Status of Mosquito-Borne Encephalitis in the United States

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MOSQUITO-BORNE encephalitis infections are relatively new diseases from the standpoint of definite knowledge; however, such diseases probably have existed in this country and in other parts of the world for centuries. In the United States the three principal encephalitis viruses—western, eastern and St. Louis—were first isolated in the early 1930's, but there is good evidence that outbreaks of sleeping sickness among horses occurred over 100 years ago. Recently, Hanson described an epizootic of encephalitis, probably the eastern type, among 100 horses in southeastern Massachusetts in 1831 (*1*).

The present status of the human encephalitis problem is difficult to evaluate because routine reporting of "infectious encephalitis" by physicians is based on clinical symptoms rather than on cause. Encephalitis means inflammation of the brain, but such inflammation can be caused by a host of agents, living and nonliving, including chemicals, protozoa, bacteria, rickettsiae, fungi, and viruses. Our most accurate information on distribution of the mosquito-borne encephalitis viruses and the magnitude of the problem they create is obtained by special studies when epidemics and epizootics occur. During the past 25 years, severe outbreaks in horses and man have occurred periodically in various parts of the United States.

Present knowledge concerning the natural history of these virus diseases indicates that wild birds are the principal reservoirs of infection and mosquitoes are the vectors. Normally the infection chain is limited to birds and mosquitoes. Under certain conditions, the virus spills over to horses, humans, and other mammals. Man and equines appear to be dead-end hosts and are not important in the natural cycle. There is no evidence that either is a significant source for mosquito infection.

The encephalitis viruses cause infections in a wide range of vertebrate hosts, but in many hosts, such as birds, the infections are apt to be mild or entirely asymptomatic; among humans and horses, they range from the clinically inapparent type to a severe, highly fatal involvement of the central nervous system.

Common symptoms of mild cases in man include a brief fever, malaise, or a headache. Among the more prominent signs and symptoms of severe infections are an abrupt onset, high fever, severe headache, stiff neck, irritability, drowsiness and coma, muscular twitching, and convulsions. The symptoms sometimes are confused with those of poliomyelitis, but residual paralysis is not common.

In this paper the current status of knowledge concerning western encephalitis (WE), eastern encephalitis (EE), and St. Louis encephalitis (SLE) will be briefly reviewed.

Western Encephalitis

The western encephalitis virus has been most active in States west of the Mississippi River and in Wisconsin and Illinois. The "cerebrospinal meningitis" in Idaho in 1897 and the famous Kansas-Nebraska horse plague of 1912 undoubtedly were caused by WE; but it was

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not until 1930 that Meyer and his co-workers isolated the etiological agent (*2*). The largest outbreaks of equine encephalitis ever recorded occurred in 1937 and 1938 when over one-third of a million equine cases with a mortality of 20 percent were reported from the 22 States west of the Mississippi River. Iowa holds the all-time record of 66,000 cases in 1 year (1938). During a recent 5-year period (1952–56), reported cases of equine encephalitis for the 22 western States averaged 1,350 cases per year, according to U. S. Department of Agriculture reports.

When Howitt recovered the WE virus from a child's brain in 1938, it was proved that the term "western equine encephalomyelitis" was a misnomer (*3*). Three years later the greatest human epidemic of WE occurred. At least 3,000 persons were attacked, including 1,100 in North Dakota. No large epidemic of WE has occurred since 1952, when California reported more than 700 human cases, of which 375 were confirmed as WE (*4*).

The fatality rate for WE averages from 20 to 30 percent in horses and 5 to 15 percent in humans. Severe and permanent aftereffects can follow the disease, especially in infants. Many cases of this disease occur in infants and to a lesser extent in persons over 50 years old; clinical infections are usually uncommon at the other ages. However, in the 1941 epidemic in North Dakota, about 37 percent of the cases occurred in the age group 15–44 years (*5*).

