PURDUE UNIVERSITY

DIAGNOSTIC FORUM

FROM THE DIRECTOR

Good day from ADDL. As this is written, it appears spring has finally arrived. It seems that this past one has been a long winter, one that has been difficult for many of our Indiana outside farm animals.

The ADDL continues to participate in national surveillance programs for classical swine fever, avian influenza, chronic wasting disease, scrapie and foot and mouth disease. The national program for routine surveillance for exotic Newcastle disease has been discontinued. The program for surveillance for classical swine fever has been expanded to include samples collected by Indiana private practitioner veterinarians. This should markedly increase the number of tonsil samples received by the Laboratory for CSF testing.

As reported in the last *Diagnostic Forum*, we completed immunohistochemistry testing of 610 lymph node samples collected from Indiana white tail deer during the '07 hunting season for chronic wasting disease. The test samples were collected by the Indiana DNR; all samples tested "No resistant prions detected". The Indiana surveillance program was started in 2002. Since that time 9609 deer have been sampled and tested. To date, no evidence of CWD has been found in our state.

Dr. Roman Pogranichniy, head of the ADDL virology and serology sections, has made available sequencing of PRRSV and porcine circovirus to fingerprint virus isolates of these swine pathogens. This allows pinpointing the source of infection in epidemiologic studies and investigations of the disease in affected swine herds. These assays that become available in virology section may be invaluable in identifying type of virus present in affected herds.

To better acquaint various agencies with intercommunication, familiarity and responsibilities for dealing with the occurrence of a catastrophic disease outbreak in our state, ADDL participated in a table-top exercise on epidemic avian influenza in the Indiana poultry population and pandemic bird flu in Indiana human population sponsored by the Indiana Department of Health on March 28, '08 in Indianapolis. To further enhance interaction of the potential involved agencies in the instance of such a disease outbreak, a table-top exercise sponsored by the National Animal Health Laboratory Network, of which ADDL is a member, will be held at the ADDL on May 16, '08. The May 16 exercise will involve many of the same agencies as the March 28 exercise with the addition of assistance from adjacent state veterinary diagnostic laboratories' contribution to a potential catastrophic disease occurrence.

In closing, I wish you a great Indiana spring season; may the morels flourish. If there are things we can improve to better serve your veterinary diagnostic needs, please do not hesitate to let me know.

FINAL DIAGNOSIS

Fatal systemic canine herpesvirus infection in 2 puppies

History: One male and one female 8-day-old puppy were submitted to the Animal Disease Diagnostic Laboratory for necropsy following a history of 7 of 12 puppies in the litter dying within 48 hours.

Gross examination: The two puppies had similar necropsy findings. The kidneys contained multifocal hemorrhages apparent from the capsular surface, measuring 2-3 mm in diameter (see Figure 1). The renal parenchyma bulged on cut section, and the hemorrhages extended into the cortex and medulla. The pleural cavity contained transparent, yellow to red-tinged fluid with the quantity ranging from approximately 10-20 mL. The lungs were diffusely mottled red to dark red, slightly firm, and noncollapsing. The livers from both pups had multiple, pinpoint, red foci apparent from the capsular surface and on cut section.

5 mm

Figure 1: Gross photograph of one kidney, depicting multiple 2-3 mm, discrete, dark red foci apparent from the capsular surface, corresponding to necrosis and hemorrhage

Pinpoint to coalescing dark red hemorrhages were present on the serosal surface of the small intestine, as well as within the thymus.

Histopathologic examination: Sections of kidney, lung, and liver from both pups had multifocal parenchymal necrosis. In the kidney, renal tubular epithelial cells were swollen with hypereosinophilic cytoplasm and absent or pyknotic nuclei, and necrotic cell debris filled tubular lumina. Glomerular tufts contained karyorrhectic debris and increased eosinophilic matrix within the mesangium. Endothelial cells lining blood vessels were disrupted, and hemorrhage extended into the necrotic foci.

