Inside this issue:

- **Bovine**
  - Toe tip necrosis
  - Vetch toxicosis
  - Abomasal bloat

- **Mycoplasma real-time PCR panel available - ruminants**

- **Small Ruminant**
  - *Clostridium perfringens* type D—goat

- **Equine**
  - Equine Influenza subtype H3N8

- **Pig**
  - Gastric ulceration

- **Poultry/Other Avian**
  - Yolk sac infections and omphalitis - chickens
  - Hemorrhagic enteritis virus - turkeys
  - Aspergillosis - duck

- **Fish**
  - Koi herpesvirus

---

**CAHFS CONNECTION**

**June 2015**

**Bovine**

**Toe tip necrosis** was diagnosed in two, 8-month-old Angus heifers from a herd with a history of rear leg lameness and occasional animals sloughing their hooves. The herd had been moved from pasture to a feedlot two weeks before the onset of clinical signs. The lateral toe of the rear right hoof of both heifers had lesions consistent with the “toe-tip necrosis” characterized by small cracks in the soles along the white line. The cracks allowed opportunistic pathogenic bacteria to invade and cause osteomyelitis of the third phalanx and inflammation of the surrounding soft tissues. This condition is poorly understood but is believed to be traumatic, associated with animals walking on concrete floors or rocky grounds.

**Vetch toxicosis** outbreaks occurred in five separate beef cattle herds this spring. All were associated with multiple deaths and most with grazing purple vetch pasture (*Vicia benghalensis*). Cattle that die from vetch poisoning develop systemic granulomatous disease that can present with sudden death or chronic wasting with diarrhea and dermatitis. Death typically occurs in cattle 2 years and older. Heifers and calves in the herd are spared because the syndrome is thought to be associated with hypersensitivity, requiring a previous exposure to vetch. What triggers the syndrome by a plant that is considered normal forage is unknown.

**Abomasal bloat** and emphysematous abomasitis associated with *Sarcina* sp. was the cause of sudden death or brief anorexia prior to death in three 15-day-old Holstein calves fed milk replacer on one dairy. Two calves had abomasal and intestinal hemorrhage, and the abomasum had ruptured in one calf. The predisposing factors for development of this condition in calves fed milk and milk products are unknown. *Sarcina*, a Gram-positive anaerobic soil bacteria belonging to the Clostridia family, is typically seen in cases of abomasal bloat.

---

**MYCOPLASMA REAL-TIME PCR PANEL NOW AVAILABLE**

CAHFS is now offering a Mycoplasma real-time PCR panel for use on ruminant samples, which is highly sensitive and specific for these organisms. Testing is performed on an enrichment broth from tissues, swabs, or milk samples and can detect as few as 240 cfu/ml. Use of the enrichment broth has been shown to increase sensitivity of detection and decrease the likelihood of PCR inhibition from sample components while facilitating differentiation of live bacteria from non-viable DNA. The panel screens all samples for *Mycoplasma* sp. in general and for *M. bovis* specifically. If a non-*M. bovis* Mycoplasma is detected, culture will be performed to determine if other Mycoplasma can be recovered and identified. The cost of the panel is $25.00 which includes both PCR tests as well as culture if indicated. Please contact the Davis lab (530-752-8700) or Dr. Kris Clothier (kaclothier@ucdavis.edu) with any questions.

---

**HOLIDAY SCHEDULE**

CAHFS will be closed on Friday, July 3, 2015 in observance of Independence Day. Please contact your laboratory to plan your testing needs accordingly.
Equine

**Equine Influenza subtype H3N8** was diagnosed in a 1-year-old Pony gelding with acute onset of fever, leukopenia and bilateral bloody nasal discharge. The pony had severe broncho-interstitial pneumonia, with hyaline membrane formation, tracheitis, laryngitis and rhinitis. Influenza virus PCR was positive and the virus was isolated from the lung and typed as H3N8. This virus is endemic in horses, birds and dogs, and has been associated with severe outbreaks due to the short incubation period (1-3 days). Transmission of the virus to humans has not been reported.

Small Ruminant

**Clostridium perfringens type D** caused diarrhea and chronic wasting in goats on one premises. A 3-year-old La Mancha goat submitted for necropsy had multifocal erosive, fibrinohemorrhagic typhlocolitis. Epsilon toxin was detected by ELISA and **Clostridium perfringens** type D was isolated from the colon. Of 96 goats purchased four months earlier, most were affected and 18 had died. However, resident goats were not affected. In this situation, all remaining goats and new goats that come onto the premises in the future should be vaccinated with **Clostridium perfringens** type D toxoid with a booster 4-6 weeks later and then boosted every 4-5 months due to environmental contamination. Frequent boosters are required because immunity produced by type D vaccines in goats is of shorter duration than in sheep.

Pig

**Blood loss from gastric ulceration** of the pars esophagæa portion of the stomach caused the death of three, 4- to 5-month-old FFA and high school pigs from different premises. Two pigs were found dead with no prior signs. The third pig had lost 4 lbs in one week and was lethargic prior to death. A large amount of blood was found in the stomach and intestine. One pig also had black feces. Small-particle-size diets have been shown to increase the prevalence of ulcers compared to coarsely ground diets in pigs.

Poultry and Other Avian

**Yolk sac infections and omphalitis** resulted in increased mortality in 2- to 3-day-old chicks. The submitted birds had wet or open navels, and enlarged yolk sacks with reddening of the capsular wall and congestion of vessels. **E. coli** was isolated from affected yolk sacs. **E. coli** and **Enterococcus** are common pathogens observed in omphalitis and retained yolk sacs. These are often associated with management issues arising from the hatchery.

**Hemorrhagic enteritis virus (HEV)** caused a three-fold increase in mortality in a house of 13,000, 10-week-old turkeys with no other clinical signs. Birds had mild hemorrhagic enteritis and pale spleens at necropsy. Adenovirus inclusion bodies were seen in the intestine, spleen and kidney and several of the birds submitted had positive titers to HEV. Concurrent crop mycosis and **E. coli** airsacculitis were found in a few birds.

**Aspergillosis** was the cause of death of a 3-month-old, Indian runner duck, submitted with a clinical history of marked difficulty breathing. At necropsy, the bird had a localized accumulation of a yellowish exudate at the bifurcation of the trachea and syrinx, from which **A. fumigatus** was isolated. Histologically, severe inflammation and large numbers of fungal hyphae obstructed the tracheal lumen and infiltrated the cartilage rings. The litter material covered by a blackish mold in the coop of this bird was likely the source of infection.

Fish

**Koi herpesvirus** caused deaths, gill flaring and respiratory distress within days after adding new koi to a pond. Both koi fish submitted had necrosis and hemorrhage of the gills, and one fish also had skin ulcers and ascites. Gill samples were positive for Koi Herpesvirus by PCR (a new test offered at CAHFS). Koi herpesvirus is a systemic and often fatal disease of koi and carp. Affected fish present with gill necrosis and hemorrhage and ulcers of the skin. The virus often occurs when pond water temperatures reach 70°F-80°F.