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A Quarterly Newsletter from the Indiana Animal Disease Diagnostic Laboratory
at Purdue University, West Lafayette, Indiana 47907 (765-494-7440)



From the Director
Dr. Stephen B. Hooser

When I wrote this, the high temperature in West Lafayette was -1°F! The air outside is cold, but it is January in Indiana and the weather will change soon. However, the faculty and staff of the ADDL are on fire to provide the best diagnostic service possible to the citizens of the State. Despite the economic trials that are facing us all, the world-class ADDL diagnosticians continue to provide prompt and accurate service and somehow find time to develop the new tests needed by Indiana animal owners. Soon we will be feeling the warm mists of spring and admiring the tiger lilies. Don't forget that these lilies are poisonous to cats!

Hot Topics

- Online results are available via the ADDL website – p.3
- Laboratory results can be emailed to you – p.2
- Voicemail – p.2
- Charge instituted for brucellosis and pseudorabies serology testing – p. 2
- Accession fee for serology cases instituted – p.2
- BVD PCR on pooled ear notch samples – p. 3
- New tests for *Brachyspira*, malnutrition, chocolate poisoning – p. 2
- Final report and billing accelerated by standard shipping charges – p.2

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Focus on Histology

The histology laboratory, overseen by Histology Section Head Dr. José Ramos-Vara, is responsible for the preparation of the microscopic slides which are evaluated by ADDL pathologists. In addition to routine H and E and special stains, technicians prepare slides for immunohistochemistry, a procedure used to further characterize neoplastic and infectious diseases.

Technicians are..

Paula Brost, Lab Supervisor, 33 years at ADDL, certified by the American Society of Clinical Pathology

Dawn Burns, Technician, 4 years at ADDL

Dee DuSold, Technician, 5 years at ADDL

Charlene Evans, Technician, 3 years at ADDL, certified by the American Society of Clinical Pathology

Tony Scalone, Technician, 3 years at ADDL



Left front: Dee DuSold
Left back: Paula Brost

Right front: Charlene Evans
Right middle: Tony Scalone
Right back: Dawn Burns

New Serology Charges

By Indiana law, the ADDL does not charge for tests that are mandated by the State. Therefore, in the past, the ADDL has not charged for Brucellosis or Pseudorabies serology testing, nor has it charged its standard accession fee for samples submitted to its Serology section. As these tests are no longer mandated by the State, ADDL will begin charging \$1.50/sample for Brucellosis and \$2.00/sample for Pseudorabies testing in order to recover the cost for these tests. These charges will begin for cases received at ADDL on or after March 1, 2009. In addition, the standard accession fee of \$7.00/case will be charged to all cases submitted to the ADDL, including cases submitted to the Serology section.

Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2009.

May 25.....Memorial Day
 July 3.....Independence Day
 September 7.....Labor Day
 November 26-27.....Thanksgiving
 December 24-25.....Christmas
 December 31-January 1.....New Year

New at ADDL

- ADDL has simplified shipping and sped up final reports by instituting the following standard shipping charges for samples requiring mail-out.

Ground	\$10.00
Priority overnight	\$40.00

Charges for overnight mailing of EIA charts will remain the same

Weekday delivery	\$12.00
Saturday delivery	\$25.00

These shipping charges were determined by averaging all shipping charges over the past year. Institution of a standard charge will increase accessioning efficiency and allow for speedier final reports and billing.

- New tests**

Serology

Brucellosis (existing test)	\$1.50
Pseudorabies ELISA (existing test)	\$2.00
Bovine leukosis cELISA	\$7.00
Porcine parvovirus cELISA	\$4.00
PRRS European strain IFA	\$5.00
PRRS NA strain IFA	\$5.00

Virology

PCV2 ORF sequencing only	\$100.00
PCV full genome sequencing	\$200.00

Toxicology

Methylxanthines (chocolate)	\$25.00
Sulfur in feed	\$15.00

Bacteriology

<i>Brachyspira</i> culture	\$10.00
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Molecular Diagnostics

Avian influenza	\$40.00
BVD PI pooled ear notches	\$25.00

(See p. 3 for details)

- All telephones of faculty and professional staff are now equipped with **voicemail**.
- Laboratory results and estimated billing reports can be **emailed** to you. If you prefer this method of reporting, please call us at 765-494-7440 and let us know your email address.
- Your laboratory results are available via the **internet**. Please see page 6 for instructions.

