

Vector-borne viral diseases in horses

Sunutcha Suntrarachun^{1*}

Abstract

Vector-borne viral diseases are major threats to equine health worldwide. These infections are transmitted by arthropod vectors such as mosquitoes, biting midges, and horseflies, resulting in clinical manifestations ranging from mild febrile illness to severe neurological, respiratory, circulatory, or hematologic disorders with high mortality rates. Major equine vector-borne viral diseases include African horse sickness (AHS), equine infectious anemia (EIA), equine encephalomyelitis (Eastern: EEE, Western: WEE, and Venezuelan: VEE), West Nile virus (WNV), and Japanese encephalitis (JE). Each disease is caused by a distinct viral pathogen and transmitted by specific vectors that influence their epidemiology and geographic distribution. Transmission occurs through either biological replication within the vector or mechanical transfer via contaminated mouthparts, depending on the virus involved. Recent climate change, environmental alterations, and increasing international animal movement have contributed to the expansion of competent vectors and the emergence of these diseases in previously non-endemic regions. Effective prevention and control strategies rely on vaccination, vector control, surveillance, movement restriction, and early laboratory diagnosis. Cooperation among veterinary, vector, and environmental sectors is important for early outbreak detection and effective disease control.

Keywords: arthropod vector, equine, horse, viral disease

¹*Department of Research and Development, Queen Saovabha Memorial Institute Thai Red Cross Society, Bangkok 10330, Thailand*

***Correspondence:** *sunutcha@yahoo.com, sunutcha.s@redcross.or.th* (S. Suntrarachun)

Received November 13, 2025

Accepted May 26, 2026

<https://doi.org/10.56808/2985-1130.3966>

Introduction

Horses have long played important roles in transportation, agriculture, sport, therapy, and biomedical applications. In addition to their economic and cultural value, horses are essential for the production of antivenoms and immunotherapeutic agents, making equine health a matter of both veterinary and public health importance (Akesowan and Suntrarachun, 2007; Kumar *et al.*, 2018).

Among infectious diseases affecting horses, vector-borne viral diseases are emerging threats due to their rapid spread, high mortality, and zoonotic potential. Hematophagous arthropods, including mosquitoes, biting midges (*Culicoides* spp.), and horseflies (*Tabanus* spp.), transmit these diseases as biological or mechanical vectors. Clinical outcomes range from mild febrile illness to severe neurological, respiratory, or circulatory disease. Importantly, several equine arboviruses are zoonotic, posing risks not only to equine populations but also to human health (Onmaz *et al.*, 2013; Toribio, 2022).

Transmission mechanisms vary among viruses and influence epidemiology and control strategies. Biological transmission involves viral replication within the vector before transmission to a new host, whereas mechanical transmission occurs through contaminated mouthparts without viral replication. Major equine vector-borne viral diseases include African horse sickness (AHS), equine infectious anemia (EIA), Eastern, Western, and Venezuelan equine encephalomyelitis (EEE, WEE, and VEE), West Nile virus (WNV) infection, and Japanese encephalitis (JE), all of which have substantial impacts on equine health and the global equine industry (Kumar *et al.*, 2018).

In recent decades, the geographic expansion of these viruses has accelerated due to climate change, globalization, and alterations in land-use patterns. Rising temperatures can shorten the extrinsic incubation period of viruses within vectors, thereby increasing transmission efficiency. Increased rainfall and flooding create additional breeding sites for mosquitoes and biting midges, while high humidity enhances vector survival. Conversely, drought conditions may concentrate hosts and vectors around limited water sources, facilitating transmission. These environmental changes have enabled vectors and pathogens to expand into previously non-endemic regions. Importantly, outbreaks of AHS have been reported beyond traditional endemic zones, while WNV and JE have expanded into temperate regions, reflecting the growing influence of environmental factors on vector ecology (Toribio, 2022).

Vaccination, vector surveillance, environmental management, and biosecurity measures must all be included in combined strategies to effectively prevent and control horse vector-borne virus infections. However, significant challenges remain, including limited vaccine availability for certain diseases, viral genetic diversity, and the need for improved international coordination. Addressing these challenges is essential for reducing the impact of these diseases on animal and human health (Onmaz *et al.*, 2013; Kumar *et al.*, 2018).

With a focus on their transmission mechanism, clinical symptoms, laboratory diagnosis, and preventative measures, this review attempts to summarize the current understanding of the main horse vector-borne viral infections. A comprehensive understanding of these factors is essential for protecting equine health, supporting the equine industry, and ensuring the sustainability of biopharmaceutical production derived from horses.

African Horse Sickness

African Horse Sickness (AHS) is a highly lethal viral disease caused by African horse sickness virus (AHSV), a double-stranded RNA virus belonging to the genus *Orbivirus* within the family *Reoviridae*. The disease is transmitted primarily by *Culicoides* biting midges and affects equids, including horses, donkeys, mules, and zebras. Among these, horses are the most susceptible, with mortality rates ranging from 50% to 95% in naïve populations during outbreaks. There are nine different serotypes of AHSV, each requiring specific immunity for protection. Serotypes 1 to 8 are generally restricted within Africa, whereas serotype 9 is more widely distributed. Due to its high mortality, rapid spread, and significant economic impact, AHS is classified as a major transboundary animal disease (Carpenter *et al.*, 2017; World Organisation for Animal Health, 2023).

Transmission mechanism: AHSV is transmitted through biological transmission by *Culicoides* midges (Fig. 1A). The virus is consumed during blood feeding on a viremic host and then replicates inside the vector, especially in the salivary glands and midgut. The virus spreads to a new host through saliva during successive blood meals after an extrinsic incubation time. This replication within the vector enhances transmission efficiency and contributes to rapid outbreak dynamics, especially under favorable environmental conditions (Dennis *et al.*, 2019; Hebo *et al.*, 2022; Emiyu, 2023).

