Saint Louis Encephalitis: A Florida Problem

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St. Louis encephalitis, abbreviated SLE, was the mosquito-transmitted virus disease of greatest medical importance in North America prior to the introduction of West Nile (WN) virus in 1999. SLE was first recognized in 1933 in St. Louis, Missouri, and epidemics have occurred sporadically and unpredictably in the subsequent decades. A large outbreak occurred in the Ohio-Mississippi River Basin in 1975, a year when close to 2000 cases were reported nationwide. During epidemics large numbers of people become seriously ill, sometimes fatally. Major SLE epidemics occurred in Florida in 1959, 1961, 1962, 1977, and 1990. Since the Florida outbreak in 1990, when 223 cases were reported, only sporadic cases of the disease have occurred in the State, with the largest report of 9 cases occurring in 1997. 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 that are actually infected with SLE virus become ill. “SLE virus” is more common and widespread 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 conditions that lead to large epidemics of SLE disease are not known with certainty. Different strains of SLE virus vary substantially in their ability to cause disease, suggesting that development of epidemics could be favored by the local appearance of a “hot” strain of virus. Though risk of epidemics may increase when there are exceptionally large numbers of mosquitoes that are capable of transmitting SLE virus, it is also true that extremely high populations of such mosquitoes are seen several times per year in Florida without causing outbreaks of disease.

Disease in Humans

The occurrence and severity of SLE in humans are 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 SLE epidemics involves an “amplification phase” in which the proportion of wild
birds infected with SLE virus grows rapidly due to a convergence of favorable rainfall patterns, mosquito activity, and bird nesting activity. 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. 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 humans, they are poor hosts of the virus; they produce little SLE virus in the blood and are “dead ends” for further virus transmission. As ever greater numbers of susceptible birds recover from SLE infections and become immune, new infections in mosquitoes dwindle. 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 played by small mammals (such as rodents) in maintaining virus is also uncertain.

**Virus Infection of Mosquitoes**

Unlike birds and humans, mosquitoes infected with SLE virus remain infected for life. Fortunately, it is surprisingly difficult for an individual mosquito to become infected with SLE virus and transmit that virus by biting another bird or human. 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 mosquito species that occur in Florida are equally susceptible to infection with SLE virus, and some are incapable of ever transmitting the virus. When a susceptible mosquito takes a blood meal from an infected bird, its stomach cells become infected with the virus. 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 animal 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. However, the latter species likely play a role whenever SLE activity occurs in the Florida panhandle and other northern counties.

During past Florida SLE epidemics, several additional mosquito species 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 humans 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. This is a direct consequence of the well-documented association between rainfall events and synchronized bursts of egg-laying and blood-feeding by older *Cx. nigripalpus* mosquitoes. Unlike the mosquito transmitters prominent in northern states, this tropical mosquito is a so-called “flood-water” mosquito and is not dependent on stagnant pools of water as breeding sites.

**Monitoring the Problem**

Many mosquito control districts or county health departments participate in a state-wide encephalitis surveillance program organized by the Florida Department of Health in 1978. The same surveillance system also monitors two other important mosquito-transmitted encephalitis viruses, Eastern equine encephalitis (EEE) and WN virus. SLE virus activity is monitored by taking regular blood samples from established “sentinel chicken flocks.” Blood samples are tested for the presence of antibodies to SLE, WN, and EEE viruses by the DOH Laboratory. Presence of these antibodies to any of these viruses indicates that the chicken was recently infected by a mosquito bite, and the sentinel chicken system can provide important evidence of seasonal and annual patterns of virus activity. When judiciously used by local surveillance personnel, background levels of virus transmission characteristic of normal years can be distinguished from abnormal transmission patterns associated with epidemic activity. The monitoring of virus transmission to sentinel chickens is the most reliable means of detecting periods of elevated risk in Florida, especially when coupled with appropriate monitoring of local mosquito populations.

**Prevention and Control of Disease**

There is no vaccine that can protect against SLE virus infection. Prevention must, therefore, concentrate on minimizing contact between humans and potentially transmitting mosquitoes. This is usually done by suppressing populations of suspected transmitters of SLE virus. Unfortunately, *Cx. nigripalpus* is by far the most common and abundant mosquito throughout central and south Florida since it uses a wide variety of semi-permanent or temporary water sources as larval habitats. These mosquito production sites are often so extensive that control of larval populations of *Cx. nigripalpus* is impractical. It is instead necessary to attempt control of flying adult mosquitoes by insecticide application, especially during periods of elevated risk of virus transmission. However, the explosive nature of epidemics often makes it difficult to evaluate the effectiveness of emergency insecticide applications.

**Additional Information**

Need more information on SLE and its control in Florida? Contact the mosquito control district in your county or Dr. Roxanne Rutledge, UF/IFAS Extension Medical Entomologist, Florida Medical Entomology Laboratory, 200 9th Street S.E., Vero Beach, FL 32962.