BVD PCR on pooled ear notches

A PCR test for Bovine Viral Diarrhea Virus (BVDV) on ear notches or serum, on pools of up to 25 samples, will now be offered at the ADDL at a cost of \$25.00/pooled sample. If the pooled sample is positive, ear notches or serum samples in that pool will be re-tested individually by antigen capture ELISA at a cost of \$3.00/individual sample. Submit individual fresh samples in 5 ml sterile snap cap tubes, labeled with the animal identification and a completed ADDL submission form. Specify that you are requesting the pooled BVD PCR. **Samples will be pooled at ADDL.**

It is strongly recommended that ear notchers be disinfected in 10% bleach after each sample is collected. Do not vaccinate or tattoo animals at the same time ear notches are taken.

ADDL test results are available on the Internet.

To set up an account:

- Log on to our website www.addl.purdue.edu
 - Click Online Reports tab
 - Click Request Info and follow instructions
- Or
- Call ADDL at 765-494-7440 and speak to the Computer Systems Manager



Bone marrow fat test for malnutrition

Definitive diagnosis of animal malnutrition can be challenging. The Analytical Toxicology section has developed a quantitative test which can help in the diagnosis of malnutrition. By measuring the amount of fat in the bone marrow and comparing it to the normal amounts, the percentage of bone marrow fat can be determined and be used to support necropsy diagnosis.

Final Diagnosis:

Lawsonia intracellularis in a horse

History: A 4-month-old Tennessee Walking horse filly was submitted dead to the ADDL for necropsy. The filly was hypothermic and had a history of diarrhea of a few days duration. The filly became depressed and began exhibiting neurological signs such as head pressing, mydriasis, and

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

decreased menace response. Fluid therapy, as well as plasma and flunixin, were administered. Clinical laboratory abnormalities included hypoproteinemia, azotemia, neutrophilia, lymphocytosis, and several electrolyte imbalances. The horse was euthanized.

Gross findings: Segmental areas of jejunal serosa were purple, with prominent serosal and mesenteric veins. Throughout the jejunum and ileum, intestinal mucosa was markedly thickened, assuming a cerebriform appearance. Multiple areas of jejunal mucosa were covered with a thin layer of tan fibrin. In severely affected regions, intestinal wall, including tunica muscularis, was markedly thickened, measuring over 1 cm in thickness. Circular foci of mucosa, ranging in size from 1-2 cm, were slightly raised and red; duodenal mucosa was diffusely red to dark red. The large colon, small colon, and cecum contained copious amounts of malodorous, dark brown, liquid feces. Nematodes consistent with *Parascaris equorum* were found within the intestine.



The thorax contained approximately 1-2 liters of clear, straw-colored fluid. The cranioventral portions of both cranial lung lobes were wet and heavy, with interlobular septa expanded by edema fluid. The abdomen contained approximately 1 liter of clear, straw-colored fluid.

Histologic findings: Ileal mucosa was markedly expanded by hyperplastic crypts which contained numerous mitotic figures and decreased goblet cells. Several crypts were tortuous and branching. Many crypts were dilated and filled with necrotic debris and degenerate leukocytes. Large foci of mucosa were necrotic, characterized by diffuse loss of tissue architecture that extended into underlying submucosa. Numerous leukocytes, including lymphocytes and neutrophils, expanded lamina propria replacing some intestinal crypts. Peyer's patches contained decreased numbers of lymphocytes and karyorrhectic lymphocytes. Submucosa was diffusely expanded by clear edema fluid. Similar changes were observed within the duodenum and jejunum, and were consistent with proliferative and necrotic enteritis.