Basophilic to pale, eosinophilic structures were present within nuclei of tubular epithelial cells and glomerular mesangial cells, reminiscent of intranuclear inclusion bodies. In the lung, disrupted alveolar septa were replaced by karyorrhectic debris and fibrin strands. Fibrin, hemorrhage, and foamy alveolar macrophages filled alveoli in affected areas. Pale basophilic to eosinophilic intranuclear inclusion bodies were rarely identified in pneumocytes and bronchiolar epithelial cells at the periphery of necrotic foci. Necrotic hepatocytes in the liver were swollen with hypereosinophilic to vacuolated cytoplasm, and either lacked apparent nuclei or contained pyknotic nuclei. The lesions were not associated with inflammatory infiltrate, but hemorrhage was occasionally present. Rare, viable hepatocytes at the periphery of necrotic foci contained 4-6 micron, lightly basophilic intranuclear inclusion bodies causing margination of chromatin. Non-staining edema fluid expanded centrilobular connective tissue.

Multifocal necrosis was also identified in the cerebrum, myocardium, adrenal cortex, and small intestinal mucosa.

Ancillary tests: Fluorescent antibody tests for canine herpesvirus were positive on submitted lung samples and on pooled samples of spleen and kidney. In addition, canine herpesvirus was isolated from the lung.

Final diagnosis: Fatal systemic neonatal infection with canine herpesvirus type 1

Comment: The age, clinical history, gross lesions, and microscopic lesions were all characteristic of systemic neonatal herpesvirus infection in this case. The diagnosis was confirmed via fluorescent antibody tests and virus isolation. Microscopic identification of intranuclear inclusion bodies can assist in the diagnosis, but inclusions are often rare or absent in canine herpesvirus as compared to herpesviral infections of other domestic species.

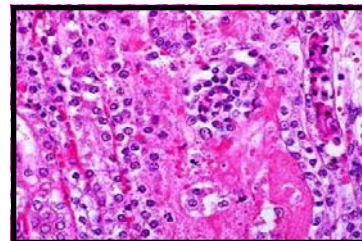-by Dr. Pam Mouser, ADDL Gradaute Student

photo from www.afip.org

Parelaphostrongylus tenuis infection in Llamas

The meningeal worm, *Parelaphostrongylus tenuis*, cases neurologic disease that can result in death of llamas and alpacas. White-tailed deer are considered the natural definitive host for the meningeal worm but seldom show signs of infection. *P. tenuis* migrates much more extensively in the central nervous system of incidental definitive hosts, often causing severe, disabling neurologic disease. There are numerous reports of infection in both domestic and wild ungulates living in close proximity to white-tailed deer. Among domestic livestock, the llama is the most sensitive to the development of clinical disease. Owners of llamas, alpacas, and other small ruminants in eastern North America, where the disease is endemic, must use proven preventative measures to control outbreaks and inevitable death in their herd.

Adult *P. tenuis* nematodes reside in the subdural space of the central nervous system and in the associated blood vessels and sinuses. The life cycle begins when adult females lay their eggs in the venous vessels and the eggs hatch in the capillaries of the lungs. First stage larvae (L1) enter the alveolar sacs and are coughed up and swallowed. L1 larvae leave the host in the mucus covering of fecal pellets, then actively penetrate gastropods residing in the pasture. The larvae molt twice in their intermediate host. Accidental ingestion of the snails containing infective L3 larvae continues the life cycle. L3 larvae leave the gastrointestinal tract of the host and enter the central nervous system in approximately ten days. Larvae develop in gray matter of the dorsal horn of the spinal cord and migrate to the subdural space 40 days later. In aberrant hosts, the parasite persists in the parenchyma of the central nervous system instead of migrating to the subdural space. Disease is caused by physical trauma to the parenchyma of the central nervous system by developing and migrating worms.

White-tailed deer are the natural host of *P. tenuis;* however, other wild and domestic ungulates have been identified as aberrant hosts and may develop severe neurologic disease. In response to infection, clinical signs usually reflect focal, asymmetrical spinal cord lesions and include ataxia, stiffness, muscular weakness, hypermetria, posterior paresis, paralysis, head tilt, arching neck, circling, blindness, gradual weight loss, depression, seizures, and death. Clinical signs generally begin in the hind limbs and progress to the front limbs. The disease may be acute or chronic, with death within days to ataxia that lasts months to years.

