17 3

Summer 2006

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

The lazy, hazy days of summer in Indiana. Afraid not in the ADDL. Not much lazy or hazy here. Although the testing requirements for exhibition animals in the State have changed so that blood testing of cattle and swine is no longer necessary for county fairs, the void created by the reduced testing requirements in the Lab has been replaced by testing development and requests in other lab areas. Under the direction of Dr. Roman Pogranichniy, the virology and serology sections now have new tests available, including hemagglutination inhibition assay for H1N1 and H3N2 influenza virus infection in swine serum; ELISA test for IBR in bovine serum; virus neutralization test for BRSV antibodies in bovine serum, virus neutralization for porcine enterovirus of serogroups 1-7; IFA test for porcine circovirus antibodies; antigen capture ELISA for canine parvovirus or rotavirus in feces and sequencing of viral isolates of PRRS. The toxicology section is now offering two new tests; one for definitive evidence of starvation by analyzing bone marrow fat and, the second, a test for juglone which is a chemical in black walnut shavings (in past years, it was thought that juglone was the chemical responsible for black walnut toxicity but it has since been disproven). The presence of juglone, however, is thought to substantiate the presence of black walnut in shavings and is of assistance in establishing accurate diagnosis when this toxicity is suspected.

In cooperation with a funded national survey, all eligible swine necropsy submissions to ADDL will be tested for Classical Swine Fever by the PCR method. This method will also be used to test any suspicious birds for Avian Influenza which is getting widespread publicity as human health threat at this time. In assistance with another national survey to be conducted the Indiana DNR, we will also be testing at least 500 birds, most to be submitted during the fall hunting season, for AI. The bacteriology and serology sections are and will be assisting with performing fecal culture and serum ELISA tests for the Indiana Johne's Disease Surveillance program. A change in this program this year will be the requirement of scheduling submissions for fecal culture to accomodate the limited volume of these samples that can be handled by the lab.

The molecular diagnostics area of the Lab continues to increase capacity, particularly for real time PCR testing. Testing using this methodology include PRRS, PCAD, FMD, CSF, END and AI. If a foreign animal disease is introduced to our state or nation, testing in this area of the Lab is predicted to escalate markedly.

Lastly, we are happy to welcome our latest addition to the ADDL faculty, Dr. Steve Lenz. Dr. Lenz joined our faculty June 12 as senior diagnostic pathologist. Steve is a 1981 DVM graduate of the Purdue School of Veterinary Medicine and a 1991 PhD graduate of the Purdue anatomic pathology program. He was board certified by examination by the American College of Veterinary Pathologists in 1992 and has been pathologist/faculty member of the College of Veterinary Medicine, Auburn University, since receiving the PhD degree. We realize the loss to the Auburn faculty effected by Dr. Lenz' departure and for that I apologize, but we also welcome Dr. Lenz to our faculty with enthusiasm; he is a valuable asset to our accomplished ADDL faculty.

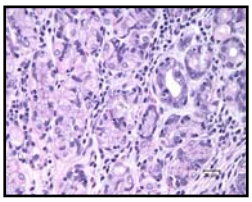
Final Diagnosis:

***Helicobacter* gastritis in a dog**

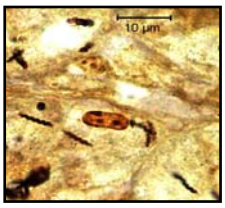
In each issue, we feature a case submitted to ADDL that we hope will be of interest to you.

History: This four-year-old, spayed female, Chinese crested dog had been losing weight for one year. No vomiting or diarrhea had been observed by the owner and the dog had been clinically normal otherwise. There were no apparent findings in the stomach on gross post mortem examination.

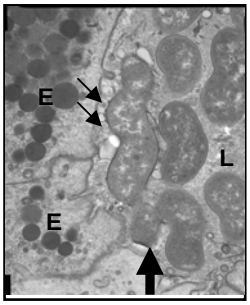
Histopathologic description: There was irregular thickness of the gastric pyloric mucosa. The lamina propria of the pylorus was markedly diffusely infiltrated by lymphocytes and plasma cells with scattered lymphoid follicle formation in the deeper mucosa. The pyloric glandular epithelium was often attenuated (degeneration) and/or infiltrated by few lymphocytes and plasma cells. Rarely, pyloric glands were dilated with mucus and scanty cell debris. In the pylorus, some gastric pits and glands had varying numbers of 6-10 µm long, spiral, gram-negative bacteria. In the fundus, mucosal inflammation and bacterial colonization were minimal.



