Chapter 20

Eastern Equine Encephalomyelitis

Synonyms

EEE, eastern encephalitis, EE, eastern sleeping sickness of horses

Cause

Eastern equine encephalitis (EEE) is caused by infection with an RNA virus classified in the family Togaviridae. The virus is also referred to as an "arbovirus" because virus replication takes place within mosquitoes that then transmit the disease agent to vertebrate hosts such as birds and mammals, including humans. The term arbovirus is shortened nomenclature for arthropod (insect) borne (transmitted) viruses. *Culiseta melanura* is the most important mosquito vector; it silently (no disease) transmits and maintains the virus among birds. However, several other mosquito species can transmit this virus, including the introduced Asian tiger mosquito. New hosts become infected when they enter this endemic natural cycle and are fed upon by an infected mosquito. Therefore, the presence of mosquito habitat, the feeding habits of different mosquito species, and the activity patterns of vertebrate hosts are among the important factors for disease transmission.

Distribution

This disease is primarily found in eastern North America especially along the Atlantic and Gulf Coasts, and the disease range extends into Central and South America. The causative virus has been isolated from eastern Canada to Argentina and Peru, and it is maintained in a mosquito-wild bird cycle as an endemic (enzootic) focus of infection in nature that is usually associated with freshwater marshes. Wild bird die-offs from EEE have been limited to captive-rearing situations. Die-offs have occurred in pheasants in coastal States from New Hampshire to Texas, where they have been raised, in chukar partridge and whooping cranes in Maryland, and in emus and ostriches in Louisiana, Georgia, Florida, and Texas.

Species Affected

EEE virus produces inapparent or subclinical infections in a wide range of wild birds (Fig. 20.1). However, EEE virus has caused mortality in glossy ibis and in several bird species that are exotic to the United States, including pigeon, house sparrow, pheasants, chukar partridge, white Peking ducklings, and emu. The infection rate in penned emus in the United States has reached 65 percent with a case mortality rate of 80 percent. In the past, extensive losses have
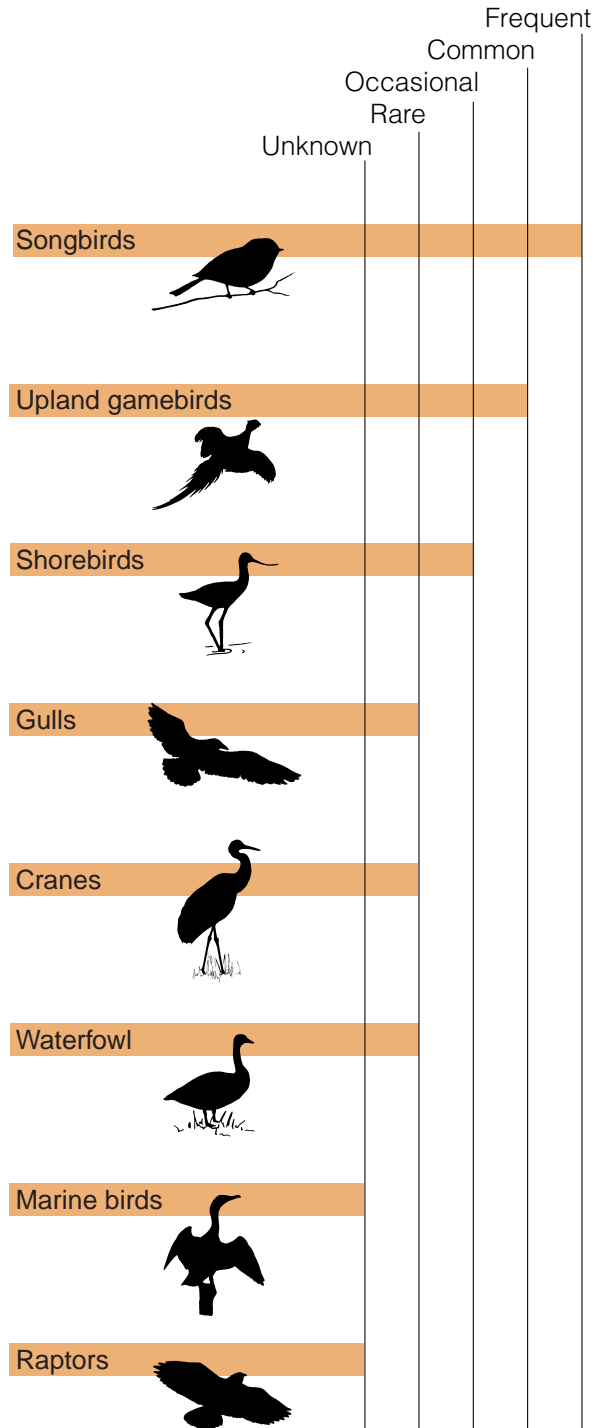

