

**VENEZUELAN EQUINE ENCEPHALOMYELITIS
STANDARD OPERATING PROCEDURES:
1. OVERVIEW OF ETIOLOGY AND ECOLOGY**

FAD PReP

**Foreign Animal Disease
Preparedness & Response Plan**



**United States
Department of
Agriculture**

United States Department of Agriculture • Animal and Plant Health Inspection Service • Veterinary Services

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The Foreign Animal Disease Preparedness and Response Plan (FAD PReP) Standard Operating Procedures (SOPs) provide operational guidance for responding to an animal health emergency in the United States.

These draft SOPs are under ongoing review. This document was last updated in **August 2013**. Please send questions or comments to:

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Venezuelan Equine Encephalomyelitis (VEE)

Etiology Quick Summary

Disease

Venezuelan equine encephalomyelitis.

Mortality & Morbidity

Depending on strain and transmission cycle, VEE can cause very high morbidity in humans and equines. Mortality is 50-70% in horses and less than 1% in humans.

Susceptible Species

All equid species and humans. Domestic rabbits, goats, dogs, and sheep are also potentially at risk.

Zoonotic Potential

High.

Reservoir Hosts

Equids and small mammals, depending on serotype.

Transmission

VEE virus is transmitted by hematophagus insects, primarily mosquitos. Black flies and ticks are also capable of being mechanical vectors.

Persistence in the Environment

VEE virus is vulnerable to heat and drying, but can survive in blood, exudates, and other cool, damp, and dark conditions.

1.1 Introduction

Venezuelan equine encephalomyelitis (VEE) is caused by the VEE virus (VEEV). The vector-borne virus, predominantly transmitted by mosquitoes, primarily affects both domestic and wild equid species and humans; the life cycle of VEEV is complex and affects many different mammalian and mosquito species. The disease is characterized in horses by fever, loss of appetite, and disorders of the central nervous system, such as muscle impairment, blindness, and convulsions.

The spread of VEE varies in speed and intensity according to the viral subtype and densities of mosquito populations, as transmission occurs by the vector biting an infected animal and then feeding on a new host. Epizootic subtypes of VEEV are amplified in equids, may spread rapidly, and can be highly pathogenic in horses, donkeys, mules, and other equids. In humans, the disease results in flu-like symptoms for healthy individuals but can cause severe illness and death in immune compromised, young, or older people.¹

Outbreaks of VEE can be severe, causing high morbidity in humans and equines. VEE has the potential to cause high mortality in equids. There has not been an epizootic outbreak of VEE in the United States since 1971.²

1.1.1 Goals

As a preparedness goal, the Animal and Plant Health Inspection Service (APHIS) will provide etiology and ecology summaries for VEE, and update these summaries at regular intervals.

As a response goal, the Unified Command and stakeholders will have a common set of etiology and ecology definitions and descriptions, to ensure proper understanding of VEE when establishing or revising goals, objectives, strategies, and procedures.

1.1.2 Further Information

This document is intended to be an overview, focusing on the threat of VEE to domestic equine and human populations. Additional resources on VEE, as well as the articles cited in this standard operating procedure (SOP), are listed in [Attachment 1.A](#). Case definitions and laboratory criteria are provided in the VEE Draft Case Definition from the APHIS Centers for Epidemiology and Animal Health, National Surveillance Unit. Case definitions are under ongoing review. This document does not comprehensively discuss vaccination, or its effects on immunity.

Publicly available documents are available here:

http://www.aphis.usda.gov/animal_health/emergency_management/materials_ref.shtml, and they are also on the APHIS Intranet (<http://inside.aphis.usda.gov/vs/em/fadprep.shtml>) for APHIS employees.

¹ Estrada-Franco JG, Navarro-Lopez R, Freier JE, Cordova D, Clements T, Moncayo A, Kang W, Gomez-Hernandez C, Rodriguez-Dominguez G, Ludwig GV, Weaver SC. 2004. "Venezuelan Equine Encephalitis Virus, Southern Mexico." *Emerging Infectious Diseases*. 10(12): 2113-2121.

² USDA APHIS. 2013. Draft Case Definition of Venezuelan Equine Encephalomyelitis.

