ST. LOUIS ENCEPHALITIS VIRUS DISEASE
(St. Louis Encephalitis, SLE)

REPORTING INFORMATION

- **Class B1**: Report by the end of the next business day in which the case or suspected case presents and/or a positive laboratory result to the local public health department where the patient resides. If patient residence is unknown, report to the local public health department in which the reporting health care provider or laboratory is located.

- **Reporting Form(s) and/or Mechanism**:
  - Ohio Confidential Reportable Disease form (HEA 3334, rev. 1/09), Positive Laboratory Findings for Reportable Disease form (HEA 3333, rev. 8/05), the local public health department via the Ohio Disease Reporting System (ODRS) or telephone.
  - The Centers for Disease Control and Prevention (CDC) Mosquito borne Illness Case Investigation worksheet is available for use to assist in local disease investigation. Information collected from the form should be entered into ODRS and not sent to ODH, unless otherwise requested. If requested, the mailing address for this form is: Ohio Department of Health, Outbreak Response and Bioterrorism Investigation Team, 246 North High Street, Columbus, Ohio 43215.

- Additional reporting information, with specifics regarding the key fields for ODRS reporting, can be located in Section 7.

AGENT

St. Louis encephalitis (SLE) virus is a flavivirus and cross reacts serologically with other flaviviruses (e.g. dengue virus, West Nile virus, yellow fever, Japanese encephalitis virus).

**Infectious dose**: A single bite of an infectious mosquito.

CASE DEFINITION

**Clinical Description**
Most arboviral infections are asymptomatic. Clinical disease ranges from mild febrile illness to severe encephalitis. For the purposes of surveillance and reporting, based on their clinical presentation, arboviral disease cases are often categorized into two primary groups: neuroinvasive disease and non-neuroinvasive disease.

**Clinical Criteria for Diagnosis**

- **Neuroinvasive disease**: A clinically compatible case of neuroinvasive arboviral disease is defined as follows:
  - Fever (≥100.4°F or 38°C) as reported by the patient or a health-care provider and
  - Meningitis, encephalitis, acute flaccid paralysis or other acute signs of central or peripheral neurologic dysfunction, as documented by a physician and
  - Absence of a more likely clinical explanation.

- **Non-neuroinvasive disease**: A clinically compatible case of non-neuroinvasive arboviral disease is defined as follows:
  - Fever (≥100.4°F or 38°C) as reported by the patient or a health-care provider and
  - Absence of neuroinvasive disease and
  - Absence of a more likely clinical explanation.
Laboratory Criteria for Diagnosis

- Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, cerebrospinal fluid (CSF) or other body fluid or
- Four-fold or greater change in virus-specific quantitative antibody titers in paired sera or
- Virus-specific immunoglobulin M (IgM) antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen or
- Virus-specific IgM antibodies in CSF and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred or
- Virus-specific IgM antibodies in CSF or serum.

Case Classification

**Probable:**
- Neuroinvasive case: a case that meets the above clinical criteria for neuroinvasive disease and with virus-specific IgM antibodies in CSF or serum but with no other testing.
- Non-neuroinvasive case: a case that meets the above clinical criteria for non-neuroinvasive disease and with virus-specific IgM antibodies in CSF or serum but with no other testing.

**Confirmed:**
- Neuroinvasive case: a case that meets the above clinical criteria for neuroinvasive disease and one or more of the following laboratory criteria for a confirmed case:
  - Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, CSF or other body fluid or
  - Four-fold or greater change in virus-specific quantitative antibody titers in paired sera or
  - Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen or
  - Virus-specific IgM antibodies in CSF and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred.
- Non-neuroinvasive case: a case that meets the above clinical criteria for non-neuroinvasive disease and one or more of the following laboratory criteria for a confirmed case:
  - Isolation of virus from, or demonstration of specific viral antigen or nucleic acid in, tissue, blood, CSF or other body fluid or
  - Four-fold or greater change in virus-specific quantitative antibody titers in paired sera or
  - Virus-specific IgM antibodies in serum with confirmatory virus-specific neutralizing antibodies in the same or a later specimen or
  - Virus-specific IgM antibodies in CSF and a negative result for other IgM antibodies in CSF for arboviruses endemic to the region where exposure occurred.

Comment

Interpreting arboviral laboratory results:
- **Serologic cross-reactivity:** In some instances, arboviruses from the same genus produce cross-reactive antibodies. In geographic areas where two or more closely-related arboviruses occur, serologic testing for more than one virus may be needed and results compared to determine the specific
causative virus. For example, such testing might be needed to distinguish antibodies resulting from infections within genera, e.g., flaviviruses such as West Nile, St. Louis encephalitis, Powassan, Dengue, or Japanese encephalitis viruses.