Geographic distribution: AHS is endemic in sub-Saharan Africa, where outbreaks usually take place during warm and wet seasons that promote vector reproduction. The geographic distribution of the disease is closely linked to the ecology of *Culicoides* midges, which are highly sensitive to environmental conditions such as temperature, humidity, and rainfall. Increased precipitation creates suitable breeding habitats, particularly through the formation of temporary water bodies and the saturation of organic-rich soil, providing ideal substrates for larval development. Higher temperatures (typically above 25°C) not only accelerate vector development but also enhance viral replication within the midge, significantly shortening the extrinsic incubation period (EIP). This rapid viral turnover, combined with high humidity that increases adult midge survival, facilitates explosive outbreak dynamics. Traditionally limited to tropical and subtropical regions of Africa, AHS has spread into North Africa, the Middle East, and Southern Europe. Recent outbreaks have been reported in parts of Asia, including India, Pakistan,

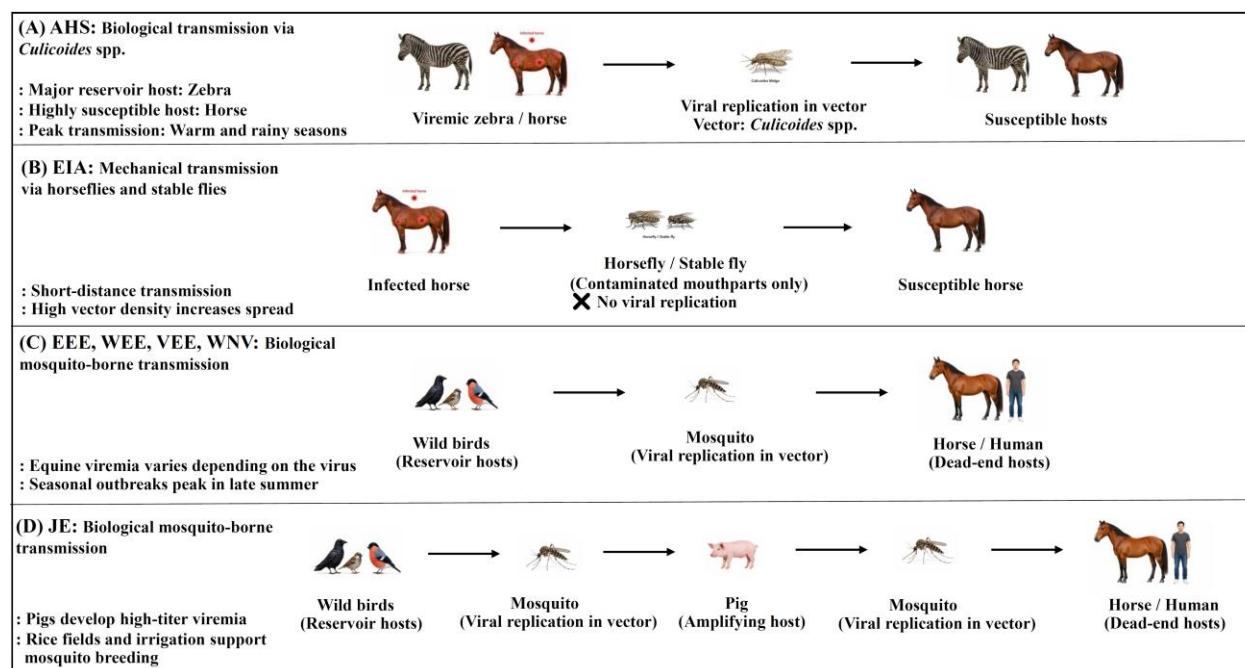


Figure 1 Overview of transmission cycles of major equine vector-borne viral diseases, including biological transmission of African Horse Sickness (A), EEE/WEE/VEE/WNV (C), JE (D), and mechanical transmission of EIAV (B).

Thailand, and Malaysia, indicating a continuing eastward spread. Climate change, increased animal movement, and vector adaptation are key drivers of this expansion, elevating the global risk of AHS transmission (Emiyu, 2023; World Organisation for Animal Health, 2023).

Clinical manifestations: Zebras are relatively resistant to AHS and serve as asymptomatic reservoir hosts, whereas donkeys and mules have reduced susceptibility compared to horses. In horses, AHS presents with a wide range of clinical manifestations, ranging from severe to mild or even subclinical cases, depending on the viral serotype, host immunity, and exposure. The pulmonary form is the most severe, characterized by high fever, respiratory distress, and frothy nasal discharge, often leading to death within 24–48 hours. The cardiac form causes fever, swelling of the head and neck, and mucosal congestion, with death occurring within 4–8 days. The mixed form combines features of both and is commonly observed in partially immune animals. Mortality in susceptible horses ranges from 50% to 95%, making AHS one of the deadliest equine viral diseases (Fig. 2A). Post-mortem findings typically include pulmonary edema, fluid in the chest cavity, and hemorrhages in the heart and lungs (Dennis *et al.*, 2019; Hebo *et al.*, 2022; Rais, 2022).

Laboratory diagnosis: Accurate laboratory diagnosis is essential for confirming AHS, distinguishing it from other equine diseases, and supporting outbreak control. Diagnostic approaches include virological, serological, and molecular techniques (Fig. 3) (Hebo *et al.*, 2022). Virus isolation can be achieved using cell cultures or embryonated eggs, followed by identification through immunofluorescence or antigen detection assays. Molecular techniques such as RT-PCR and real-time RT-PCR are the most sensitive and specific methods for detecting viral RNA and

determining AHSV serotypes. Serological assays, including ELISA, are widely used for detecting antibodies or antigens in serum samples, supporting both diagnosis and surveillance. Virus neutralization tests (VNTs) remain the gold standard for serotype confirmation. In endemic areas, monitoring equine populations as indicators through serological tests enables the early detection of viral circulation. Rapid and accurate diagnosis is critical for effective control, including movement restrictions and outbreak suppression (Emiyu, 2023; World Organisation for Animal Health, 2023).