Figure 20.1 *Relative frequency of EEE virus isolation or presence of antibodies in birds.*
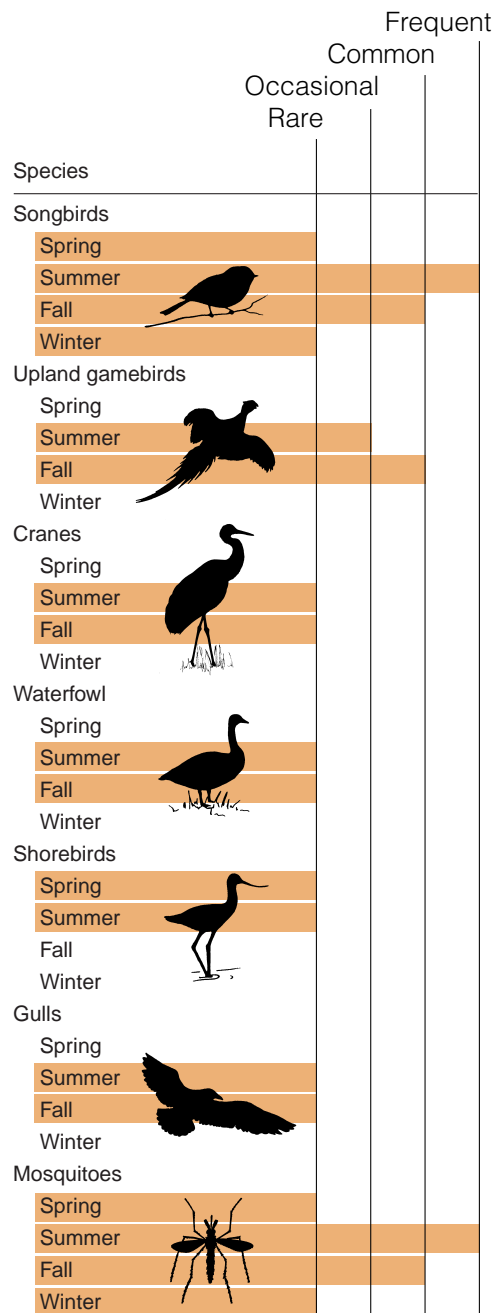
Figure 20.2 Seasonality of virus isolation from birds and mosquitoes.

occurred in ring-necked pheasant being reared in captivity for sporting purposes, including one outbreak in a South Dakota pheasant farm. Large-scale mortalities in captive pheasants are perpetuated by bird-to-bird disease spread through pecking and cannibalism after EEE has been introduced by mosquitoes, usually of the genus *Culiseta*. Outbreaks have not been reported during recent years.

In September and November of 1984, EEE virus was associated with the deaths of 7 of 39 captive whooping cranes at the Patuxent Wildlife Research Center in Laurel, Maryland. Sandhill cranes coexisting with the whooping cranes did not become clinically ill or die.

Passerines (perching songbirds), some small rodents, and bats are highly susceptible to infection and they often die from experimental infections. Horses are highly susceptible and they often die from natural infections.

Seasonality

EEE is associated with the early summer appearance of *C. melanura* mosquito populations (Fig. 20.2). Nestling birds, such as passerines and other perching birds, are the amplification hosts for the virus, producing high concentrations of virus in their blood or viremia following mosquito infection. New populations of emerging mosquitoes become infected when they feed on the viremic birds. *C. melanura* and other species of infected mosquitoes can transmit the virus to other species of birds susceptible to disease (Fig. 20.3).

The summer-fall transmission cycle is followed by little virus transmission during the winter and spring months. The overwintering mechanism for virus survival is not known. Infected mosquitoes, other insects, cold-blooded vertebrate species, or low levels of virus transmission by mosquitoes are among current theories for virus cycle maintenance in milder climates. It is also believed that bird migration spreads the virus to higher latitudes in the spring.

Field Signs

Clinical signs do not develop in most native species of wild birds infected with EEE virus. Clinical signs for non-indigenous birds (including pheasants) include depression, tremors, paralysis of the legs, unnatural drowsiness, profuse diarrhea, voice changes, ataxia or loss of muscle coordination, and involuntary circular movements (Fig. 20.4). Some of the EEE-infected whooping cranes became lethargic and incoordinated or ataxic, with partial paralysis or paresis of the legs and neck 3–8 hours prior to death; other cranes did not develop clinical signs before they died.

