



Epidemiology and Ecology of Eastern Equine Encephalomyelitis

United States Department
of Agriculture

Animal and Plant Health
Inspection Services

Veterinary Services

Centers for Epidemiology
And Animal Health

Center for Emerging Issues

April 2004

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Acknowledgements

The following reviewers are acknowledged for their editorial input and for images contained in this publication:

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Epidemiology and Ecology of Eastern Equine Encephalomyelitis

Table of Contents

List of Abbreviations.....	IV
List of Figures.....	IV
Tables.....	List of V
Summary.....	6
Introduction.....	7
“Micro-level” Arbovirus Transmission.....	7
“Macro-level” Arbovirus Transmission.....	9
Ecology of EEE.....	12
Epizootiological Investigations of EEE.....	13
Risk Factors Associated with EEE.....	23
Impact of Climate Change on EEE.....	24
Economic Burden of EEE.....	25
Bibliography.....	29

Epidemiology and Ecology of Eastern Equine Encephalomyelitis

List of Abbreviations

EEE, Eastern equine encephalomyelitis
EEEV, Eastern equine encephalomyelitis virus
LAC, LaCrosse encephalitis
NOAA, National Oceanic and Atmospheric Administration
SLE, St. Louis encephalitis
VEE, Venezuelan equine encephalomyelitis
VEEV, Venezuelan equine encephalomyelitis virus
WEE, Western equine encephalomyelitis
WEEV, Western equine encephalomyelitis virus

List of Figures

Figure 1. Sequential steps required for a competent female mosquito to transmit an arbovirus after ingestion of an infective blood meal.

Figure 2. Transmission cycles for arboviruses.

Figure 3. Equine cases of eastern equine encephalomyelitis, United States, January 1, 1994 through December 31, 1994.

Figure 4. Epizootic curves of four outbreaks of eastern equine encephalomyelitis in horses in Michigan.

Figure 5. Temporal distribution of eastern equine encephalomyelitis in Florida, years 1982 and 1983.

Figure 6. Clinical signs of eastern equine encephalomyelitis in a horse during the EEE epizootic in Wisconsin, USA, 2001.

Epidemiology and Ecology of Eastern Equine Encephalomyelitis

List of Tables

- Table 1.** Geographical location of the predominant hardwood trees that are found in swamps with endemic eastern equine encephalomyelitis virus.
- Table 2.** Annual amount of precipitation and mean temperature in Michigan during the year preceding an outbreak and the year of an outbreak of disease in horses that was attributed to eastern equine encephalomyelitis virus.
- Table 3.** Categories of factors, specific factors, presence or absence of factors, and extent of factors potentially associated with eastern equine encephalomyelitis in horses in Michigan during five outbreaks, between years 1972 and 1991.
- Table 4.** Comparison of eastern equine encephalomyelitis (EEE) horse incident cases among five geographic zones in Florida during years 1982 and 1983, as reported from mail survey responses received from 126 private equine or mixed-practice veterinarians.
- Table 5.** Comparison of eastern equine encephalomyelitis (EEE) horse incident cases among five geographic zones in Florida during years 1982 and 1983, as reported by the Florida Animal Disease Diagnostic Laboratory.
- Table 6.** Hypothesized management and environmental risk factors potentially associated with eastern equine encephalomyelitis (EEE)-affected equids during an outbreak of EEE in Michigan in 1991.
- Table 7.** Confirmed management and environmental risk factors associated with eastern equine encephalomyelitis-affected equids during an outbreak in Michigan in 1991.
- Table 8.** Medical interventions used by veterinarians who had observed horses in Florida that survived eastern equine encephalomyelitis during years 1982 and 1983.
- Table 9.** Estimated cost of clinically apparent cases of EEE in horses in Florida during years 1982 and 1983.

Summary

Eastern equine encephalomyelitis viruses (EEEV) are members of the *Alphavirus* genus, family *Togaviridae*. EEEV can be transmitted to equines and humans during the bite of an infected mosquito. In addition to horses and humans, EEEV can produce severe disease in common livestock species such as swine and cattle. EEEV, or virus-specific antibodies, have been recovered from birds such as the turkey, pheasant, quail, ostrich, and emu, among others. Other animals in which EEEV have been found are the turtle, snake, hamster, and fish. In addition to the mosquito, EEEV have been isolated from the horse fly, blackfly, mite, lice, and *Culicoides* spp. The majority of EEEV isolates have been from only 27 species of mosquito, and a high percentage of the isolates have been from a single species of mosquito, *Culiseta melanura*.

The primary habitats for EEEV are lowlands. Considered to be enzootic EEEV habitats, swamps located in Atlantic coastal states (e.g., Florida, Maryland, New Jersey, New York) and in Michigan are characterized by muck-peat soils that are dominated by hardwood trees. These hardwood trees have a preference for wet, mucky habitats, and they provide a root system that supports oviposition and larval development of *C. melanura*. The dispersal of arboviruses such as EEEV from enzootic foci is determined by the arthropod vectors and vertebrate hosts involved in the natural cycles of arboviruses. “Uncomplicated”, “intermediate”, and “complicated” cycles of arbovirus transmission have been described. The uncomplicated cycle involves a single vertebrate host and a single primary vector species. In the intermediate cycle, the virus is maintained in an enzootic cycle, and humans or other vertebrates are involved tangentially, mostly as dead-end hosts. The complex cycle has enzootic and epizootic subcycles with different vectors and vertebrate hosts in each subcycle. Humans and domestic animals are involved tangentially from the epizootic portion of the complicated cycle, again as dead-end hosts.

Naturally-occurring outbreaks of EEE usually are observed during periods of hot, rainy weather. These weather conditions are ideal for expansion of *C. melanura* and other mosquito populations. Outbreaks of EEE in horses and humans are expected to occur from midsummer to late summer, with August being the peak month of incident cases in much of the US. The duration of these outbreaks is from two to three months in the eastern US and three to five months in the southeastern US. The percent case-fatality due to EEE in horses in Florida was 75% to 100%. An evaluation of EEE in horses in Michigan during five outbreaks between years 1972 and 1991 showed that the factors associated potentially with the outbreaks were incomplete drainage-lake type hydrographic regions as a type of water distribution, *C. melanura*, *Coquilletidia perturbans*, and *Aedes vexans* as vectors, wild and domestic birds as reservoir hosts, humans and commercial poultry flocks as incidental hosts, and an increase in the state-wide annual precipitation and an increase in region-specific precipitation, both as weather conditions. An increase in the state-wide annual temperature was not an apparent factor. An epidemiological investigation of EEE in Michigan in 1991 showed that having swamp land on the premises and having wooded land on the premises were associated with increased risk of EEE. Annual vaccination of horses against EEEV and using insect repellents were associated with decreased risk of EEE.

Epidemiology and Ecology of Eastern Equine Encephalomyelitis

Introduction

The Eastern equine encephalomyelitis viruses (EEEV) are members of the *Alphavirus* genus, family *Togaviridae*. EEEV can be transmitted to equines and humans by the bite of an infected mosquito. Equine epizootics in North America have been recorded as far back as 1831, although the enzootic cycle probably was present much earlier than 1831. In addition to equines (Morris, 1988), and humans (Centers for Disease Control and Prevention, 2000), EEEV can produce severe disease in common livestock species such as swine (Elvinger et al., 1994; Elvinger et al., 1996), calves (Scott et al., 1988), and cows (McGee et al., 1992). EEEV, or antibodies against the EEEV, have been recovered from birds such as the turkey, pheasant, quail, ostrich, emu, Peking duck, chukar, pigeon, whooping crane, owl, glossy ibis, and the snowy egret (Morris, 1988; Tully et al., 1992, Morris, 1994). Other animals in which EEEV have been found are the turtle, snake, hamster, and fish. Regarding insects and arthropods, EEEV has been isolated from the horse fly, blackfly, mite, lice, and *Culicoides* spp. However, the majority of EEEV isolates were from 27 species of mosquitoes, and 80 percent of the isolates were from a single species of mosquitoes, *Culiseta melanura* (Morris, 1994).

