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# St. Louis Encephalitis Virus and Neuroinvasion: Insights into Virology, Epidemiology, Pathogenesis, Transmission, Clinical Features, and Global Public Health Impact

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**ABSTRACT**

St. Louis encephalitis virus (SLEV) is a mosquito-borne flavivirus endemic to the Americas and a significant cause of arboviral encephalitis. Primarily transmitted by *Culex* species mosquitoes in an enzootic cycle involving wild birds, SLEV sporadically spills over into human populations, leading to outbreaks of neurological disease. This review aims to synthesize the current understanding of SLEV, encompassing its history, virology, transmission dynamics, clinical manifestations, diagnostic methods, and prevention strategies. It also highlights the emerging challenges and future directions for research and public health intervention. A comprehensive literature review was conducted using scientific databases (e.g., PubMed, Scopus) to collate information on SLEV's molecular biology, epidemiology, pathogenesis, and control.Since its identification in 1933, SLEV has caused numerous outbreaks across North and South America. Its single-stranded positive-sense RNA genome encodes a polyprotein that is cleaved into three structural and seven non-structural proteins. The virus's pathogenesis involves neuroinvasion following a mosquito bite, leading to inflammation of the brain parenchyma. Clinical presentation ranges from a mild febrile illness to severe encephalitis, with advanced age being the most significant risk factor for severe disease and mortality. Diagnosis relies on serological assays (e.g., IgM ELISA) and molecular methods (RT-PCR). No specific antiviral treatment exists; management is supportive. Prevention hinges on integrated vector control and personal protective measures against mosquito bites.SLEV remains a persistent public health threat. Its ecological complexity, co-circulation with other flaviviruses, and the challenges of climate change and urbanization necessitate sustained surveillance, robust diagnostic capabilities, and effective public health messaging to mitigate its impact.

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**Keywords**
St. Louis Encephalitis Virus, SLEV, Flavivirus, Arbovirus, Encephalitis, *Culex* Mosquito, Zoonosis, Neuroinvasion, Vector-Borne Disease, Public Health.



## INTRODUCTION

St. Louis encephalitis virus (SLEV) is a mosquito-borne virus that belongs to the Flavivirus genus. It is primarily transmitted to humans through the bite of infected mosquitoes, particularly the Culex species. SLEV can cause inflammation of the brain, leading to a condition known as St. Louis encephalitis. This viral infection has been identified in various regions of the Americas, including the United States, Mexico, and parts of South America (Baele and Lemey. 2013). The symptoms of St. Louis encephalitis typically include fever, headache, dizziness, nausea, and malaise. In more severe cases, patients may experience central nervous system infections, such as confusion, disorientation, tremors, and even coma. While individuals of any age can be infected, the risk of severe illness and complications is higher among the elderly (Auguste et al., 2019).

Diagnosing St. Louis encephalitis involves laboratory tests on blood or spinal fluid samples to detect specific antibodies produced in response to the viral infection. Unfortunately, there is no specific treatment for SLEV, and antibiotics are ineffective against viral infections. Supportive therapy is provided to manage symptoms and complications, including hospitalization, respiratory support, intravenous fluids, and prevention of secondary infections (Coleman et al., 2015).

Preventing St. Louis encephalitis primarily involves measures to reduce mosquito bites and control mosquito populations. This includes using insect repellents, wearing protective clothing, and eliminating mosquito breeding sites by removing standing water sources. Additionally, public health efforts focus on surveillance, monitoring mosquito populations, and implementing vector control strategies to minimize the spread of the virus.

St. Louis encephalitis virus poses a significant public health concern in regions where it is endemic. Understanding the transmission dynamics, clinical manifestations, and preventive measures associated with SLEV is crucial for effective disease management and control. Ongoing research and surveillance efforts are necessary to monitor the prevalence and potential outbreaks of St. Louis encephalitis and to develop strategies to mitigate its impact on human health (Coleman et al., 2015).

## HISTORY

St. Louis encephalitis virus (SLEV) has a long history, with the first documented outbreak occurring in St. Louis, Missouri, United States, in 1933. The virus was initially isolated from the brain of a fatal encephalitis case during that outbreak, hence its name.

