Reprinted from the JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, Vol. 187, No. 11, Pages 1248-1249.

Equine herpesvirus type 1 abortion in an onager and suspected herpesvirus myelitis in a zebra

—*Richard J. Montali, DVM, George P. Allen, PhD, John T. Bryans, PhD, Lyndsay G. Phillips, DVM, and Mitchell Bush, DVM, Departments of Pathology and Animal Health, National Zoological Park, Smithsonian Institution, Washington, DC 20008, and the Department of Veterinary Science, University of Kentucky, Lexington, KY 40506*

IN FEBRUARY 1984 at the National Zoological Park (NZP) in Washington, DC, a male onager fetus (*Equus hemionus onager*) was aborted after approximately 10 months of gestation. At necropsy, sanguinous cavitary effusions, pleuropulmonary petechiae, splenomegaly, and white spots on the liver were seen. Histologic examination of the tissues indicated necrosis of the liver (Fig 1) and splenic white pulp (Fig 2), with intranuclear inclusion bodies in hepatocytes (Fig 1, inset) and in splenic lymphocytes. The inclusion bodies also were in cells of the bronchioles, thymic medulla, and adrenal cortex, but not in the placenta. The onager dam of the fetus had not been exposed directly to domestic horses nor had she been vaccinated against equine herpesvirus type 1 (EHV-1). She originally came from the west coast and previously had 4 healthy foals while at the NZP Conservation and Research Center (CRC), in Front Royal, Va. The onager dam, the sire of the fetus, and 2 herdmates had been brought to NZP grounds from CRC 4 months before the abortion.

Cytopathic virus isolates recovered from the lung, liver, and spleen of the aborted onager fetus were identified as EHV-1 on the basis of virus neutralization by rabbit antiserum to EHV-1 stock virus.[1] The isolates were serotyped as the abortogenic subtype-1 on the basis of their antigenic reactivity pattern with a panel of EHV-1 subtype-specific monoclonal antibodies.[2] A subtype-specific monoclonal antibody (16H9) that reacted with 10 epizootiologically nonrelated equine isolates of EHV-1 did not react with the virus isolated from the onager. The endonuclease cleavage patterns indicated that the virus isolated from the onager fetus had a unique restriction profile that was not seen in the DNA of >200 epizootiologically nonrelated horse isolates of EHV-1 (Fig 3).[3]

One week after the onager abortion, a 9-month-old male zebra (*Equus burchelli*), located in a pen adjacent to the onagers, developed weakness, posterior ataxia, and partial rectal prolapse. The zebra was leukopenic and developed moderate dehydration. He was treated with corticosteroids, antibiotics, and fluids and the rectal prolapse was repaired surgically. After one week, the neurologic deficit improved, but the zebra developed cystitis associated with beta-hemolytic streptococcus, which responded to trimethoprim and ampicillin therapy. The zebra was clinically healthy one month after the onset of illness. A tentative diagnosis of herpesvirus myelitis was made on the basis of the clinical features of the zebra's illness and the zebra's juxtaposition to the onager mare that aborted.

The onager dam and sire of the aborted fetus, and the zebra with

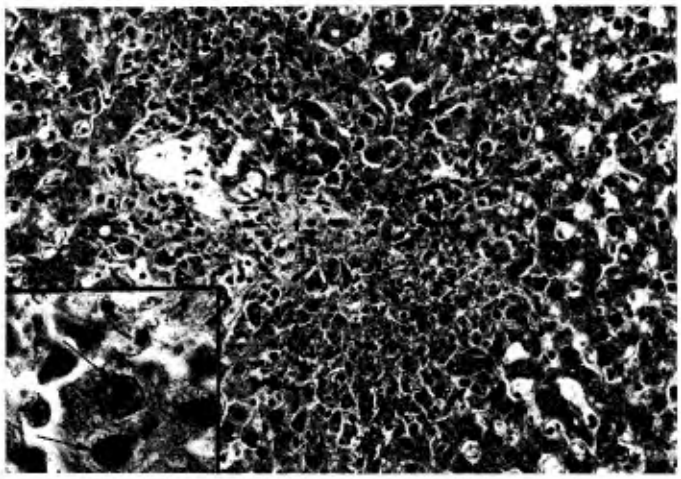

Fig 1—Photomicrograph of the liver from an aborted onager fetus, indicating a focus of hepatocellular necrosis. H&E stain; × 210. Inset—Hepatocytes with intranuclear herpetic inclusion bodies. H&E stain; × 680.

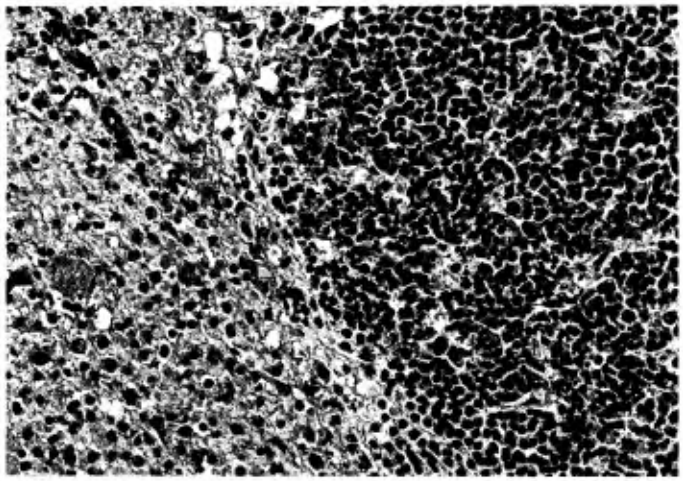

Fig 2—Photomicrograph of the spleen from an aborted onager fetus, indicating congestion of the red pulp (left) and lymphoid follicular necrosis (right). H&E stain; × 210.

