Equine Viral Arteritis

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KEYWORDS

• Equine viral arteritis • Equine arteritis virus • EAV • EVA

KEY POINTS

- Equine arteritis virus (EAV) is the causative agent of equine viral arteritis (EVA).
- EVA is a respiratory and reproductive disease of the horse that occurs throughout the world.
- Most EAV infections are inapparent (or subclinical); however, acutely infected animals may develop a wide range of clinical signs.
- Virus causes abortion in pregnant mares and a high proportion of acutely infected stallions become persistently infected and shed the virus in semen.
- EAV infection can cause a severe fulminating interstitial pneumonia and a progressive pneumoenteric syndrome in young foals.

VIRUS

EAV was first isolated from the lung of an aborted fetus after an extensive outbreak of respiratory disease and abortion on a Standardbred breeding farm near Bucyrus, Ohio, in 1953.[1,2] After isolation of the causative virus (EAV) and description of characteristic vascular lesions, EVA was identified as an etiologically distinct disease of the horse.[1] EAV is a small enveloped, positive-sense, single-stranded RNA virus that is the prototype virus in the family *Arteriviridae* (genus *Arterivirus*), order *Nidovirales*, a taxonomic grouping that includes porcine reproductive and respiratory syndrome virus, simian hemorrhagic fever virus, lactate dehydrogenase-elevating virus of mice, and recently identified wobbly possum disease virus of free-ranging Australian brushtail possums (*Trichosurus vulpecula*) in New Zealand.[3,4]

The molecular properties of EAV were reviewed by Balasuriya and colleagues[5,6] (2013 and 2014). Briefly, the EAV genome length varies between 12,704 and 12,731 base pair among different viral strains and includes a 5′ leader sequence (224 nucleotides) and at least 10 open reading frames (ORFs).[5] The 2 most 5′-proximal ORFs (1a and 1b) occupy approximately three-fourths of the genome and encode

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2 replicase polyproteins (pp1a and pp1ab). These precursor proteins are extensively processed after translation into at least 13 nonstructural proteins (nsp1–12, including nsp7 α/β) by 3 viral proteases (nsp1, 2, and 4).[5] The structural proteins of EAV include seven envelope proteins (E, GP2, GP3, GP4, ORF5a protein, GP5, and M [encoded by ORFs 2a, 2b, 3-4, 5a, 5b, and 6]) and a nucleocapsid protein (N [encoded by ORF7]). All the structural protein encoding ORFs are located at the 3 proximal quarter of the genome (**Fig. 1**).[5,7] Three of the minor envelope glycoproteins (GP2, GP3, and GP4) form a heterotrimer in the EAV particle, and the M (nonglycosylated) and GP5 (glycosylated) proteins form a disulfide-linked heterodimer.[5,7]

Organ samples and tissue culture fluid containing EAV can be stored frozen (−70°C to −80°C) for decades without significant loss of virus infectivity. The virus also survives in cryopreserved semen samples and embryos for many years. EAV remain infectious 75 days at 4°C, between 2 to 3 days at 37°C, and 20 to 30 minutes at 56°C. EAV is readily inactivated by lipid solvents (ether and chloroform) and by common disinfectants and detergents.

CLINICAL SIGNS

The clinical signs displayed by EAV-infected horses depend on a variety of factors, including the genetics, age, and physical condition of the horses; challenge dose; route of infection; strain of virus; and environmental conditions.[8–11] Although there is only 1 known serotype of EAV, there is significant variation in virulence phenotype between EAV field strains.[5,11] Based on the clinical severity of the disease during natural outbreaks, EAV field strains could be segregated into viruses that cause moderate to severe disease (eg, EAV KY84, EAV AZ87, EAV IL93, and EAV PA96), mild disease (eg, EAV SWZ 64, EAV AUT68, EAV IL94, and EAV CA97) and asymptomatic infection (EAV KY63, EAV PA76, EAV KY77, and EAV CA95; Moore and colleagues,[12] 2002). Similarly, laboratory and vaccine strains differ significantly in their virulence phenotype from the highly virulent, horse-adapted Bucyrus strain (virulent Bucyrus

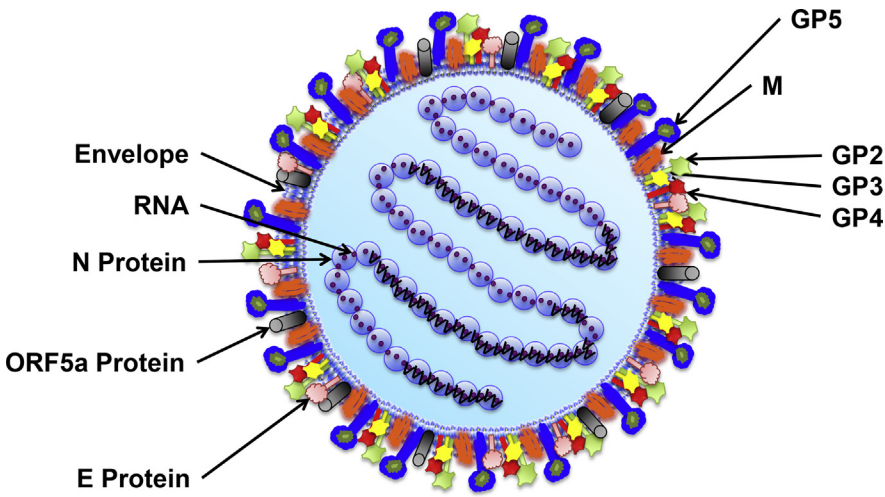

Fig. 1. Virion architecture. EAV particle consists of a nucleocapsid (N) and 7 envelope proteins, which include 2 major envelope proteins (GP5 and M form a dimer), 3 minor envelope glycoproteins (GP2, GP3, and GP4 form a trimer), and 2 other minor envelope proteins (E and ORF5a protein).