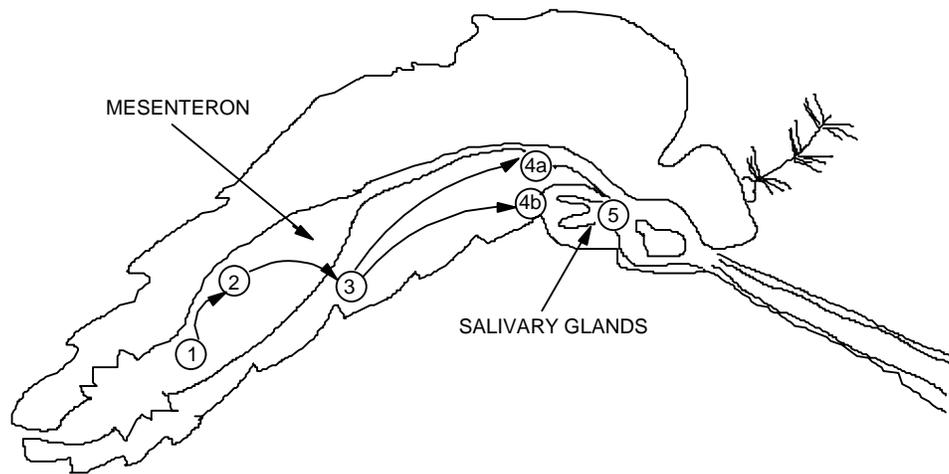
While the core proteins of all strains of EEEV are similar, structural differences among the strains from different biological sources and different geographical sources have led to the multiplicity of groupings such that EEEV strains have been grouped over the years by various investigators into 3, then 4, then 2, then 7 groups (Morris, 1994). There are biological differences among epidemic/epizootic and endemic/enzootic strains. There also are differences in the virulence of strains from different geographical regions.

“Micro-level” Arbovirus Transmission

The following discussion of micro-level and macro-level arbovirus transmission applies not only to EEEV, but to arboviruses in general. A complex series of interactions among the virus, arthropod vectors, and vertebrate hosts, in concert with a range of ecological conditions, are necessary for successful transmission of arboviruses (Calisher, 1994). The number of “unknown” factors probably exceeds the number of “known” factors. A biological transmission cycle of an arbovirus begins with an uninfected, susceptible, and competent hematophagous arthropod consuming a blood meal from a virus-infected vertebrate (**Figure 1**). If there is no mechanism that prevents the virus from moving from the arthropod’s gut into other tissues, the ingested virus will replicate in the gut and then be disseminated to other tissues.

Arboviruses are exposed to two vastly different biological environments: vertebrate host cells and arthropod cells. The ability of arboviruses to process enzymes from the different host types influences the capacity of the viruses to tolerate such disparate environments. Viral glycoproteins frequently are necessary for viruses to attach to the host cell membrane. Processing of LaCrosse virus (LAC, a bunyavirus) glycoproteins removes one glycoprotein from the viral surface,

Figure 1. Sequential steps required for a competent female mosquito to transmit an arbovirus, after ingestion of an infective blood meal (Reproduced from Hardy, 1988. With permission).



1. Infectious blood meal ingested into the midgut.
2. Virus infects and multiplies in mesenteronal epithelial cells.
3. Virus released from mesenteronal epithelial cells into the body cavity.
- 4a. Virus infects salivary glands after secondary amplification in other cells/tissues.
- 4b. Virus infects salivary glands without secondary amplification in other cells/tissues.
5. Virus released from salivary gland epithelial cells and is transmitted during blood feeding.

but a second glycoprotein remains intact; thus, “attachment” of LAC bunyavirus to host cells may be disrupted only minimally by its host. If an arbovirus traverses the gut wall successfully, it is transported along nerves to organs and tissues, including the salivary gland, where it replicates. Then, the arthropod will transmit virus to a vertebrate host on which the arthropod feeds. The arthropod vector’s genes determine an arthropod’s susceptibility to arboviruses and its ability to transmit the virus further. Five sequential steps are required for a competent female mosquito to transmit an arbovirus after ingestion of an infective blood meal (Hardy, 1988).

These steps are:

1. Infectious blood meal ingested into the midgut.
2. Virus infects and multiplies in mesenteron epithelial cells.
3. Virus is released from mesenteron epithelial cells into the body cavity.
- 4a. Virus infects salivary glands after secondary amplification in other cells/tissues.
- 4b. Virus infects salivary glands without secondary amplification in other cells/tissues.
5. Virus is released from salivary gland epithelial cells and is transmitted during blood feeding.

Several factors influence the ability of arthropod vectors to transmit arboviruses successfully. Some of these factors include: (1) the blood feeding preferences of the vector, (2) attractiveness of the susceptible vertebrate host to the arthropod, (3) degree of tolerance of the vertebrate host to being fed upon by vector, (4) geographical distribution of vertebrate hosts, (5) abundance of vertebrate hosts, and (6) characteristics of the climate and local habitat (Scott, 1988).

After a vertebrate host has been fed upon by an infected arthropod, the vertebrate host itself becomes infected, develops viremia, and produces antibodies. Antibody production by the host has benefits and drawbacks, the benefit being suppression of viremia, and the drawback being modification of the arbovirus population via genetic selection of more hardy genotypes of virus. Birds are the primary vertebrate hosts of EEE, western equine encephalomyelitis (WEE), and St. Louis encephalomyelitis (SLE) viruses.

“Macro-level” Arbovirus Transmission

Dispersal of arboviruses from enzootic foci, or from more recently established foci, is determined by the arthropod vectors and vertebrate hosts involved in the natural cycles of arboviruses (Turell, 1988). “Uncomplicated”, “intermediate”, and “complicated” cycles of arbovirus transmission have been described (Figure 2A, 2B, 2C). The uncomplicated cycle involves a single vertebrate host and a single primary vector species (2A). In the intermediate cycle, the virus is maintained in an enzootic cycle; humans or other vertebrates are involved tangentially, mostly as dead-end hosts (2B). The complex cycle has enzootic and epizootic subcycles with different vectors and vertebrate hosts in each subcycle. Humans and domestic animals are involved tangentially from the epizootic portion of the cycle, again primarily as dead-end hosts (2C).

Figure 2A, 2B. Transmission cycles for arboviruses. A, “uncomplicated” cycle. B, “intermediate” cycle. (From Turell, 1988. With permission).