Prevention and control: Effective prevention and control of AHS rely on coordinated vaccination, vector management, movement regulation, and surveillance. Vaccination remains the primary preventive strategy in endemic regions, with live attenuated polyvalent vaccines widely used, while safer inactivated and recombinant vaccines are under development. Vector control aims to reduce exposure to *Culicoides* midges through insect-proof stabling with fine mesh screens (≤ 0.6 mm), housing horses during peak vector activity at dusk and dawn, and the use of high-velocity fans, which can reduce midge activity due to their weak flight ability. Application of synthetic pyrethroid repellents and environmental management, including elimination of standing water and organic-rich breeding sites, further helps reduce vector populations (Hebo *et al.*, 2022; World Organisation for Animal Health, 2023).

During outbreaks, strict movement restrictions, zoning, and quarantine measures are essential to limit disease spread. A notable example of practical implementation was observed during the 2020 AHS outbreak in Thailand, where the government enforced an immediate nationwide movement standstill and established strict protection zones. This was coupled with a mandatory mass vaccination campaign and the

requirement for insect-proof stabling with fine mesh, which were pivotal in controlling the spread and eventually regaining AHS-free status. Continuous serological surveillance and vector monitoring support early detection and rapid response. Long-term control strategies should incorporate vaccination programs, vector ecology studies, and climate-based risk assessments to reduce recurrence and support global disease management (Dennis *et al.*, 2013; Castillo-Olivares, 2021; Rais, 2022).

Equine Infectious Anemia

Equine infectious anemia (EIA) is a persistent viral disease caused by equine infectious anemia virus (EIAV), a lentivirus belonging to the family Retroviridae. Similar to human immunodeficiency virus (HIV), EIAV is a retrovirus that can cause a persistent, lifelong infection in equids. It is an enveloped RNA virus characterized by its ability to integrate its genetic material into the host's genome, leading to lifelong infection. EIAV mainly affects macrophages and monocytes, causing persistent viremia and immune-mediated damage. The virus shows high genetic variability due to frequent mutations, allowing it to evade the host's immune response (Suntrarachun *et al.*, 2010; World Organisation for Animal Health, 2022).

Transmission mechanism: EIAV is transmitted mechanically by blood-feeding insects, specifically horseflies (*Tabanus* spp.) and stable flies (*Stomoxys calcitrans*). When feeding is interrupted, contaminated blood adhering to the insect's mouthparts can be transferred from an infected horse to a susceptible host (Fig. 1B). Unlike biologically transmitted arboviruses, EIAV does not replicate within the vector, and transmission efficiency depends on factors such as the volume of residual blood, the interval between feedings, and vector density. This type of mechanical transmission results in sporadic yet significant spread, especially in environments with high biting-fly activity (Akesowan and Suntrarachun, 2007; World Organisation for Animal Health, 2022).

Geographic distribution: EIA occurs worldwide in horses, mules, and donkeys, except in regions with strict eradication programs. The disease remains endemic in parts of the Americas, Africa, Asia, and some areas of Europe, with outbreaks commonly associated with animal movement and inadequate vector control. In Europe, intensive surveillance and testing programs have greatly reduced disease prevalence, although sporadic outbreaks still occur. In contrast, continued viral circulation persists in many Asian and African countries due to limited surveillance and high vector density. Climate change also influences the geographic expansion of EIA by affecting vector ecology. Increased temperature, rainfall, and humidity enhance the survival, reproduction, and breeding habitats of horseflies and stable flies, resulting in greater vector density and prolonged transmission seasons. These environmental changes may facilitate the spread of EIAV into previously non-endemic regions, highlighting the

importance of continuous surveillance and vector management. In Thailand, EIA is classified as a controlled disease under the Department of Livestock Development (DLD), with a reported seroprevalence of approximately 0.5–1% in monitored populations. Mandatory serological testing and certification prior to transport, trade, or equestrian events are required to prevent disease spread and identify inapparent carriers (Suntrarachun *et al.*, 2010; Suntrarachun *et al.*, 2013; Carvelli *et al.*, 2024).

Clinical manifestations: EIA presents with a wide range of clinical outcomes, ranging from acute and chronic disease to inapparent infection. Acute infection is characterized by high fever, depression, anorexia, anemia, thrombocytopenia, and edema, particularly in the limbs and ventral abdomen. Chronic cases show recurrent fever, progressive weight loss, weakness, and intermittent edema with periods of temporary recovery. Many infected horses become inapparent carriers, remaining clinically normal while serving as lifelong reservoirs of infection (Fig. 2B). The prevalence of subclinical or inapparent infections varies significantly; for instance, studies in endemic regions of Southeast Asia and South America have reported seroprevalence rates ranging from 2% to over 15% in asymptomatic populations, depending on management practices and vector density. In Thailand, historical records indicate a low but persistent seroprevalence of approximately 0.5–1% in monitored populations, though this can spike in areas with high horsefly concentrations (Suntrarachun *et al.*, 2013; Carvelli *et al.*, 2024). Clinical signs may resemble other infectious or parasitic diseases, making laboratory confirmation essential. Persistent viremia and cyclic clinical episodes reflect the virus's ability to evade host immune responses (Cook *et al.*, 2013; Onmaz *et al.*, 2013).

Laboratory diagnosis: Accurate diagnosis is critical for managing the spread of EIAV. The Agar Gel Immunodiffusion (AGID) test, known as the Coggins test, remains the gold standard for antibody detection due to its high specificity. In this assay, EIAV antigen and test serum diffuse through a specialized agar plate; a positive result is identified by a visible precipitation line formed where antibodies and antigens meet. Conversely, the absence of this line indicates a negative status (Fig. 3). For early detection or in seronegative carriers, molecular techniques such as PCR are used to detect viral nucleic acids. In endemic regions, regular serological testing is mandatory for identifying inapparent carriers and certifying disease-free status prior to movement, trade, or events. A comprehensive strategy combining serological screening with molecular diagnostics ensures maximum accuracy and prevents iatrogenic transmission through blood products (World Organisation for Animal Health, 2022).