Alveoli and interlobular septa within the lung were expanded by lightly eosinophilic material, consistent with pulmonary edema. Clear space surrounded arterioles

within cerebral white matter, giving adjacent neuropil a lacy appearance.

Histologic changes in the brain were consistent with edema.

Ancillary findings: Two potential inhabitants of the gastrointestinal system, *E. coli* and *Aeromonas caviae*, were cultured from the intestine. *Salmonella* culture was negative. Fecal flotation found numerous ova consistent with *Parascaris equorum*.

A section of jejunum tested positive for *Lawsonia intracellularis* via PCR. A Warthin-Starry stain was applied to sections of jejunum and ileum, and numerous intracytoplasmic bacteria were located in the apical portion of enterocytes lining hyperplastic crypts.

Discussion: Characteristic gross and histopathologic lesions, coupled with positive PCR, were consistent with proliferative enteropathy in this foal. The causative agent is an obligate intracellular and gram-negative bacteria that is most often associated with proliferative ileitis in swine. To date, several species have reportedly developed disease due to *Lawsonia*, including horses, hamsters, dogs, and rabbits. Although the histopathologic diagnosis “proliferative enteropathy” was made in a foal as early as 1982, the first reported association between this disease in foals and *Lawsonia* was made by authors from Kentucky in 1996.



Lawsonia- caused proliferative enteritis occurs sporadically in horses, with both individual cases and outbreaks on breeding farms. Foals from 3-13 months are most commonly affected. The most common clinical signs include diarrhea, colic, weight loss, and ventral or submandibular edema, all of which can be fairly acute. Common clinical pathologic abnormalities usually reflect a marked hypoproteinemia due to loss of protein through affected intestine. Leukocytosis is also a common abnormality. Antemortem diagnosis of this uncommon equine disease requires exclusion of other, more common, causes of diarrhea and colic in foals. If proliferative enteritis is suspected after other causes have been excluded, fecal PCR for *Lawsonia intracellularis* and serology can be used to aid in diagnosis.

The above cases differ from previous reports of proliferative enteritis as this foal rapidly developed severe neurological signs such as head pressing. Because of previous farm history and recent diagnoses on the same farm, *Lawsonia intracellularis* was the likely cause of diarrhea in this foal. Indeed, *Lawsonia* was confirmed histologically and via PCR. Clinical pathology and gross lesions were consistent with severe hypoproteinemia; thus, cerebral edema was suspected as the underlying mechanism for manifestation of neurologic signs. Histopathologic examination of the brain supported this hypothesis as lesions suggested cerebral edema. No other cause of neurologic disease was observed.

-by Dr. Grant Burcham, ADDL Graduate Student

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Feline Heartworm Disease

Heartworm disease is a widely discussed topic in veterinary clinics throughout Indiana. It is one of the main diseases about which dog owners are informed, and measures are taken to prevent its occurrence. Heartworm disease is not addressed as often when speaking to cat owners, even though feline heartworm disease has been increasing in incidence over the past 10 years.

Heartworm disease requires the mosquito vector in order to develop. The adult worm, located in the pulmonary artery, right atrium, and/or right ventricle, releases microfilariae that circulate through the blood stream. These microscopic larvae can be ingested by the mosquito during a feeding. Within the mosquito, the larvae undergo further development to the L3 stage, and are re-released into the circulation of another animal during a subsequent feeding by the mosquito. These L3 larvae migrate and develop into L4 and finally L5 stages to become mature heartworms.

In general, the prevalence of feline heartworm disease is about 5-10% of that in dogs in a given area. The rate of infection depends on the mosquito species in that area, most notably the *Culex* spp. and *Aedes* spp., as well as the mosquito's preference for feeding on dogs versus cats. In addition, cats appear to be more resistant to infection by the causative agent of heartworm disease, *Dirofilaria immitis*. Cats typically have only a short, transient phase of microfilaremia and low worm burdens in contrast to dogs that typically have higher adult worm burdens and a much longer period of microfilaremia. Reports also show that cats are more susceptible to aberrant migration of L4



larvae which have been found in the central nervous system and cutaneous tissues. The presence of a bacterium, *Wolbachia*, which is consistently found in heartworms, may have a role in the immune response against *D. immitis* in both dogs and cats, especially after death of an adult worm or during release of microfilariae.