The pyloric lamina propria is infiltrated by many lymphocytes and plasma cells. (HE, bar = 20µm



Spiral bacteria are present in the lumen and intercellular spaces of pyloric glands (Warthin-Starry silver stain, bar = 10µm



Spiral bacteria in the pyloric glandular lumen (L) have periplasmic fibrils (smaller arrows) and flagella (larger arrow). Pyloric glandular epithelial cells (E) contain numerous electron-dense secretory granules. Transmission electron microscopy, bar = 1µm

Discussion: Severe lymphoplasmacytic gastritis in this dog was associated with intralesional spiral bacteria. Light and electron microscopic characteristics of this bacteria are compatible with *Helicobacter* species. The gastritis might have played a role in the emaciation and weight loss of this dog.

Genus *Helicobacter* encompasses gram-negative, microaerophilic, curved to spiral-shaped bacteria. Chronic gastric inflammation due to *H. pylori* has been associated with increased risk of gastric adenocarcinoma and other malignancies in humans and some animal species.

Several gastric *Helicobacter* species have been isolated from apparently healthy animals or animals with clinical signs of gastrointestinal problems. In dogs, four gastric *Helicobacter* species have been described and the prevalence of these bacteria is generally very high regardless of concurrent clinical signs. *H. (Flexispira) rappini (*sheep, humans, dogs) 4-5 µm, is fusiform, entwined with multiple periplasmic fibers, and has multiple bipolar sheathed flagella. *H. felis* (cats, dogs, humans) is 7-10µm with superficial, sparse periplasmic fibers and multiple bipolar sheathed flagella. *H. heilmannii* and *H. Gastrospirilium hominis* as synonyms (dogs, cats, humans, nonhuman primates, pigs) is 7-10 µm long, has multiple bipolar sheathed flagella, but lacks periplasmic fibers. *H. salomonis* (dogs) is 5-7 µm long with tufts of sheathed flagella at each end. Based on this classification, the majority of bacteria in this dog's stomach are most consistent with *H. felis.*

The majority of gastric *Helicobacter* species infections in animals has been reported to be asymptomatic; however, some animals may show intermittent vomiting, weight loss, or diarrhea. Clinical signs less frequently seen include pica, belching, anorexia, or emaciation. The mode of transmission of *Helicobacter* species is yet to be elucidated although fecal-oral or oral-oral transmission is likely. Until now, there has been no report pointing to a direct relationship between human infection by animal *Helicobacter* species and gastric disorders in humans, even in the case of *H. pylori.*

Diagnosis of *Helicobacter* species infection is usually made by histologic examination of endoscopic or postmortem stomach specimens through demonstration of mucosal inflammation accompanied by organisms. Endoscopic collection of gastric mucus using a brush has been reported to be equally or more sensitive in detection of the organisms although the degree of gastric inflammation is difficult to assess. Commercial rapid urease testing on gastric mucus samples has been used although sensitivity and specificity are variable. Culture of these fastidious bacteria requires modification of routine methods and, thus, is not practical. Electron microscopy and PCR amplification of 16S ribosomal RNA amplicons can be used to differentiate *Helicobacter* species.

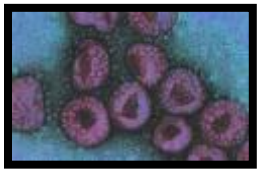
Pathogenicity of gastric *Helicobacter* species relies on their urease production, which increases pH just around the bacterial wall and provides them with an appropriate milieu. Interestingly, because not all animals and humans that harbor these bacteria develop clinical signs, both bacterial virulence and host genetic diversity are thought to be involved in disease initiation, progression and, in some instances, carcinogenesis. The reason the pylorus was selectively inflamed and colonized by *Helicobacter* species in this dog is not clear, although the difference in intragastric distribution of inflammation and bacterial colonization has often been described in human and animal infections.

-by Dr. Ikki Mitsui, ADDL Graduate Student

References:

1. Fox JG: 2006. Gastric *Helicobacter* infections. In: Infectious Diseases of the Dog and Cat. Ed. Greene CE, 4th ed W.B. Saunders Company, Philadelphia. PP 343-351.
2. Happonen I et al: 1996. Occurrence and topographical mapping of gastric *Helicobacter*-like organisms and their association with histological changes in apparently healthy dogs and cats. J Vet Med 43:305-315.
3. Jenkins CC, Bassett JR: 1997. *Helicobacter* infection. The Compendium 19:267-309.
4. Neiger R, Simpson KW: 2000. *Helicobacter* infection in dogs and cats: facts and fiction. J Vet Intern Med 14: 125-133.
5. Peek Jr RM, Crabtree: 2006. *Helicobacter* infection and gastric neoplasia. J Pathol 208:233-248.
6. Simpson KW et al: 1999. *Helicobacter felis* infection in dogs: effect on gastric structure and function. Vet Pathol 36:237-248.