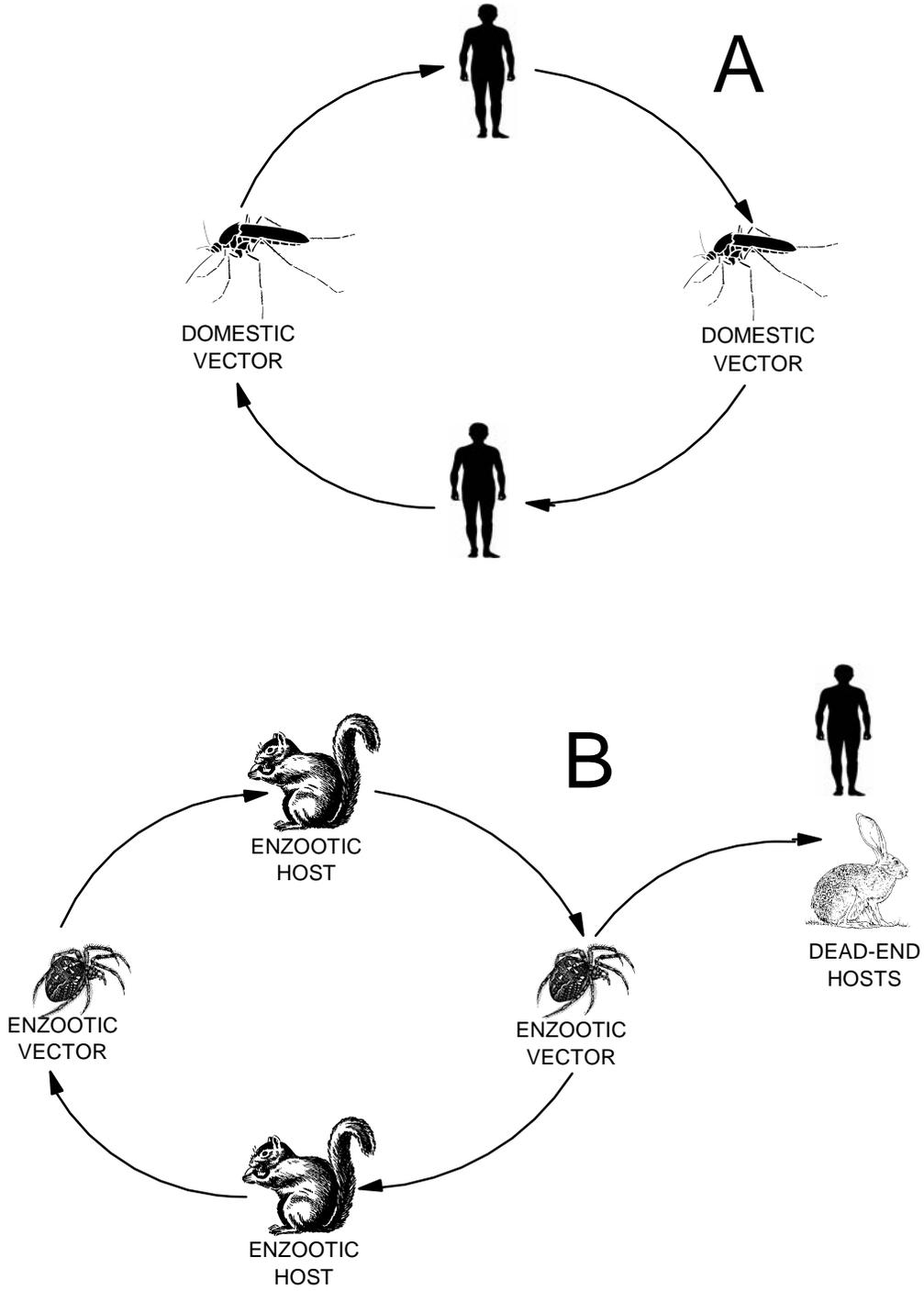
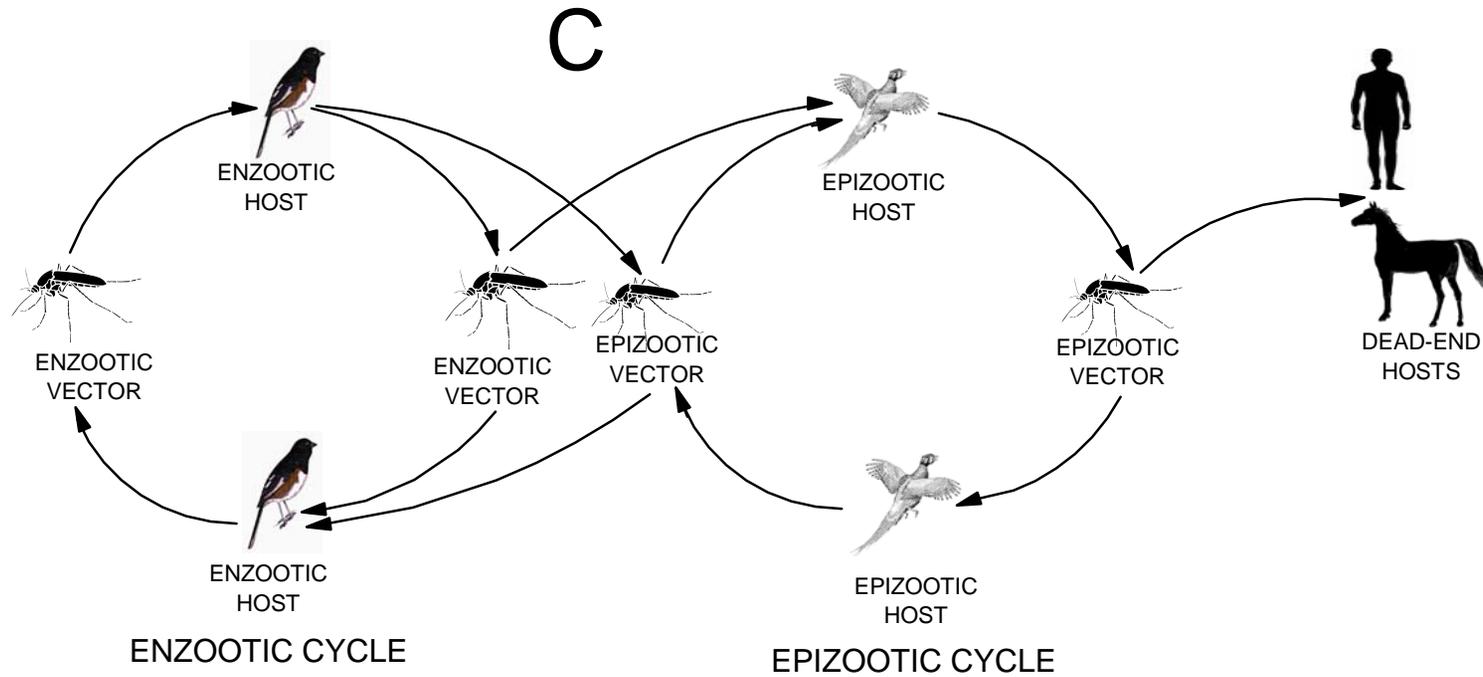


Figure 2C. Transmission cycles for arboviruses. C, “complicated” cycle (From Turell, 1988. With permission).



Ecology of EEE

The primary habitats for EEEV are lowlands, although the virus may inhabit some upland areas (Morris, 1994). Endemic EEE swamps located in Atlantic coastal states (e.g., Florida, Maryland, New Jersey, New York) and in Michigan are characterized by muck-peat soils that are dominated by hardwoods (Table 1). These hardwoods have a preference for wet, mucky habitats, and they provide a root system that supports oviposition and larval development by *C. melanura*. The larvae require a darkly shaded source of water that contains a high concentration of organic matter located at the bottom. The larvae that survive the overwintering phase develop into pupae. Then adult mosquitoes emerge, first in February and March in Florida, and in April through May in the northern US. A second emergence of larvae occurs in late June and early July in the northern US. EEEV isolations are more prevalent in mosquitoes from the second emergence. Thus, it has been suggested that the first and second broods may differ genetically and physiologically, and that these differences may influence the vector potential of each brood (Morris, 1988). The daily survival rate of the adult female mosquitoes is 0.89. This indicates that by 24 hours after a given population of adult mosquitoes has emerged, only 89 percent will have survived, and for each day thereafter, only 89 percent of the respective, remaining population can be expected to survive (Morris, 1994).

Table 1. Predominant hardwoods that are found in various North American, EEE endemic foci.	
Country or State	Predominant Hardwoods
Canada, New York, Massachusetts, Michigan	red maple, hornbeam
New Jersey, Maryland	red maple, cedar
Florida	loblolly bay, swamp and sweet bay, black gum, sweet gum
Source: Morris, 1994.	

