

# THE RISE AND FALL OF ST. LOUIS ENCEPHALITIS VIRUS IN FLORIDA

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## ABSTRACT

St. Louis encephalitis virus has had a fascinating history in Florida. The virus was introduced into the Miami area in the early 1950s. This introduction resulted in two human outbreaks, one in 1952 and a second in 1958. Three urban SLE epidemics were reported in the Tampa Bay area in 1959, 1961, and 1962. The virus virtually disappeared from the state until 1977 when a widespread rural SLE epidemic was reported in 20 Florida counties with 110 confirmed human cases. An almost identical rural SLE epidemic was reported in 1990 when 226 human SLE cases were reported in 28 Florida counties. Following the introduction of West Nile virus into Florida in 2001, reports of SLEV transmission to sentinel chickens and humans decreased dramatically. Except for a 2014 focal outbreak of two SLE cases in Duval County, only sporadic transmission of SLEV to humans and sentinel chickens was reported in the state between 1998 and 2022.

**Key words:** St. Louis encephalitis virus, arbovirus, mosquito-borne virus, sentinel chicken arboviral surveillance

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## THE INTRODUCTION OF SLEV INTO FLORIDA

The first isolation of St. Louis encephalitis virus (SLEV, genus *Flavivirus*) in Florida was from a 1952 human case in Miami (Bond et al. 1963). Six years later in 1958, the first multiple-case outbreak of SLE was reported in the same geographic region of Miami as the 1952 case when five humans were diagnosed with SLE (Ehrenkranz et al. 1963). Onset dates for these cases ranged from September 23 through December 29, 1958. Ages of the cases ranged from 32 to 77 and four were female. The 1958 Miami SLE outbreak occurred coincidentally with human SLE epidemics in the Caribbean Basin and Central America. Human cases were reported in Panama in February, Jamaica in August, and Trinidad and Panama in September (Ehrenkranz et al. 1963).

In 1957 sporadic SLEV activity was detected in the Tampa Bay area when the virus was isolated from cerebrospinal fluid of a man suffering from mild aseptic meningitis (Brody and Murray 1957). This case foreshadowed the Tampa Bay area urban SLE epidemics described below.

## THE URBAN SLE EPIDEMICS OF 1959, 1961, AND 1962

The virus reemerged as a series of human epidemics in the Tampa Bay area in 1959, 1961, and 1962. In 1959, a focal outbreak of human neurologic disease was reported in Pinellas County, Florida (Figure 1). Between August and November 1959, 68 cases of neurologic illness (19 aseptic meningitis and 49 clinical encephalitis (48 SLE and one eastern equine encephalitis virus)) and five deaths were reported. The epidemic peak occurred in late October. Intense vector control efforts began on 10/26/1959 and 10/30/1959, well after most of the cases were infected. Most cases in this outbreak were reported in urban St. Petersburg (southern Pinellas County) (Bond et al. 1963). A follow-up serosurvey conducted in January 1960 documented an estimated 5,000 subclinical cases of SLE for a subclinical to clinical ratio of 68:1 for the 1959 outbreak. The known urban vector of SLEV, *Culex quinquefasciatus*, was virtually absent during this epidemic while the rural vector, *Cx. nigripalpus* was far more abundant (Bond et al. 1963). The human SLE epidemic vector was not documented for the 1959 outbreak.

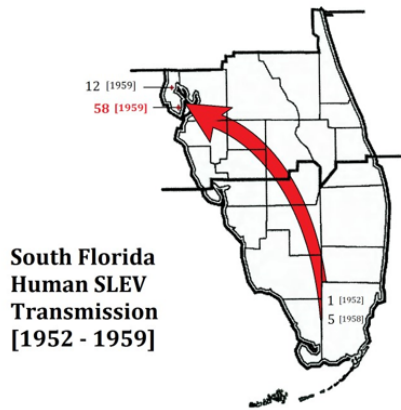


Figure 1. The early years of human SLEV transmission in Florida.