The typical clinical presentation of heartworm disease in the cat is quite different than what one would expect in the dog. While dogs typically show a progression of pulmonary and cardiac clinical signs, cats often present in an acute dyspneic crisis and some may acutely collapse and die. Signs in cats are primarily associated with the initial migration of immature worms through the pulmonary arteries as well as with the death of an adult worm. In the interim, there are often no clinical signs. Most heartworm disease lesions in the cat are found in the lungs at necropsy. They include muscular hypertrophy of the pulmonary arteries and arterioles, diffuse infiltration of large numbers of inflammatory cells within the intima of the pulmonary arteries, interstitial fibrosis, and increased macrophages within the alveoli. It is not uncommon to find these pulmonary changes in cats even when no worms are found at necropsy. In areas of endemic feline heartworm disease, only 4-5% of cats that tested serologically positive for heartworm infection actually had worms present at necropsy. Cats appear to have more dramatic hypersensitivity reactions to initial infection of larvae and, therefore, may have permanent pulmonary changes even if they have been able to reject a full-blown heartworm infection.

It can be difficult to diagnose heartworm disease in the cat as the serological tests often used in dogs need to be interpreted differently in the case of a feline infection. The antigen tests detect a protein found in the reproductive tract of the adult female worm. Sensitivity for antigen tests tends to be lower in the cat due to the lower heartworm burden and the frequent single-sex or even single worm infections. However, the antigen tests do have a high specificity, so few false positives will occur. The interpretation of the feline heartworm antibody can also be problematic. The antibody tests may be positive simply due to antibody response to previously circulating microfilariae or a previous adult worm infection. Therefore, false positives are more likely with the antibody test. To increase both sensitivity and specificity, it is recommended to combine both the heartworm antigen and feline heartworm antibody tests, along with thoracic radiography and potentially echocardiography.

Heartworm disease in the cat is typically self-limiting, and therefore is often treated symptomatically with corticosteroids, oxygen therapy, and furosemide. Heparin or aspirin are used as anti-thrombotic agents because heartworm-positive cats can present with pulmonary thromboembolism. Surgical removal of worms is also a possibility; however a life-threatening, acute anaphylactic reaction is highly probable if a worm is damaged during the procedure.

Monthly heartworm preventative is commonly prescribed for dogs in Indiana, but it is also recommended for cats living in endemic heartworm regions. Indoor cats are just as likely to be infected with heartworm disease as outdoor cats. There does not appear to be a predilection for gender or age, and infection can occur despite a healthy immune system.

Recent literature has shown that heartworm disease continues to be an important topic in both canine and feline medicine, and it should be considered when cats present with acute dyspnea or even sudden death.

-by Sarah College, Class of 2009

-edited by Dr. Abigail Durkes, ADDL Graduate Student

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Percent resistance to antimicrobials from selected animal pathogens --data supplied by Bacteriology Section, ADDL, Purdue University (periods of Jan.-June and July-Dec. 2008) Please note that when the veterinary break points are not available, the human ones are used.

