St. Louis Encephalitis—The Role of Chickens [1]

Gary D. Butcher and Richard Miles[2]

St. Louis encephalitis (SLE) was first recognized in 1932 in Paris, Illinois. One year later, a large epidemic occurred in St. Louis and Kansas City, Missouri (thus "St. Louis" encephalitis) where over 1,100 cases of the disease were diagnosed. Since then, the disease has remained endemic—constantly present in a particular locality—and has spread widely throughout the United States.

St. Louis encephalitis occurs seasonally with most outbreaks in the summer and early fall, coinciding with greatest mosquito activity. The incidence of the disease in man declines rapidly as cool weather approaches and the mosquito population diminishes. It has been demonstrated that species of the *Culex* mosquito transmit the virus and are the principal vectors (carriers) of the SLE virus.

Birds have received much attention as possible hosts for the SLE virus because of the known feeding preference of the *Culex* mosquito for birds. SLE virus has been isolated from several species of wild and domestic birds. However, it must be noted that chickens do not develop the disease and that the degree of viremia or blood virus level produced by chickens is generally low. Thus, chickens do not play a significant role in the bird-mosquito transmission cycle. There are obviously many questions and concerns regarding chickens and their role in SLE outbreaks. These concerns are unfounded. The SLE virus is "mosquito" transmitted. There is no danger of becoming infected with the SLE virus from consuming poultry products including meat and eggs. Possibly this confusion has resulted because health officials throughout the state of Florida are using chickens as sentinels (test animals) in SLE virus suspect areas. These test chickens are exposed to mosquitoes in suspect areas. Blood samples are then evaluated to determine if the test chickens have been exposed to the virus and have developed antibodies in their blood against the virus. Remember, chickens do not get the disease. Chickens have been selected as test animals for these studies because they are relatively cheap, they are easy to handle, the mosquito has a preference for birds, and chickens do not normally have antibodies against the SLE virus. However, I want to stress that poultry products continue to be very high quality, nutritious, and safe; and there is no danger of becoming infected with the SLE virus from their consumption.

The clinical features of the disease in man include a sudden onset with headache, high fever, neck stiffness, nausea, mental confusion, and tremors. This is usually followed in 1 to 4 days by encephalitis (inflammation of the brain), with depression and sleepiness in some or excitement and sleeplessness in others. The disease may become complicated by pneumonia, bacterial blood infection, pulmonary embolism, or gastrointestinal hemorrhage. The severity of the disease increases with advancing age, with persons over 60 years of age most severely affected. The length of time from when a SLE virus-infected mosquito bites a person to when clinical signs of disease appear varies from four to 21 days. Domestic animals including the dog, cat, horse, chicken, etc. do not develop clinical signs of disease after being bitten by an SLE virus-infected mosquito.

1. This document is VM71, one of a series of the Veterinary Medicine-Large Animal Clinical Sciences Department, UF/IFAS Extension. Original publication date November 1991. Revised December 2017. Visit the EDIS website at http://edis.ifas.ufl.edu.

2. Gary D. Butcher, poultry veterinarian; and Richard Miles, professor emeritus, Animal Sciences Department; UF/IFAS Extension, Gainesville, FL 32611.

Reviewed: 03/2025

The incidence of SLE is seasonal as described earlier. The method of "overwintering" of the virus has not been clearly defined. The virus has been isolated from hibernating adult *Culex* mosquitoes. Serologic surveys show evidence for early springtime infections in wild bird populations. There is also evidence that female *Culex* mosquitoes can pass the virus to their offspring by way of the mosquito egg (transovarian transmission). The virus has also been shown to be present for long periods in the blood of bats. However, for the SLE virus to cause outbreaks in the human population, several conditions have to be present. As one studies the epizootiology of SLE, it becomes clear that many factors influence the rate of spread of the virus and determine the cause and extent of epidemics. An amplified transmission of the SLE virus begins in the springtime and early summer with the re-emergence of the *Culex* mosquitoes. Remember, the SLE virus is always present, but if conditions are favorable for the development of large populations of mosquitoes, a rapid increase in the virus transmission cycle follows. The virus may then enter the human population and cause an epidemic. Wild birds serve as the main virus carriers (in the blood) in the cycle. If the rate of virus transmission between wild birds and mosquitoes is sufficiently high, humans and other mammals may become infected. However, the blood virus level in man and domestic mammals is low. Thus, they do not serve as hosts for further virus spread.

Presently, no vaccine for the prevention of SLE is available. Reduction of the mosquito population remains the most widely used method for the prevention and control of SLE outbreaks. Surveillance programs focusing on early detection of increased virus activity using serologic testing of avian sera are also beneficial. In addition, avoiding contact with mosquitoes is recommended.