There was no evidence of SLEV transmission in the Tampa Bay region in 1960 (Figure 2). This is interesting considering the 1959, 1961, and 1962 sequence of human SLE epidemics in the region. One possible explanation for this is that Hurricane Donna that passed through the Tampa Bay area as a Category 2 hurricane on September 10<sup>th</sup>, 1960. Hurricanes tend to disrupt vector-borne disease transmission cycles because they disperse mosquito vectors and avian amplification host and disrupt mosquito and avian breeding cycles.

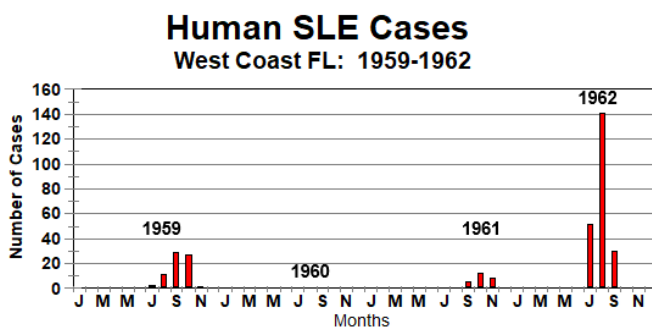


Figure 2. The temporal distribution of human SLE cases during the Tampa Bay area epidemics of 1959, 1961, and 1962.

The second of three SLE epidemics occurred in 1961 when 25 cases and seven deaths were reported in the Tampa Bay area (Chamberlain 1980). Unlike 1959 when the cases were restricted to southern Pinellas County, the cases in 1961 were reported in three adjacent counties: Pinellas (9 cases), Manatee (10 cases), and Sarasota (6 cases) (Waters et al. 1963). Onset dates for

these cases ranged from 10/3/61 through 12/17/61. The peak transmission of human SLEV was reported in mid-November. The outbreak began in southern Pinellas County in early October.

This outbreak was unusual for three reasons. First, the case-fatality ratio of 28% was unusually high. Second, the outbreak was unusually short, lasting only seven weeks. Finally, the onset of disease was two months later than normal. Mosquito control in all three counties was discontinued in August because of a lack of mosquito biting complaints, no rainfall 40 days prior to the beginning of the outbreak, and low mosquito catches. However, all patients interviewed remembered mosquito bites prior to disease onset (Waters et al. 1963).

The final and most extensive SLE epidemic, in terms of the number of cases and the spatial distribution of the cases, in the Tampa Bay area occurred in 1962 (Figure 2). From mid-July through mid-October, 222 confirmed cases and 43 deaths (case-fatality ratio of 19.4%) were reported in Hillsborough (20 cases), Manatee (16), Pinellas (171), and Sarasota (15) Counties. The epidemic began in urban St. Petersburg in mid-July and spread north, east, and south into Clearwater (Pinellas County), Tampa (Hillsborough County), Bradenton (Manatee County), and Sarasota (Sarasota County) in mid-August with the last case reported in Hillsborough County during the second week of October (Bond et al. 1965).

It was during this epidemic that *Culex nigripalpus* Theobald was incriminated as the SLE epidemic vector in Florida. Forty-two SLEV isolates were made from 64,000 mosquitoes collected in chicken-baited miniature light traps. Most (75%) of the mosquitoes were *Cx. nigripalpus* from which 40 SLEV isolates were made. An isolate of SLEV was made from one pool of *Anopheles crucians* Wiedemann and from one pool of an unidentified *Culex (Melanoconion)* spp. (Taylor 1969). In addition to the SLEV isolates from mosquito pools, SLEV isolates were also made from four human SLE cases in 1962.