Mark-recapture techniques have been used to verify the potential role of arthropods in dispersing arboviruses from enzootic foci. Mosquitoes of the species *Culiseta* (*C. melanura*, *C. morsitans*) were found as far as 9.8 km from the site where they were released, indicating that significant spread of vectors by vector flight alone does occur (Howard et al., 1989). Increases in arthropod and vertebrate host populations, movements of foraging mammals, movements of birds, the hatch and birth of susceptible clutches of birds, and abundance of some foods may support virus amplification and transmission of infection. Winds may disperse infected arthropods from enzootic and epizootic sites. The wind velocity, wind trajectory, ambient temperatures, rainfall, barometric pressures, and other meteorologic factors have been studied, but there is insufficient evidence that these factors serve as effective passive mechanisms for dispersal of arbovirus infections. Extensive ecological investigations to assess the effects of meteorologic factors and global warming on arbovirus infections and investigations of interactions between virus, vector, and vertebrate host genes may help to forecast the onset of vector-borne viral diseases (Shope, 1991).

The normal geographical distribution of EEE viruses in horses in the US is east of the Mississippi River, typically including the coastal states of Alabama, Florida, Georgia, Louisiana, Maryland, Mississippi, New Jersey, North Carolina, and South Carolina, and the midwestern states of Indiana, Michigan, and Ohio (**Figure 3**). Similar patterns were observed during 1994-1996 (USDA APHIS, 1995,1996,1997). At least one sporadic incident of EEE in a horse has been reported in California, but the origin of the incident was not established (Franklin et al., 2002).

Epizootiological Investigations of EEE

Michigan

Some of the more thorough and more recent investigations of the epizootiology of EEEV were done in Michigan. Epizootics of disease attributable to EEEV in horses in Michigan and the environmental patterns and weather conditions that were associated with these epizootics were evaluated extensively and intensively by Ross and Kaneene (Ross and Kaneene, 1996). Their goal was to identify the factors that may predispose horses in specific geographical regions of Michigan to infections with EEEV. The data for this retrospective epidemiological analysis were acquired from reports of epizootics of EEE during five different years. These epizootics were reported during years 1973, 1980, 1981, 1982, and 1991. Hydrographic regions (i.e., water drainage), land cover, vectors of EEEV (e.g., *C. melanura*, *Coquilletidia perturbans*, and *Aedes vexans*), wild-bird reservoir hosts, incidental hosts (e.g., horses, humans), weather conditions (e.g., precipitation, ambient temperature), and season of the year (e.g., summer, winter) were among the classes of variables that were evaluated during their investigation.

Season

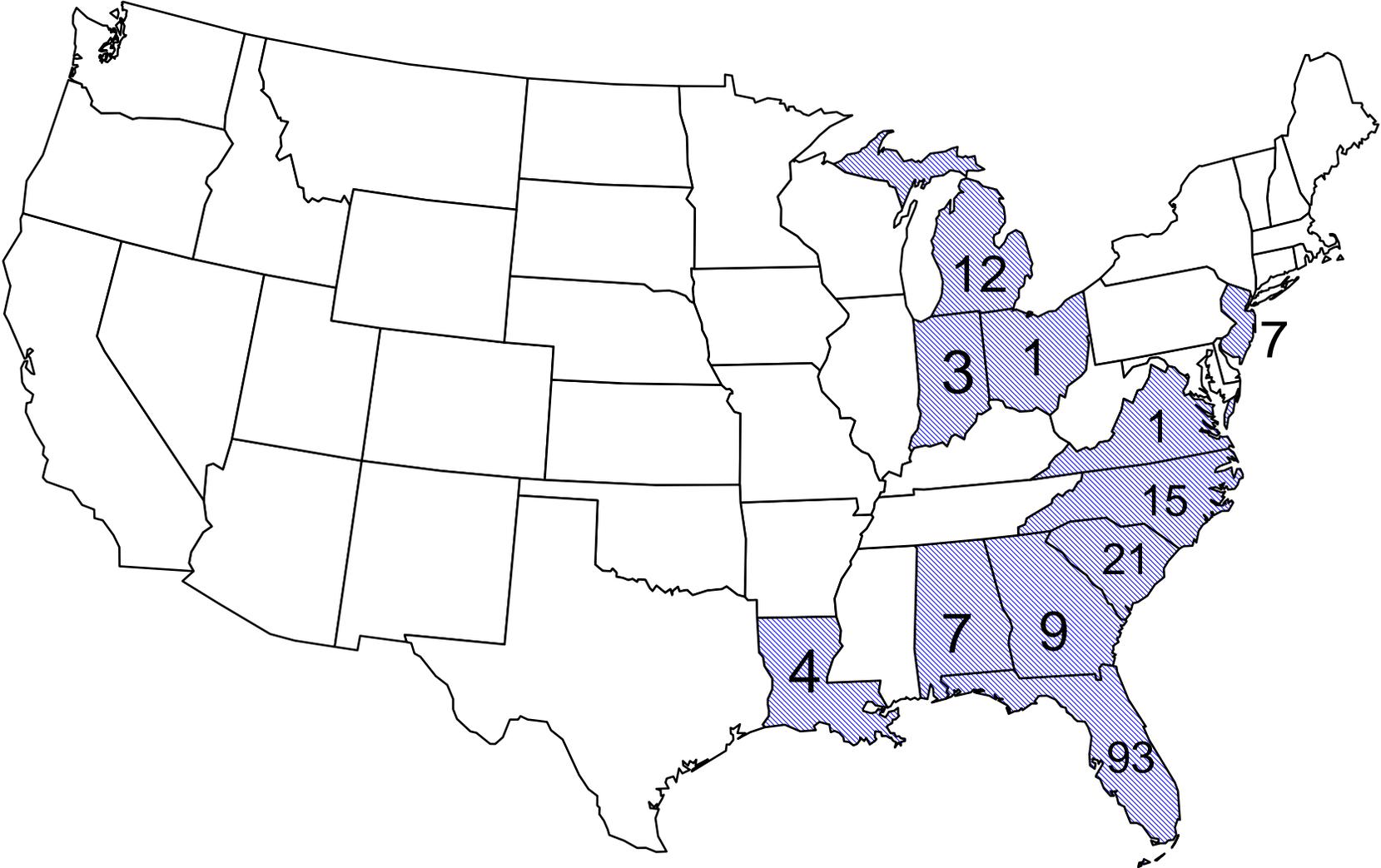
Specific weather conditions that precipitate epizootics attributable to EEEV have not been established clearly. However, epizootics usually are observed during periods of hot, rainy weather; these conditions are ideal for expansion of *C. melanura* and other mosquito populations (Walton, 1992; Nasci et al., 1993). Epizootics of EEE are expected to occur from midsummer to late summer, with August being the peak month of incident cases (Gibbs, 1976; Hanson, 1972). The epizootiological investigations of EEE in Michigan showed that the incident equine cases were most frequent during September (**Figure 4 a, b, c, d**), and these cases were reported only from the lower peninsula of Michigan (Ross and Kaneene, 1996). The duration of the epizootics was from two months to three months.