Gross Lesions

Gross lesions in whooping cranes included fluid accumulation in the abdominal cavity or ascites, intestinal mucosal discoloration, fat depletion, enlarged liver or hepatomegaly, enlarged spleen or splenomegaly, and visceral gout (Fig. 20.5).

Diagnosis

Because of human health hazards, field personnel should not dissect birds suspected of having died from EEE. Whole carcasses should be submitted to diagnostic laboratories capable of safely handling such specimens. EEE can be diag-
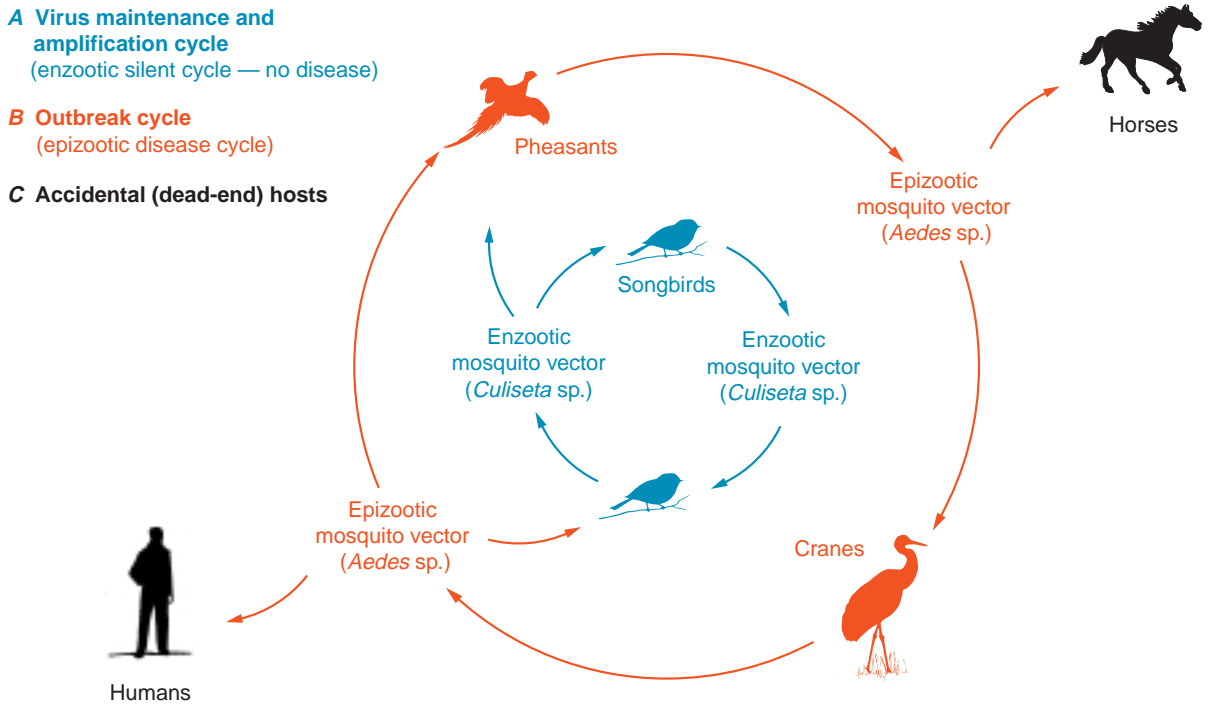
A **Virus maintenance and amplification cycle**
(enzootic silent cycle — no disease)

B **Outbreak cycle**
(epizootic disease cycle)

C **Accidental (dead-end) hosts**

Pheasants

Horses

Epizootic mosquito vector (*Aedes* sp.)

Songbirds

Enzootic mosquito vector (*Culiseta* sp.)

Enzootic mosquito vector (*Culiseta* sp.)

Epizootic mosquito vector (*Aedes* sp.)