**THE RURAL SLE EPIDEMICS OF 1977 AND 1990**

No human SLE cases were reported in Florida from 1962-1976 except for 1969 when three human infections were reported in Polk County with onset dates in late August (2 cases) and early September (1). In 1977 a dramatic shift in the SLE epidemic transmission pattern was observed in Florida. Prior to 1977, SLE epidemics were primarily urban with transmission foci in Miami, St. Petersburg, Clearwater, Tampa, Bradenton, and Sarasota. The 1977 SLE epidemic in Florida marked a shift from urban transmission to rural transmission (Day 2001). The

index case (first person infected) for the 1977 epidemic lived in Fellsmere, Florida (Indian River County). The infected individual had an onset date of August 8, 1977. The approximate infection date for this individual was July 30, 1977. What followed was an epidemic of 110 confirmed human SLE cases and 8 deaths (7.3% case-fatality ratio) in 20 south-central Florida Counties (Figure 3). The onset date for the final case in the epidemic was 12/5/77, in an outbreak that was more widespread than any previously reported SLE epidemic in Florida (Nelson et al. 1983).

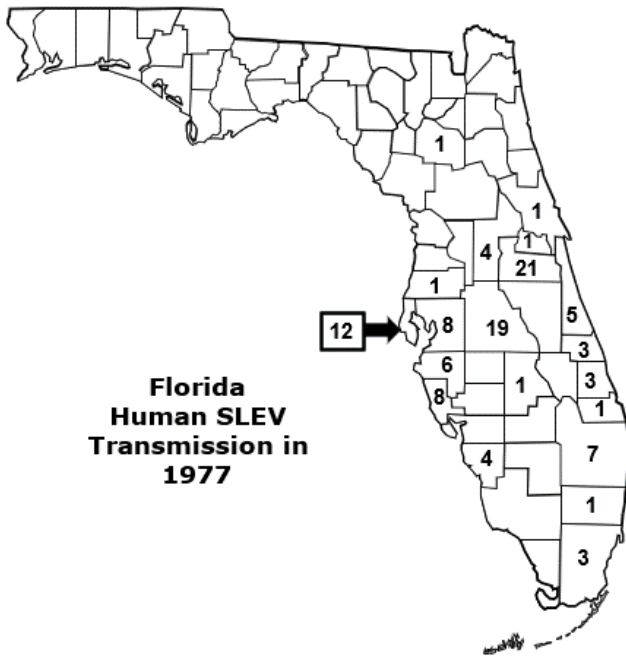


Figure 3. The spatial distribution of 110 human SLE cases in 20 Florida counties during 1977.

Sporadic human SLE cases were reported in South Florida between 1979 and 1985 with the notable exception of 1980 when a cluster of five human SLE cases was reported in Okaloosa County in the Florida Panhandle. The transmission of SLEV to humans in the Florida Panhandle is unusual with only six confirmed SLE cases reported between 1952 and 2022. The suspected epidemic vector for this outbreak was *Culex quinquefasciatus* Say (McCaig et al. 1994).

In 1990, a second widespread rural SLE epidemic that was remarkably similar to the 1977 outbreak was reported in South Florida (Meehan et al. 2000). The index case for the 1990 SLE epidemic was a 22-year-old male who, like the index case for the 1977 SLE epidemic, lived in Fellsmere, Florida (Indian River County). The onset date for the 1990 index case was July 28, 1990, with an approximate infection date of July 19. The 1990 SLE epidemic continued

for 25 weeks in 28 Florida counties (Figure 4) where 226 laboratory-confirmed human cases and 11 deaths (case-fatality ratio of 4.9%) were reported. The final human case was a 72-year-old male from Polk County with an onset date of 1/8/90 and an approximate infection date of 12/30/90 (Day 2001).