Type A Influenza H5N1



Introduction: A zoonotic disease at the center of the world's attention is Type A Influenza H5N1. It has been labeled "Asian flu" or "Bird flu". Popular press has been quite alarmist! There is significant concern for this disease not only because of its potential impact on the worldwide poultry industry and its markets but, more importantly, its significance to public health.

Influenza, both Type A and Type B, commonly referred to as the "flu", manifests itself in susceptible humans with cold-like symptoms aggravated by fever, malaise, muscle and joint aches, nausea and vomiting. Immuno-compromised, the young, and the elderly are particularly susceptible if unprotected.

Type A influenzas have been subtyped into 16 H and 9 N surface proteins. Type B influenzas, are thought to be more stable, and are more amenable to becoming vaccine candidates. Type A influenza reservoirs are considered to be migratory waterfowl. Among poultry veterinarians, migratory waterfowl are considered to be in "4-F" status – "fowl feathered flu factories". Influenza breaks in domestic poultry have largely been traced to migratory waterfowl or live bird markets to the extent that some state plans for emergency disease planning in poultry discourage poultrymen and service personnel from hunting and processing migratory waterfowl due to the asymptomatic shedding of type A viruses and the ease of fecal-oral transmission to poultry. In poultry, signs of influenza may vary from negligible, with no effect on growth or laying, to extreme mortality, leaving a decimated, cyanotic flock with digestive and respiratory tract hemorrhages.

In 1997, six human deaths in Hong Kong were attributed to a pathogenic strain of type A influenza H5N1. Because of intermingling of migratory and domestic waterfowl and live bird markets, concerns for genetic mutations allowing bird to human transmission were raised. Since 2003, there have been 225 confirmed cases of H5N1 influenza in humans with 128 fatalities. As of this writing, human to human transmission has not been demonstrated.

As of June 1, 2006, H5N1 has been reported from wild and domestic birds in 49 countries in Asia, the middle East, Africa and some parts of Europe.

Etiology: Influenza viruses are of the Orthomyxoviridae family and have three types: type A (found in both humans and animals), type B (found only in humans), and type C (uncommon strain found only in humans). Type A may be subtyped according to two surface glycoproteins. There are 16 H or hemagglutinins and 9 N or nueraminadases, allowing 144 different subtype combinations. For the human, type A provides a more ominous threat due to the assortment of virus components. Recurrent epidemics of type A flu are a constant challenge to the CDC in predicting future subtype candidate viruses.

Epidemiology: Type A influenza viruses in domestic poultry are categorized into Low Pathogenic AI (LPAI), which is the most common, and Highly Pathogenic AI (HPAI), or the lethal form, uncommon in US poultry.

LPAI viruses cause mild infections (no weight loss or respiratory signs) in domestic birds and are not considered a threat to public health. Generally, world marketing agreements do not turn away finished products from an LPAI geographic area.

HPAI virus strains H5 and H7 can cause alarming losses with 90-100% mortality in commercial poultry. HPAI has been a problem in live bird markets of some east coast US cities where the USDA has continually monitored the eradication efforts. H5N1 subtype is an HPAI. It has cost the poultry industry of Asia millions of dollars in bird losses and marketing barriers have been established. H5N1 influenza has been isolated from domestic cats, captive tigers, and

leopards in Asia. In 2005, there was an unusually high death loss of migratory birds due to H5N1 in central China. In the past 30 years, there have been 24 outbreaks of HPAI worldwide; most of these were confined to a single flock or farm.

International vigilance in monitoring avian influenza outbreaks is occurring because LPAI H5 and H7 viruses are capable of evolving into HPAI. Likewise, there is fear that HPAI viruses could transmit to humans through genetic mutation. At present, extensive monitoring for H5N1 virus among waterfowl migrating to US flyways is being carried out across the United States.

Diagnosis: Detection of type A influenza is dependent upon diagnostic tests. Antigen capture screening tests are available. Nasopharyngeal, tracheal or cloacal swabs are used, and results can be determined in a short period of time. Positives are subtyped. ADDL has continuous settings of SPF chicken eggs for virus isolation attempts from tracheas, bronchi, cloacal, or nasopharyngeal swabs. Embryo harvest is followed by electron microscopy, antigen capture immunoassay, and subtyping.

Real time Reverse Transscription Polymerase Chain Reaction (RT-PCR) test is available at ADDL. This test allows greater sensitivity and is useful for large scale screening during epidemics. Results are available quickly. Advantages to this method are small sample size requirement, timeliness, and the ability to subtype. Suitable samples to submit for this test are tracheal and nasopharyngeal swabs of Dacron on plastic sticks (1 swab per tube with 1 ml viral transport media), with no blood contamination, shipped on cold paks.