Proof that the WE virus could be transmitted by mosquitoes (*Aedes aegypti*) was obtained by Kelser's classic experiments in 1933 (*6*). It was not until 1941, however, that Hammon and his associates reported the first WE virus isolation from a mosquito in nature, *Culex tarsalis* (*7*). Since 1941, *C. tarsalis* has been considered the primary vector of WE. To date, the WE virus has been isolated from several hundred pools of this species, collected in 11 States and the Province of Manitoba, Canada. The geographic distribution of *C. tarsalis* corresponds to the general distribution of the WE virus. This vector is common west of the 95th meridian, roughly the 17 western States, and in the entire State of Iowa, where it breeds in great numbers in seeps and grassland surface pools. *C. tarsalis* fits the role of a primary vector very well because of its abundance in western areas; its tendency to feed readily on a wide range of hosts, including birds, cows, horses, and man (*8*); and its remarkable laboratory vector efficiency in transmitting the WE virus (*9*).

Studies made since 1941 show that wild birds are commonly infected with the WE virus. Virus recoveries have been made from approximately 20 species in nature. Domestic fowl also may serve as natural hosts from which mosquitoes become infected.

Factors favoring outbreaks of WE include an abundance of *C. tarsalis*, particularly early in the season. With respect to the epochal outbreak of encephalitis in North Dakota in 1941, it may be significant to note that the year 1941 was the wettest year in the climatologic history of North Dakota, according to a personal communication from F. J. Bavendick, U. S. Weather Bureau, Bismarck, N. Dak. The California epidemic of 1952 was preceded by one of the heaviest snow packs ever recorded on the mountain ranges of this State; the subsequent melting of the snow, coupled with moderate winter and springtime temperatures, resulted in a flood which caused an early season buildup of vector populations (*10*).

Eastern Encephalitis

Eastern encephalitis was first recognized as a distinct disease entity in 1933 (*11*, *12*). It has since caused intermittent outbreaks among horses, humans, and birds in various sections of eastern United States, principally in coastal States from Massachusetts to Texas.

Eastern encephalitis was first recognized as a disease of man in 1938 in Massachusetts (*13*, *14*). Since 1938, there have been reported less than 100 human cases for the entire Nation, but 50 of these cases have occurred in Massachusetts—34 in 1938, 4 in 1955, and 12 in 1956 (*15*)—and 18 in Louisiana. In 1957, Florida reported two confirmed human cases of EE for the first time (personal communication from J. O. Bond, Florida State Board of Health, Jacksonville).

Most of the outbreaks among horses have comprised not more than a few hundred cases. One exception was the epizootic in 1947 in Louisiana, when 14,334 cases and 11,722 deaths were reported.

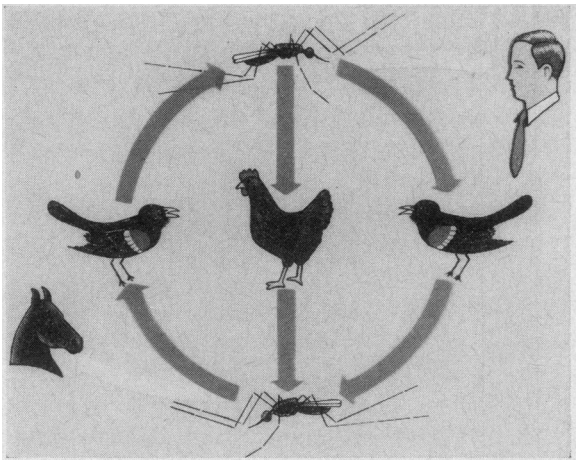
In Florida, a total of 2,219 cases among horses and mules was reported by the U. S. Department of Agriculture for the 23-year period 1935–57. The largest number of cases (368) was reported in 1957. The disease has been reported in all but 2 of the 67 counties of the State, but the majority of cases have occurred in the fresh-water lake region of central Florida.