- **Rise and fall of IgM antibodies:** For most arboviral infections, IgM antibodies are generally first detectable at 3 to 8 days after onset of illness and persist for 30 to 90 days, but longer persistence has been documented (e.g., up to 500 days for West Nile virus). Serum collected within 8 days of illness onset may not have detectable IgM and testing should be repeated on a convalescent-phase sample to rule out arboviral infection in those with a compatible clinical syndrome.

- **Persistence of IgM antibodies:** Arboviral IgM antibodies may be detected in some patients months or years after their acute infection. Therefore, the presence of these virus-specific IgM antibodies may signify a past infection and be unrelated to the current acute illness. Finding virus-specific IgM antibodies in CSF or a fourfold or greater change in virus-specific antibody titers between acute- and convalescent-phase serum specimens provides additional laboratory evidence that the arbovirus was the likely cause of the patient’s recent illness. Clinical and epidemiologic history also should be carefully considered.

- **Persistence of IgG and neutralizing antibodies:** Arboviral IgG and neutralizing antibodies can persist for many years following a symptomatic or asymptomatic infection. Therefore, the presence of these antibodies alone is only evidence of previous infection and clinically compatible cases with the presence of IgG, but not IgM, should be evaluated for other etiologic agents.

- **Arboviral serologic assays:** Assays for the detection of IgM and IgG antibodies commonly include enzyme-linked immunosorbent assay (ELISA), microsphere immunoassay (MIA) or immunofluorescence assay (IFA). These assays provide a presumptive diagnosis and should have confirmatory testing performed. Confirmatory testing involves the detection of arboviral-specific neutralizing antibodies utilizing assays such as plaque reduction neutralization test (PRNT).

- **Other information to consider:** Vaccination history, detailed travel history, date of onset of symptoms and knowledge of potentially cross-reactive arboviruses known to circulate in the geographic area should be considered when interpreting results.

**SIGNS AND SYMPTOMS**
St. Louis encephalitis viral infection can produce a febrile illness of variable severity associated with neurologic symptoms ranging from headache to aseptic meningitis or encephalitis. Arboviral encephalitis cannot be distinguished clinically from other central nervous system (CNS) infections. Symptoms can include headache, confusion or other alteration in sensorium, nausea and vomiting. Signs may include fever, meningoismus, cranial nerve palsies, paresis or paralysis, sensory deficits, altered reflexes, convulsions, abnormal movements and coma of varying degree. [See also the Aseptic Meningitis chapter.]

**DIAGNOSIS**
Laboratory diagnosis of arboviral infections is generally accomplished by testing of serum or CSF to detect virus-specific IgM and neutralizing antibodies. During an acute infection, certain viruses can be isolated through culture or detected by nucleic acid amplification.
In fatal cases, nucleic acid amplification, histopathology with immunohistochemistry and virus culture of autopsy tissues can also be useful. Only a few state laboratories or other specialized laboratories, including those at CDC, are capable of doing this specialized testing.

EPIDEMIOLOGY

Source
The vector in Ohio is the northern house mosquito, *Culex pipiens*. Birds are the amplification host. Humans are dead-end hosts.

Occurrence
SLE is only known in the western hemisphere. Virus isolations and a small number of human cases are documented from Central and South America, but outbreaks are not known from this region. In North America, widespread epidemics of SLE have occurred. The elderly experience more morbidity and mortality from SLE than children, giving SLE epidemics a distinctive age distribution. However, all age groups are affected. The most recent and largest epidemic of SLE occurred in 1975 in the Midwestern states, resulting in 1,815 cases with 416 cases and 29 fatalities, from Ohio. In the post-epidemic years, 1976-2010, Ohio has documented 19 cases.

Mode of Transmission
SLE virus is transmitted to humans through the bite of infected *Culex* species mosquitoes. Summer amplification of virus occurs in avians. The over-wintering mechanism is not understood.

Period of Communicability
Humans are dead-end hosts for the virus, i.e., they do not circulate sufficient numbers of the SLE virus in the bloodstream to infect a mosquito. The disease cannot be spread from person to person.

Incubation Period
5 to 15 days.

PUBLIC HEALTH MANAGEMENT

Case
Investigation
With serologic identification of SLE infection, a complete travel history for the three weeks prior to onset is obtained. Sites of outdoor exposure and after-dark activities can be evaluated for *Culex* potential by standard mosquito collection techniques (shelter collections, light traps, biting and larval samples, bait traps and oviposition [gravid] traps). Mosquitoes collected should be immediately placed on dry ice in sealed air-tight tubes and sent to the Zoonotic Disease Program (ZDP) for arboviral assay. The geographic extent of SLE activity can be estimated by an avian serosurvey.