Presented at the annual meeting, American Association of Zoo Veterinarians, Louisville, Ky, October 1984.

suspected herpetic myelitis had antibody titers to EHV-1 (detected by ELISA) >10,480, which is indicative of active infection in domestic horses.[2]

Enzyme-linked immunosorbent assays performed on sera stored from the equid collection at the

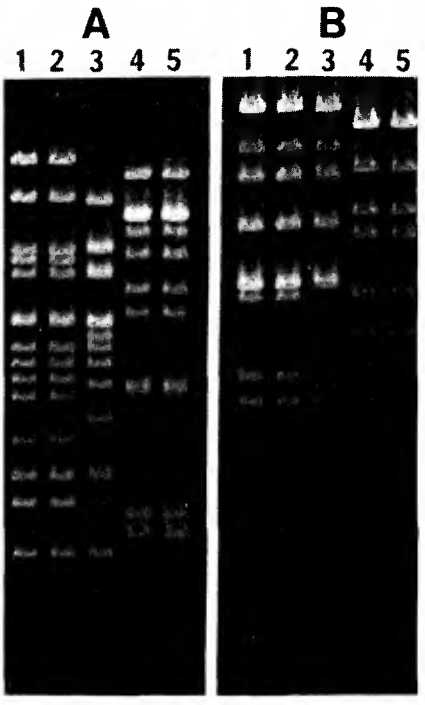

Fig 3—Agarose gel electrophoresis of the BamHI (A) or EcoRI (B) restriction endonuclease fragments of the DNA from isolates of equine herpesvirus type 1 (EHV-1). No. 1 and 2 are reference strains of subtype-1 EHV-1; 3 is the onager fetal virus isolate; and 4 and 5 are reference strains of subtype-2 EHV-1.

NZP and the CRC since 1982 indicated prior exposure to EHV-1 in 9 of 10 zebras, 3 of 9 onagers, and 1 of 6 Przewalski horses *(Equus przewalskii)* evaluated. Titers were 640 to 10,240 in the zebras, and 2,560 to 5,120 in the onagers.

Before the abortion of the onager and the onset of ataxia in the zebra, EHV-1 had not been considered a disease threat in the equid collection. Upper respiratory tract illnesses resembling rhinopneumonitis of horses or illnesses attributable to herpesvirus infection had not been observed in the equids. The gross and microscopic findings, herpetic inclusions in the onager fetus, and isolation of an abortogenic strain of EHV-1 from the fetal tissues indicated virus-induced abortion in the onager, typical of the virus abortion seen in domestic horses associated with EHV-1. The diagnosis of herpesvirus CNS disease in the zebra was made on the basis of clinical signs of myelitis and serologic evidence of active herpesvirus infection during the illness, followed by a reversion of the convalescent titer to 2,560 nine months after the onset of illness. Also, virus isolates from domestic horses with EHV-1-related CNS disease have been the subtype-1 abortogenic strain,[2] as was isolated from the onager fetus.

The DNA fingerprint analysis of the virus isolate from the onager indicated that the virus was unique from the strains of subtype-1 isolated from domestic horses. Virus abortion only has been reported in one other nondomesticated horse, a Konik mare (*Equus caballus* syn *ferus*) from a zoo in Germany[4]; however, this species is regarded as a feral horse that is not truly representative of an exotic equid. Herpesviruses closely antigenically related to EHV-1 have been isolated from fallow deer[5]; therefore, examination of the white-tailed deer population that was in close proximity to the equids at CRC may have helped determine the nature and source of the EHV-1 titers in the equids at CRC.

One of the older zebra stallions at CRC had evidence of exposure to EHV-1 early in 1982. The recent serologic recrudescence of EHV-1 in this zebra in July 1984 indicated that he may have been a potential source of the infection in the collection. Also, this zebra was the sire of the zebra with the suspected herpesvirus myelitis.

More information concerning EHV-1 in exotic equine species is necessary before recommendations for control measures can be made; however, results of the present study indicate that different equine species probably should be kept separated from each other, particularly when breeding programs are anticipated.

1. Bryans JT. Immunity to disease caused by equine herpes virus-1. *J Am Vet Med Assoc* 1969;155:294–300.

2. Allen GP, Bryans JT. Molecular epizootiology, pathogenesis, and prophylaxis of equine herpesvirus-1 infections. *Prog Vet Microbiol* 1985; in press.

3. Allen GP, Yeargan MR, Turtinen LW, et al. Molecular epizootiologic studies of equine herpes virus-1 infections by restriction endonuclease fingerprinting of viral DNA. *Am J Vet Res* 1983;44:263–271.

4. Kast VA, Kruedener RV. Virus abortion of a mare in a zoo, in *Proceedings*. 12th Int Symp Dis Zoo Anim, Budapest, 1970; 91–93.

5. Kinyili JH, Thorsen J. Antigenic comparisons between herpes viruses isolated from fallow deer in Alberta and the viruses of infectious bovine rhinotracheitis, equine rhinopneumonitis and DN-599, a non-IBR bovine herpes virus. *J Wildl Dis* 1979; 15:339–341.