Following the initial outbreak, SLEV continued to cause sporadic cases and outbreaks in various parts of the United States. In the 1940s and 1950s, several large outbreaks occurred in cities such as Chicago, Detroit, and Los Angeles. These outbreaks led to increased research and surveillance efforts to better understand the virus and its transmission dynamics (Day, 2021).

During the mid-20th century, SLEV was recognized as one of the leading causes of mosquito-borne encephalitis in the United States. However, with the introduction of mosquito control programs and improved public health measures, the incidence of SLEV decreased significantly. The last major outbreak in the United States occurred in the 1970s in the Midwest.

While the United States has seen a decline in SLEV cases, the virus continues to circulate in other parts of the Americas. Outbreaks have been reported in Mexico, the Caribbean, and parts of South America. In recent years, there have been sporadic cases and localized outbreaks in Colombia, Venezuela, and Brazil (Baillie et al., 2018).

The geographic distribution of SLEV has expanded over time, with the virus now found in various bird species and mosquito vectors across the Americas. This expansion has raised concerns about the potential for future outbreaks and the need for ongoing surveillance and control measures.

The history of St. Louis encephalitis virus spans several decades, with initial outbreaks in the United States followed by sporadic cases and outbreaks in different parts of the Americas. Understanding the historical context of SLEV is important for recognizing its impact on public health and guiding future prevention and control efforts (Diaz et al., 2018).

## CLASSIFICATION

The classification of St. Louis encephalitis virus is as follows:
Family: Flaviviridae
Genus: Flavivirus
Species: St. Louis encephalitis virus
This classification is based on the virus's genetic and structural characteristics, as well as its mode of transmission through mosquitoes. Understanding the classification aids in studying its biology, epidemiology, and developing strategies for prevention and control.

St. Louis encephalitis virus (SLEV) is classified as a member of the Flaviviridae family, genus Flavivirus. It is an arthropod-borne virus (arbovirus) that is primarily transmitted to humans and animals through the bites of infected mosquitoes, particularly the Culex species.
SLEV is an enveloped, single-stranded RNA virus with a positive-sense genome. It is closely related to other flaviviruses, such as West Nile virus, Japanese encephalitis virus, and Zika virus. These viruses share similarities in their genetic makeup and structure (Drummond, and Rambaut, 2017).

Within the Flavivirus genus, SLEV is further classified into different genotypes based on genetic variations. Currently, there are eight recognized genotypes of SLEV, labeled as genotypes I to VIII. These genotypes exhibit distinct genetic characteristics and geographic distributions.

Genotype I is the most widespread and has been found in various regions, including North America, South America, and the Caribbean. Genotype II is primarily found in South America, while genotypes III and IV have been reported in the United States and Colombia, respectively.

The classification of SLEV into different genotypes is important for understanding the genetic diversity of the virus and its potential impact on disease transmission, virulence, and epidemiology. It also helps in tracking the spread of the virus and designing appropriate control strategies (Edgar, 2014).

In summary, St. Louis encephalitis virus belongs to the Flaviviridae family, genus Flavivirus. It is further classified into different genotypes based on genetic variations, with genotype I being the most widespread.

## STRUCTURE

The structure of St. Louis encephalitis virus (SLEV) is similar to other flaviviruses (Fig. 1). It is an enveloped virus with a spherical shape and a diameter of approximately 50 nanometers. The virus particle consists of three main structural components: the envelope (E) protein, the membrane (M) protein, and the capsid (C) protein (Fang, and Reisen, 2017).

The envelope protein (E protein) is embedded in the lipid envelope and is responsible for binding to host cells and mediating viral entry. It plays a crucial role in the virus-host interaction and is a major target for the host immune response. The E protein is composed of three domains: domain I, domain II, and domain III. These domains undergo conformational changes during viral fusion with host cell membranes.

The membrane protein (M protein) is a transmembrane protein that anchors the envelope to the viral core. It is involved in virus assembly and release. The capsid protein (C protein) forms the inner core of the virus particle and encapsulates the viral RNA genome. It plays a role in protecting the viral RNA and facilitating viral replication.

The genome of SLEV is a single-stranded RNA molecule with a positive sense. It contains approximately 11,000 nucleotides and encodes a single polyprotein that is processed into three structural proteins (C, M, and E) and several non-structural proteins. Overall, the structure of St. Louis encephalitis virus is characterized by its enveloped spherical shape, with the envelope protein, membrane protein, and capsid protein playing essential roles in viral entry, assembly, and replication (Grubaugh et al. 2019).