A unique feature of the eastern virus is that it may cause clinical illness in birds, particularly confined ring-necked pheasants. Since 1938, when the virus was first isolated from the brains of pheasants reared on farms in Connecticut (*16*) and New Jersey (*17*), the disease has caused more than 100 outbreaks among pheasant flocks in 8 States, including 1 outbreak in Florida. An interesting epidemiological feature of the malady in pheasants was the finding that the virus can be transmitted mechanically from bird to bird during acts of feather picking and cannibalism, common vices among penned pheasants (*18*, *19*).

Of the three encephalitis viruses, EE is the most virulent. Fatality averages about 90 percent for horses and 60 percent for humans. Approximately two-thirds of the human cases occur in children under 10 years of age. Of the survivors, most do not recover completely.

Recent studies on vectors of EE have implicated the bog mosquito *Culiseta melanura* as the primary vector for maintaining the basic infection chain in nature (bird-mosquito-bird). Proof is lacking, however, that *C. melanura* is responsible for human and horse outbreaks of EE. Certain other mosquitoes, such as *Aedes*, *Psorophora*, and *Mansonia*, are suspected vectors (*20*). *C. melanura* has been shown to be a laboratory transmitter of the EE virus (*21*), and 17 natural isolations have been made from pools of this species in Louisiana (*22*), New Jersey (*23*, *24*), Massachusetts (*25*), and Alabama (personal communication from R. W. Chamberlain, Public Health Service, Montgomery, Ala.). This mosquito feeds extensively upon birds (*24*), but the extent to which it feeds on man and horses under natural conditions is unknown.

C. melanura is extremely selective in its breeding places and tends to choose secluded shady sites with cool and acid water in permanent fresh-water swamps. Some of its principal habitats are sphagnum bogs and cedar swamps (*24*). In general, the distribution of major fresh-water swamps in this country coincides with the range of the EE virus and *C. melanura*, the principal area being the low-lying coastal plains.



Infection chain of the mosquito-borne viral encephalitides.

Wild birds are considered to be natural reservoirs of the EE virus. During outbreaks of the disease, approximately one-half of the birds sampled may show immunity against EE, as evidenced by the presence of neutralizing antibody (*26*). Virus recoveries have been made from about 20 species of wild birds. Domestic fowl are not considered to be important reservoirs of the eastern virus.

An abundance of summer rainfall seems to be a contributory factor in epidemics and epizootics of EE. This is exemplified by outbreaks in Massachusetts: In June and July 1938, the rainfall totaled 16 inches, and in August 1955, following hurricanes Connie and Diane, 17 inches occurred. Normal summer rainfall is 3 to 3.5 inches per month (*27*). Conversely, prevailing dry conditions tend to inhibit outbreaks. In 1957, when Massachusetts experienced an extreme summer drought, there were no verified clinical cases of EE in pheasants, humans, or horses (*28*).

St. Louis Encephalitis

St. Louis encephalitis is regarded by some as the most important mosquito-borne disease in

the United States. This type of encephalitis derives its name from the serious epidemic which was centered in St. Louis and St. Louis County, Mo., during the late summer of 1933. More than 1,100 human cases were reported in the city and county.

Distribution of the SLE virus is not so clearly delineated as that of the WE and EE types since the horse does not serve as a sentinel animal for SLE infections. In general, the virus occurs throughout the western and central States. An unusual record was obtained last summer, when the virus was recovered from the spinal fluid of a patient in Plant City, Fla. (personal communication from J. O. Bond, Florida State Board of Health, Jacksonville).

St. Louis encephalitis outbreaks appear to be of two types, rural and urban (*29*). The rural outbreaks have been mostly in irrigated areas extending from the central valley of California to western Texas. One of the more recent epidemics occurred in the high plains of Texas in 1956; there were at least 250 cases. One characteristic of rural SLE is that it is frequently mixed with WE infections.