Prevention and control: In the absence of an effective vaccine or antiviral treatment, EIA control relies on early detection, strict management of infected horses, and prevention of mechanical transmission. Routine serological screening, particularly using the Coggins

test, is essential for identifying infected animals before movement or introduction into disease-free populations. Seropositive horses are typically isolated or euthanized according to national regulations, and maintaining a separation distance of at least 200 meters can help reduce mechanical transmission by biting flies. Strict hygiene practices, including the use of individual needles and surgical instruments, are necessary to prevent iatrogenic spread. Vector control methods such as insect repellents, fly traps, insect-proof stabling, and environmental sanitation help reduce exposure to horseflies and stable flies. Movement restrictions, quarantine protocols, and surveillance programs are particularly important in endemic regions, including Thailand. Combined strategies that incorporate diagnostics, vector control, biosecurity, and regulatory enforcement are crucial to reduce the effects of EIA on equine health and the equine industry (Kumar *et al.*, 2018; World Organisation for Animal Health, 2022; Carvelli *et al.*, 2024).

Eastern, Western, and Venezuelan Equine Encephalitis

Eastern, Western, and Venezuelan equine encephalomyelitis (EEE, WEE, and VEE) are mosquito-borne viral diseases caused by alphaviruses within the family *Togaviridae*. These enveloped, single-stranded RNA viruses are capable of rapid replication and are responsible for severe neurological disease in both horses and humans. They are maintained in enzootic transmission cycles involving birds (EEE and WEE) or small mammals (VEE), with spillover transmission to equines and humans occurring via mosquito vectors (Azar *et al.*, 2020). Among these viruses, EEE is the most virulent, with case fatality rates of up to 90%, followed by VEE (40–80%), while WEE generally causes milder disease. Viral replication in the central nervous system leads to inflammation, neuronal damage, and severe neurological dysfunction. Due to their zoonotic potential, high virulence, and outbreak capability, these viruses represent significant threats to both veterinary and public health (Arechiga-Ceballos and Setien, 2015; Knox and Beddoe, 2021).

Transmission mechanism: EEE, WEE, and VEE are transmitted via biological transmission through mosquitoes. The virus is acquired during blood feeding on viremic reservoir hosts, such as birds (EEE and WEE) or rodents (VEE), and then replicates within the mosquito vector. After an extrinsic incubation period, the virus spreads to the salivary glands and is transmitted to a new host during later blood meals. Horses and humans are generally considered dead-end hosts for EEE and WEE because they do not develop sufficient viremia to sustain transmission. In contrast, horses infected with certain VEE strains may develop high-titer viremia and can serve as amplifying hosts during outbreaks (Fig. 1C). This distinction has important implications for outbreak dynamics and control strategies (Arechiga-Ceballos and Setien, 2015; Toribio, 2022).

Geographic distribution: EEE, WEE, and VEE are found throughout different regions of the Americas. EEE is mainly distributed in eastern North America, including the United States and Canada, with occasional outbreaks in the Caribbean and South America. WEE is found primarily in western North America and parts of South America, although its incidence has declined in recent decades. VEE remains endemic in Central and South America and the Caribbean, with periodic outbreaks extending into northern Mexico and the southern United States. Transmission patterns are strongly influenced by environmental and ecological factors. Warm temperatures accelerate mosquito development and viral replication, while rainfall and flooding increase mosquito breeding habitats and vector density. High humidity enhances mosquito survival and prolongs transmission periods, particularly during late summer and early autumn when outbreaks commonly peak. The abundance of reservoirs and amplifying hosts also contributes to regional outbreak dynamics. Continuous surveillance of both equine populations and mosquito vectors is essential for early detection and prevention of regional spread (Mackay and Tannerre, 2022; Toribio, 2022).

Clinical manifestations: The incubation period for EEE, WEE, and VEE in horses is typically 5–14 days following exposure to mosquitoes. Early clinical signs include fever, anorexia, lethargy, and stiffness, followed by neurological manifestations such as ataxia, head pressing, circling, muscle tremors, and visual impairment. Severe cases may progress to paralysis, seizures, coma, and death (Fig. 2C) (Azar *et al.*, 2020). Mortality rates are highest in EEE (75–90%), followed by VEE (40–80%), while WEE generally causes milder disease with lower mortality (20–50%). Subclinical or mild infections are relatively common, particularly in endemic regions. Serological studies have shown that approximately 20–40% of horses in endemic areas may possess antibodies without a history of clinical disease, indicating silent viral circulation. Surviving horses may develop long-term neurological effects, including behavioral abnormalities, weakness, and impaired coordination. Due to the overlap of clinical signs with other equine neurological diseases, especially West Nile virus infection, laboratory confirmation is essential for accurate diagnosis (Go *et al.*, 2014; Arechiga-Ceballos and Setien, 2015).

Laboratory diagnosis: Diagnosis of EEE, WEE, and VEE relies on a combination of serological, molecular, and virological techniques (Fig. 3). Virus isolation can be performed in cell cultures or laboratory animals but requires high-containment facilities due to biosafety concerns. Serological assays, such as ELISA, hemagglutination inhibition (HI), and plaque reduction neutralization tests (PRNT), are used to detect virus-specific antibodies, with IgM-capture ELISA indicating recent infection. Molecular techniques such as RT-PCR provide rapid and sensitive detection of viral RNA in blood, tissues, or cerebrospinal fluid and allow differentiation among EEE, WEE, and VEE. Due to potential cross-reactivity in serological assays, particularly in endemic regions,

molecular confirmation is often required. Routine laboratory surveillance, combined with clinical observation, supports early outbreak detection and informs timely intervention strategies (Knox and Beddoe, 2021; World Organisation for Animal Health, 2021).

Prevention and control: Vaccination, mosquito control, and environmental oversight are essential for preventing and controlling EEE, WEE, and VEE. Inactivated and live-attenuated vaccines are effective and commonly used in horses. A two-dose primary vaccination series administered 4–6 weeks apart is recommended, followed by annual boosters; however, biannual vaccination may be necessary in endemic or high-risk regions to maintain protective immunity during peak mosquito seasons. Booster vaccination of pregnant mares 4–6 weeks before foaling is recommended to enhance maternal antibody transfer to foals. Mosquito control includes stabling horses in screened barns during peak mosquito activity at dusk and dawn, using fans, repellents, and permethrin-treated protective equipment. Environmental management, such as eliminating standing water, cleaning water troughs, and applying larvicides, is essential for reducing mosquito breeding sites. Integrated strategies combining vaccination, vector surveillance, environmental control, and rapid reporting of neurological cases are critical for minimizing outbreak risk and supporting a One Health approach to disease prevention (World Organisation for Animal Health, 2021; Mackay and Tannerre, 2022).