Serological type A screening, using the Agar Gel Precipitin (AGP) or Agar gel Immunodeficiency (AGID) method, requires a minimal amount of serum and can be read within 1-2 days. In some instances where only eggs were available, yolk antibodies have been tested.

Surveillance and Control (Biosecurity): Surveillance of type A influenza viruses in the Indiana poultry industry is based on monitoring sera for the presence of type A antibodies. The USDA provides antigen and antibody to perform the AGP test to private and public laboratories upon request. The National Poultry Improvement Plan (NPIP) provides protocol and uniform procedures that are acceptable according to world standards. Seropositive samples are submitted to the National Veterinary Services Laboratory (NVSL) for subtyping. Commercial poultry farms exercise biosecurity precautions in housing that limits access by wild birds, unwanted visitors, vermin, and rodent entry. Visitors are required to put on disposable suits, head, hand and shoe covers before entry and removal at exit. Delivery traffic and utility personnel respect biosecurity protocols. Commercial killed vaccines have been advocated and stockpiled by the USDA for commercial poultry use. In a 2004 outbreak of type A influenza (LPAI-H7N2) among commercial poultry in the eastern United States, eradication was the response of choice.

On a regional basis, biosecurity concerns are implemented according to the location of disease. A Level 1 would be the minimum amount of acceptable biosecurity for poultry, while Level IV would indicate that poultry are in imminent threat and biosecurity is at its highest level. A communication network between the Indiana State Poultry Association and poultry producers is being updated in order for sound and current information to be made available. Outdoor poultry that can be exposed to migratory waterfowl, directly or indirectly, as well as farm ponds, remain a threat to disease prevention programs.

Strict surveillance of poultry populations is enforced in most countries in an attempt to contain the H5N1 outbreaks. Continuous research on methods of identifying the constantly changing AI virus is key to finding a solution to this impending global problem.

-by Maria Solacito, ECFVG Student
-edited by Dr. Tom Bryan, Avian Diagnostician, Heeke ADDL

References:

1. Avian Influenza. Field Manual of Wildlife Diseases: General Field Procedures and Diseases of Birds. National Wildlife Health Center/US Geological Survey: 1999. Friend M and Franson C (eds.). 22:181-184.
2. Centers for Disease Control: 2005. Spread of Avian Influenza Viruses Among Birds. www.cdc.gov/flu/avian/gen-info/spread.htm
3. Centers for Disease Control: 2006. New Laboratory Assay for Diagnostic Testing of Avian Influenza A/H5 (Asian lineage). MMWR February 3. pp 55.
4. Lee CW, Senne DA, Linares JA, Woolcock PR, et al: 2005. Characterization of recent H5 subtype avian influenza viruses from US poultry. Avian Path 33(3): 288-297.
5. Newton DW, Mellen CF, Baxter BD et al: 2002. Practical and Sensitive Screening Strategy for Detection of Influenza Virus. J Clin Microbiol 40(11): 4353-4356.
6. Ng EK, Cheng :K, NgAy et al: 2005. Influenza A H5N1 detection. Emerg Infect Dis 11(8)
7. Payungporn S, Chutinimitkul S, Chaisingh A et al: 2005. Single step multiplex real time RT-PCR for H5N1 Influenza A virus detection. J Virol Meth 131: 143-147.
8. Tiensin T, Chaitaweesub P, Songserm T et al: 2005. Highly Pathogenic Avian Influenza H5N1, Thailand, 2004. Emerg Infec Dis 11(11): 1664-72.
9. USDA/APHIS: 2006. Screening for Highly Pathogenic H5N1 Avian Influenza in Migratory Birds. USDA Fact Sheet. March www.aphis.usda.gov.

(references continued p. 4)

10. World Health Organization (Writing Committee of WHO Consultation on Human Influenza A/H5). Avian Influenza A (H5N1) Infection in Humans. New England J Med 353 (13): 1374-85.
11. Xu X, Jim M, Yu Z et al: 2005. Latex Agglutination Test for Monitoring Antibodies to Avian Influenza Virus Subtype H5N1. J Clin Microbiol 43(4): 1953-1955.

Feline Ocular Sarcoma

Feline ocular sarcomas are malignant intraocular neoplasms that are often associated with a history of ocular trauma. Cats appear to be the only species predisposed to the development of this neoplasia and the mean age is 12 years. Affected cats often have a history of penetrating ocular injury resulting in the perforation of the lens, but trauma is not necessary. The duration of ocular disease prior to the detection of the neoplasm ranges from several months to years.

