

California Animal Health and Food Safety Laboratory System

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Holiday Schedule

In observance of Independence Day, CAHFS will be closed on Wednesday, July 4, 2012.

CAHFS CONNECTION

June 2012

Bovine

Lead toxicosis was diagnosed in two, 12-month-old heifers submitted for necropsy six days after the first death occurred. Two additional animals were showing neurologic signs and blindness. In total, thirteen animals died. Deaths began three days after the heifers were given access to a new pasture where a bucket of lead grease, which appeared to have been licked by the heifers, was found. Lead was detected in both heifers and the grease contained 180,000 ppm of lead. Most of the lead in animals that die of acute lead poisoning is concentrated in the liver and kidneys and not in the bone and muscle, so rendering without the organs is acceptable.

Delayed anaphylaxis caused respiratory distress (coughing and foaming at the mouth), ataxia and lethargy observed three hours post-vaccination in 15, 10- to 12-month-old, Holstein heifers from a group of 200 heifers vaccinated with an inactivated Gram-negative bacteria and modified live virus product. Animals were treated with epinephrine intravenously and all except four recovered. Severe pulmonary edema and foam in the trachea was found on necropsy examination of the two animals submitted. The changes are consistent with a delayed acute anaphylactic reaction. These reactions are seen occasionally in young (<14-month-old) Holstein cattle given vaccines containing a Gram-negative bacteria, but are not associated with excessive endotoxin. Classic anaphylactic reactions occur within five to 30 minutes after administration whereas delayed reactions may take up to six hours to appear. All adverse product events should be reported to both the manufacturer and the Center for Veterinary Biologics (using a form available on their website at: http://www.aphis.usda.gov/animal health/vet biologics/vb adverse event.shtml

Poultry

Enterococcus cecorum was isolated from bone and trachea, and E. coli was isolated from multiple organs including bone, in a group of 48-day-old chickens experiencing increased condemnations at slaughter. E. cecorum can be associated with vertebral and femoral bone infections in chickens that are down and unable to rise or showing signs of lameness. The organism has also been isolated from trachea, air sacs, and liver of both turkeys and chickens and synovial membrane and blood of chickens.



Vertebral abscess and spinal cord compression

CAHFS Lab Locations

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Your feedback is always welcome. To provide comments or to get additional information on any of the covered topics or services, please contact Sharon Hein at slhein@ucdayis.edu.

We're on the Web www.cahfs.ucdavis.edu

Exotic Avian

Pasteurella multocida Infection was diagnosed in a 3-year-old male African Grey parrot presented for necropsy with a history of weakness, not perching and eyes closed. Necropsy revealed enlarged dark liver and spleen. Histopathology revealed severe inflammation in liver, spleen, brain, spinal cord and other organs associated with numerous Gram negative bacteria. Pasteurella multocida was isolated from the liver. During a follow up inquiry with the owner, it was learned that a dog had attacked the bird the day before. Psittacines are highly susceptible to P. multocida and the most common source of this bacterium is from a bite by a cat or a dog.

Small Ruminants

Recently three premises experienced outbreaks of **caprine Mycoplasmosis**. One farm reported that 2- to 3- week-old kids were unable to stand five days prior to death. Mycoplasma septicemia and joint infections were found at necropsy. The second premise had Mycoplasma septicemia and meningoencephalitis in 4- to 6-week-old kids with a history of respiratory signs unresponsive to antibiotics. On the third premise Mycoplasma was isolated from joints and milk from multiple does with arthritis and mastitis. In all cases, the Mycoplasma grew rapidly as is typical of goat types and did not react with bovine serotyping. Owners elected not to have the isolates sent out for typing.

Porcine

Postweaning multisystemic wasting syndrome associated with porcine circovirus-2 {PCV-2} was diagnosed on two farms with unresponsive respiratory disease progressing to weight loss. Lymph node lesions were typical with mild pneumonia. PRRS virus was a co-infection on one farm. The other farm had pigs with rear limb incoordination due to meningoencephalomyelitis, a rarely reported lesion, and widespread vasculitis. Vasculitis is most commonly seen with the dermatitis and nephropathy syndrome associated with PCV-2.

Testing for enteric clostridial toxins available at CAHFS

The diagnosis of enteric diseases produced by *Clostridium perfringens* and *Clostridium difficile* relies in most cases on detection of the major toxins of these microorganisms in intestinal content and/or feces. CAHFS offers three ELISAs for this purpose:

ELISA for *C. difficile* toxins A and B: this test detects toxin A, B or both. Detection of either of these toxins is confirmatory for *C. difficile* infection.

ELISA for *C. perfringens* alpha, beta and epsilon toxins. This test detects any of these toxins. Detection of beta toxin is confirmatory in cases of *C. perfringens* type C necrotizing enteritis of foals, piglets, calves, kids and lambs, while detection of epsilon toxin is confirmatory for *C. perfringens* type D enterotoxemia (overeating disease) in sheep and goats. Alpha toxin, although included in the panel, is of little diagnostic value, as this toxin can be frequently found in intestinal content of normal animals.

ELISA for *C. perfringens* enterotoxin. This test detects enterotoxin, a toxin responsible for food poisoning in humans, but not very frequently associated with animal disease. Dogs and occasionally other animal species are affected by this toxin.

For each of these three tests a minimum of 1 ml of feces or intestinal content (refrigerated or frozen) is required. Materials should be sent in a plastic or glass container but already removed from the intestine as the enzymes present in the intestinal wall may destroy the clostridial toxins.