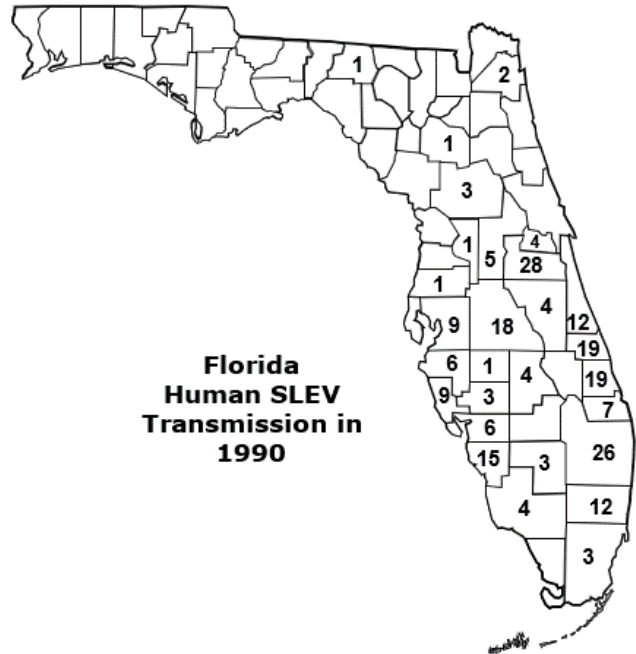


Figure 4. The spatial distribution of 226 human SLE cases in 28 Florida counties during 1990.

The 1990 Florida SLE epidemic marked the final reported epidemic of this virus in Florida. Between 1991 and 1999 focal (1993) and sporadic (1997) outbreaks of SLE in humans were reported in the state (Day and Stark 2000). However, no significant transmission of SLEV has been reported in Florida since 1990. Exact reasons for this decline are not known and some possible explanations are discussed below.

**POSSIBLE REASONS FOR THE URBAN TO RURAL SHIFT AND THE DECLINE OF SLEV IN FLORIDA**

The shift from an urban SLE transmission pattern (1958, 1959, 1961, and 1962) to a rural transmission pattern (1977 and 1990) was dramatic. One consistent factor between both types of transmission was the epidemic vector, *Cx. nigripalpus* (Dow et al. 1964, Nelson et al. 1983, Shroyer 1991). *Culex nigripalpus* is a subtropical mosquito that is extremely abundant in South Florida. The species is most abundant during the summer rainy season and

population peaks follow heavy rainfall events (Day and Curtis 1994). This species is an opportunistic blood feeder that oviposits in a wide variety of aquatic habitats found in urban and rural habitats (Provost 1969). The rural SLE epidemics of 1977 and 1990 mimicked past SLE epidemics in the western United States where *Cx. tarsalis* Coquillett was the primary epidemic vector and epidemics were associated with agricultural irrigation practices (Hammon 1941, Sciple et al. 1968). It is significant that a majority of human SLE cases in 1977 and 1990 occurred in regions of Florida where citrus farming and its associated irrigation patterns were abundant (Day and Curtis 1994).

The marked decline in the transmission of SLEV in Florida from 1998 to 2022 may have several explanations. Environmental factors may be important. All six of the Florida SLE epidemics (1958, 1959, 1961, 1962, 1977, and 1990) were preceded by severe winter freezes (Day and Shaman 2009). There have been no major freezes in South Florida since the Christmas freeze of 1989.

West Nile virus (WNV, genus *Flavivirus*) was first detected in Florida in 2001 (Blackmore et al. 2003). West Nile virus and SLEV are serologically related flaviviruses that circulate between *Culex* spp. mosquitoes and wild avian amplification hosts in well-defined transmission foci. Reports of a decline in transmission of SLEV in Florida and California suggest that there may be an immunological interaction between WNV and SLEV in wild avian amplification hosts reducing the transmission of SLEV in areas where the viruses overlap. It has been proposed that cross-neutralization in avian amplification hosts may explain the reduction of SLEV transmission after the introduction of WNV (Reisen et al. 2003, Fang and Reisen 2006, Reisen et al. 2008). However, Maharaj et al. (2018) have presented genetic evidence that, along with the co-circulation of WNV and SLEV in Arizona in 2015 (Yang et al. 2028), demonstrates pre-existing cross-neutralization immunity in avian amplification host populations does not eliminate the co-circulation of these two viruses.

There has been speculation since 1952 about how SLEV entered Florida and how the virus is dispersed around the state. In 1963 Ehrenkranz et al. proposed the possibility that migrant birds using the Mississippi and Atlantic flyways may serve as a means of SLEV introduction into Florida. Large numbers of migrating birds travel through Florida in the autumn during their southward migration and return through Florida in the spring during their northern migration. Introduction and dispersal of SLEV during both migrations is possible. Bond et al. (1963) suggest that migrant birds, along with their periodic delays due to environmental conditions (the migration

pulling-up effect) may be an important factor in the re-introduction and dispersal of SLEV in Florida. Likewise, Jennings et al. (1963) maintain that migrant birds were important to SLEV transmission in the Tampa Bay area in 1959 and 1961. They observed that areas surrounding most of the known human cases were heavily vegetated and contained ideal roosting sites, not only for resident wild birds, but also for migrants.