Although this neoplasia appears to be rare, any cat with a history of ocular trauma or chronic ocular disease should be evaluated if there are any changes in the eye. Abnormalities include white discoloration (opacity), bulging and firmness of the diseased eye. Sometimes cats show blindness and neurological signs resulting from neoplastic infiltration of the optic chiasm and brain via the optic nerve.

Feline ocular sarcomas are locally invasive and have metastatic potential. Tumors occupy the posterior iris and expand diffusely into the ciliary body, posterior chamber, retina and choroid. Extraorbital invasion is common and may begin at the limbus or occur through the optic nerve. The lens is invariably destroyed and significant inflammation accompanies tumor growth. Whether this occurs because of the initial traumatic event or is secondary to ocular neoplasia is unclear. The specific histologic diagnosis of tumor types is variable. While fibrosarcoma is the most common diagnosis, osteosarcoma or undifferentiated sarcoma may occur. There does not appear to be any variation in biologic behavior for different histopathologic types. While the cell of origin has previously been unknown, Zeiss CJ et al reported recently that some of these tumors are of lens epithelial origin while others are of myofibroblastic origin. Lens epithelial transformation appears to be limited to cats as these tumors have not been identified in dogs and humans. Also, according to this study, feline ocular sarcomas demonstrate morphologic similarities to feline vaccine-associated sarcomas. The suggested links between the two diseases include trauma, chronic inflammation with cell proliferation, and neoplastic transformation.

Ocular ultrasonography may allow evaluation of the globe prior to surgery, although CT and MRI are more accurate imaging modalities. Aspiration cytology and biopsy of regional (submandibular and retropharyngeal) lymph nodes should be performed if they are enlarged. Metastases to regional lymph nodes have been seen in some cats. Thoracic radiography should be obtained as pulmonary metastases have also been described.

Enucleation is the treatment of choice for feline ocular sarcoma. However, these are very invasive tumors; surgery may be ineffective in cats with extensive local spread. Imaging is important to identify good surgical candidates. When performing enucleating surgery, care must be taken to avoid placing strong traction on the optic nerve. Several anecdotal accounts suggest that animals can become blind due to optic nerve dysfunction in the remaining eye following a routine enucleation. A working hypothesis for this unfortunate outcome is mechanical damage to the contralateral optic nerve at the level of optic chiasm due to surgical traction.

After surgery, metastasis or extension involving the central nervous system can be seen. Radiation therapy may have a role in advanced, invasive ocular sarcomas, although the high dosages that are necessary require sophisticated computer planning equipment to minimize damage to the CNS. Chemotherapy has not proven effective in the treatment of soft tissue sarcoma to date. Doxorubicin and carboplatin could be offered for palliation. However, euthanasia is often elected for affected cats due to the neurologic signs and poor prognosis.

-by Kyunga An, ECFVG Student
-edited by Dr. Pam Mouser, ADDL Graduate Student

References:

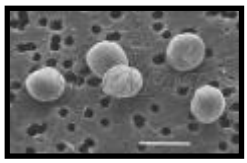
1. Dubielzig RR, Everitt J, Shadduck JA, Albert DM: 1990. Clinical and morphologic features of post-traumatic ocular sarcomas in cats. Vet Pathol 27: 62-65.
2. Dubielzig RR et al: 1994. Morphologic features of feline ocular sarcomas in 10 cats: Light microscopy, ultrastructure and immunohistochemistry. Prog Vet Comp Ophthal 4:7-12.
3. Ogilvie GK and Moore AS: 2001. Feline oncology. Veterinary Learning Systems. 266-267.
4. Peiffer RL and Simons KB: 2002. Ocular tumors in animals and humans. Iowa State Press. 291-292.

Diarrhea in Calves Induced by *Cryptosporidium parvum*

Diarrhea is a common manifestation of intestinal/ systemic homeostatic alteration in neonatal calves, lambs, and kids. Neonatal diarrhea may cause acute dehydration and death or lead to malnutrition and emaciation. *Cryptosporidium parvum* is highly infectious and highly resistant to inactivation in the environment. There is no routinely successful form of therapy available. There is also a zoonotic implication in humans handling the animals, especially in immunocompromised humans.

Etiology and Pathogenesis: Neonatal calf diarrhea usually involves the association of more than one pathogen. The most common implicated pathogens are *E. coli*, rotavirus, coronavirus, and *Cryptosporidium parvum*. *Cryptosporidium parvum* (disease name Cryptosporidiosis) is a protozoan parasite transmitted by fecal-oral contamination. These protozoa invade the apical surface (brush border) of the enterocyte in the distal small intestine and proximal colon and form parasitophorus vacuoles where development occurs. Infection results in crypt and submucosal inflammation, necrosis of microvilli, villous atrophy, and decreased mucosal enzyme activity. This results in decreased absorptive ability of the intestinal tract, fermentation of nutrients within the lumen, and osmotic diarrhea.

