Vesicular Stomatitis virus (VSV) New Jersey outbreak in Southern California

Vesicular Stomatitis virus (VSV) New Jersey strain infection was confirmed by the National Veterinary Services Laboratory (reference laboratory) on May 17th in a horse from San Diego county. Since then, 24 new VSV affected equine premises have been identified in San Diego and Riverside counties. The virus causes vesicles and erosions mostly in the mouth, but it can also affect the udder, feet (coronary bands), and ears. Other livestock species including ruminants and swine, and several wildlife species such as white-tailed deer and raccoons, are susceptible to VSV. The disease may be transmitted to humans so the use of personal protective equipment is recommended when dealing with animals suspected to be infected by VSV. The virus is transmitted by insects, especially black flies, sand flies, and Culicoides midges. Eliminating breeding grounds for insects by daily removal of manure, elimination of standing water and the use of fly spray, are good preventative practices. Please contact your local California Department of Food and Agriculture office or veterinarian if you notice any suspicious lesions.

Tulare Lab update

We remain closed for receipt of live animals, carcasses and fresh tissues until we finish renovating the old necropsy facility within our current home at the Veterinary Medicine Teaching and Research Center (VMTRC). We can receive swabs, culture plates, blood and serum samples, fecal samples, formalin-fixed tissues and some other specimens at our VMTRC location for Histopathology, Bacteriology, Biotechnology, Immunology and Parasitology testing either here in Tulare or at the other CAHFS branches. Please call the laboratory with any questions about current testing capabilities [(559) 688-7543]. Our field necropsy service will continue until the weather stops cooperating, with our preference to provide necropsies in the morning while temperatures remain safe. To schedule a field necropsy, call the lab during normal business hours. We do not have a firm date for resumption of necropsy service at our VMTRC location, but we hope to be up and running sometime over the summer.

Holiday Schedule:

CAHFS laboratories will be closed on June 19th in observance of Juneteenth
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Equine

Clostridial hepatitis was diagnosed in a 13-year-old Quarter Horse gelding that was euthanized after a 5-day history of colic, fever and abdominal distension. On necropsy, the carcass was severely icteric and there was tricavitary serosanguineous effusion in the body cavities. A dense fibrin mat pooled on the cranial abdomen, mostly over the diaphragmatic aspect of the liver, which had a focal large brown/black depressed lesion with gas bubbles. Histologically, there was necrosis, thrombosis, and gram-positive bacilli with subterminal spores in sections of this liver lesion. *Clostridium novyi* was detected by fluorescent antibody technique and immunohistochemistry. There are two clostridial diseases that may be associated with hepatitis and both may affect horses with a similar clinicopathologic presentation: Infectious necrotic hepatitis, caused by *Clostridium novyi* type B, and bacillary hemoglobinuria, which is caused by *Clostridium haemolyticum* (also known as *C. novyi* type D). This case was confirmed to be caused by *C. haemolyticum* by PCR.

Liver of a horse with clostridial hepatitis showing multiple gram positive rods (*Clostridium haemolyticum*) responsible for this disease

Small Ruminant

Caseous lymphadenitis was the cause of progressive weight loss of a 5-year-old Hampshire and Suffolk cross sheep. Despite eating normally, the animal became emaciated and had to be euthanized. Multiple abscesses were detected in the liver, mesentery and thyroid glands. *Corynebacterium pseudotuberculosis* was isolated and an antibody titer of 1:128 (compatible with active infection) for this bacterium was detected in serum. Caseous lymphadenitis may present in two different forms: external, which affects peripheral lymph nodes; and internal, which affects visceral lymph nodes and parenchymal organs. The latter is considered among the diseases contributing to the so-called “thin ewe syndrome”.

Chlamydia and Toxoplasma abortions and stillbirths were diagnosed in a group of 180 goats over a 5-week period. Placenta submitted from a 1.5-year-old doe had placentitis caused by *Chlamydia* spp. based on PCR, while 3 stillborn fetuses submitted later were positive for *Toxoplasma* spp. antibodies in fetal fluids; *Toxoplasma* spp. was detected by immunohistochemistry in the brain of one of 2 fetuses that had encephalitis. No placentas were received on the second submission so a concurrent *Chlamydia* spp. infection could not be ruled out. Marked copper (3ppm, normal 25-100ppm in liver) and selenium deficiency (0.14ppm, normal 0.25-1.5ppm in liver) was also detected in one of the submitted fetuses.

*Listeria monocytogenes* encephalitis was the cause of neurologic signs in two goats and one ewe from 3 separate premises in one week. A 4-year-old Dorper ewe with a two-day history of nystagmus, circling, inability to stand, eat or drink, and fever was one of 4 affected in a group of 6 that had possible exposure to moldy silage. A 3-year-old crossbred goat with staggering, opisthotonus, nystagmus and seizures for under 24 hours was one of 2 affected in a group of 8 on a second farm. A 2-year-old Nigerian dwarf goat with nystagmus of 3 days duration was the only one affected on the third farm. All 3 animals had typical histologic brain lesions of listeriosis. *L. monocytogenes* was isolated from the brainstem of one goat and the organism was confirmed by immunohistochemistry in the other two animals.

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