West Nile Virus

West Nile virus (WNV) is a flavivirus transmitted by mosquitoes that belongs to the Flaviviridae family. It is a single-stranded RNA virus with an envelope and various genetic lineages that demonstrate differing degrees of virulence (Hale, 2015). Transmission occurs primarily through *Culex* mosquitoes, especially *C. tarsalis*, *C. pipiens*, *C. restuans*, and *C. quinquefasciatus*. WNV is maintained in a bird-mosquito transmission cycle, where wild birds serve as reservoir hosts and develop high-titer viremia. Horses and humans are incidental dead-end hosts because they generally do not develop sufficient viremia to transmit the virus back to mosquitoes (Suntrarachun *et al.*, 2013; Boukraa *et al.*, 2016).

Transmission mechanism: WNV is transmitted through biological transmission by mosquitoes. Following ingestion of viremic blood from infected birds, the virus replicates within the mosquito midgut and disseminates to the salivary glands during an extrinsic incubation period. Transmission to a new host occurs via saliva during subsequent blood feeding. The efficiency of transmission is influenced by vector competence, environmental temperature, and the duration of the extrinsic incubation period. Horses and humans are considered dead-end hosts, as their level of viremia is insufficient to infect feeding mosquitoes, thereby limiting their role in maintaining the transmission cycle (Fig. 1C) (Ostlund *et al.*, 2000; Suntrarachun *et al.*, 2013).

Geographic distribution: WNV was first discovered in Uganda in 1937 and later emerged in North America in 1999, rapidly spreading throughout the United States and Canada. A major equine outbreak in 2002 caused more than 14,000 reported equine cases in the United States, highlighting the virus's potential for rapid geographic expansion. WNV is currently distributed across Africa, the Middle East, Europe, North America, and parts of Oceania, with transmission closely linked to migratory bird flyways. Environmental and climatic factors strongly influence WNV transmission dynamics. Elevated temperatures accelerate mosquito development and viral replication, while rainfall, irrigation, and standing water increase mosquito breeding habitats. Drought conditions may also enhance transmission by concentrating birds and mosquitoes around limited water sources. In temperate regions, transmission peaks during late summer and early autumn, whereas tropical regions may experience year-round circulation with seasonal fluctuations. Despite its wide distribution, WNV has not been reported in the United Kingdom (Suntrarachun *et al.*, 2013; Mackay and Tannerre, 2022).

Clinical manifestations: The incubation period for WNV in horses ranges from 3 to 15 days. It is estimated that less than 10% of infected, non-vaccinated horses develop clinical neurological disease, while the vast majority, approximately 90%, undergo subclinical seroconversion (Hale, 2015). Clinical signs consist of weakness, ataxia, stumbling, lethargy, anorexia, dysphagia, circling behavior, and hyperexcitability. In severe cases, neurological involvement may progress to facial paralysis, hindlimb weakness, recumbency, seizures, blindness, and coma (Fig. 2C). The case fatality rate in horses exhibiting neurological signs is approximately 33–40%, which is lower than EEE but remains a significant veterinary concern (Ostlund *et al.*, 2000). Surviving horses may show residual neurological deficits. Due to overlapping clinical signs with other equine neurological diseases, including rabies, botulism, Japanese encephalitis, equine protozoal myeloencephalitis, and other arboviral encephalitides, laboratory confirmation is essential for a conclusive diagnosis (Hale, 2015; Ostlund *et al.*, 2000).

Laboratory diagnosis: The diagnosis of WNV infection relies on both serological and molecular techniques. Serological assays, including IgM-capture ELISA and plaque reduction neutralization tests (PRNT), are commonly used to detect virus-specific antibodies, with PRNT providing higher specificity. Molecular techniques such as RT-PCR, including nested and multiplex formats, enable rapid and sensitive detection of viral RNA in blood, tissues, or cerebrospinal fluid (Fig. 3). Distinguishing it from other flaviviruses, especially Japanese encephalitis virus, is essential in endemic regions due to potential serological cross-reactivity. WNV-specific antibodies may persist for several months; therefore, molecular detection is particularly valuable during the early viremic phase prior to seroconversion. Combining serological and molecular approaches improves diagnostic accuracy and supports effective surveillance and outbreak

response (Cavalleri *et al.*, 2022; Suntrarachun *et al.*, 2013).

Prevention and control: Vaccination is the primary strategy for preventing WNV infection in horses. Available vaccines include both inactivated and recombinant canarypox-vectored types, and the American Association of Equine Practitioners (AAEP) advises WNV vaccination as a fundamental immunization. A primary two-dose series administered 3–6 weeks apart is followed by annual boosters, ideally given before peak mosquito season, in high-risk areas, booster vaccination every 6 months may be recommended. Vaccination significantly reduces the risk of neurological disease. Mosquito control is also essential and includes stabling horses during peak mosquito activity at dusk and dawn, using fans, and applying repellents or permethrin-treated protective equipment. Environmental management, such as eliminating standing water, cleaning water containers, and applying larvicides, helps reduce mosquito breeding sites. Surveillance of mosquito vectors, bird reservoirs, and equine neurological cases supports early detection and outbreak prevention. Integrated strategies combining vaccination, vector control, and surveillance are critical for minimizing the impact of WNV and reducing zoonotic risk (Suntrarachun *et al.*, 2013; Cavalleri *et al.*, 2022).

Japanese Encephalitis

Japanese encephalitis (JE) is caused by the Japanese encephalitis virus (JEV), a mosquito-borne flavivirus belonging to the family Flaviviridae. JE is a single-stranded, enveloped, positive-sense RNA virus encoding structural (C, prM/M, E) and nonstructural (NS1–NS5) proteins essential for viral replication and immune evasion. The virus is primarily transmitted by *Culex* mosquitoes, particularly *C. tritaeniorhynchus*, which breed in irrigated rice fields and flooded environments and exhibit peak activity during dusk and dawn. Additional vector species include *C. vishnui*, *C. gelidus*, and *C. pipiens*, with occasional involvement of *Aedes* species (Igarashi, 1992; Ellis *et al.*, 2000; World Health Organization, 2022).