Life cycle: Infection begins by ingestion of oocysts from feces. These oocysts contain four sporozoites and initiate infection following excystation. The organism replicates asexually and then sexually to produce new oocysts that are shed into the environment in feces or reinfect the host. The definitive hosts include many mammals such as cattle, dogs, cats, and humans.



Epidemiology: *C. parvum* oocysts are commonly found in the feces of healthy calves. The cause of diarrhea depends on multiple factors such as the degree of virulence of the pathogenic strain, the presence of more than one pathologic agent, and the success of passive transfer of colostral immunoglobulins. Calves with low immunity are highly susceptible to enteropathogenic infections leading to severe and often fatal diarrhea. Also, the lack of specific antibodies in the dam and the use of specific vaccines may interfere in the immunoglobulin transfer to the calf. Stress factors, poor environmental conditions, exposure to contaminated maternal feces as well as feces from healthy calves, and inappropriate diet also increase the risk for disease. Transmission is by fecal-oral contact and fecal aerosol.

Diagnostics: Fecal samples from untreated calves should be submitted during early diarrheic stages. Sugar flotation can be performed to identify the oocysts. Bacteriology and virology cultures can be performed to verify the presence of other agents. The microbiologic interpretation may be difficult because of mixed infection and because some potential enteropathogens are commonly present in healthy calves. Ideally, live representative calves should be presented for necropsy examination so that fresh intestinal sections can be prepared to identify the presence of organisms at the surface of epithelial cells.

Treatment: There is no effective chemotherapeutic agent for routine treatment of cryptosporidiosis, but supportive care is recommended. Paromycin has been used with limited success in cats and human patients, but its efficacy in other species has not been determined. Oral Bovine Serum concentrate has been used in calves with experimentally induced cryptosporidiosis. Hunt and Armstrong determined that there was a 33% reduction in the volume of diarrhea at the peak of illness, 30% reduction in intestinal permeability, and enhanced ideal crypt depth and villous surface area, as compared to untreated, infected calves.

Prevention and control: Good hygiene during management of the entire herd is important in reducing the incidence of cryptosporidiosis. Isolation of sick calves to a separate area to reduce contamination is also important. The dam and calf should be provided good nutrition, and administration of high quality colostrum within the first six hours of birth helps reduce infection.

-by Jorge Araque, ECFVG Student
-edited by Dr. Michael Owston, ADDL Graduate Student

References:

1. Holland RE: 1990. Some infectious causes of diarrhea in young farm animals. Clin Microbiol Rev 3:345-75.

2. Harp JA, Goff, JP: 1995. Protection of calves with vaccine against *Cryptosporidium parvum.* J Parasitol 81:54-57.

3. Hunt E, Fu Q, Armstrong MU: 2002. Oral Bovine Serum Concentrate Improves Cryptosporidial Enteritis in Calves. Pediatric Res 51:370-376.

4.www.cal.vet.upenn.edu/dxendopar/parasitepages_protozoa/c_parvum.htm

5.www.cal.vet.upenn.edu/dxendopar/techniques/comfecal.html

6. www.merckvetmanual.com/mvm

7.www.vetmed.wisc.edu/pbs/zoonoses/Glk9fel/crypto.html

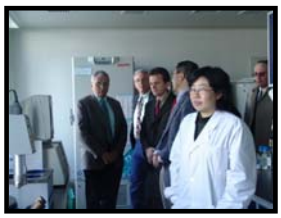
Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2006

July 4, ...Independence Day
September 4..... Labor Day
November 23-24.....Thanksgiving
December 22-26.....Christmas

Purdue Animal Disease Diagnostic Laboratory West Lafayette, IN

Heeke Animal Disease Diagnostic Laboratory Dubois County, IN

On the Road



In March, 2006, **Dr. Roman Pogranichniy**, ADDL Virologist, participated in an Iowa State/USDA/ Purdue University funded trip to China where he and a group from Iowa met with officials from the Ministry of Agriculture. He visited four Universities, including the China Agricultural University and met with faculty, scientists, and potential graduate students to discuss their diagnostic capabilities and potential collaboration. Dr. Pogranichniy learned that faculty members from Zhejiang University were already familiar with Purdue University and had established collaborations.

Dr. Greg Stevenson, ADDL pathologist, traveled to Germany, Portugal, Greece, Hungary, the Czech Republic and Poland as an invited speaker at scientific meetings sponsored by Virtac Animal Health, Carros Cedex, France.

Mary Woodruff (Virology) and **Ron Gillespie** (Bacteriology) attended the Heartland Chapter meeting of the Association of Veterinary Microbiologists in Reynoldsburg, Ohio, April, 2006.

