

SAINT LOUIS ENCEPHALITIS - A FLORIDA PROBLEM

Donald A. Shroyer, PhD., Florida Medical Entomology Laboratory

St. Louis encephalitis, abbreviated SLE, is the mosquito-transmitted virus disease that is generally considered to be of greatest medical importance in North America. SLE was first recognized in 1933 (in St. Louis, Missouri), and epidemics have occurred sporadically and unpredictably in the subsequent decades. During epidemics large numbers of people become seriously ill, sometimes fatally. Major SLE epidemics occurred in Florida in 1959, 1961, 1962, and 1977. SLE virus also occurs in Central and South America, but rarely causes human disease in those regions.

It is important to distinguish between the human disease that we call SLE, and the mosquito-transmitted virus that may cause such disease. Even during epidemics, only a small proportion of individuals actually infected with SLE virus become ill. SLE VIRUS is more common and widely distributed than SLE DISEASE. The virus is a permanent resident of Florida and can be found in some south Florida counties nearly every year.

However, the causes of epidemics of SLE disease are not known with certainty. Different strains of SLE virus vary substantially in their ability to cause disease. This suggests that epidemics might be caused by the local appearance of a “hot” strain of virus. Risk of epidemics increases when there are exceptionally large numbers of mosquitoes that are capable of transmitting SLE virus.



Historic distribution of SLE in the U.S.

DISEASE IN MAN

The occurrence and severity of SLE in man is strongly dependent on age. During epidemics, incidence of disease in people older than 60 is generally 5-40 times greater than in those less than 10 years old. Frequency of encephalitis (the most severe symptom associated with SLE) is also age-dependent, increasing from 56% for those age 20 or younger, to 87% for those over 60. Risk of death shows the same trend. Mortality is 7-24% among those with SLE and over 50, and less than 5% for those under 50. It is not uncommon for those surviving severe cases of SLE to suffer long-term residual neurological damage (known as “sequelae”), which may include paralysis, memory loss, or deterioration of fine motor skills.

It bears repeating that substantial numbers of people are infected with SLE virus, but do not develop recognizable disease. However, those who do become ill face a very serious threat to life. The incubation period from the infective mosquito bite to the first symptoms of SLE is 4-21 days.

TRANSMISSION CYCLE OF SLE VIRUS

The TRANSMISSION CYCLE leading to the SLE epidemics is thought to accompany infection of various species of wild birds. A bird infected by the bite of a mosquito can later produce enough virus in its blood to infect other susceptible mosquitoes that might feed upon it. It takes 1-2 days after infection for the bird to produce significant quantities of virus in its blood, and this virus rapidly disappears 1-3 days later as the bird recovers from the infection. Thus, there is a narrow “window of opportunity” for the mosquitoes to pick up the virus and then further spread (“amplify”) it. After experiencing one SLE infection, an individual bird is forever immune from another SLE infection. SLE virus is not known to cause disease in birds, and birds cannot infect one another.

Under suitable conditions, SLE virus is sufficiently amplified in local bird populations that virus is by chance also transmitted to man and other incidental hosts of the virus. Although SLE virus can produce severe disease in man, humans are poor hosts of the virus; they produce little SLE virus in the blood and are “dead ends” for further virus transmission. As even greater numbers of susceptible birds recover from SLE infections and become immune, new infections in mosquitoes dwindle. Since mosquitoes are short-lived, mosquitoes with “old” SLE infections rapidly disappear as well, and the epidemic rapidly ends.

The TRANSMISSION CYCLE described above is just one part of the, largely unknown, annual LIFE CYCLE of SLE virus in Florida. We know that the virus is maintained in parts of Florida even in years when we do not detect the virus in birds and mosquitoes.

There are mosquito species in Florida that are not suspected of playing a part in the TRANSMISSION CYCLE leading to epidemics, yet may nonetheless be important in

maintaining the SLE virus LIFE CYCLE. The role small mammals, such as rodents, play in maintaining virus is also unknown.

VIRUS INFECTION OF MOSQUITOES

Unlike birds and man, mosquitoes infected with SLE remain infected for life. Fortunately, that life rarely exceeds a week or two. It is surprisingly difficult for an individual mosquito to become infected with SLE virus and transmit that virus by biting another bird or man. Much has to happen before a mosquito can be converted from an uninfected mosquito to a dangerous transmitter of SLE virus.

