Congratulations to Dr. Santiago Diab, Davis pathologist, for receiving the 2016 C.L. Davis/S.W. Thompson Foundation Annual Award for the Best Diagnostic Exercise. The Diagnostic Exercises of the Davis/Thompson Foundation are published monthly on-line and distributed to diagnosticians and pathologists throughout the world. Dr. Diab’s exercise described a case of bovine pneumonia caused by Mycoplasma bovis.

Congratulations also to Dr. Rahul Dange, Davis pathology resident, and Dr. Arya Sobhakumari, Davis toxicology resident, who received Trainee Travel Awards from the American Association of Veterinary Laboratory Diagnosticians, for their abstracts on “Proliferative thrombo-vascular necrosis of the pinnae in dogs” and “Lead contamination in backyard chicken flocks – incidence and exposure assessment in positive cases”, respectively.

Equine

**Equine herpesvirus-1 (EHV1)** infection is ubiquitous in most equine populations, with about 60% of healthy horses typically harboring the virus. Latent infection can be reactivated during periods of stress, causing clinical disease and viral shedding. Two outbreaks of neurological disease attributed to EHV1 have been detected in the last month in California. The outbreaks involve two different strains of EHV1. Although both strains cause neurologic disease, only one strain harbors the EHV1 neuro-pathogenic marker in the genome of the virus. This is consistent with previous reports that up to 25 percent of EHV1 neurologic disease is caused by strains without the neuro-pathogenic marker. Outbreaks of EHV1 are best controlled by early diagnosis and prevention of further spread. Because of the high prevalence of EHV1 in healthy horses, testing for EHV1 in horses without clinical signs is not recommended.

**Myeloencephalopathy** due to equine herpesvirus type 1 (EHV; non-neuro-pathogenic form) was diagnosed in a Saddlebred mare with a history of 24 hours of ataxia. This horse was part of one of the two currently ongoing outbreaks of EHV infection in California. On post-mortem examination this horse had severe hemorrhagic necrosis in the spinal cord. This lesion was responsible for the clinical signs observed. The etiology was confirmed by PCR for EHV on the brainstem.

Bovine

**Bacillary hemoglobinuria (BH)** was diagnosed in a Jersey cow on pasture that died 48 hours after developing diarrhea. Another cow had died 2 weeks prior on the same property. All cows were vaccinated with Clostridial 8-Way as heifers. Tissues submitted from a field necropsy revealed severe multifocal hepatitis. *Clostridium haemolyticum* was isolated from the liver. No evidence of liver fluke was seen in the liver and the predisposing factor for BH in this case remains undetermined.

Continued
Leptospirosis was diagnosed in a 6-month-old Holstein heifer. Three animals had died without prior clinical signs in the same pen over the preceding week. The heifer was markedly icteric and had hematuria. Histological examination revealed mild interstitial nephritis. The presence of Leptospira spp. was confirmed by PCR on kidney. The animal also had locally extensive bronchopneumonia caused by Pasteurella multocida, Histophilus somni and Mycoplasma bovis.

Small ruminants

Nitrate toxicosis was diagnosed in 6-month-old lambs of which 204 died and 100 were lethargic the day after the flock of 500 animals arrived in California and were placed on a field of black-eyed peas. Aqueous humor had nitrate levels of 540 and 700 ppm in two animals, thus confirming nitrate toxicity. Levels above 25 ppm are considered toxic. The source of the nitrates was not confirmed as the client declined to submit feed and water for testing.

Poultry and Other Avian

Sarcocystis falcata was caused by the sudden death of a 15-year-old Palm Cockatoo in an aviary. On gross examination there was severe pulmonary edema and enlarged mottled liver and spleen. Microscopically there was marked pneumonia, hepatitis and splenitis, with intralesional protozoa that were identified as Sarcocystis falcata by immunohistochemistry. This is a protozoal parasite that is shed in the feces of North American opossums. Birds in outdoor aviaries are more at risk since they can come into contact with opossum scat, but insects that ingest opossum fecal matter, such as cockroaches, can also carry the parasite and infection occurs when the birds ingest these insects. Infection by Sarcocystis falcata is not transmitted from bird to bird.

Severe coccidiosis caused by Eimeria maxima was diagnosed in two flocks of ~1-month-old broilers. Sick birds were submitted with a history of increased mortality, unevenness in size and diarrhea with sloughed intestinal mucosa. On postmortem examination, the jejunum and ileum were dilated and flaccid, with many petechiae on the thickened mucosa. Large numbers of coccidia oocysts were detected on wet preparations of the intestinal mucosa. Histologically there was severe enteritis associated with large numbers of coccidia in different stages including large oocysts, characteristic of Eimeria maxima.

Fowl cholera caused respiratory signs and increased mortality in a house of 10-week-old turkeys. Necropsy revealed air sacculitis, pneumonia, splenitis and arthritis. Pasteurella multocida, the agent of fowl cholera, was isolated from multiple tissues of these birds. Although commercial vaccines are available for the most common serotypes (1, 3 and 4) of P. multocida, prevention and control of the disease is best accomplished with stringent biosecurity protocols, including pest control and sanitation of water troughs and feeders. Survivors of fowl cholera can become carriers and continually shed the bacteria.

Pigeon circovirus infection was diagnosed as the cause of mass mortality in an aviary from which five birds were submitted. All pigeons had non-specific lesions, mostly consistent with systemic infections, including lymphoid depletion in the bursas, which prompted testing for an underlying viral infection. Pigeon circovirus was detected by in situ hybridization in the spleen of two of the birds, confirming the infection.