activities—by the community and by individuals—so that the work would be justified economically. In view of our lack of precise information concerning the vectors of EEE, the control program should include virtually *all* species of mosquitoes. In summation, mosquito control for the community and protection against mosquito bites for the individual are the most reasonable prophylactic measures against this dread disease.

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THE EFFECTS OF DESICCATION ON THE EGGS OF *ANOPHELES QUADRIMACULATUS* SAY

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Anopheline eggs apparently have been found to be fairly resistant to desiccation under experimental conditions. Mayne (1926) reported survival of *Anopheles quadrimaculatus* eggs for as long as 16 days, and survival of *A. crucians* eggs for 21 days when subjected to drying mud. Stone and Reynolds (1939) obtained larvae of *A. punctimacula, A. albimanus,* and *A. aquasalis* from Canal Zone mud which had been devoid of standing water for about one month. Deane and Causey (1943) found some *A. gambiae* eggs able to survive for 18 days in drying sand. Darrow (1949), in a series of critical studies, showed that eggs of *A. quadrimaculatus* had to be kept moist for between 0.2 and 0.3 of the total period of embryonic development in order for them to resist desiccation. She further suggested that eggs on a dry substratum would continue to develop provided the relative humidity was 100 percent.

The present studies were undertaken