## THE FUTURE OF SLEV IN FLORIDA

It is evident that the transmission of SLEV in Florida has declined since 2000. The Florida Sentinel Chicken Arboviral Surveillance Program has been in place since 1978 (Day and Lewis 1992). During the lifetime of this program the Florida Department of Health has issued weekly, monthly, and annual summaries concerning arboviral transmission within the state including summaries for the sentinel chicken program. For the 23-year period from 1978 through 2000 an annual average of 173 SLEV antibody-positive isolates were made from sentinel chickens in Florida. In contrast, during the 22-year period from 2001 to 2022 an annual average of 24 SLEV antibody-positive isolates were made. Similar post-WNV declines in SLEV transmission were reported in Arizona and California (White et al. 2016).

It appears that the post-WNV transmission pattern of SLEV in Florida will be one of focal reintroduction, probably by migrating birds. This type of viral reintroduction and transmission has been reported in Arizona and California (White et al. 2016). The reintroduction and transmission of SLEV in Florida since 2001 has been focal. For example, in 2014 two human SLE cases were reported in Duval County during a year when 106 SLEV antibody-positive sentinel chickens were reported in 13 Florida counties. Most (84, 79%) of the antibody isolates were from chickens maintained in the central Florida peninsula.

Likely, the future of SLEV in Florida will center on migrating birds, local amplification of reintroduced SLEV in resident birds, and, in rare cases, infection of vector mosquitoes to a level where viral spillover to humans is possible. The 2022 arboviral transmission season in Florida illustrates this. During 2022, 19 SLEV antibody-positive sentinel chickens were reported in 11 Florida counties (Figure 5). One positive sentinel chicken was reported in Okaloosa County in March and likely represents the reintroduction of SLEV into Okaloosa County during spring migration. There were no antibody-positive sentinel chickens reported in Florida during April-June, a period when amplification is usually reported in breeding resident bird populations (Day

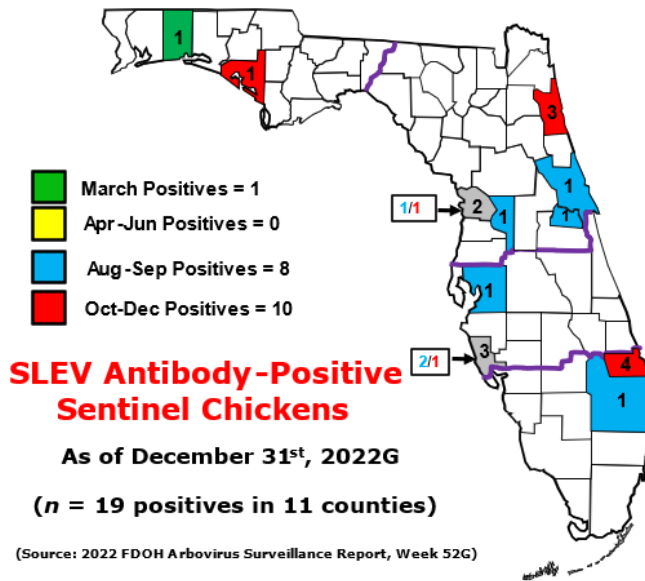


Figure 5. The spatial and temporal distribution of 19 SLEV antibody-positive sentinel chickens in 11 Florida counties during 2022.

1999). Eighteen SLEV antibody-positive sentinel chickens were reported from August-December, a period that corresponds to fall migration in Florida. Likely, large rural SLE epidemics are probably a thing of the past in Florida. Future SLE outbreaks will likely be urban and restricted to focal introductions by migrating birds followed by local amplification and spillover of SLEV to humans in small transmission foci.

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