Microscopic lesions include scattered foci of hemorrhagic necrosis. Acute lesions are characterized by focal parenchymal loss with hemorrhage in and around the area of injury. Most chronic lesions have no hemorrhage, but varying numbers of large, foamy macrophages, some containing gold pigment consistent with hemosiderin. Around some necrotic foci there can be swollen axons. The microscopic lesions seen are most compatible with lesions caused by a migrating parasite.

The use of cerebrospinal fluid for diagnosis of *P. tenuis* infection is valuable, especially since hematologic abnormalities are often not found with meningeal worm infection Eosinophilia in the cerebrospinal fluid is a common, although inconsistent, finding in aberrant hosts.. Leukocytosis and vacuolated monocytoid cells are often found. CSF eosinophilic pleocytosis is not always associated with cerebrospinal parelaphostrongylosis, and other parasites can cause eosinophilic meningitis in South American camelids.

The only antemortem test for diagnosing *P. tenuis* is the Baerman technique, which relies on the detection of L1 larvae in the feces of infected animals by microscopic examination. Aberrant hosts rarely shed larvae within their feces, thus this test is unreliable even when repeated. Experimental ELISA-based antigen-antibody tests in goats and elk have shown promise but this test is not currently available. Additionally, an antigen-capture ELISA has been developed that can detect antigens of *P. tenuis* in cerebrospinal fluid, but this test is not commercially available.

The definitive diagnosis of meningeal worm currently requires demonstration of larval or adult *P. tenuis* in the brain or spinal cord of an affected animal at necropsy. Nematodes are identified on the basis of their size and the following features: lateral cord cells broader at the base than at the apex, multinucleated intestinal cells, with no more than two cells per cross section, and polymyarian coelomyarian musculature. A presumptive diagnosis may be based on clinical signs, exposure, and response to treatment.

Recommendations for the prevention of meningeal worm infections include the exclusion of white-tailed deer from llama and alpaca pastures in endemic areas and clearing thick ground cover to discourage establishment of snail intermediate hosts. Prophylactic treatment with ivermectin is more effective against early larval stages because the drug does not cross the blood brain barrier. Anti-inflammatory drugs are also important for reduction of the inflammation associated with migrating larvae and the subsequent inflammatory response to killed larvae. Use of anti-inflammatory drugs is especially important to prevent the clinical signs from worsening after treatment.

The prognosis of suspected meningeal worm infection is guarded. Some clinicians suggest that animals that are only able to stand with support have a much poorer prognosis than those who are able to stand without assistance. Some animals suffer permanent neurologic damage but remain otherwise healthy members of the herd.

Meningeal worm infection may be severely debilitating and potentially fatal, but can be effectively prevented. Simple steps such as routine deworming every 4-6

4-6 weeks, minimizing cohabitation with white-tail deer, and a clean, dry environment unfavorable for the growth of snails and slugs will considerably reduce the herd's risk of infection with meningeal worm.

-by Abby Durkes, Class of 2008
-edited by Dr. Grant Burcham, ADDL Graduate Student

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Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2008.

May 26..Memorial Day
July 4......................................Independence Day
September 1...Labor Day
November 27-28.............................Thanksgiving
December 25-26.................................Christmas

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Microsporidiosis in Animals: Encephalitozoonosis and the Importance for Public Health

Introduction: Microsporidiosis is a diverse group of diseases caused by microsporidian parasites in a wide range of invertebrate and vertebrate species, including insects, fish, birds and mammals. Traditionally, they were considered protozoa, but recently have been reclassified as fungi based on molecular biology and genomics. These parasites have been reported as causing economic losses in honeybee, fish, mink, and other fur-bearing animals. They also cause problems in the establishment of colonies of specific pathogen free (SPF) laboratory animals for biomedical studies. Nowadays, they are emerging as opportunistic infection parasites in immunocompromised individuals, especially in AIDS patients.

  The phylum Microsporidia is subdivided into a variety of families and genera, which includes the genus *Encephalitozoon* as an important study target*,* especially for human beings. This genus includes the species *E. cuniculi, E. hellem, E. intestinalis* and *E. lacerate*. The latter species was isolated only from reptiles, while the former three species (*E. cuniculi, E. hellem, E. intestinalis),* together with *Enterocytozoon bieneus*, were identified as human pathogens. They were reported as causing disease in AIDS patients, organ transplant recipients, children and elderly. Microsporidian spores have been identified in meat and water sources; they are relatively resistant in the environment, and strains of animals were already isolated from humans, raising the concern for the zoonotic potential of microsporidiosis.