Notable epidemics of SLE in urban and suburban areas since the historic 1933 outbreak include the following:

Date	*Place*	*Number of cases*
1937	St. Louis, Mo	500
1954	Hidalgo County, Tex	1,000
1955	Calvert City, Ky.[1]	13
1956	Louisville, Ky	110
1957	Cameron County, Tex	130

[1] Population 1,500.

Although all age groups are susceptible to the SLE virus, in many epidemics the incidence has been highest in individuals over 50 years of age. Mortality during recent years has ranged from less than 2 percent to 11 percent.

Current knowledge indicates that *C. tarsalis* is the principal vector of rural SLE in the west and *Culex pipiens-quinquefasciatus* the primary vector for the urban form of the disease in central United States. Several hundred virus isolations have been made from *C. tarsalis* in 8 States. Lumsden, in 1933, implicated *C. pipiens* as the vector of encephalitis in St. Louis (*30*); and SLE virus isolations were made from the *C. pipiens* complex during outbreaks in Yakima Valley, Wash., 1942 (*31*); in Hidalgo County, Tex., 1954 (*32*); and in Calvert City, Ky., 1955 (*33*).

C. pipiens-quinquefasciatus mosquitoes are frequently referred to as "dirty water" breeders since they show a predilection for polluted water. Outbreaks of SLE in the lower Ohio Valley in 1955 were associated with heavy production of *C. pipiens* caused by improper disposal of industrial wastes (*33*). During the Louisville outbreak in 1956, excessive populations of these mosquitoes were produced from an estimated 20,000 to 25,000 street catch basins situated throughout the city.

It is paradoxical that droughts and outbreaks of urban SLE seem to be related. Climatic conditions in the St. Louis area in 1933 were most unusual. According to official Weather Bureau records, the rainfall in St. Louis for the months of June, July, and August was the lowest in its history, since 1837 (*34*). It should be noted that a dearth of rainfall is not necessarily deleterious to the production of *C. pipiens*, a species that breeds prolifically in sewage disposal areas and other manmade breeding places which may receive an artificial supply of water.

Epidemiological studies have revealed that both wild and domestic birds are commonly infected with the SLE virus. Most of the evidence, however, is based on antibody studies rather than on virus recoveries. To date, the virus has been reported from only two birds in the United States, a mourning dove in California and a flicker in Kentucky (*33*).

Current Investigations and Control

Intensive investigations on mosquito-borne encephalitis are now underway in various sections of the United States. The primary purpose of these investigations is to acquire detailed information on the natural history of these viruses so that practical control measures can be developed. Some pertinent questions that need to be answered are: What causes the periodic epizootics and epidemics? Is each virus continuously present in an area or is it reintroduced? What is the most feasible approach to the control of encephalitis? What is the epidemic potential for the future?

Cause of Periodic Outbreaks

Current information suggests that epidemics and epizootics of mosquito-borne encephalitis are due to a fortuitous set of ecologic conditions, including high prevailing temperatures, heavy mosquito populations, and high rates of virus infections among an abundance of wild birdlife.

High sustained temperatures during the summer months seem to favor outbreaks of encephalitis. Normally the peak months for these outbreaks are July, August, and September. In mild climates, such as Florida and Texas, cases may occur as early as April, May, or June.

Many investigators believe that the most important basic factor in determining whether or not an epidemic would occur in an area is prevalence of the mosquito vector. There is much evidence that a rapid increase in vector populations is usually accompanied by a corresponding increase in virus infection among birds.

A large population of susceptible birds and efficient vectors in the vicinity of man and horses would set the stage for explosive outbreaks of encephalitis. Birds appear to be ideal natural hosts because, as Buescher points out, they are prolific and short-lived (*35*). This results in a rapid turnover of population with large numbers newly susceptible each year. Smaller birds and nestlings, which circulate high levels of virus in their blood, are probably more important than the larger and older birds in the transmission cycle. In a recent study, it was shown that *C. tarsalis* feeds much more readily on nestling birds than on adults of the same species (*36*).