Antibiotic	Canine						Equine						Feline																			
	E. Coli		Psc. aeruginosa		Staph. aureus		Staph. intermedius		E. Coli		Salmonella sp.		Staph. aureus		Staph. epidermidis		Strep. equi		Strep. zooepidemicus		E. Coli		Enterococcus sp.		Psc. aeruginosa		Staph. aureus					
	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	July-Dec.		
Amikacin	3	0	64	64	0	0	9	11	0	17	7	7	0	0	47	33	18	0	0	100	7	0	0	0	0	100	0	0	0	20		
Amoxicillin/Clavulanic acid	22	20	29	24	100	100	13	19	0	17	19	7	0	13	40	0	36	30	0	0	0	0	0	13	4	100	50	100	100	0	0	
Ampicillin	43	50	36	32	100	100	79	70	67	67	39	26	57	13	100	100	55	40	0	0	0	7	0	25	25	100	50	100	100	50	0	
Cefazolin	24	24	82	68	100	100	13	19	0	17	16	15	57	13	40	0	32	30	0	0	0	0	0	19	9	100	100	100	100	0	0	
Cefoxitin	21	19	89	88	100	100	13	19	0	17	19	7	0	13	40	0	32	30	0	0	0	0	0	13	4	100	100	100	100	0	0	
Cefpodoxime	24	24	93	80	100	100	15	20	0	17	13	19	0	13	40	0	46	30	0	0	7	0	0	13	9	100	100	100	100	0	0	
Ceftiofur	22	23	89	76	100	100	14	19	0	17	13	15	0	13	40	0	41	30	0	0	21	0	0	6	4	100	100	100	100	0	0	
Cephalothin	31	27	78	76	100	100	13	19	0	17	19	19	57	13	40	0	23	30	0	0	7	0	0	19	9	100	100	100	100	0	0	
Chloramphenicol	16	20	21	8	78	70	3	5	0	17	16	15	0	13	0	0	14	0	0	0	0	0	0	13	4	0	25	0	0	0	0	
Clindamycin	100	100	93	84	100	100	42	40	0	67	100	100	100	100	53	33	67	60	0	0	8	0	0	100	100	100	100	100	50	60	0	
Enrofloxacin	24	29	57	44	17	22	24	18	0	0	7	7	0	0	0	0	41	10	0	100	7	0	0	6	8	100	50	0	0	0	0	
Erythromycin	100	100	54	48	100	100	41	41	0	50	100	100	100	100	53	33	59	60	0	0	36	43	0	100	100	75	100	100	50	60	0	
Gentamicin	18	16	43	36	6	0	8	11	0	17	26	15	0	0	47	33	18	0	0	0	0	0	0	13	8	0	100	0	0	20	0	
Imipenem	0	0	39	28	0	4	72	69	0	17	0	0	0	0	80	22	41	30	0	0	0	0	0	0	4	100	50	0	0	100	80	
Marbofloxacin	23	29	nt	nt	nt	nt	26	18	0	0	7	7	0	0	0	0	41	10	0	0	7	0	0	6	9	nt	nt	nt	nt	0	0	
Orbifloxacin	24	29	46	24	28	26	26	18	0	0	nt	nt	nt	nt	nt	nt	nt	nt	0	nt	nt	nt	nt	6	9	100	50	0	0	0	0	
Oxacillin + 2% NaCl	100	100	89	92	100	87	13	19	0	17	100	100	100	100	40	0	23	30	0	0	7	0	0	100	100	100	100	100	100	0	0	
Penicillin	100	100	36	24	100	100	100	69	67	100	100	100	100	100	100	100	55	40	0	0	7	0	0	100	100	50	100	100	100	100	0	
Rifampin	98	92	18	28	100	100	0	2	0	17	100	100	100	100	0	0	5	30	0	0	7	7	0	100	78	100	75	100	100	0	0	
Tetracycline	32	34	65	72	100	100	42	39	33	83	36	30	57	13	47	33	32	20	0	0	43	14	0	31	13	100	75	100	100	0	40	
Ticarcillin	39	47	32	24	17	22	18	19	0	17	29	22	57	0	50	0	23	30	0	0	7	0	0	25	22	100	50	0	0	0	0	
Ticarcillin/Clavulanic Acid	14	15	36	24	6	9	13	19	0	17	0	7	57	13	40	0	23	30	0	0	0	0	0	6	0	100	50	0	0	0	0	
Trimethoprim/Sulphamethoxazole	26	31	18	24	89	83	19	19	0	17	42	37	0	13	40	11	33	10	0	0	0	0	0	7	13	100	75	100	0	20	0	
number of isolates	110	132	28	25	18	23	100	131	3	6	31	27	7	8	15	9	22	10	0	1	14	14	16	23	1	4	1	4	1	2	4	5