Treatment
There is no specific therapy for SLE. Supportive care is indicated.

Isolation and Follow-up Specimens
Since the diagnosis of SLE is often not known until after patient discharge, enteroviral precautions (i.e. fecal, respiratory) are usually indicated for encephalitis. A plaque-reduction neutralization test is required for confirmatory testing.
Public Health Significance
Significant. Identification of a single case of SLE during summer months might signify that an outbreak is developing. A statewide epidemic of SLE occurred in 1975.

Contacts
No treatment or prophylaxis of contacts is indicated.

Prevention and Control
Vaccination
There is no vaccine.

Special Information
The risk of exposure to SLE virus is statewide because the northern house mosquito is abundant and has been found in every county.

Vector Investigation
Likelihood of SLE transmission is reduced if populations of the vector species, Culex pipiens, are kept under control by larviciding and control of breeding sites, including catch basins and backyard containers (tires, cans, bottles) in urban areas. Sewage-polluted ditches and stagnant water are more important in the rural setting. Education of the public about backyard breeding sites, screening of windows and personal protection are also recommended as a means of preventing cases. Surveillance of urban avians for seropositivity rates greater than 5% for SLE is useful in detecting an impending outbreak. For advice on vector assessment, contact the Zoonotic Disease Program 614-752-1029.

Because of the potential for epidemic SLE, the diagnosis of a single human case should be followed by control of adult mosquitoes by aerosol application (ultralow volume cold fog or thermal fog) of an approved pesticide. This is required to break the transmission cycle.
Ohio Department of Health Fact Sheet  
St. Louis Encephalitis (SLE)

**What is SLE?**
It is a mosquito-borne illness caused by a virus. SLE is one of a group of similar illnesses, including eastern equine encephalitis (EEE) and La Crosse encephalitis (LAC), which can affect the central nervous system in people and cause severe complications or even death.

SLE is found primarily in the Midwest and the southern United States, although occasional cases occur along the East Coast as far north as Connecticut and New York. There have also been a few documented human cases from Central and South America.

Nationally, widespread epidemics of SLE have occurred. The largest and most recent one occurred in 1975 in the Midwestern states, resulting in 1,815 cases, 416 of those, including 29 fatalities, were from Ohio. Between 1976 and 2010, Ohio has documented only 19 cases.

**How is SLE transmitted?**
SLE is acquired through the bite of an infected Culex mosquito. Mosquitoes become infected when they feed on birds carrying the SLE virus. Infected mosquitoes then transmit the SLE virus to other birds and to humans when they bite them. It can not be transmitted directly from person to person.

**How long after infection before symptoms appear?**
Symptoms generally appear within 5 to 15 days after being bitten by an infected mosquito.

**What are the symptoms of SLE?**
Mild infections occur without apparent symptoms other than fever with headache. More severe infection is marked by headache, high fever, neck stiffness, disorientation, coma, tremors, occasional seizures (especially in infants) and paralysis. 3 to 30% of cases are fatal, with most deaths occurring in the elderly.

**How is SLE diagnosed?**
Specific antibodies can be found in the blood or spinal fluid. Other tests are available to confirm infection, but results can take weeks. Care must be taken in interpreting test results due to possible cross-reactions with other similar viruses such as those causing West Nile encephalitis and dengue fever.

**Can SLE be treated?**
There is no specific treatment for SLE. Antibiotics are not effective against viruses, and no effective anti-viral drugs have been discovered. Patient care centers on treatment of symptoms and complications.

**Is there a vaccine against SLE?**
There is no human vaccine, and none are currently being researched.

**How can I prevent SLE?**
Prevent mosquito bites. It only takes one bite from an infected mosquito to transmit disease.

Avoid mosquito bites.
• Avoid areas where mosquitoes are active.
• Avoid outdoor activities during the peak mosquito biting times of dawn, dusk and early evening.
• When outdoors, apply mosquito repellant as directed to clothing and exposed skin.
• Reapply mosquito repellant as needed especially if swimming or sweating.
• Clothing will help protect you from mosquito bites. If weather permits, wear long pants, long sleeves, and/or socks.
• Install or repair window and door screens to keep mosquitoes outside.

Eliminate mosquito breeding sites.
• At least once or twice a week, empty water from flower pots, pet food and water dishes, birdbaths, swimming pool covers, buckets, barrels and cans.
• Check for clogged rain gutters and clean them out.
• Remove discarded tires and other items that could collect water.
• Be sure to check for containers or trash in places that may be hard to see, such as under bushes or under your home.

For more information please visit these Web sites:


CDC insect repellant use and safety