Dr. Tsang Long Lin, ADDL Pathologist, attended the Midwest Fish and Wildlife Committee meeting in Madison, Wisconsin, May, 2006.

ADDL NEWS

ADDL is pleased to introduce our newest faculty pathologist, Dr. Steve Lenz, who joined our staff in June. Dr. Lenz completed his pathology training at Purdue in 1991, was board certified by the American College of Veterinary Pathologists in 1992, and has since been a veterinary pathologist at Auburn University.

Please join us in welcoming Dr. Lenz and his family back to West Lafayette.

ADDL test results are available on the Internet.

Call 765-494-7440 to set up an account or log on to www.addl.purdue.edu
Click on blue "Online Reports tab"
Click on "Request Info" on left navigation bar

From the Virology/Serology Section

The ADDL Virology section is now offering **sequence analysis of PRRSV ORF5 viral isolates.** This test is performed on viruses isolated in cell culture. Cost is $150.00/sample

The ADDL Serology section is now offering a new **cELISA test for detecting antibodies to TGE and PRCV**. Cost is $8.00/sample.

Percent of Micro-organisms that are Resistant to Selected Antibiotics from Jul.- Dec. 2005 and Jan.-June 2006.

Antibiotic	Beef								Dairy										Swine							
	E. coli		Man. haemolitica		Past. multocida		Salmonella sp.		E. coli		Man. haemolitica		Past. multocida		Staph. aureus		Salmonella sp.		Haemophilus sp.		E. coli		Salmonella sp.		Strep. suis	
	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June
Ampicillin	42	35	0	17	0	11	0	89	55	65	33	9	0	11	11	31	56	33	21	16	58	63	64	40	4	4
Ceftiofur	16	13	0	0	0	0	0	89	33	40	0	0	0	0	0	15	38	37	0	0	26	28	46	23	6	2
Chlortetracycline	74	48	0	0	0	0	0	89	79	90	33	0	0	0	nt	nt	63	37	0	2	92	93	85	80	93	76
Clindamycin	0	100	100	100	100	100	100	100	100	98	100	91	86	100	nt	nt	100	100	15	25	100	99	100	100	96	90
Danofloxicin	0	0	0	0	0	0	0	0	0	0	0	0	0	0	nt	nt	0	0	0	0	0	0	0	0	0	0
Enrofloxacin	5	7	0	17	0	0	0	0	13	20	33	9	0	0	nt	nt	0	0	3	0	1	1	0	0	3	1
Erythromycin	100	100	0	33	0	22	100	100	100	97	17	9	18	33	0	0	100	100	15	20	100	98	100	97	86	85
Florphenicol	100	100	0	33	0	56	100	100	100	97	33	0	14	22	nt	nt	97	100	12	0	100	99	100	93	56	54
Gentamicin	11	17	0	33	0	0	0	11	54	49	0	0	14	11	nt	nt	16	10	0	0	14	15	13	13	6	6
Neomycin	26	33	63	67	33	100	0	89	73	78	50	55	29	44	nt	nt	56	30	9	14	45	53	44	17	23	27
Oxytetracycline	84	70	38	50	33	44	0	89	81	93	0	27	43	22	nt	nt	75	47	24	34	92	95	92	90	97	86
Penicillin	100	100	63	33	17	0	100	100	100	97	33	64	36	22	nt	nt	100	100	48	50	100	99	100	100	18	9
Sulphadimethoxine	63	43	75	67	50	89	0	100	61	76	0	55	55	78	89	47	94	87	18	27	74	82	85	87	66	61
Spectinomycin	26	37	100	100	33	56	100	89	70	71	0	91	43	44	nt	nt	72	77	45	42	63	57	87	93	32	16
Sulphachloropyridazine	84	65	0	17	100	78	50	100	94	93	50	9	71	100	nt	nt	94	87	18	16	91	85	92	80	69	65
Sulphathiazole	63	43	63	67	67	56	0	89	75	90	0	55	71	33	nt	nt	75	47	61	59	69	79	46	77	69	63
Tiamulin	100	98	13	50	83	67	100	100	100	98	0	0	71	56	nt	nt	100	100	21	33	100	99	100	100	31	25
Tilmicosin	100	100	0	33	0	56	100	100	99	95	0	9	29	22	nt	nt	100	100	9	3	98	96	100	100	90	86
Triple Sulfa	11	22	0	17	0	22	0	44	67	73	0	9	14	0	nt	nt	28	17	3	2	23	27	10	17	3	2
Tylosin	100	100	100	100	67	100	100	100	100	98	0	91	86	89	nt	nt	100	100	nt	nt	100	99	100	100	nt	nt
number of isolates	19	46	8	6	6	9	2	9	89	78	6	11	11	10	9	13	32	30	33	64	133	175	39	30	112	160

nt - not tested

Percent of Micro-organisms that are Resistant to Selected Antibiotics from Jul.- Dec. 2005 and Jan.-June 2006.