1.2 Purpose

This document provides responders and stakeholders with a common understanding of the disease agent.

1.3 Etiology

1.3.1 Name

This disease is called Venezuelan equine encephalomyelitis, and it is also often called Venezuelan equine encephalitis. Some of the enzootic viral species within the VEE family are known as Mosso das Pedras virus, Everglades virus, Mucambo virus, Tonate virus, Pixuna virus, Cabassou virus, and Rio Negro virus.³

1.3.2 Virus Characteristics

According to the International Committee on Taxonomy of Viruses, this disease has the following characteristics:

- Family: *Togaviridae*
- Genera: *Alphavirus*
- Baltimore Classification: Group IV (+) ssRNA.⁴

1.3.3 Morphology

The VEEV is a single-stranded ribonucleic acid (RNA) virus, approximately 70 nm in diameter, with icosahedral symmetry. The 5' end of the genome encodes four nonstructural proteins, nsP1, nsP2, nsP3, and nsP4, and the 3' end is responsible for three structural proteins, the capsid and the E1 and E2 envelope proteins. The nonstructural proteins are involved in replicating the viral genome and functions within the host's cytoplasm.⁵

1.3.4 VEEV Serotypes and Strains

There are 6 subtypes (I-IV) within the VEEV complex. The epizootic strains are distributed within subtype I, serotypes I-AB and I-C, and are generally thought to be responsible for major epizootics and epidemics in equid species and humans; serotypes I-AB and I-C are believed to have descended from enzootic strains. Subtypes ID and IE are comprised of enzootic, equine-avirulent strains, but some are known to cause disease and death in humans. Recently, a subtype IE strain in Mexico also caused neurologic signs in equine hosts but did not produce high-titer viremia.⁶

³ Weaver SC, Barrett ADT. 2004. "Transmission cycles, host range, evolution and emergence of arboviral disease." *Nature Reviews Microbiology*. 2: 789-801.

⁴ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

⁵ Weaver SC, Ferro C, Barrera R, Boshell J, Navarro J-C. 2004. "Venezuelan Equine Encephalitis." *Annual Review of Entomology*. 49: 141-174.

⁶ Weaver et al., 2004.

Table 1-1. The VEE Complex of Viruses⁷

Subtype	Species	Serotype	Transmission Pattern
I	VEE virus	AB	Epizootic
	VEE virus	C	
	VEE virus	D	
	VEE virus	E	
	Mosso das Pedras virus	F	
II	Everglades virus		Enzootic
III	Mucambo virus	A	
	Tonate virus*	B	
	Mucambo virus	C	
	Mucambo virus	D	
IV	Pixuna		
V	Cabassou virus		
VI	Rio Negro virus		

Source: Weaver et al., 2004

*Bijou bridge virus is a well-known strain of Tonate virus that has been isolated in Colorado swallow bugs.

Serotype I-D viruses can alter via mutations in the E2 envelope glycoprotein to serotype I-AB or I-C viruses that are able to amplify in equid hosts and cause epizootic outbreaks.⁵ For example, the enzootic I-D strains differs from the epizootic I-C strain by only a few nucleotides, thus epizootic, equine virulent disease can arise where enzootic disease is endemic.⁸ Changes in the E2 protein are responsible for the higher equine virulence and host specificity that enable epizootic outbreaks of VEE.⁹

⁷ Weaver et al., 2004.

⁸ Rico-Hesse R, Weaver SC, de Siger J, Medina G, Salas RA. 1995. "Emergence of a new epidemic/epizootic Venezuelan equine encephalitis virus in South America." *PNAS*. 92: 5278-5281.

⁹ Weaver et al, 2004.