Vectors

Seventy-seven (93 percent) of the 83 counties in Michigan reported at least one of the three species of mosquitoes that are vectors for EEEV. Of the 78 counties in which *C. perturbans* and *A. vexans* were found, both species were found in 62 counties, *C. perturbans* alone was found in 6 counties, and *A. vexans* alone was found in 10 counties. *C. melanura*, *C. perturbans*, and *A. vexans* were identified in the geographical vicinity of incident equine cases

Figure 3. Equine Cases of Eastern Equine Encephalomyelitis *

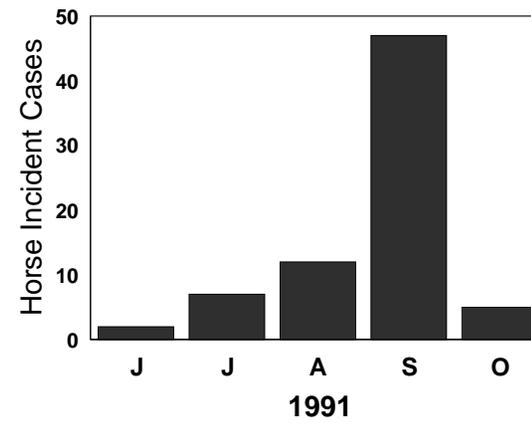
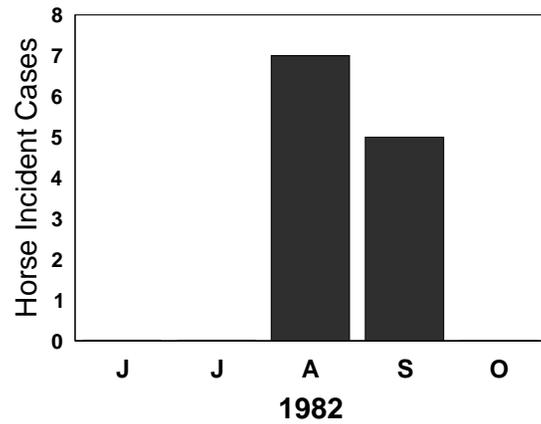
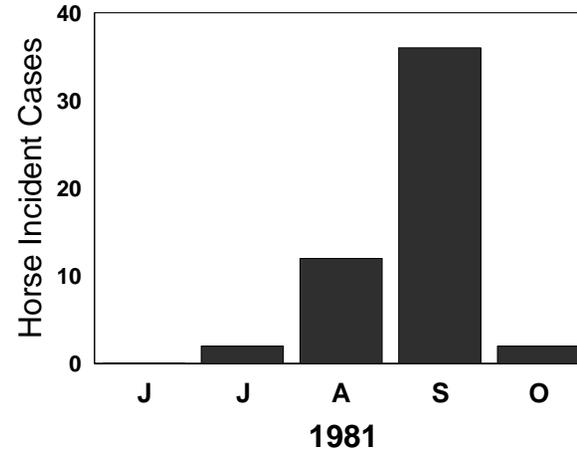
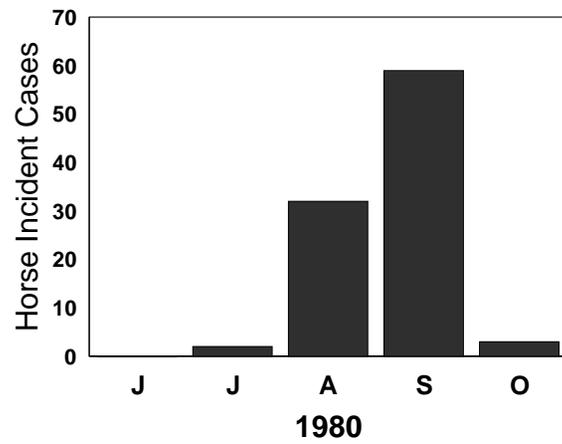
January 1, 1994 through December 31, 1994



* Revised from USDA APHIS Veterinary Services, 1995.

* Data Reported by National Veterinary Services Laboratories and Centers for Disease Control and Prevention.

Figures 4A,4B,4C,4D. Epizootic curves of four outbreaks of eastern equine encephalomyelitis in Michigan (From Ross and Kaneene, 1996. With permission).



during most of the reported epizootics of EEE. All three species were positive for EEEV via virus isolation during three of the EEE investigations. *C. melanura* was identified in 11 counties in the lower peninsula, and affected horses were located in eight of these 11 counties. *C. melanura* was identified only within or immediately bordering incomplete drainage-lake type hydrographic regions in the lower peninsula. These two findings support the role of *C. melanura* as a potential EEEV vector. *C. melanura* was not found in the upper peninsula where there were no cases of EEE, not even in the incomplete drainage-lake type hydrographic region. Thus, the absence of *C. melanura* from the upper peninsula may explain the absence of affected horses from this region.

Wild-bird reservoir hosts and incidental hosts

Wild-bird reservoir hosts were found during 5 of the epizootics, and exposure of wild-bird reservoir hosts to EEEV during the epizootics was verified, either by virus isolation or by virus neutralization tests. Humans, as well as domestic species including chickens and pheasants, were positive for EEEV, either by virus isolation or by virus neutralization tests.

Precipitation

An annual excess in precipitation of 20-plus centimeters for two consecutive years, (1) the year preceding the epizootic and (2) the year of the epizootic, has been reported as a predictor of EEE epizootics, but the source of these data was limited to a few states in the U.S. (Grady et al., 1978; Letson et al., 1993). The state-wide, annual amounts of precipitation in Michigan were greater than normal during each of the years prior to and each of the years of an epizootic of EEE; specifically, the annual precipitation in Michigan was 10 centimeters (approximately 8 percent) greater than expected during each of the years prior to and each of the years of an epizootic of EEE. Even after the state was stratified into its five National Oceanic and Atmospheric Administration (NOAA) geographic regions, the annual amounts of precipitation within each geographic region remained greater than expected during these same years. For example, annual precipitation in the southwestern lower peninsula was 14 to 15 centimeters greater than expected. However, the amount of precipitation in no region of Michigan reached the EEE-predictive level of 20-plus centimeters that had been referred to in previous investigations (Grady et al., 1978) (Table 2).

Similar findings were reported from an analysis of data from 11 states located along the Atlantic and Gulf Coasts of the US that were involved in an epizootic of EEE in 1989 (Letson et al., 1993). For example, in Massachusetts and New Jersey, there were epizootics of EEE when there was unusually heavy rainfall during late summer and early fall of the year preceding the outbreak and during the summer of the year of the epizootic. A similar, season-specific analysis of precipitation data from Michigan was done also. The analysis of the Michigan data showed that precipitation also was increased during late summer and early fall of the year preceding an EEE epizootic, and it was increased during the summer of an EEE epizootic (Ross and Kaneene, 1996).

Table 2. Annual amount of precipitation and mean temperature in Michigan

during the year preceding an epizootic and the year of an epizootic of disease in horses that was attributed to eastern equine encephalitis virus.				
Year	Annual Precipitation* (cm)		Annual Temperature* (°C)	
	Total	Deviation‡	Mean	Deviation‡
1972	92.3	6.9	11.4	-1.4
1973	88.0	9.0	7.1	0.7
1979	82.2	7.0	0.7	-1.1
1980	87.9	7.2	6.1	-0.9
1980	87.9	7.2	6.1	-0.9
1981	89.0	7.8	7.5	-0.3
1981	89.0	7.8	7.5	-0.3
1982	89.4	7.6	7.9	-0.5
1990	98.9	8.7	17.3	0.9
1991	93.1	8.9	8.1	1.0

*Values reported are computed normal annual means that were determined for the years 1951-1980. ‡Deviation from the computed normal annual amount of precipitation. †Deviation from the computed normal annual mean temperature. [Source: Ross and Kaneene, 1996. Reprinted with permission.](#)

Environmental temperature

There have been attempts in the laboratory environment to develop thermal models to predict the emergence of *C. melanura* in natural environments ([Mahmood and Crans, 1998](#)). The embryonic development and emergence of *C. melanura* were correlated with ambient temperatures within the range of 10°C to 34°C. The thermal minimum for embryonic development was 9.4°C. Embryonic development progressed slowly at 10°C; however, the amount of time required for eclosion (i.e., progression from the egg stage to the adult stage) decreased as the temperature was increased from 10°C to 28°C. Temperatures between 29°C and 32°C were lethal to the eggs. The thermal minimum for larval development was 8.5°C and, as in the case of embryonic development, the thermal minimum for larval development decreased also as the temperatures were increased. The water temperature in the subterranean habitats may regulate the northern geographic limit for EEEV amplification each year ([Mahmood and Crans, 1998](#)).