Not all of the 70 mosquito species that occur in Florida are equally susceptible to infection with SLE virus. Some are incapable of ever transmitting this virus. When a susceptible mosquito takes a blood meal from an infected bird, its stomach cells become infected. After a few days, the virus goes on to infect other organs, including the salivary glands. When salivary glands begin to produce SLE-enriched saliva, the mosquito has been transformed into a dangerous insect that can transmit virus to each host that it bites.

SUSPECTED MOSQUITO TRANSMITTERS

The mosquito *Culex nigripalpus* is a common Florida mosquito that has been linked to past SLE epidemics in the state. It is a highly efficient transmitter and its preference to take blood meals from birds favors its involvement in the SLE TRANSMISSION CYCLE in Florida. *Cx. nigripalpus* is a tropical species and does not occur in most of North America; other *Culex* mosquitoes serve as the principal SLE transmitters in northern states. Three of these, *Cx. quinquefasciatus*, *Cx. salinarius* and *Cx. restuans*, are common in Florida but have not, as yet, been implicated in SLE transmission in Florida.

During past Florida SLE epidemics, several additional mosquitoes were found infected in nature, but it is unknown whether these species are important transmitters. While it is currently believed that *Culex nigripalpus* is the most important SLE-transmitting mosquito in Florida, it is unwise to ignore the likelihood that other species also contribute to the SLE problem.

SEASONAL OCCURRENCE

SLE virus transmission to birds – and to man during epidemics – is most likely from August through November. Populations of *Cx. nigripalpus* also tend to reach their annual peak during this period. While it is impossible to reliably predict epidemic activity, transmission to birds in Florida seems to increase when long periods of drought are broken by subsequent heavy rains. Efforts to monitor SLE virus, and to control *Cx. nigripalpus* in Florida have traditionally focused on August-November.

MONITORING THE PROBLEM

Many mosquito control districts or county health departments participate in a statewide encephalitis surveillance program organized by the Florida Department of Health & Rehabilitative Services. This surveillance system was begun in 1978, with the hope that it would indicate when the threat of an SLE epidemic is especially severe. The same surveillance system also monitors another mosquito-transmitted encephalitis virus, Eastern Encephalitis.

SLE virus is monitored by establishing “sentinel chicken flocks.” Blood samples are taken from chickens in each flock on a regular basis and tested for the presence of antibodies to SLE virus. The presence of these antibodies indicates that the chicken recovered from a recent SLE infection acquired by a mosquito bite.

For a variety of reasons, a sentinel-based surveillance system cannot be expected to reliably predict the occurrence of an SLE epidemic. Unfortunately, the absence of infections in a small sentinel flock can provide a false sense of security. Although there is a need for more effective means of monitoring risk of SLE epidemics, the present sentinel chicken system can provide important evidence of seasonal and annual patterns of virus activity.

An often over-looked potential benefit of sentinel flocks is their value, not in predicting epidemics, but in confirming that a developing epidemic of encephalitis is due to SLE virus infection, and not to one of many other microorganisms that can cause encephalitis in man.

There is no vaccine that can protect against SLE virus infection, and like all virus infections, SLE cannot be cured. Prevention must, therefore, concentrate on minimizing contact between man and potentially transmitting mosquitoes. This is usually done by suppressing populations of suspected transmitters of SLE virus. Unfortunately, *Cx. nigripalpus* uses a wide variety of semi-permanent or temporary water sources as larval habitats which are often difficult to find or eliminate.

Spraying to control adult populations is appropriate in certain circumstances, especially during an epidemic. Unfortunately, the explosive nature of epidemics often means that knowledge that an epidemic is underway may come only after peak transmission to man has already passed. This makes it nearly impossible to assess the effectiveness of emergency insecticide applications.

Need more information...

...on SLE and its control in Florida? Contact the mosquito control district in your county or **Dr. Roxanne Rutledge at the Florida Medical Entomology Laboratory, IFAS-University of Florida, 200 9th Street Southeast, Vero Beach, FL 32962.**