  In animals, the most common and important pathogen is *E. cuniculi.* This microorganism is distributed world-wide and present especially in the domestic rabbit population. It may cause different clinical and pathologic presentations, depending on the immune status and species of the affected animal. Although serological studies have been extensively performed and diagnostic tests and treatment have quickly developed during the last few years, further studies are required to completely understand the epidemiology and pathobiology of microsporidiosis.

Etiologic agent: Microsporidia are unicellular, spore-forming, obligatory intracellular eukaryotes that are highly resistant to environmental conditions. They characteristically lack obvious mitochondrial structures and have a polar filament, which is a unique feature of these organisms and is important for host cell infection.
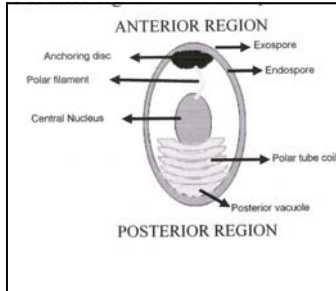
Fig. 1. Diagram of microsporidian spore which has walls composed of glycoprotein, chitin and a trilaminar membrane system. (Font: Modified from Wasson & Peper. 2000)

As mentioned above, microsporidians are nowadays considered to be more closely related to fungal than to protozoal organisms. Similar to fungi, they have prokaryote-like ribosomes (16S and 23S) and α-and β-tubulin gene sequences, as well as the largest subunit of RNA polymerase II and mitochondrial HWP70. Additional similarities with fungi include certain features of the cell cycle, chitin as one of the endospore constituents, and trehalose as an energy source. The microsporidian are classified based on the natural host, mode of cell and nuclear division, and ultrastructural features, such as size, nuclear arrangement, number of coils of the polar filament, and developmental stages in the host cell. Typically, *Encephalitozoon* spp. replicate by binary division within a parasitophorous vacuole, forming 1.5 to 4.0μm, small oval spores containing approximately 4-9 polar filaments coils with single row arrangement. All three *Encephalitozoon* spp. considered to be human pathogens have morphologically indistinct spores. In the specific case of *E. cuniculi,* three strains are being recognized based on the animal species from which they were originally isolated and based on the number of 5"GTTT-3" nucleotide sequences: strain I or "rabbit strain", strain II or "mouse strain" and strain III or "dog strain". These have three, two and four 5"-GTTT-3" nucleotide repeats respectively; experimental infection studies failed to prove a host specificity of these strains however.

Life cycle: The life cycle is simple and direct. Most of the infections are acquired through ingestion or inhalation of infective spores, but transplacental transmission of *E. cuniculi* has also been reported. Rarely, the infection may be transmitted through traumatized epithelium or direct contact. The spore germination occurs when microenvironmental stimuli, such as change in pH or in osmotic pressure, induce posterior vacuole and polarplast alterations, which consequently result in extrusion of the polar filament. This structure is responsible for injecting the sporoplasm into the host cells, which are commonly epithelial cells, macrophages, and endothelial cells. The cycle continues with the development of proliferative forms (meronts) which consequently

result in extrusion of the polar filament. This structure is responsible for injecting the sporoplasm into the host cells, which are commonly epithelial cells, macrophages and endothelial cells. The cycle continues with the development of proliferative forms (meronts), which undergo binary division and differentiate into sporoblasts and sporonts. In *Encephalytozoon* spp., the cycle occurs within a parsitophorous vacuole (PV) in the host cell, with immature meronts attached to the PV membrane and mature sporonts present in the center. Eventually, the PV and host cell membranes rupture and release spores into the extracellular space. Dissemination may occur by direct extension into adjacent cells or by invasion of the vascular system. The primary sites of infection will depend on the route of transmission, but commonly affected tissues are small intestine, respiratory tract and placenta, with secondary dissemination to kidneys, liver and brain; however, every organ may be affected. The infective spores may be shed through feces, urine or respiratory secretions. This is again dependent on the infection site.