Figure 1-1. Countries with Confirmed VEE from 2007-2012



Source: World Animal Health Information Database (WAHID) 2012: Disease Timelines

1.3.5 Differential Diagnoses

VEE presents similarly to many neurologic diseases and other equid disorders, so laboratory confirmation is necessary for a definitive diagnosis. According to the OIE, rule-outs for VEE include Eastern and Western equine encephalomyelitis, Japanese encephalitis, West Nile fever, rabies, tetanus, African horse sickness, leucoencephalomalacia (*Fusarium* species), bacterial meningitis, and toxic poisoning.¹⁰

1.4 Ecology

1.4.1 General Overview

There have been 21 outbreaks of VEE since the 1930s, ranging from South America to the United States.¹¹ VEE continues to remain a naturally emerging disease due to mutations of the enzootic and endemic viral strains that circulate as a vector-borne disease among mammalian host populations, particularly in forest and swamp habitats.^{12,13} Historically, VEE has ranged from Argentina to midway through the Rocky Mountain Range in the United States. The enzootic strain known as Everglades virus, of subtype II, is currently endemic in southern Florida, and subtype III-B viruses have been found in Colorado and South Dakota.¹⁴

Since its identification in 1938, VEE complex viruses have been found throughout North,

¹⁰ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

¹¹ Weaver et al., 2004.

¹² Johnson KM, Martin DH. 1974. "Venezuelan equine encephalitis." *Advances in Veterinary Science and Comparative Medicine*. 18: 79-116.

¹³ Estrada-Franco et al., 2004.

¹⁴ USDA APHIS. 2013. Draft Case Definition of Venezuelan Equine Encephalomyelitis.

Central, and South America (Figure 1-2). Most epizootic VEE outbreaks have occurred in northern South America (Table 1-2), even though areas where enzootic strains are known to circulate cover a wider geographic range. In many cases, subtypes of VEE complex viruses are geographically distinct, and habitat type can play a significant role in the emergence of VEE or a particular subtype. For example, in regions where VEE is endemic, an outbreak is more likely to occur after heavy rainfall due to increased mosquito populations.¹⁵ Enzootic subtypes have been generally linked to lowland, wet, and/or forested habitats, but as tropical forests increasingly disappear to make way for farming, enzootic VEEV may be taking advantage of alternate vector species that are more suited to the changing landscape; this in turn leads to new patterns of epizootic VEEV emergence.¹⁶ Epizootic VEEV is more often associated with desert habitats after heavy rain has fallen.¹⁷ Additionally, climate change may impact enzootic VEEV habitat distribution, vector species abundance, and reservoir hosts.

Figure 1-2. Historical Distribution of VEE by Country (1925–Present)



Source: Weaver et al., 2004

¹⁵ Weaver et al., 2004.

¹⁶ Estrada-Franco et al., 2004.

¹⁷ Weaver and Barrett, 2004.

Table 1-2. Dates and locations of epizootic VEE outbreaks

Date	Location	Subtype
1925–1938	Colombia, Venezuela	IAB
1925–1946	Ecuador, Peru	IAB
1941–1943	Colombia, Venezuela	IAB
1942–1946	Peru	IAB
1949	Colombia, Venezuela	IAB
1950	Ecuador, Peru	IAB
1952	Colombia, Venezuela	IAB
1958	Ecuador, Peru	IAB
1959	Colombia, Venezuela	IAB
1962–1964	Colombia, Venezuela	IC
1967–1969	Colombia, Venezuela	IAB
1969	Ecuador, Peru	IAB
1969–1972	El Salvador, Guatemala, Honduras, Nicaragua	IAB
1971	Mexico, United States	IAB
1973	Colombia, Venezuela, Ecuador, Peru	IAB
1992–1993	Colombia, Venezuela	IC
1993	Mexico	IE
1995	Colombia, Venezuela	IC
1996	Mexico	IE

Source: Weaver et al., 2004

1.4.2 Susceptible Species

Equids and humans are highly vulnerable to epizootic/epidemic viral outbreaks. All members of the Equidae family are susceptible, and horses are the most common amplifying host for the virus.¹⁸ Cattle, swine, chickens, and canids can become infected but generally do not show signs of illness. Additionally, domesticated rabbits, goats, and sheep can be at risk for fatal disease during an epizootic.¹⁹

The circulation of enzootic VEEV subtypes in sylvatic mammals and other wildlife is significant in the emergence of epizootic disease. Many wildlife species are susceptible, and evidence of infection has been found in rodents, bats, opossums and other small mammals, gray fox, and wild bird species. Sylvatic rodents are thought to be the primary host species for enzootic VEEV

¹⁸ Center for Food Security and Public Health, Iowa State University. 2008. Equine Encephalomyelitis. Available at http://www.cfsph.iastate.edu/Factsheets/pdfs/easter_wester_venezuelan_equine_encephalomyelitis.pdf. Accessed July 2013.