Although the eclosion of *C. melanura* has been correlated with the environmental temperature in the laboratory, the environmental temperature has **not** been shown to be a valuable predictor for EEE epizootics in nature. The annual mean environmental temperatures in Michigan were approximately normal during each of the years prior to and each of the years of an outbreak of EEE (**Table 2**). Approximately normal temperatures were reported during the

spring and summer seasons of the years in which epizootics of EEE were studied, there being just a single exception to this pattern.

Regions and land cover

Unlike weather conditions, a specific type of land cover has been associated with the habitat of EEEV vectors and wild-bird reservoir hosts for EEEV (Morris et al., 1980; Emord et al., 1984). Hydrographic (versus geographic) regions are used to describe the distribution of various bodies of water including lakes, rivers, and oceans and their drainage patterns. The distribution of hydrographic regions in Michigan is associated with the surface characteristics of glacial deposition. These hydrographic regions that are classified as ‘incomplete drainage-lake type’ hydrographic regions are regions in which the glaciated surface does not permit the streams to drain completely. The regions are characterized by numerous small lakes, swamps, and bogs that are connected by streams, and that usually have dry land between the lakes, swamps, and bogs. Incident equine cases were reported from four of the five incomplete drainage-lake type hydrographic regions in Michigan, with the exception of the one incomplete drainage-lake type hydrographic region in the upper peninsula. Incident cases of EEE were reported in six contiguous counties in southwestern Michigan with these characteristics during most of the epizootics of EEE in Michigan. Epizootics of EEE also have been associated with similar hydrographic regions in New York and in Ohio (Morris et al., 1980; Nasci, 1993). These regions were described as hardwood forests and swamps that contain maple, beech, white pine, hickory and oak trees. The regions also were located in close proximity to agricultural lands.

Summary of EEE in Michigan

- The peak number of horses that were affected by EEE in Michigan occurred in September, not in August, as has been reported by other investigators (Gibbs, 1976; Hanson, 1972).
- Horses in the lower peninsula, specifically the southeastern and south-central Michigan, were most consistently involved in the EEE epizootics in Michigan. No horses from the upper peninsula were affected by EEE.
- EEE cases in horses in Michigan were consistently associated with incomplete drainage-lake type hydrographic regions.
- The three species of mosquitoes that are associated most frequently with the EEE virus life cycle, *C. melanura*, *C. perturbans*, and *A. vexans*, also were identified in Michigan.
- Each species of mosquito was identified in geographic locations in which EEE cases in horses were identified. Also, each species of mosquito was identified in the incomplete drainage-lake type hydrographic regions.
- *C. melanura*, the primary vector of EEEV, was not identified in the upper peninsula of Michigan. Neither were there EEE cases in horses in the upper peninsula.
- Wild-bird reservoir hosts of EEEV were found in geographic locations in which cases of EEE in horses also were found in Michigan.

- The annual precipitation in Michigan was increased during years in which there were EEE epizootics in horses, but the annual precipitation did **not** reach the 20-plus centimeter level, one criterion that has been reported to be “predictive” of EEE epizootics.
- The annual temperatures in Michigan were **lower** than expected during each year prior to an EEE outbreak. These temperatures were approximately normal during the spring and summer of the years in which there were outbreaks of EEE.

The factors that potentially predisposed horses to EEE during five outbreaks in Michigan between 1972 and 1991 are listed in **Table 3**.

Table 3. Category, description, and presence or absence of factors potentially associated with eastern equine encephalomyelitis in horses in Michigan during five outbreaks during the 20-year interval 1972 to 1991.		
Factors		
Category	Description	Present or Absent
Hydrographic region	Incomplete drainage-lake type	Present
Vectors of EEEV	<i>Culiseta melanura</i>	Present
	<i>Coquilletidia perturbans</i>	Present
	<i>Aedes vexans</i>	Present
Bird reservoir hosts	Wild birds	Present
	Domestic birds (chickens, pheasants)	Present
Incidental hosts	Humans	Present
	Commercial poultry flocks	Present
Weather conditions	Increased state-wide annual precipitation	Present
	Increased region-specific precipitation	Present
	Increased state-wide annual temperature	Absent

[Source: Ross and Kaneene, 1996.](#)

Florida

An investigation of the epizootology and, to a limited extent, the economic burden of EEE in horses in Florida was done in 1982 and 1983. This investigation was undertaken in response to a perceived increase in the incidence of EEE in general, as well as an increase in the incidence of EEE in vaccinated horses (Wilson et al., 1986). The results of a mail survey were used to estimate EEE prevalence, vaccination status, month and year of outbreak, and the geographical location of the outbreak. The state was divided into five geographical zones in accordance with state government administrative regions.

Contrary to the original goal of estimating prevalence, the prevalence of EEE in horses could not be determined because there was no valid census of the horse population in Florida in 1983. With regard to the incidence of EEE, incident horse cases were reported more frequently during June, July, and August (**Figure 5A, 5B**). The seasonal distribution of these cases was the

same as the incident cases from an investigation of EEE in horses in Florida during the years 1955 to 1974 in which approximately 66 percent of the clinically diagnosed cases, serologically confirmed cases, and virus isolations of EEE from horses were reported during May through August (Bigler et al., 1976). Veterinarians in Florida reported a higher incidence of EEE in specific geographic locations within their practices. These locations were in close proximity to bodies of water such as marshes and rivers (Wilson et al., 1986). On a larger geographical scale, the zone-specific proportional mortality data indicated that there were fewer cases in northwestern and southern Florida (Table 4 and Table 5). The proposed explanation for the smaller number of cases in southern Florida is based on the geographical distribution of *C. melanura*, the principal vector of EEEV during the enzootic bird cycle in Florida. Both *C. melanura* and EEEV have been recovered during all seasons in swamps that were located in central and northern Florida. However, *C. melanura* was recovered much less frequently in southern Florida.

California

An outbreak of EEE that involved a single incident case in a horse in California represents an atypical geographical distribution of EEE in the US (Franklin et al., 2002). The 14-month-old gelding was admitted to a veterinary referral hospital in Southern California in year 2000 because of sudden onset of quadraparesis. The gelding and 27 stable mates had been vaccinated with a multidose, multivalent vaccine containing formalin-inactivated EEEV as well as WEEV, influenza virus, and tetanus toxoid. The horses had been vaccinated seven days prior to the onset of illness.

Laboratory specimens were submitted to the National Veterinary Services Laboratories. Diagnostic tests for the equine encephalomyelitides included virus isolation and serology for WEEV, EEEV, VEEV, *Equid herpesvirus 1*, and West Nile virus. Virus preparations were identified as EEEV by a complement fixation test with reference antisera. The serum neutralizing antibody titer versus EEEV was greater than 100 with the plaque reduction neutralization test, but no titer was detectable against either WEEV or VEEV. The serum also was positive in an immunoglobulin (Ig) M-capture enzyme-linked immunosorbent assay for EEEV with a titer greater than 1000. Isolation of EEEV was successful at two other diagnostic laboratories.

EEEV in the US is diagnosed typically in states that are located from New England to Florida and along the Gulf Coast. EEEV is diagnosed less frequently in upper-midwestern states, including Michigan and Wisconsin. Four hypotheses were investigated to explain the occurrence

Figure 5A, 5B. Temporal distribution of eastern equine encephalomyelitis in horses in Florida in 1982 and 1983. (From Wilson et al., 1986 . With permission).