Transmission mechanism: JEV is maintained in a zoonotic transmission cycle involving mosquitoes, reservoir hosts, and amplifying hosts. Wild birds serve as natural reservoir hosts that maintain viral circulation in the environment. Domestic pigs are the primary amplifying hosts because they develop high-titer viremia sufficient to infect feeding mosquitoes and sustain transmission cycles. In contrast, horses and humans are considered dead-end hosts, as they typically develop viremia levels too low to infect mosquitoes. Following ingestion of viremic blood, JEV replicates within the mosquito midgut, disseminates to the salivary glands during the extrinsic incubation period, and is subsequently transmitted to a new host during blood feeding (Fig. 1D). This mosquito–host transmission cycle underlies both endemic circulation and outbreak dynamics (Harshani and Abeynayake, 2025).

Geographic distribution: JEV is endemic in Southeast Asia, South Asia, and parts of the Western Pacific, with geographic expansion reported into regions such as northern Australia. In temperate climates, transmission is seasonal, typically occurring from late spring to early autumn (May to October), with peak incidence in late summer. In tropical regions, transmission may occur year-round but intensifies during rainy seasons due to increased mosquito breeding. Environmental factors strongly influence transmission dynamics: rainfall and irrigation create larval habitats, warm temperatures accelerate mosquito development and viral replication, and agricultural practices such as rice cultivation enhance vector–host contact. Expansion of irrigated agriculture, climate variability, and land-use changes have contributed to the spread of JEV into new geographic areas (Harshani and Abeynayake, 2025).

Clinical manifestations: The incubation period in horses ranges from approximately 4 to 14 days. Most equine infections are subclinical, with estimates indicating that less than 1% (around 0.1–1%) of infected horses develop overt neurological disease, although the apparent morbidity rate may be higher in immunologically naïve populations during outbreaks (Mackay and Tannerre, 2022). Mild cases may present with transient fever, anorexia, lethargy, and mucous membrane congestion, typically resolving within a few days. Neurological disease, when it occurs, is characterized by ataxia, dysphagia, neck rigidity, visual impairment, paresis, and paralysis (Fig. 2C). Severe cases may involve high fever, disorientation, behavioral abnormalities, and rapid progression to recumbency and death within 24 to 48 hours. Although clinical disease is relatively uncommon, its potential severity and zoonotic implications warrant continued surveillance and preventive measures (Igarashi, 1992; Mackay and Tannerre, 2022).

Laboratory diagnosis: Diagnosis of JE relies on serological and molecular techniques. Serological assays include hemagglutination inhibition, IgM-capture ELISA, virus neutralization tests, and immunofluorescence; however, cross-reactivity with other flaviviruses, particularly West Nile virus, may complicate interpretation. Molecular techniques, including RT-PCR and real-time RT-PCR, provide high sensitivity and specificity for detecting viral RNA in blood, tissues, or cerebrospinal fluid and are preferred for early diagnosis (Fig. 3). Multiplex real-time RT-PCR platforms have enhanced surveillance capabilities by enabling simultaneous detection of multiple flaviviruses in both vertebrate hosts and mosquito vectors (Suntrarachun *et al.*, 2013; Cavalleri *et al.*, 2022).

Prevention and control: Prevention of JEV in horses relies on vaccination, mosquito control, and management of amplifying hosts. Both live-attenuated and inactivated vaccines are used in endemic regions to reduce the risk of clinical disease, while vaccination of pigs helps decrease viral amplification. Vector control measures include stabling horses in screened enclosures during peak mosquito activity, applying repellents, and reducing mosquito breeding sites

through proper environmental management. To minimize exposure, equine facilities should ideally be located away from intensive pig farming operations, as pigs serve as major amplifying hosts. Where separation is not feasible, enhanced vector control measures, including physical barriers and targeted mosquito management, should be implemented to reduce

transmission risk. Surveillance of mosquito populations, pig herds, and equine neurological cases remains essential for early detection and outbreak prevention under a One Health approach (World Health Organization, 2022; Harshani and Abeynayake, 2025).