Cranes

Humans

Figure 20.3 *Transmission of eastern equine encephalomyelitis. **(A)** Virus circulates in songbird populations by being transmitted by mosquitoes. Those birds are susceptible to infections, but they do not become clinically ill or die. **(B)** The outbreak cycle is started either when an infected mosquito from the enzootic cycle feeds on highly susceptible birds such as pheasants or cranes, or when another species of mosquito, that primarily feeds on these same birds, becomes infected after feeding on songbirds in the enzootic cycle and transmits the virus. The epizootic cycle is maintained by the second mosquito species. **(C)** The broader host feeding range of the second mosquito results in exposure of horses and humans. No disease cycle is maintained between these species by mosquitoes.*

Photo by Milton Friend

Figure 20.4 *A hen pheasant with EEE exhibiting neurologic signs.*
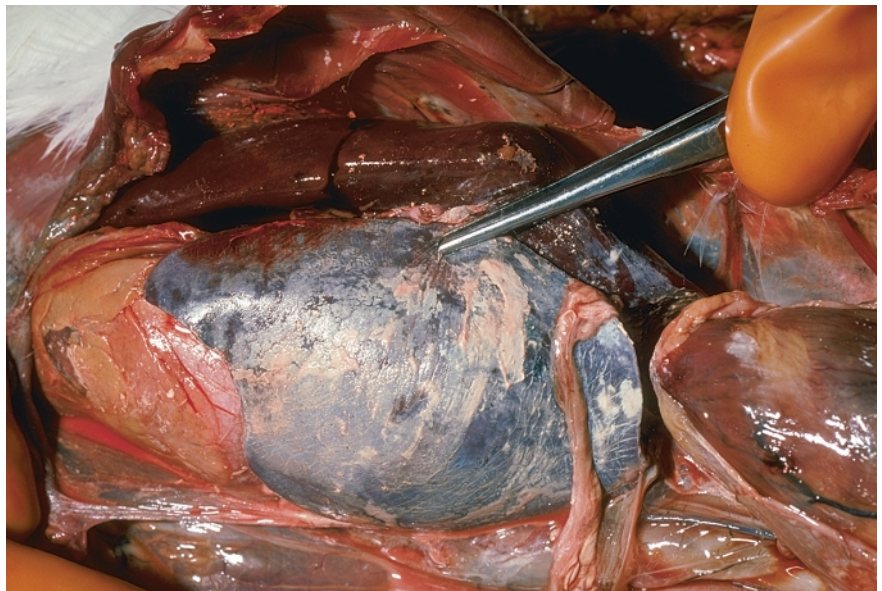

Photo by James Runningen

Figure 20.5 *The white, grainy material on the liver of this whooping crane is evidence of visceral gout.*

nosed by virus isolation from infected whole blood or brain and other tissues from dead birds. Diagnosis of virus activity can be made from surviving birds because they will have virus neutralizing antibodies in their blood serum. The rise and fall in serum antibodies that occurs after virus exposure can be used to assess infection rates and the relative timing of exposure before antibody levels reach nondetectible levels. Most native birds do not suffer clinical infections.

Control

There are two approaches to protecting susceptible animals from infection from vector-borne diseases. The first approach includes separating mosquitoes from animals at risk. This requires eliminating mosquito breeding and resting sites in an endemic area or protecting animals from mosquito contact by maintaining them in an insect-proof enclosure. In the second approach, vaccination is used to render the animal immune. A killed-virus vaccine was used in captive whooping cranes to protect the rest of the breeding flock following the 1984 outbreak. Vaccination has also been used to protect whooping cranes released into an area where EEE is prevalent in mosquito populations. Field data suggest that immunity in those cranes is being boosted by natural infections after their release.

Human Health Considerations

Humans are susceptible to EEE and human cases typically arise after the disease has appeared in horses. EEE is a significant disease in humans with a case fatality rate of between 30–70 percent and it often causes severe permanent neurological disorders among survivors. Aerosol infection is possible but rare. Laboratory personnel have been infected with this virus. Human pre-exposure vaccination is recommended for people who may handle infected tissues.

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Supplementary Reading

Dein, J.F., Carpenter, J.W., Clark, G.G., Montali, R.J., Crabbs, C.L., Tsai, T. F., and Docherty, D.E., 1986, Mortality of captive whooping cranes caused by eastern equine encephalitis virus: The Journal of American Veterinary Medical Association, v. 189, p. 1,006–1,010.

Gibbs, E.P.J., and Tsai, T.F. 1994, Eastern encephalitis, *in* Beran, G.W., and Steele, J.H., eds., Handbook of zoonoses (2d ed.), Section B: Viral: Boca Raton, Fla., CRC Press, p. 11–24.

Kissling, R.E., Chamberlain, R.W., Sikes, R.K., and Eidson, M.E., 1954, Studies on the North American arthropod-borne encephalitides III. eastern equine encephalitis in wild birds: American Journal of Hygiene, v. 60, p. 251–265.