Maintenance of Viruses

A major problem that still needs to be solved is how the viruses maintain themselves during the nonepidemic season. One leading theory is that they are harbored in overwintering mosquitoes; another possibility is that they are maintained by latent infections in birds. There is considerable evidence that both these survival mechanisms are involved. For example, the WE virus has been isolated from naturally infected *C. tarsalis* collected in Kern County, Calif., in all months of the year except December (*37*), and it can survive through the winter in experimentally infected *C. tarsalis* (*38*). Also, this virus has been isolated from *C. tarsalis* in Colorado in December (*39*). In support of the avian theory are laboratory studies showing that birds may have a recurrent viremia and persisting organ infection for 1 to 10 months after infection with the WE virus (*40*). The hypothesis that migratory birds annually reintroduce encephalitis into this country has fallen into disrepute. Results of recent studies by Kissling and co-workers on the EE virus indicate the presence of stationary foci of infection (*41*).

Control

At present, prevention of encephalitis consists of two principal approaches, immunization and mosquito control. Vaccination is recommended as a prophylaxis for equines, but human immunization is not considered economically feasible; furthermore, suitable vaccines for human use are now not available.

Employment of measures to minimize vector mosquito production in areas where encephalitis may be endemic or epidemic appears to be the most practical approach to prevention. Present knowledge indicates that control efforts should be focused on *C. tarsalis*, *C. pipiens-quinquefasciatus*, and *C. melanura*. An alternative possibility, which needs further study, would be the control of avian reservoirs of the viruses.

Epidemic Potential

Because of the lack of specific therapeutic treatment and lack of suitable human vaccines, coupled with the apparent widespread infection among birds and mosquitoes, the encephalitis viruses have a high epidemic potential. Hence, it behooves all public health workers to keep well informed on the epidemiology of this group of diseases and to maintain continuous vigilance. It is hoped that the intensive investigations now underway will provide a practical answer for adequate protection against the mosquito-borne encephalitis infections.

Summary

During the past 25 years, severe epidemics of western encephalitis (WE) and St. Louis

encephalitis (SLE) have occurred throughout western and central United States, but to date no major human outbreaks of eastern encephalitis (EE) have been recorded.

Because of grossly inaccurate and incomplete reporting, the actual incidence of "infectious encephalitis" is unknown, but this disease is being recognized as an increasingly serious health problem in the United States.

Information accumulated regarding the natural history of the WE, SLE, and EE viruses indicates that wild birds, particularly small species and nestlings, are the principal reservoirs of infection, and mosquitoes the vectors. The basic infection cycle normally is limited to birds and mosquitoes, with humans and horses as incidental entries.

Primary vectors of encephalitis include *Culex tarsalis* for both WE and rural SLE in the west and the *Culex pipiens* complex for urban SLE in central United States. Definite knowledge regarding the vectors of EE is lacking, but current evidence implicates *Culiseta melanura* for maintaining the basic infection chain in nature (bird-mosquito-bird) and possibly *Aedes*, *Psorophora*, and *Mansonia* mosquitoes for transmitting the disease to horses and humans.

Epidemics appear to be due to a fortuitous set of ecologic conditions, including high prevailing temperatures, heavy mosquito populations, and high rates of virus infections among nestling and adult birds.

The only known possible approach to the control of encephalitis in the United States is one of prevention, the employment of measures to minimize mosquito production in areas where encephalitis may be endemic or epidemic.

Owing to the constant and serious threat of encephalitis epidemics, it behooves all public health workers to keep well informed on the epidemiology of this group of diseases and to maintain continuous vigilance.

EDITOR'S NOTE: Mr. Beadle in this paper omits from western and eastern encephalitis the term "equine" on the ground that in the light of present knowledge this designation is misleading.

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