¹⁹ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

due to their continually high rates of infection, their immunity, and their moderately high levels of viremia.²⁰

1.4.3 Introduction and Transmission of VEE

VEE is primarily transmitted by mosquito vectors, but the virus has been identified in black flies and ticks as well. Humans are typically infected by mosquitos that have contracted an epizootic virus from an equid source. Although some human viraemias may reach levels high enough to infect mosquitos, people do not play a significant role in the transmission of VEEV. The high viral titers produced in horses and other equids are essential to the transmission of VEEV in an outbreak, because it leads to the infection of less competent mosquito hosts. Generally, arbovirus hosts are small in size, such as the mammalian reservoirs of enzootic virus. The comparatively large size of equine hosts enables epizootic VEE viruses to spread widely and quickly. Additionally, outbreaks can be wider still if vector populations are simultaneously high due to seasonal patterns or wet weather events.²¹

1.4.3.1 Vector Transmission

As a group of arboviruses, the VEE complex exploits multiple mosquito vectors, with different species and genera of mosquito transmitting disease depending on the viral subtype and infected species. The mosquito genera *Ochlerotatus*, *Anopheles*, *Culex*, *Deinocerites*, *Mansonia*, and *Psorophora* have all been found to be infected with epizootic strains of VEEV. Enzootic VEE has been found to be transmitted by mosquito subgenus *Culex*, but epizootic outbreaks are more commonly associated with floodwater species, such as those of the *Ochlerotatus* and *Psorophora* genera.²² In the one epidemic in the United States, in Texas (1971), *Psorophora confinnis* was implicated as the most important VEE vector species.²³

The transmission cycles of VEEV between enzootic and epizootic hosts and vectors are complex. Figure 1-3 (adapted from Weaver et al., 2004) depicts the relationship between enzootic and epizootic VEE hosts, vectors, and susceptible species; the dotted arrow reflects the mutation of enzootic VEEV subtypes to epizootic, equine-virulent subtypes.²⁴

²⁰ Weaver et al., 2004.

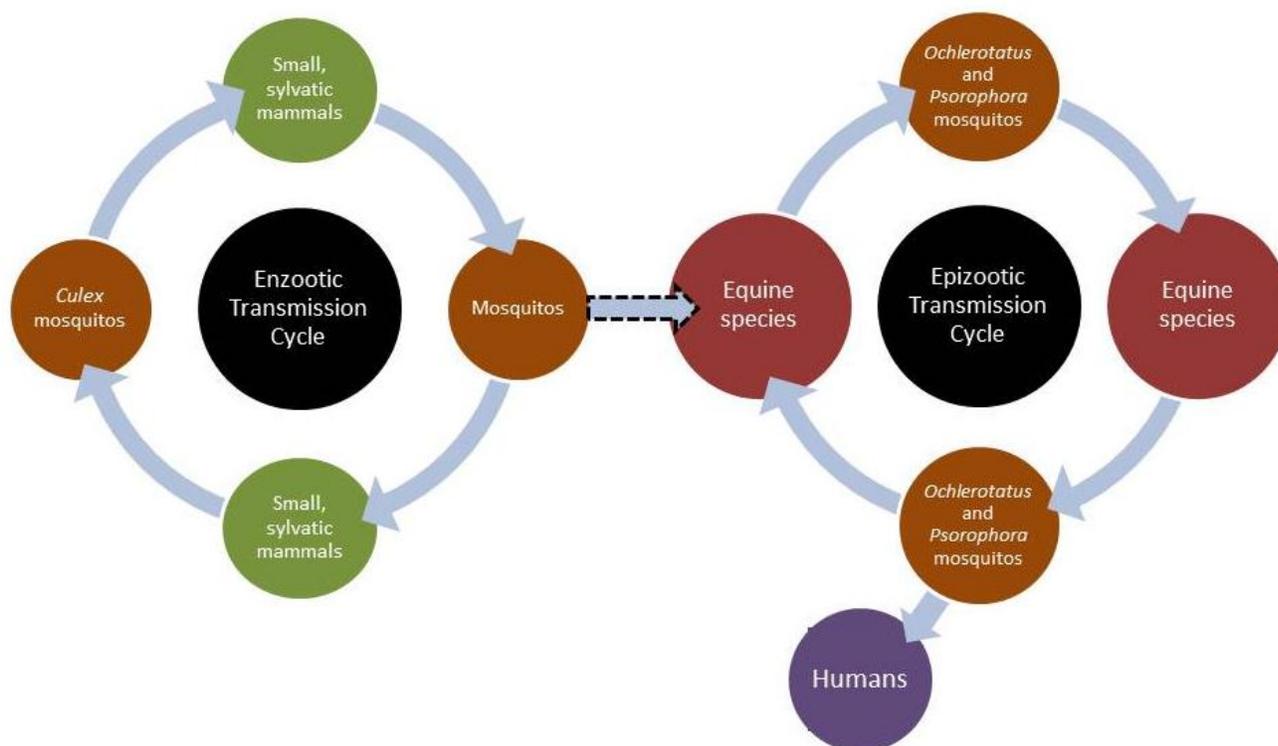
²¹ Weaver et al., 2004.