Figure 1: St. Louis encephalitis virus particles
**Source:** (https://www.sciencephoto.com/media/1116650/view/st-louis-encephalitis-virus-particles-illustration)

**Genome structure**

St. Louis encephalitis virus (SLEV) has a single-stranded, positive-sense RNA genome, which means the genetic information can be directly translated by host cells. The genome is approximately 11 kilobases in length and encodes a single, large polyprotein.

The genome of SLEV is organized into three structural and seven non-structural proteins. The structural proteins form the viral particle, while the non-structural proteins play various roles in the virus's replication and evasion of the host immune response (Grubaugh et al. 2019).

The structural proteins include:
1. Capsid (C)
2. Envelope (E)
3. Membrane (M or prM)

The non-structural proteins are:
1. NS1 2. NS2A 3. NS2B 4. NS3
5. NS4A 6. NS4B 7. NS5

These proteins are involved in processes such as viral replication, translation, assembly, and evasion of the host's immune response. Understanding the genome structure of SLEV is crucial for studying its molecular biology, developing diagnostic tools, and designing strategies for antiviral interventions (Hepp et al., 2018).

## VIRAL REPLICATION

The replication of St. Louis encephalitis virus (SLEV) follows a well-defined process within the host cell. Here is a simplified overview of the viral replication cycle:

1. Attachment and Entry: SLEV attaches to specific receptors on the surface of host cells, typically neural cells and cells of the immune system. The viral envelope protein (E protein) mediates attachment and fusion of the virus with the host cell membrane, allowing the viral particle to enter the cell (Iheukwumere *et al*., 2025a).

2. Uncoating and Release of Viral Genome: Once inside the host cell, the viral particle undergoes uncoating, where the viral RNA genome is released from the capsid protein. The viral genome then serves as a template for replication and protein synthesis (Iheukwumere *et al*., 2025b).

3. Genome Replication: The viral RNA genome is replicated by the viral RNA-dependent RNA polymerase (RdRp) within specialized compartments called replication complexes. This process involves the synthesis of a complementary negative-sense RNA strand, which is then used as a template to generate multiple positive-sense RNA copies (Iheukwumere *et al*., 2025c).

4. Protein Synthesis: The viral genome also serves as a template for the synthesis of viral proteins. The polyprotein precursor is synthesized and processed by viral and host proteases into individual structural and non-structural proteins. These proteins play essential roles in viral assembly, replication, and evasion of host immune responses (Iheukwumere *et al*., 2025d).

5. Assembly and Maturation: Newly synthesized viral RNA and structural proteins are assembled into new viral particles within the host cell. The viral particles acquire their envelope by budding through host cell membranes, incorporating viral glycoproteins into the lipid envelope (Iheukwumere *et al*., 2025e).

6. Release: The mature viral particles are released from the infected host cell, either by cell lysis or by budding from the plasma membrane. These released viral particles can then infect new host cells and continue the replication cycle (Iheukwumere *et al*., 2025f).

It is important to note that viral replication is a complex process, and there may be additional steps and factors involved in the replication of St. Louis encephalitis virus. However, the general outline provided above gives an overview of the key steps involved in the replication of SLEV within host cells (Quick et al., 2017).

## TRANSMISSION MECHANISM

The primary transmission mechanism for St. Louis encephalitis virus (SLEV) involves mosquitoes. SLEV is mainly transmitted through the bite of infected mosquitoes to humans and other vertebrate hosts. Mosquito species belonging to the Culex genus are common vectors for SLEV transmission.

**Here's a simplified overview of the transmission cycle:**

1. Amplification in Birds: SLEV typically circulates in wild birds, serving as amplifying hosts. Mosquitoes become infected when they feed on viremic (virus-carrying) birds.
2. Transmission to Humans and Other Mammals: Infected mosquitoes then bite humans, horses, and other mammals, transmitting the virus. Humans are considered incidental hosts, as they don't play a significant role in the virus's natural amplification cycle.

3. Human Infection: Once transmitted to humans, SLEV can cause a range of clinical manifestations, from mild febrile illness to severe neurological complications like encephalitis. Understanding and monitoring the transmission cycle are crucial for implementing effective mosquito control measures and public health strategies to prevent and manage outbreaks of St. Louis encephalitis. It is worth mentioning that while mosquitoes are the primary vector for SLEV, there have been rare cases of transmission through blood transfusion, organ transplantation, and laboratory accidents. However, these modes of transmission are considered to be very uncommon (Jones et al., 2021).