Clinical signs: The clinical signs will depend on the immune status and the species affected, as well as on the pathogenicity of the microsporidian strain. Most of the infected individuals do not become ill and may appear clinically healthy for years. In animals, *E. cuniculi* is the most important microsporidian pathogen. Serological studies revealed a worldwide range of exposed mammalian species, most of which were asymptomatic. *E. cuniculi* infection was experimentally reproduced in mice, rabbits and guinea pigs. The clinical signs were worse in immunocompromised animals, such as athymic mice or mice treated with immunosuppressive corticosteroid doses. In rabbits, encephalitozoonosis is a well recognized disease, especially in the domestic rabbit population. Infection probably occurs through ingestion of spores. Occasionally, these animals may develop neurological signs of ataxia, opisthotonos, torticollis, paralysis and head tilt, but more often these clinical signs are a result of bacterial otitis interna. *E. cuniculi* infection has been described also as fatal infection in newborn puppies and in squirrel monkeys, and as a cause of abortion in horses and non-human primates. In chronically infected dogs, a hyperimmune response may lead to progressive renal failure because of immunocomplex deposition. Severe disease caused by *E. cuniculi*, characterized by disseminated infection as well as milder clinical presentations such as kerato-conjunctivitis, have been recognized in immune compromised humans. The other pathogenic Encephalitozoon spp., *E. hellem* and *E. intestinalis*, are more often associated with persistent diarrhea in AIDS patients and organ transplant recipients.

Pathologic findings: Gross examination commonly fails to demonstrate lesions in cases of encephalitozoonosis. Usually, the lesions are characterized by lymphoplasmacytic or granulomatous interstitial cell infiltration in multiple sites, particularly in the kidney, liver and brain, but also in the heart, brain, skeletal muscle, and placenta. Especially in immune-

compromised hosts, the inflammatory lesions are often accompanied by necrosis and, consequently, by suppurative infiltrate. In fatal cases, these necrotic areas are larger and surrounded by epitheloid macrophages. In cases with chronic liver and kidney involvement, multifocal depressed irregular areas may be present on the renal surface and multifocal pale to yellow areas, characteristic of multifocal hepatitis, may be noted in the parenchyma of the liver grossly. At light microscopy, the organisms may be seen in the center of the necrotic areas; however, parasites may also be visualized without surrounding tissue inflammation, either as small ovoid intracytoplasmatic structures of free within the interstitium. Especially in chronic infections, it may be impossible to find the parasite because it is sparsely distributed and more likely to be disseminated throughout multiple organs. This raises the possibility of underdiagnosis of encephalitozoonosis during routine examination.

Definitive diagnosis and complementary exams: Demonstration of organisms in tissue sections, isolation in tissue culture, inoculation into laboratory animals, electron microscopy, serological tests and molecular techniques such as PCR, are all ways to diagnose encephalitozoonosis. As mentioned above, visualization of the parasite during the histological exam may be difficult in some cases. Gram, periodic acid-Schiff, Warthin-Starry and Giemsa stains have been successfully used to stain microsporidia in tissue sections. Transmission electron microscopy is still the gold standard to identify these organisms based on the detection of the polar filament and PV visualization. Serological exams with determination of antibody titers are useful in epidemiological animal studies and in the control of colonies of SPF laboratory animals. It is important to note, however, that the antigens for such serological assays are available only for microsporidian species maintained in cell culture such as *E. cuniculi.* Especially when used to evaluate human exposure, the real efficacy of these tests is questionable as there seems to be a risk of nonspecific reaction when spore walls are used as the antigen. More than this, cross reaction may occur, especially in patients with tropical protozoal diseases and, at this time, there are no serological assays that allow the differentiation between current infection and previous exposure to the microorganism. Efforts are being made to use a recombinant protein of the polar filament as the antigen which gives a highly specific result. The most sensitive diagnostic method to identify microsporidian organisms is still the PCR assay.