²² Weaver and Barrett, 2004.

²³ Bowen GS, Calisher CH. 1976. "Virological and serological studies of Venezuelan equine encephalomyelitis in humans." *Journal of Clinical Microbiology*. 4(1): 22-27.

²⁴ Weaver et al., 2004.

Figure 1-3. Transmission Cycles of VEE



While their role in outbreaks is unknown, tick species from *Amblyomma* and *Hyalomma* can be experimentally infected with enzootic and epizootic VEEV, and *Simulium* species of black flies can act as mechanical vectors.²⁵

1.4.3.2 Air/Windborne Transmission

While not naturally transmitted through the air, VEEV is highly infectious as an aerosol and is easily produced in this form in large quantities. For this reason, the virus had been developed as a biological weapon by the United States during the Cold War.²⁶

1.4.4 Incubation Period

The incubation period for VEE is reported to range from 12 hours to 5 days in equids and 1 to 6 days in humans; high fever is typically observed within 1 day.²⁷ The OIE defines the incubation period as 5 days and the infective period as 14 days.²⁸

1.4.5 Morbidity and Mortality

Equine morbidity and mortality rates from epizootic outbreaks vary widely and are estimated to be from 10 to 100 percent and 50 to 70 percent.²⁹ Enzootic VEEV almost never causes disease or

²⁵ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

²⁶ Croddy EC, J Hart, C Perez-Armendariz. 2002. *Chemical and Biological Warfare*. Springer-Verlag, New York.

²⁷ CFSPH, 2008

²⁸ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

²⁹ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

fatalities in equids. Human morbidity rates are extremely high (90 to 100 percent), but fatalities are rare, seen in less than 1 percent of cases, and severe neurological signs are seen in only 4 to 14 percent of infections; however, more severe illness and higher mortality rates are observed in children and the elderly.³⁰

1.4.5.1 Clinical Signs

VEE is generally characterized by febrile illness that can progress to include neurological signs.

1.4.5.1.1 Equids

Horses and donkeys typically present with fever, tachycardia, depression, and anorexia appearing 2–5 days after infection. Fever can be present as early as 12 hours. Encephalitis occurs after 5–10 days accompanied by circling, ataxia, and hyperexcitability. With a fatal case, death will usually occur after 1 week; in rare instances of severe disease, an animal may survive.

There are four typical presentations of clinical disease, as defined by the OIE:³¹

- Subclinical: Cases where clinical disease is absent are commonly associated with enzootic VEEV strains.
- Moderate: The main signs are fever, lack of appetite, and depression.
- Severe, non-fatal: Fever, anorexia, and depression continue along with an increase in neurological signs that may include muscle spasms, lack of coordination, blindness, circling, rocking on limbs, paddling (in fallen or recumbent animals), stupor, and convulsions. Diarrhea and colic may occur in some cases.
- Fatal: Severe disease concluding in death, which may be sudden and occur shortly after neurological signs appear. Alternately, prolonged disease can result in death from dehydration and deteriorated condition.