nt - not tested

Antibiotic	Beef						Dairy						Swine															
	E. coli		Man. haemolítica	Past. multocida	Salmonella sp.	E. coli	Man. haemolítica	Past. multocida	Staph. aureus	Salmonella sp.	Haemophilus sp.	E. coli	Salmonella sp.	Strep. suis														
	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.												
Ampicillin	39	47	0	0	0	33	33	33	33	76	68	0	0	100	0	0	0	0	13	18	41	70	52	48	2	2		
Ceftiofur	15	7	0	0	0	33	33	33	33	46	34	0	0	0	0	0	0	0	52	63	36	24	19	21	2	3		
Chlortetracycline	78	43	0	0	0	33	33	33	33	94	81	0	33	100	33	nt	nt	52	74	19	45	94	90	77	69	93	87	
Clindamycin	100	100	100	2	100	100	100	100	100	100	100	100	100	100	100	100	100	100	100	13	27	100	100	100	100	100	85	81
Enrofloxacin	12	29	0	0	0	33	0	33	0	23	23	0	0	0	0	0	0	0	4	0	9	0	2	0	0	5	6	
Florphenicol	61	43	0	0	0	33	33	33	33	73	58	0	33	0	33	nt	nt	57	66	0	27	59	46	52	38	0	1	
Gentamicin	17	0	0	0	0	33	0	33	0	58	37	0	0	0	0	0	0	0	4	0	63	36	21	23	7	17	8	
Neomycin	22	29	0	1	0	50	33	0	33	89	67	67	67	100	100	nt	nt	35	69	56	82	55	41	29	24	27	25	
Oxytetracycline	81	43	0	1	33	50	33	33	33	97	84	33	67	100	100	nt	nt	57	74	50	55	95	92	81	69	97	92	
Penicillin	100	100	nt	0	0	100	100	100	100	100	100	nt	0	100	100	nt	nt	100	100	100	55	100	100	100	100	100	7	13
Spectinomycin	34	29	0	2	0	50	67	0	67	79	67	33	33	100	0	nt	nt	57	40	33	64	57	64	68	45	17	19	
Sulphadimethoxine	68	47	33	0	0	100	100	100	100	78	69	50	67	100	60	0	10	83	94	13	9	81	78	94	79	70	77	
Tiamulin	95	86	0	0	0	100	100	100	100	97	100	0	0	0	33	nt	nt	100	100	0	9	100	93	100	100	10	19	
Tilmicosin	100	100	0	0	0	100	100	100	100	100	100	0	33	0	67	nt	nt	100	100	6	18	100	100	100	100	100	81	78
Triple Sulfa	22	21	0	0	0	33	0	33	0	77	61	0	33	100	33	nt	nt	13	6	0	18	26	27	10	14	7	6	
Tylosin	nt	nt	nt	2	100	nt	nt	nt	nt	nt	nt	nt	100	100	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt
number of isolates	11	14	3	2	3	4	4	4	3	62	57	4	3	10	10	10	9	10	23	35	16	11	106	93	31	29	59	118

nt - not tested

DIAGNOSTIC FORUM

Diagnostic Forum is a quarterly newsletter of current services, regulations and research 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, Purdue ADDL, 406 S. University St., West Lafayette, IN 47907 or email to addl@purdue.edu.

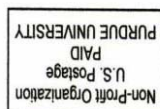
ADDL SECTION HEADS

Director: Steve Hooser, DVM, PhD
Assistant to Director: Linda Hendrickson, BS, MA
Avian: Tsang Long Lin, DVM, PhD
Avian: Pat Wakenell, DVM, PhD
Bacteriology: Ching Ching Wu, DVM, PhD
Business Manager: Tonya Byrd, BS
Computer Services: Steve Vollmer, BS

Histology: Josè Ramos-Vara, DVM, PhD
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