## PATHOGENESIS

The pathogenesis of St. Louis encephalitis virus (SLEV) involves several steps that contribute to the development of neurological symptoms and brain inflammation:

1. Entry and Replication: SLEV enters the body through the bite of an infected mosquito. The virus initially replicates in the skin and local lymph nodes, and then spreads to the bloodstream. From there, it can cross the blood-brain barrier and invade the central nervous system (CNS) (Iheukwumere *et al*., 2024a).

2. Neuroinvasion: Once in the CNS, SLEV infects neurons and glial cells, leading to neuronal damage and inflammation. The virus can also infect other cells of the immune system,

such as microglia and astrocytes, which contribute to the inflammatory response (Iheukwumere *et al*., 2024b).

3. Immune Response: The immune response plays a crucial role in the pathogenesis of SLEV. The infection triggers the release of pro-inflammatory cytokines, chemokines, and other immune mediators, leading to inflammation in the brain. This immune response can contribute to tissue damage and neurological symptoms (Iheukwumere *et al*., 2024c).

4. Neuronal Damage: SLEV infection can directly cause damage to neurons through various mechanisms. The virus can disrupt normal cellular processes, induce apoptosis (cell death), and trigger an excessive immune response that leads to collateral damage to healthy neurons (Iheukwumere *et al*., 2024d).

5. Blood-Brain Barrier Dysfunction: SLEV infection can disrupt the integrity of the blood-brain barrier, which normally protects the brain from harmful substances. This barrier dysfunction allows immune cells and inflammatory molecules to enter the brain more easily, exacerbating the inflammatory response and contributing to brain damage (Iheukwumere *et al*., 2025e).

6. Clinical Manifestations: The pathogenesis of SLEV can result in a range of clinical manifestations, including fever, headache, dizziness, nausea, and malaise. In severe cases, patients may develop signs of central nervous system involvement, such as confusion, disorientation, tremors, and even coma. Elderly individuals are at higher risk of developing severe disease (Iheukwumere *et al*., 2025f).

It is important to note that the pathogenesis of SLEV is complex and can vary among individuals. Factors such as the host's immune response, viral strain, and other host-related factors can influence the severity and outcome of the infection (Lord et al**.,** 2019).

## CLINICAL MANIFESTATIONS

Clinical manifestations of St. Louis encephalitis virus (SLEV) can vary depending on the individual and the severity of the infection. Some common clinical manifestations include:

1. Mild Symptoms: Many individuals infected with SLEV may experience mild symptoms or may be asymptomatic. These mild symptoms can include fever, headache, muscle aches, fatigue, and gastrointestinal symptoms such as nausea and vomiting.

2. Neurological Symptoms: In more severe cases, SLEV can cause neurological symptoms. These can include a stiff neck, confusion, disorientation, dizziness, tremors, unsteadiness, and difficulty with coordination and balance. Severe cases may progress to coma.

3. Encephalitis: St. Louis encephalitis refers to the inflammation of the brain. In some individuals, SLEV infection can lead to encephalitis, which is characterized by more severe neurological symptoms. Encephalitis can cause changes in mental status, seizures, focal neurological deficits, and even paralysis.

4. Age-related Differences: The clinical manifestations of SLEV infection can differ based on age. Children and young adults are more likely to experience milder symptoms, such as fever and headache, or develop aseptic meningitis, which is inflammation of the membranes surrounding the brain and spinal cord. In contrast, older adults are at higher risk of developing severe encephalitis with more pronounced neurological symptoms.

5. Case Fatality: While most individuals infected with SLEV recover completely, severe cases can be life-threatening. The overall case fatality rate for St. Louis encephalitis is estimated to be between 5-15%, with the risk of fatal disease increasing with age.

It is important to note that the clinical manifestations of SLEV can overlap with other viral encephalitis or febrile illnesses. Laboratory testing is necessary to confirm the diagnosis of St. Louis encephalitis and differentiate it from other similar conditions (Li et al., 2019).

