UCDAVIS
VETERINARY MEDICINE
California Animal Health and Food Safety Laboratory System

CAHFS CONNECTION

March 2012

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HOLIDAY SCHEDULE

CAHFS will be open, but will have limited services available on Friday, March 30, 2012 in observance of Cesar Chavez Day.

Please contact the laboratory to plan your testing needs accordingly.

Bovine

Bovine tuberculosis - CAHFS laboratories have provided support to CDFA and USDA for the bovine tuberculosis program since 2001. These activities include necropsy of gamma interferon or comparative cervical test reactors, suspect and exposed animals; histopathology examination of granulomas found at slaughter plants in California; and gamma interferon testing of 1) caudal fold test-positive animals from infected herds and 2) animals from uninfected herds that are tested for movement, sales or traces related to an infected herd. Necropsies involve extensive examination and collection for culture and histopathology of lymph nodes and lesions to determine if bovine tuberculosis is present. Since 2002, CAHFS has performed necropsies on cattle from 10 affected herds, 5 of which were identified from slaughter surveillance testing. The other five herds were found during trace testing from infected herds. Three of these herds to date had a single infected animal with the same strain of *Mycobacterium bovis* as a source herd while the remaining 2 herds had a single animal with a strain unrelated to the source herds. For more information about bovine tuberculosis status and history of infection in California visit the CDFA website at: http://www.cdfa.ca.gov/ahfss/animal_health/tb_info.html.

Equine

Clostridium perfringens type C is an important cause of **enteritis** and enterocolitis in newborn foals and rarely in adult horses. The disease is a classic enterotoxemia, and the enteric lesions and systemic effects are caused primarily by beta toxin, one of two major toxins produced by *C. perfringens* type C. Grossly, multifocal to segmental hemorrhage and thickening of the intestinal wall are common in the small intestine, although the colon and cecum may also be affected. The horses have variable amounts of fluid, often hemorrhagic, intestinal contents. The most characteristic microscopic lesion is necrotizing or necrohemorrhagic enteritis with thrombosis. Numerous Gram-positive rods may be seen in affected mucosa, although this is a poor diagnostic tool. A presumptive diagnosis of *C. perfringens* type C enterotoxemia is based on the history and gross and histologic lesions, while confirmation of the diagnosis should be based on detection of the beta toxin in intestinal contents by ELISA.

Submission of formalin-fixed samples to CAHFS

For **formalin-fixed** samples, choose an appropriate container (no yogurt/cottage cheese tubs) and seal it tightly. We recommend wrapping the sealed container with parafilm, and then surround it with absorbent material (paper towels, gauze, cotton or equivalent) before placing it in a double tight sealed bag. If samples have been sufficiently fixed for a period of time (usually at least 24 hours) before shipping, wrap the tissues in formalin-soaked gauze and place them in a sealable container as above for shipping, and eliminate the excess formalin. The wrapped and bagged container should then be placed in an appropriately padded box for shipping. Please do not freeze the samples; refrigeration is not required for samples submitted in formalin. Please remember that whatever you do, containers should not leak as formalin is a health hazard. If your 10% formalin, which is 3.7% formaldehyde, contains methanol, there are package label (Formaldehyde solutions, flammable, UN1198) and shipper declaration requirements. There is no requirement for methanol-free 10% formalin solutions. For information on this revised rule see July 2011 Federal Register, http://www.gpo.gov/fdsys/pkg/FR-2011-07-20/pdf/2011-17687.pdf

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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@ucdavis.edu.

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

Pigs

In December, three swine ranches experienced **pneumonia** due to ***Actinobacillus pleuropneumoniae*** in 2- to 4-month-old pigs. This organism causes severe rapidly progressive pneumonia, high fever and increased mortality. Some pigs may die with no signs of respiratory distress. Two ranches experienced up to 10% mortality in the affected age group. Concurrent agents found included Porcine Reproductive and Respiratory Syndrome virus (PRRSV) and tracheal isolation of *Bordetella bronchiseptica*, *Haemophilus parasuis* and *Streptococcus suis*.

Poultry

Infectious bursal disease (IBD) is an economically important, highly contagious disease that affects immature chickens. The causative agent, IBD virus (IBDV), targets the immune system causing immune suppression by destroying the immature B-lymphocytes primarily in the bursa of Fabricius. Without B-lymphocytes the bird does not have a learned immune response and therefore cannot produce antibodies, leaving chickens susceptible to many diseases. IBDV isolates can be differentiated into serotype 1 and 2 with clinical disease attributed only to serotype 1. Serotype 1 IBDV was originally discovered in the U.S. around 1960, and has been controlled by vaccinating birds.

Very virulent IBDV (vvIBDV) is a mutant of IBDV, which was first detected in broilers in the Netherlands in the late 1980s. The primary feature of vvIBDV is the ability to induce higher mortality in susceptible chickens than classical serotype 1 strains. Mortality rates of 100% in specific pathogen free (SPF) chickens, 60% in layers and 30% in broilers have been reported. In late 2008, vvIBDV was detected and diagnosed in chickens from Northern California which had a dramatic increase in mortality with severe lesions in multiple tissues including the bursa of Fabricius. Molecular sequence analysis of the entire viral genome and animal challenge studies in SPF birds (90 to 100% mortality) confirmed its high pathogenic potential. Ongoing surveillance efforts have shown that vvIBDV remains only in Northern California; however, there are several genetic variations of IBDV throughout the state. The genetic variability of the virus complicates diagnoses and requires a more thorough definition of the disease and the virus. The current disease definition of vvIBD includes 1) severe mortality in a flock; 2) severe lesions in tissues; 3) lack of protecting antibodies; and 4) molecular analysis of the viral genome showing compatibility with the vvIBD virus. An optional step would be to determine the mortality in SPF birds that were inoculated with the suspected virus.

Wildlife

Hair loss syndrome in California mule deer. A syndrome characterized by increased grooming behavior, patchy alopecia, poor hair coat and body condition and mortality in the fawns, yearlings and does in the winter and spring has been identified in California deer. This syndrome, which is multifactorial and associated with internal parasitism and nutritional deficiencies, had been associated with the exotic *Damalinia* louse species. Prior to 1995, exotic lice were reported sporadically in North American deer; however, following this time a syndrome of hair loss and mortality in black tailed deer associated with an exotic louse spread through Washington and down into Oregon. The syndrome appeared in California for the first time in 2009 and was associated with the death of over 200 deer in Tuolumne County. Deer were from herds experiencing hair loss from which exotic lice, *Bovicola tibialis* and *Linognathus africanus*, were identified. Since then, mortality events of deer with hair loss syndrome have occurred almost on a yearly basis. Tule elk and mule deer on the coast have hair loss syndrome associated with *Damalinia* and inland herds with *Bovicola tibialis*. In most outbreaks of hair loss syndrome in California, selenium deficiency and pediculosis are the most common findings, followed by copper deficiency; often both deficiencies are evident. The primary cause of the high mortality of the deer in this outbreak is still undetermined. *Bovicola tibialis* and copper and selenium deficient regions have been factors to which deer have been exposed and susceptible for many years, although mortality was only recently observed. The reason for susceptibility to severe pediculosis and nutritional deficiencies that lead to high mortality in deer herds is unknown. Investigation is ongoing at CAHFS as this syndrome may not only affect deer but also may be an indicator of future problems in livestock.