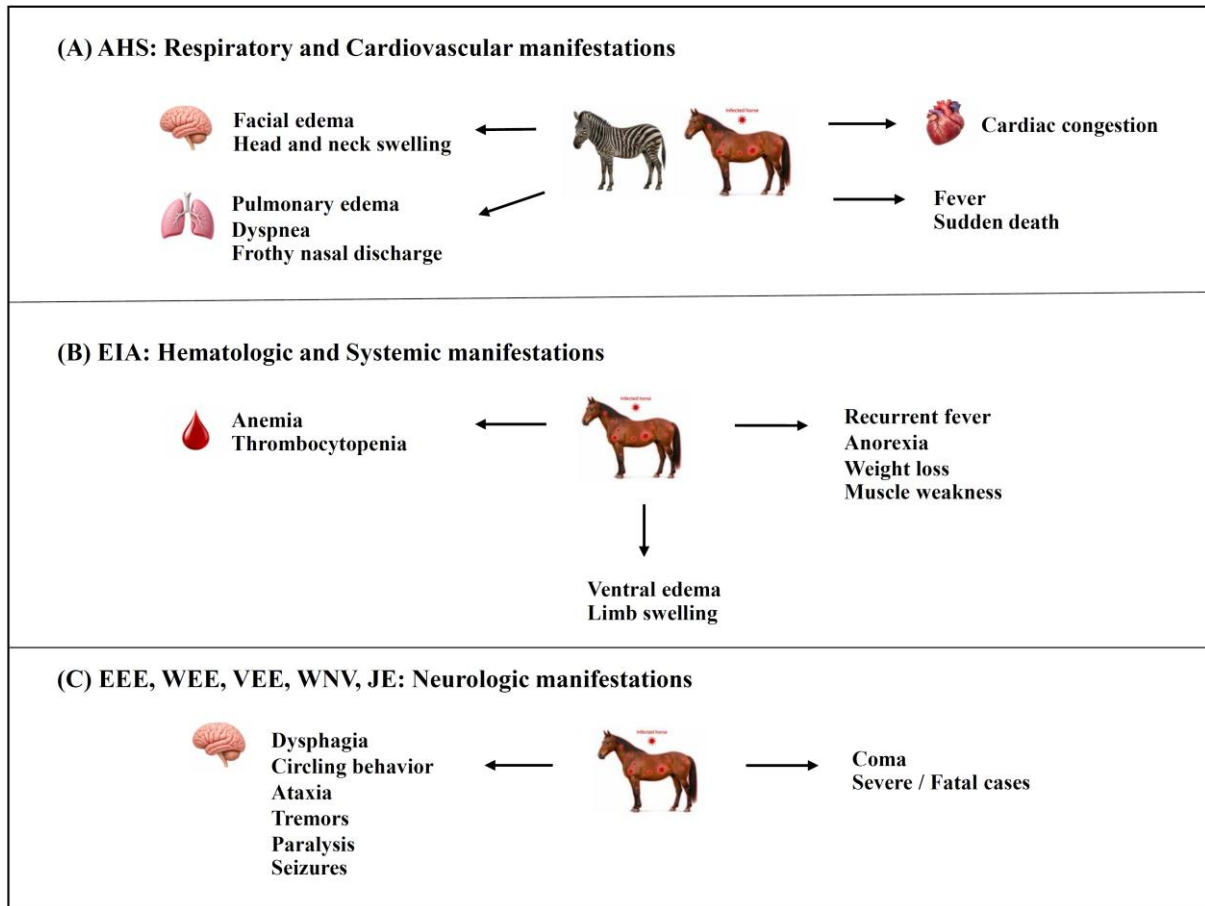


Figure 2 Clinical manifestations of major equine vector-borne viral diseases, including respiratory/cardiovascular, hematologic, and neurologic manifestations.

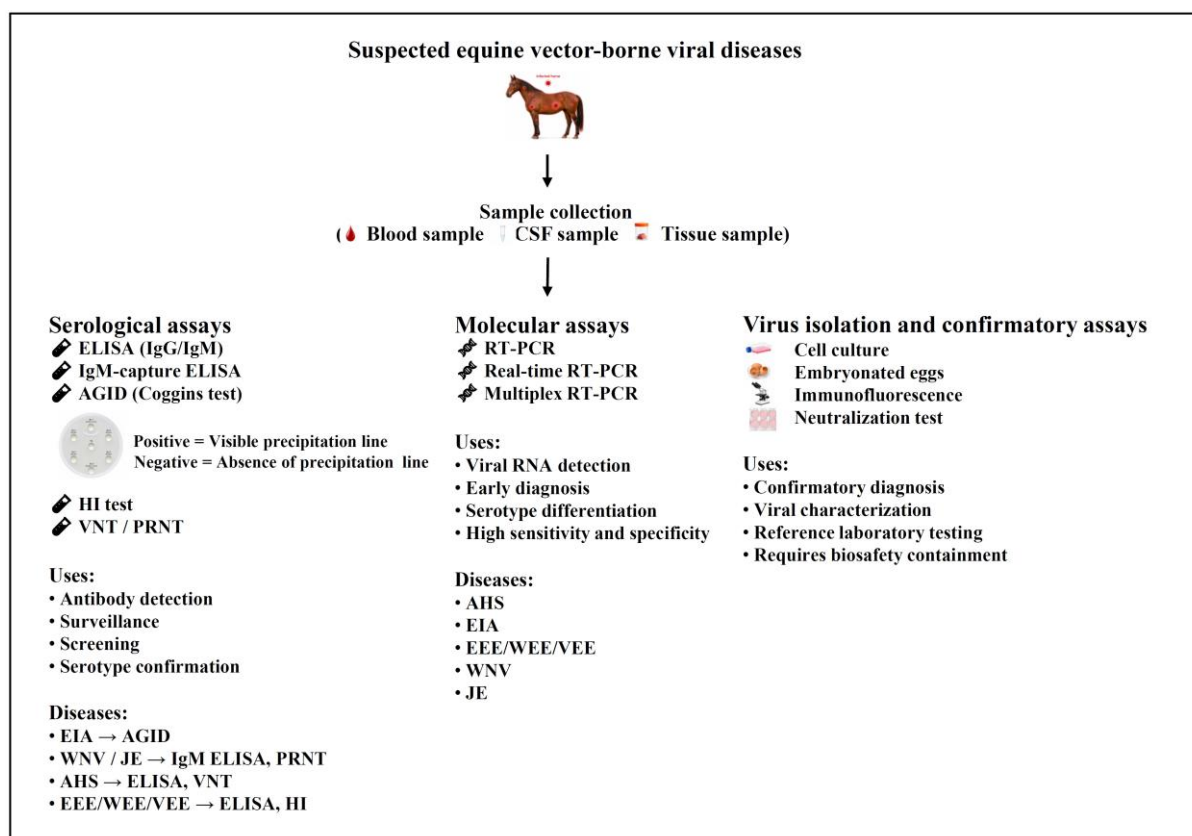


Figure 3 Overview of laboratory diagnostic approaches for major equine vector-borne viral diseases, including serological assays, molecular detection assays, and virus isolation/confirmatory techniques. AGID (Coggins test) schematic demonstrates positive and negative precipitation line patterns for equine infectious anemia diagnosis.

In conclusion, vector-borne viral diseases remain major threats to equine health worldwide, causing substantial mortality, economic losses, and impacts on industries such as sport, therapy, and biopharmaceutical production. Diseases including African horse sickness, West Nile virus infection, and equine infectious anemia illustrate the complexity of vector transmission, environmental influences, and regional epidemiology. Climate change, globalization, and land-use modification have expanded vector distributions, increasing the risk of disease emergence and transboundary spread. Environmental factors such as temperature, rainfall, and agricultural practices play key roles in shaping vector populations and transmission intensity, emphasizing the requirement for region-specific risk assessments. Effective prevention and control require integrated strategies combining vaccination, vector management, surveillance, movement control, and early laboratory diagnosis. Practical measures such as vector-proof housing, targeted vaccination programs, and routine screening are essential for reducing transmission. Future efforts should focus on the development of improved vaccines, rapid diagnostic tools, predictive outbreak models, and strengthened One Health collaboration to better protect equine and public health worldwide.