1.4.5.1.2 Humans

Early symptoms of VEEV infection in humans include flu-like symptoms such as fever, chills, malaise, severe headache, myalgia in the legs and lower back, leukopenia, tachycardia, and, in some cases, nausea, vomiting, and diarrhea. If neurological signs are present, they may include convulsions, drowsiness, confusion, and photophobia. Children are more likely to sustain permanent neurological damage. After acute disease has passed in 4 to 6 days, an infected person may feel weak and/or fatigued for several weeks. In lethal cases, diffuse congestion and edema with brain, gastrointestinal tract, and lung hemorrhaging occur, sometimes with meningoencephalitis. VEEV is also a risk to the fetus, capable of causing birth defects, stillbirth, and abortion.³²

³⁰ Center for Food Security and Public Health, Iowa State University. 2008.

³¹ OIE. 2009. Technical Disease Card for Venezuelan equine encephalitis.

³² Weaver et al., 2004.

1.5 Environmental Persistence of VEEV

VEEV is vulnerable to exposure to sunlight and heat and/or drying. It can survive in cool, moist, and dark conditions. The virus can be maintained in blood, exudates, and freeze dried materials (such as aerosols).³³

Table 1-2. Resistance of VEEV to Physical and Chemical Action

Action	Resistance
Temperature	Alphaviruses are inactivated at 58°C or higher. Further, Togaviruses cannot survive 15 minutes at 65°C.
pH	Stable in alkaline conditions (pH 7-8) but quickly inactivated by acidic environment (pH <6).
Disinfectants	Inactivated by various common disinfectants; sensitive to organic solvents and detergents 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde and formaldehyde.
Survival	VEEV is susceptible to radiant sunlight, moist or dry heat, and drying.

Source: OIE Technical Disease Card for VEE, 2009.

1.6 Risk of Introduction into the United States

Epizootic VEE could again spread into the United States from Mexico; furthermore, enzootic subtype II, Everglades virus, is already found in southern Florida, and Bijou Bridge virus has been observed in swallow bugs in Colorado. Also, an invasive mosquito species that is pervasive in many areas of the United States has recently been proven a competent VEEV vector.³⁴

During past epizootic outbreaks in Mexico, USDA APHIS has banned or restricted movement of equids from Mexico and heightened surveillance. In such an event, it should be noted that infected vectors could bring VEE via vehicles, ships, and aircraft, especially in cargo. Inactivated VEE virus vaccines are approved for use and commercially available in the United States, and some equids are routinely vaccinated against VEE. Quarantine and indoor or screened stabling during outbreaks may also help limit introduction or spread of epizootic VEEV.³⁵

³³ US Department of Health and Human Services. 2009. *Biosafety in Microbiological and Biomedical Laboratories* (5th ed.). Available at http://www.cdc.gov/biosafety/publications/bmbl5/BMML5_introduction.pdf. Accessed August 2013.

³⁴ Beaman JR, Turell MJ. 1991. Transmission of Venezuelan Equine Encephalomyelitis Virus by Strains of *Aedes albopictus* (Diptera: Culicidae) Collected in North and South America. *Journal of Medical Entomology*. 28: 161-164.

³⁵ USDA APHIS. 2013. Draft Case Definition of Venezuelan Equine Encephalomyelitis.

Attachment 1.A References and Selected Resources

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Attachment 1.B Abbreviations

APHIS	Animal and Plant Health Inspection Service
FAD PReP	Foreign Animal Disease Preparedness and Response Plan
VEE	Venezuelan equine encephalomyelitis
VEEV	Venezuelan equine encephalomyelitis virus
OIE	World Organization for Animal Health
NAHEMS	National Animal Health Emergency Management System
RNA	ribonucleic acid
SOP	standard operating procedure
TDD	telecommunications device for the deaf
USDA	United States Department of Agriculture