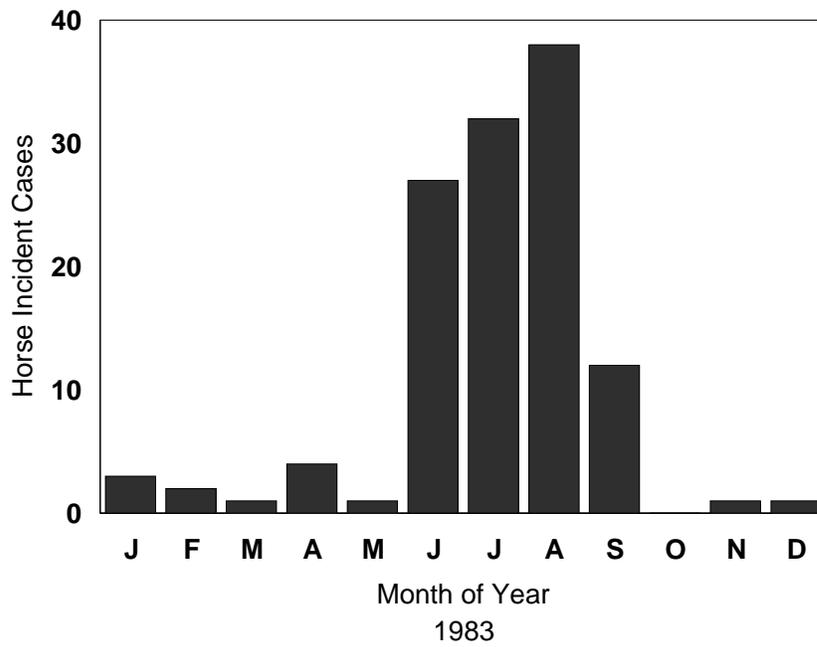
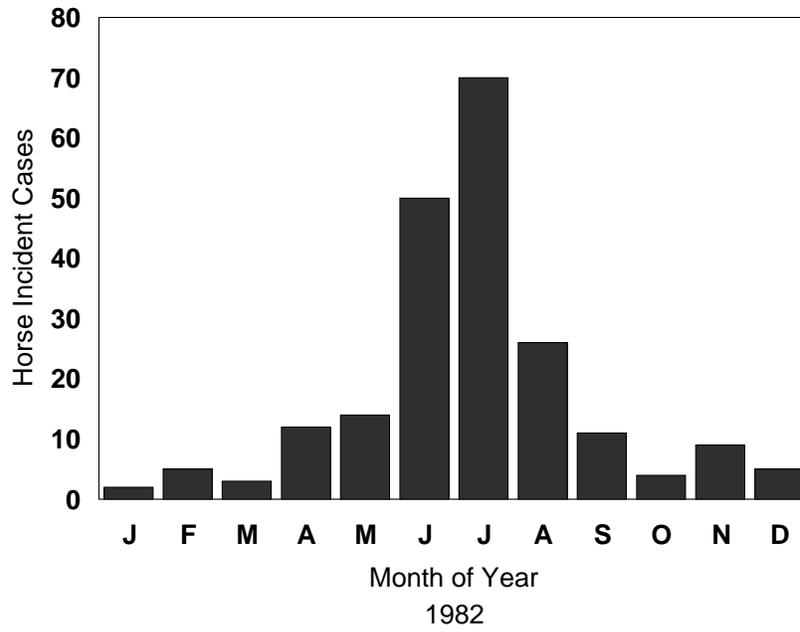


Table 4. Comparison of eastern equine encephalomyelitis (EEE) horse incident cases among five geographic zones in Florida during years 1982 and 1983, as reported from mail survey responses received from 126 private, equine or mixed-practice veterinarians.*

Year	Incident Cases Per Geographic Zone					Total
	Zone 1 (Northwest)	Zone 2 (North)	Zone 3 (East Central)	Zone 4 (West Central)	Zone 5 (South)	
1982	13	103	99	44	16	275
1983	20	143	71	39	5	278
Total	33	246	170	83	21	553

*The study population equaled 170 veterinarians. [Source: Wilson et al.,1986. Revised, with permission.](#)

Table 5. Comparison of eastern equine encephalomyelitis (EEE) horse incident cases among five geographic zones in Florida during years 1982 and 1983, as reported by the Florida Animal Disease Diagnostic Laboratory.

Year	Incident Cases Per Geographic Zone					Total
	Zone 1 (Northwest)	Zone 2 (North)	Zone 3 (East Central)	Zone 4 (West Central)	Zone 5 (South)	
1982	37	44	68	30	23	202
1983	12	40	38	22	5	117
Total	49	84	106	52	28	319

[Source: Wilson et al.,1986. Revised, with permission.](#)

of EEEV outside of its typical geographical range. The four hypotheses were: (1) imported infection from an EEEV-endemic region, 2) autochthonous transmission by locally infected mosquitoes, (3) intentional inoculation of the horse with live EEEV or intentional contamination of the vaccine, and (4) incomplete inactivation of EEEV in a commercially inactivated viral vaccine. There was no evidence that the outbreak was due to importation from an EEEV-endemic region. Autochthonous transmission refers to origination of the infection in the same region in which it was diagnosed. There was no evidence of autochthonous transmission of EEEV by local mosquito populations, based on surveillance in mosquitoes, sentinel chickens, wild birds, horses, and humans from the region. A serosurvey of 10 randomly selected stable mates of the affected horse was done to test for EEEV antibodies. The laboratory findings were compatible with recent vaccination or with the presence of maternal antibodies in the younger horses. There was no evidence of intentional contamination of the vaccine, or intentional inoculation of the incident case; nor was there an apparent motive for such actions. Attempts to isolate EEEV from vials of vaccine that had been stored at the farm were not successful. Also, attempts to isolate EEEV from stored vaccine that was derived from the same lot as the vaccine stored at the farm also were not successful. While the evidence was not convincing that the vaccine was the source of exposure for the incident case, the vaccine was not completely eliminated as the source (Franklin et al., 2002).

Risk Factors Associated with EEE

Horses

A retrospective case-control study of EEE in horses was undertaken to identify risk factors during an epizootic in Michigan in 1991 (Ross and Kaneene, 1995). The goal was to identify specific environmental and management risk factors that may have been associated with the occurrence of EEE in these horses. There were reports of 55 equids that were affected by EEE during 1991. The case group included equine herds in which there were positive or suspect EEE-affected equids. The control group included equine herds in which there were no reported EEE-affected equids during 1991. The hypothesized risk factors for EEE were assigned to one of two categories: (1) environment and (2) management (**Table 6**). The primary focus of questions about the environmental risk factors was the geographic setting of the operation, including types of land-cover on the operation proper, as well as the area that immediately surrounded the operation. The main focus of questions about the management risk factors was operation type, herd size, vaccination practices, insect repellent use, and stabling methods. Multiple logistic regression analysis was used to calculate odds ratios (OR) for management and environmental risk factors at the herd level.

Annual vaccination against EEE (OR = 0.14; $p < 0.003$) and the use of insect repellent methods (OR = 0.04; $p = 0.02$) were associated with decreased risk of EEE in Michigan equine herds. When wooded land (OR = 3.70; $p = 0.03$) and swamp land (OR = 2.38; $p = 0.14$) were found on the operation, the risk of EEE was increased (**Table 7**).

Table 6. Hypothesized management and environmental risk factors potentially associated with eastern equine encephalomyelitis (EEE)-affected equids during an epizootic of EEE in Michigan in 1991.

Category	Hypothesized Risk Factor
<i>Management</i>	Operation type
	Herd size
	Vaccination protocol
	Vaccinator
	Vaccine supply
	Vaccine storage
	Insect repellent methods
	Lighted housing at night
	Access to shelter
	<i>Environment</i>
Open land on the operation	
Wooded land on the operation	
Swamp land on the operation	
Crop land on the operation	
Pasture land on the operation	
Source: Ross and Kaneene, 1995. Revised, with permission.	