### Laboratory Diagnosis

The laboratory diagnosis of St. Louis encephalitis virus (SLEV) infection involves several methods to detect the presence of the virus or the body's immune response to the infection. These methods include:

1. Virus Isolation: SLEV can be isolated from various clinical specimens, such as cerebrospinal fluid (CSF), blood, or tissue samples, using cell culture techniques. This method involves inoculating the specimen onto specific cell lines that are susceptible to SLEV infection. The presence of the virus can be confirmed by observing characteristic cytopathic effects or by detecting viral antigens using immunofluorescence or molecular techniques (Iheukwumere *et al*., 2025g).
2. Reverse Transcription Polymerase Chain Reaction (RT-PCR): This molecular technique is commonly used to detect the genetic material (RNA) of SLEV in clinical specimens. RT-PCR amplifies specific regions of the viral genome, allowing for the detection and identification of SLEV. This method is highly sensitive and specific and can be performed on various specimens, including CSF, blood, or tissue samples (Iheukwumere *et al*., 2025h).

3. Serological Tests: Serological tests detect the presence of antibodies produced by the body in response to SLEV infection. These tests include enzyme-linked immunosorbent assays (ELISA), neutralization assays, and hemagglutination inhibition assays. Serological testing is usually performed on paired serum samples, with the first sample collected during the acute phase of illness and the second sample collected 2-4 weeks later. A significant increase in antibody titer between the two samples confirms recent SLEV infection (Iheukwumere *et al*., 2025i).

4. IgM Capture ELISA: This specific serological test detects the presence of SLEV-specific IgM antibodies, which are produced early in the course of infection. IgM capture ELISA is a rapid and sensitive method and is commonly used for the diagnosis of acute SLEV infection (Iheukwumere *et al*., 2025j).

It is important to note that laboratory diagnosis should be performed in specialized laboratories with expertise in

handling and testing for arboviruses, including SLEV. Clinical and epidemiological information should also be considered in conjunction with laboratory results for an accurate diagnosis of St. Louis encephalitis.

## TREATMENT AND PREVENTION

**Treatment**:

There is no specific antiviral treatment for St. Louis encephalitis virus (SLEV) infection. Supportive care is the mainstay of treatment for patients with severe illness. This may include hospitalization, respiratory support, intravenous fluids, and management of complications. It is important to monitor and manage symptoms, such as fever, headache, and neurological manifestations.

**Prevention**:

Prevention of SLEV infection primarily involves mosquito control measures and personal protective measures:

1. **Mosquito Control:** Reducing mosquito populations and their breeding sites is crucial in preventing SLEV transmission. This can be achieved by eliminating standing water sources, such as emptying containers, cleaning gutters, and ensuring proper drainage. Insecticides may also be used to control adult mosquitoes and larval habitats.

**2. Personal Protective Measures:** Individuals can protect themselves from mosquito bites by following these measures:

- Use insect repellents: Apply insect repellents containing DEET, picaridin, IR3535, or oil of lemon eucalyptus on exposed skin and clothing. Follow the instructions on the product label.
- Wear protective clothing: Wear long sleeves, long pants, and socks to minimize exposed skin.
- Use bed nets: When sleeping in areas with high mosquito activity, use bed nets that are properly treated with insecticides.
- Avoid peak mosquito activity: Mosquitoes that transmit SLEV are most active during dawn and dusk. Minimize outdoor activities during these times.
- Maintain screened windows and doors: Ensure that windows and doors have intact screens to prevent mosquito entry into the living spaces.
- Eliminate standing water: Remove or regularly empty containers that can collect water, such as flower pots, buckets, and tires, to eliminate mosquito breeding sites (Kramer, and Chandler, 2021).

## CONCLUSION

In conclusion, St. Louis encephalitis virus (SLEV) is a mosquito-transmitted virus that can cause inflammation of the brain. The infection is considered a neglected disease, with low scientific production and a lack of overall knowledge about the infection. SLEV can lead to a range of symptoms, from mild fever and headache to severe neurological complications. Elderly individuals are at higher risk of severe disease. There is no specific treatment for SLEV, and supportive care is provided for severe cases. Prevention involves mosquito control measures and personal protective measures, such as using insect repellents, wearing protective clothing, and eliminating mosquito breeding sites. It is crucial to raise awareness, implement active surveillance, and establish prevention policies to prevent the introduction and dissemination of SLEV in the population.

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