Antibiotic	Canine										Equine												Feline							
	E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus		Staph. intermedius		E. Coli		Salmonella sp.		Staph. aureus		Staph. epidermidis		Strep. equi		Strep. zooepidemicus		E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus	
	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June
Amikacin	0	0	36	35	0	0	0	0	0	0	4	6	0	0	0	0	0	5	0	0	11	0	0	0	29	83	0	0	0	0
Amoxycillin/Clauvulinic acid	21	22	32	13	100	100	0	19	4	3	4	13	29	13	0	0	6	5	0	0	0	10	21	14	57	33	100	100	29	33
Ampicillin	43	47	36	26	100	100	43	57	46	17	20	27	50	13	50	50	47	30	0	0	0	10	32	57	57	33	100	100	57	100
Cefazolin	22	22	68	78	100	100	0	17	2	3	4	13	29	0	0	0	6	10	0	0	5	5	18	14	71	100	100	100	29	33
Cefotaxime	23	22	86	87	100	100	0	17	2	3	4	8	29	13	0	0	6	15	0	0	0	0	18	14	71	100	100	100	29	33
Cefpodoxime	19	21	86	96	100	100	0	17	4	6	4	13	29	0	0	25	18	20	0	0	0	10	11	14	71	83	100	100	29	33
Ceftiofur	18	16	82	91	100	100	0	17	4	3	4	8	38	0	0	0	6	25	0	25	5	5	14	14	71	67	100	100	29	33
Cephalothin	34	29	68	78	100	100	0	17	2	3	12	17	29	13	0	0	6	5	0	0	0	5	25	29	71	100	100	100	24	33
Chloramphenicol	15	17	5	0	52	69	0	0	4	0	16	15	29	0	0	0	0	10	0	0	0	0	14	7	0	0	100	100	0	0
Clindamycin	99	100	82	87	100	100	29	17	14	11	100	98	100	100	0	0	6	25	0	0	5	15	100	100	86	100	100	100	29	33
Enrofloxacin	29	28	32	30	12	31	0	26	12	6	12	2	0	0	0	0	6	5	0	25	5	0	0	14	57	33	0	0	29	33
Erythromycin	95	53	18	17	100	62	29	19	20	19	96	40	0	50	0	0	24	20	0	25	11	20	86	43	14	50	100	67	29	33
Gentamicin	23	19	27	9	0	0	0	7	2	0	16	17	100	0	67	0	12	15	0	0	5	0	4	14	43	17	0	0	0	0
Imipenem	0	0	23	17	0	0	0	17	2	0	0	0	50	0	0	0	6	0	0	0	0	0	0	0	57	33	0	0	14	33
Marbofloxacin	29	26	23	26	0	8	0	26	10	6	8	2	0	0	0	0	0	0	0	25	5	0	0	14	14	33	0	0	29	33
Orbifloxacin	29	28	23	26	12	15	0	24	12	6	12	2	0	0	0	0	0	0	0	0	0	0	0	14	14	33	0	0	29	33
Oxacillin + 2% NaCl	99	100	82	74	100	100	0	17	4	6	100	100	0	100	0	0	12	5	0	0	0	5	100	100	71	100	100	67	24	33
Penicillin	100	100	27	13	100	100	43	57	52	53	100	100	100	100	50	50	41	55	0	0	0	5	100	100	57	33	100	100	57	100
Rifampin	97	94	23	9	100	100	0	0	2	0	100	100	100	100	0	0	0	5	0	0	0	0	96	93	0	17	100	100	0	0
Tetracycline	37	38	45	57	76	85	14	31	28	14	36	42	57	13	33	25	6	5	0	25	21	40	18	43	71	67	0	67	43	0
Ticarcillin	39	44	27	22	4	0	43	57	36	0	20	27	14	13	50	50	35	5	0	0	0	0	29	57	57	33	100	0	57	100
Ticarcillin/Clavulanic Acid	14	16	27	17	0	0	0	17	2	8	0	8	0	0	0	0	6	5	0	0	0	0	11	7	57	33	100	0	29	33
Trimethoprim/Sulphamethoxazole	34	33	5	4	80	92	14	10	10	6	36	38	13	0	33	0	12	5	0	0	0	0	11	21	0	17	100	67	0	0
number of isolates	146	166	22	23	25	13	7	42	50	36	25	48	7	8	6	4	17	20	2	4	19	20	28	14	7	6	1	3	7	3

nt - not tested