Public health importance: Microsporidia are recognized as an emerging opportunistic infectious agent in mammals. The incidence of their diseases is significantly increasing in humans, especially in immunocompromised individuals. Although the most common microsporidium associated with disease in humans in *Enterocytozoon bieneusi*, *Encephalitozoon* spp, were also recognized as potential human pathogens. Some authors defend the zoonotic potential of encephalitozoonosis and state that it could occur through exposure of both animals and humans to contaminated water, food or aerosols. The spores are resistant in the environment and can survive for months under humid conditions The chitinous wall of the microorganism seems to be what offers protection against environmental conditions. The identification of microsporidian organisms in water sources is further indicative of a potential waterborne transmission. Nevertheless, additional studies are required to clarify the true risks of waterborne transmission, as well as of other sources of infection such as food and insects. Better characterizations of the biology and epidemiology of these agents may help to determine risk factors, potential therapeutic drugs for humans and animals, and preventive strategies. Veterinarians are fundamental during this process. They work with a wide range of animal species and may therefore be able to conduct comparative biology studies to complement the understanding of microsporidian infection in different hosts.

-by Ana Carolina Maluenda, Extern
-edited by Dr. Inge Langohr, ADDL Graduate Student

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ADDL NEWS

Several ADDL employees recently celebrated anniversaries for their years of service to Purdue University and ADDL.

Donna Schrader
30 years
Avian Lab Assistant

Melba Campbell
25 years
Business Assistant

Mary Woodruff
40 years
Virology Lab Supervisor

Also celebrating anniversaries were (l to r) **Bob White**, Necropsy Technician (25 years)   **Bonnie Vera**, Bacteriology Lab technician (20 years) and **Charlene Evans**, Histology lab technician (10 years).

Dr. Greg Stevenson, ADDL pathologist for 17 years, left his position at Purdue ADDL to work for Life Action Ministries as Director of Outreach to faculty and content development. Please join us in wishing him well.

Scenes from farewell lunch/reception ⟶

On the Road

Dr. Roman Pogranichniy attended the American Association of Swine Veterinarians meeting in San Diego, March, 2008.

Dr. Steve Hooser participated in a National Animal Health laboratory Network governmental relations meeting with the USDA and FDA in Washington D.C., March 2008

Drs. **Ching Ching Wu, Tsang Long Lin and Tom Bryan** and graduate students **Ying-yi Chen** and **Young-yi Chen** attended the North Central Animal Disease Conference in Minneapolis, March, 2008

Dr. Roman Pogranichniy, Virginie Lazar, Hueling Wei and Ingeborg Langohr attended the Conference for Research Workers in Animal Disease in Chicago, December, 2007.

Histology technicians **Paula Brost, Dee DuSold** and **Charlene Evans** attended the Indiana Society for Histotechnology workshops in Indianapolis, Marcy, 2008.

DIAGNOSTIC FORUM

DIAGNOSTIC FORUM ois a quarterly newsletter of current services, regulations and re-search projects involving the ADDL which may be of interest to Indiana veterinarians and animal owners. It is our intention that the information provided will serve you. Please send your comments, suggestions, requests and questions to: Diagnostic Forum Editor, Animal Disease Diagnostic Laboratory, 406 S. University St., Purdue University, West Lafayette, In 47907.

ADDL SECTION HEADS

Director: H.Leon Thacker, DVM, PhD
Assistant Director: Steve Hooser, DVM, PhD Pathology:
Assistant to Director: Linda Hendrickson, BS, MA
Avian: Tsang Long Lin, DVM, PhD
Bacteriology: Ching Ching Wu, DVM, PhD
Business Manager: Tonya Byrd, BS
Computer: Steve Vollmer, BS

Histology: Jose Ramos-Vara, DVM, PhD
Molecular Diagnostics: R. Vemulapalli, DVM, PhD
Serology: Roman Pogranichniy, DVM, PhD
Toxicology: Steve Hooser, DVM, PhD
Virology: Roman Pogranichniy, DVM, PhD
Heeke ADDL: Tom Bryan, DVM
Duane Murphy, DVM, PhD

VETERINARY PATHOLOGISTS:
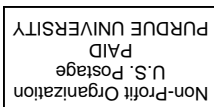Christine Holland, DVM, PhD
Steve Lenz, DVM, PhD
Tsang Long Lin, DVM, PhD
Peg Miller, DVM, PhD
Duane Murphy, DVM, PhD
Jose Ramos-Vara, DVM, PhD
Leon Thacker, DVM, PhD