Humans

Residing in rural environments and residing within five miles of freshwater swamps where the endemic bridge vector *C. melanura* breeds are two risk factors for eastern encephalitis in humans (Morris, 1994). There is some evidence that factors that promote humans to remain indoors at dusk (e.g., air conditioning in the residence, viewing television) are protective against WEE and SLE (Gahlinger et al., 1986). Whether these same factors, or analogous factors, are protective against EEE in domestic species such as horses has not been explored, but such protective factors would be plausible biologically.

Impact of Climate Change on EEE

There continues to be much debate about the potential impact of changes in the climate (e.g., global warming) on the future incidence of infectious diseases in humans and animals (Reeves et al., 1994). A change in the ambient temperature is just one factor that may increase or decrease the incidence of arbovirus transmission, depending on the direction of that change. *C. melanura* is the enzootic vector that is responsible for the spring-to-summer amplification of EEEV in a mosquito-bird-mosquito cycle of transmission (Freier, 1993). Although *C. melanura* prefers cooler ambient temperatures, the effect of a global increase in temperatures on the micro-climate and micro-habitat of this vector is uncertain. An increase in the ambient temperature will support the survival of mosquito larvae during the winter and will lead to a shorter extrinsic incubation period for infected female adult mosquitoes. This same

Table 7. Confirmed management and environmental risk factors associated with eastern equine

encephalomyelitis (EEE)-affected equids during an epizootic in Michigan in 1991.*	
Confirmed Risk Factor	Associated Impact on Risk of EEE Infection Φ
Bi-level Risk Factors	
Annual vaccination protocol	Reduced
Insect repellent methods	Reduced
Wooded land on the operation	Increased
Swamp land on the operation	Increased
Multi-level Risk Factors	
<i>Operation type</i>	
Boarding	Reference group
Breeding	Neutral
Training	Neutral
Work/draft	Neutral
Pleasure	Neutral
Other	Neutral
<i>Herd size</i>	
1 to 4 equids	Reference group
5 to 9 equids	Neutral
10 to 14 equids	Neutral
15 equids plus	Neutral
*Results from multivariable logistic regression analysis of data. Φ Based on the probability value from the Wald test statistic, odds ratios, and 95 percent confidence intervals for odds ratios. Source: Ross and Kaneene, 1995. Revised, with permission.	

increase in ambient temperature will decrease the survival of adult mosquitoes. The degradation of polar ice caps due to an increase in ambient temperature may raise the sea level and alter the breeding habitat for *C. melanura* in swamps. If the area of salt marshes were to expand, vectors such as *Aedes sollicitans* may become more abundant (Crans, 1977). A decrease in the ambient temperature may increase the density of passerine birds due to creation of cooler swamp-forests habitat that would be more attractive to passerine birds, and these birds may support conversion of EEE virus to a virulent phase.

Economic Burden of EEE

Animals

An investigation to generate rough estimates of the economic impact of EEE in horses in Florida during years 1982-1983 was undertaken in 1984 (Wilson et al., 1986). A questionnaire to solicit: (1) data about incidence of EEE, (2) recommendations for vaccination, and (3) costs of the EEE was mailed to 170 veterinary medicine practitioners who were engaged in equine or mixed-species practice in Florida. Questionnaires from 72 veterinary practices were completed,

and these 72 questionnaires represented the responses from 126 veterinarians. Those veterinarians who responded to the survey represented only 28 percent of the veterinarians in Florida who were engaged in mixed practice, or large animal practice, or equine-exclusive practice in 1983.

The percent case-fatality due to EEE was 75 to 100. Thirty-four percent of the veterinarians who had observed horses that survived EEE reported that the sequelae were severe. The sequelae included blindness, ataxia, sleepers, and mental retardation (**Figure 6**). The various interventions by those veterinarians who provided care for horses that survived EEE included supportive therapy, anti-inflammatories, antibiotics, etc. (**Table 8**). The estimated costs of morbidity, mortality, therapeutics, and prophylactics due to EEE in Florida by the practitioners who responded to the survey were \$1.6 million in 1982 and \$1.07 million in 1983 (**Table 9**). The number of cases of EEE reported by the veterinarians was substantially greater (73 percent greater) than the number of cases confirmed by the Florida Animal Disease Diagnostic Laboratory. One explanation for the disparity in the numbers is that only 50 percent of the responding veterinarians always sought laboratory confirmation of a clinical diagnosis of EEE. The disparity between the data from the practitioners and the Florida Animal Diagnostic Disease Laboratory data indicates that there may have been significant under-reporting of EEE cases to the laboratory. The actual costs of EEE in Florida in 1983 probably were much greater than indicated by these data, since the data were from a relatively small percentage of the total number of practitioners who were more likely to diagnose EEE cases.

Humans

An estimate of the economic burden of a residual case of EEE was investigated in humans ([Villari et al., 1995](#)). A laboratory diagnosis of EEE was confirmed in thirteen (n = 13) human patients during the 1980s, nine of whom survived the disease and were included in the “subject population”. The investigators were able to physically locate seven of the nine patients, and six of the seven agreed to participate in the economic analysis. Either proxies for each patient (e.g. the parents), or the patients themselves, were interviewed to collect data about three different costs: **(1)** direct medical costs (e.g., diagnosis, therapy), **(2)** direct nonmedical costs (e.g. re-design of the home, special education), and **(3)** indirect costs (e.g. decreased wages, bypassed job opportunities). For a detailed discussion of the methods used to compute the costs, refer to Villari et al., 1995. Four of the six patients were children; the remaining two patients were adults. Three of the six patients, two adults and one child, suffered from a transient episode of EEE. The three other patients, of which all were children, were victims of severe residual sequelae.

The costs were reported as year 1990 dollars. All costs associated with a transient episode of EEE were incurred within one month following the onset of symptoms. Of the total costs incurred during a transient episode of EEE, 92 percent were for direct medical services. The average value of the total costs per patient was \$21,051. All patients with severe residual



Figure 6. Clinical signs of eastern equine encephalomyelitis (EEE) in a horse during the EEE epizootic in Wisconsin, USA, 2001. Note the sleepy facial expression, the lax muzzle, and the abrasions between the eye and the base of the ear.

sequelae were children. The residual sequelae included impaired gross motor control, fine motor control, attention control, mobility, and behavioral and emotional disturbances. None of the children were expected to attain “productive employment” during their life span, due to the severity of the residual sequelae. The costs of severe residual sequelae accumulate throughout the patient’s life. The annual costs varied greatly during the first two years of the illness (maximum \$425,000) and reached a plateau of \$100,000 per year during three through six years of age. The average of the six-year total costs for a residual case of EEE was \$0.85 million, and the lifetime total costs for a residual case was an average of \$2.95 million. Although several limitations of this economic analysis were disclosed, it was believed that the analysis provided reasonably accurate estimates of the cost of a residual case of EEE in humans.

Table 8. Interventions used by veterinarians who had observed domesticated horses in Florida that survived eastern equine encephalomyelitis during years 1982 and 1983.

Intervention	Percent of Veterinarians
Supportive or symptomatic therapy	47
Corticosteroids	23
Antibiotics	22
Fluids	19
Tranquilizers, sedatives	12
Anti-inflammatory drugs	8
Dimethylsulfoxide	6
Antipyretics	3
Plasma	2
Euthanasia	22

Source: Wilson et al., 1986.

Table 9. Estimated cost of clinically apparent cases of eastern equine encephalomyelitis in domesticated horses in Florida during years 1982 and 1983. †

Category	1982	1983	Total Cost
Morbidity and Mortality*	\$587,700	\$496,750	\$1,084,450
Therapeutics and Prophylactics	\$580,763	\$580,763	\$1,161,526
Total Cost*	\$1,168,463	\$1,077,513	\$2,245,976

†Costs are reported in U.S. dollars, year 1983. *Costs represent costs only for the 126 practitioners who responded to the survey. Source: Wilson et al., 1986. Revised, with permission.

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