References

- Akesowan S and Suntrarachun S 2007. Equine infectious anemia (EIA). *J Thai Vet Pract.* 19: 25-38.
- Arechiga-Ceballos N and Setien AA 2015. Alphaviral equine encephalomyelitis (Eastern, Western and Venezuelan). *Rev Sci Tech Off Int Epiz.* 34(2): 491-501.
- Azar SR, Campos RK, Bergren NA, Camargos VN and Rossi SL 2020. Epidemic alphaviruses: Ecology, emergence and outbreaks. *Microorganisms* 8(8): 1167.
- Boukraa S, Grandiere MAL, Bawin T, Raharimalala FN, Zimmer JY, Haubruge E, Thiry E and Francis F 2016. Diversity and ecology survey of mosquito potential vectors in Belgian equestrian farms: A threat prevention of mosquito-borne equine arboviruses. *Prev Vet Med.* 124: 58-68.
- Carpenter S, Mellor PS, Fall AG, Garros C and Venter GJ 2017. African horse sickness virus: History, transmission, and current status. *Annu Rev Entomol.* 62: 343-358.
- Carvelli A, Nardini R, Carnio A, Ricci I, Rosone F, Sala M, Simeoni S, Maccarone D and Scicluna MT 2024. Equine infectious anemia: The active surveillance of an entire equid population reduces the occurrence of the infection. *Transbound Emerg Dis.* 2024: 3439871.
- Castillo-Olivares J 2021. African horse sickness in Thailand: challenges of controlling an outbreak by vaccination. *Equine Vet J.* 53(1): 9-14.
- Cavalleri JMV, Korbacska-Kutasi O, Leblond A, Paillot R, Pusterla N, Steinmann E and Tomlinson J 2022. European college of equine internal medicine consensus statement: Flavivirus infections in European equids. *J Vet Intern Med.* 36(6): 1858-1871.

- Cook RF, Cook SJ and Issel CJ 2013. Equine infectious anemia virus: Epidemiology and control. *J Equine Vet Sci.* 33(9): 545-560.
- Dennis SJ, Meyers AE, Hitzeroth II and Rybicki EP 2019. African horse sickness: A review of current understanding and vaccine development. *Viruses* 11(9): 844.
- Ellis PM, Daniels PW and Banks DJ 2000. Japanese encephalitis. *Vet Clin North Am Equine Pract.* 16(3): 565-578.
- Emiyu K 2023. Review on African horse sickness. *Austin J Vet Sci and Anim Husb.* 10(3): 1123.
- Go YY, Balasuriya UBR and Lee CK 2014. Zoonotic encephalitides caused by arboviruses: transmission and epidemiology of alphaviruses and flaviviruses. *Clin Exp Vaccine Res.* 3(1): 58-77.
- Hale D 2015. West Nile virus: An overview. *Home Healthc Now.* 33(5): 260-264.
- Harshani HBC and Abeynayake JI 2025. Japanese encephalitis in Asia: A comprehensive review. *J Vector Borne Dis.* 10.4103.
- Hebo G, Afera B and Singh K 2022. Review on African horse sickness. *Acad J Anim Dis.* 11(3): 70-79.
- Igarashi A 1992. Epidemiology and control of Japanese encephalitis. *Health Stat Q.* 45: 299-305.
- Knox A and Beddoe T 2021. Isothermal nucleic acid amplification technologies for the detection of equine viral pathogens. *Animals* 11: 2150.
- Kumar B, Manuja A, Virmani N and Tripathi BN 2018. Zoonotic viral diseases of equines and their impact on human and animal health. *Open Virol J.* 12: 80-98.
- Mackay RJ and Tannerre D 2022. "Equine arboviral encephalomyelitis." [Online]. Available: <https://www.msdsvetmanual.com/nervous-system/equine-arboviral-encephalomyelitis/equine-arboviral-encephalomyelitis>. Accessed October 16, 2025.
- Onmaz AC, Beutel RG, Schneeberg K, Pavaloju AN, Komarek A and van den Hoven R 2013. Vectors and vector-borne diseases of horses. *Vet Res Commun.* 37(1): 65-81.
- Ostlund EN, Andresen JE and Andreson M 2000. West Nile encephalitis. *Vet Clin North Am Equine Pract.* 16(3): 427-441.
- Rais G 2022. Clinical signs, treatment and prevention of African horse sickness. *J Vet Sci.* eISSN: 2581-3897.
- Suntrarachun S, Akesowan S and Khwanpech P 2013. Flavivirus encephalitis in equines with an emphasis on the Japanese encephalitis and west Nile viruses. *Asian J Biol Life Sci.* 2: 92-95.
- Suntrarachun S, Akesowan S, Tirawatnapong T and Khaw O 2010. Detection of equine infectious anemia viral nucleic acid in asymptomatic carrier horses in Thailand by nested PCR. *Asian Biomed.* 4: 1-5.
- Toribio RE 2022. Arboviral equine encephalitides. *Vet Clin North Am Equine Pract.* 38(2): 299-321.
- World Health Organization (WHO) 2022. Japanese encephalitis vaccines: WHO position paper. *Wkly Epidemiol Rec.* 97(1): 1-24.
- World Organisation for Animal Health (WOAH) 2023. African horse sickness (infection with African horse sickness virus). In: *Manual of Diagnostic Tests and*
- Suntrarachun S. / Thai J Vet Med.* 2026. 56(2): 20.
- Vaccines for Terrestrial Animals. Paris: WOA. 1-8.
- World Organisation for Animal Health (WOAH) 2021. Equine encephalomyelitis (Eastern, Western and Venezuelan). In: *Manual of Diagnostic tests and Vaccines for Terrestrial Animals.* Paris: WOA. 1-16.
- World Organisation for Animal Health (WOAH) 2022. Equine infectious anemia (infection with equine infectious anemia virus). In: *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.* Paris: WOA. 1-7.