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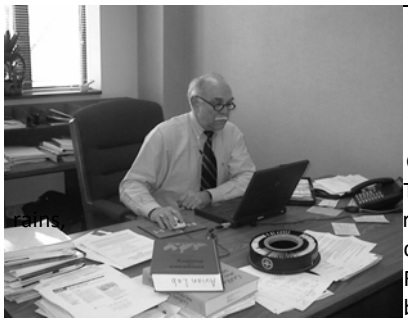
A Quarterly Newsletter from the Animal Disease Diagnostic Laboratory
Purdue University, West Lafayette, Indiana 47907 (765-494-7440)



FROM THE DIRECTOR

H. Leon Thacker

Good day from ADDL. I hope this finds each of you dry and enjoying summertime. This spring has been difficult to contend with in much of our state. Frequency of rains, flooding, crop losses, properly losses and bad haying weather has been discouraging. In much of the state, however, the corn crop looks very good. Fortunately, we have received very reports or diagnostic submissions for problems brought about by the floods. Considering the extent of the flooding losses, direct detriment to animals and birds was of limited extent.

On July 1, we welcomed four new veterinarians/students to the anatomic pathology graduate program. These individuals come to us with much enthusiasm and excellent backgrounds of veterinary education accomplishment. The students are Dr. Abby Durkes, a Purdue SVM graduate, Dr. Tiffany Reed, a graduate from the University of Georgia College of Veterinary Medicine and Drs. Chad Frank and Ryan Jennings, both graduates of the Michigan State College of Veterinary Medicine. With the nationwide shortage of veterinary pathologists, I am sure these individuals will make valued contributions to some of the need for qualified veterinary pathologists upon completion of their advanced degree programs.

Among the dilemma diagnostic cases we have had of late are a number of young captive, white tail deer with clinical signs of swollen heads; some recover with treatment, some have died. So far, it appears that this is necrobacillosis. The means of spread and means by which the clinical expression is so consistent are yet under investigation. The ADDL continues to perform necropsy examinations on all horses that die on either of Indiana's two race tracks. These examinations include checking for administration of unauthorized compounds; to date, all have tested negative. We will again be running tests on urine from winning animals of the Indiana State Fair to detect unauthorized drug administration also. Our state fair testing program in Indiana has been copied by several other states in attempt to detect and stop the drugging of show animals.

As this may be my last memo "From the Director", I would like to say that my time as Director of the Purdue/Indiana Animal Disease Diagnostic Laboratories has been an excellent run. The employees, students, and supporters of these Laboratories are exemplary in their enthusiasm, aim for perfection and dedication to providing the best veterinary diagnostic service available anywhere. I am eternally grateful to these individuals as well as to the users of the Laboratories for their dedication and commitment. I look forward to continuing on the faculty of ADDL for at least another year as a working and teaching veterinary pathologist. It is a great place to work, I appreciate it immensely.

After 23 years as Director of the Purdue ADDL, Dr. Leon Thacker is stepping down from that position as of August 1, 2008.

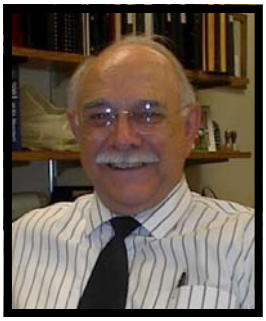
An Indiana native, Dr. Thacker earned his DVM at the Purdue School of Veterinary Medicine in 1965. After 7 years in private practice in Kentucky, he returned to graduate school at Purdue to study veterinary pathology, completing his PhD in 1976 and, in 1977, became a diplomate of the American College of Veterinary Pathologists. He became Assistant Director of the Purdue ADDL in 1978 and continued in that position until he was appointed Director in 1985.

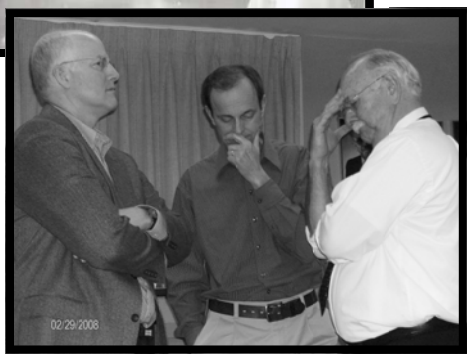
During his tenure, Dr. Thacker has served on countless community, university, state, and national committees including a term as President of the American Association of Veterinary Laboratory Diagnosticians, a national association for veterinary pathologists. AAVLD is also the accrediting body for diagnostic labs nationwide and Dr. Thacker served as Chairman of the Accreditation Committee of that organization for 10 years.

His many honors and awards include the Purdue School of Veterinary Medicine Faculty Award for Excellence in Service, the PUSVM Distinguished Alumnus award, the Raymond E. Plue Outstanding Teacher Award. the PUSVM Award for Excellence in Engagement, the Indiana Veterinary Medical Association Veterinary Service Award, the Indiana Veterinary Medical Association President's Award, and the AAVLD Pope Memorial Award.

Instrumental in planning and securing the new ADDL building, one of his proudest moments was realized at the dedication of the new West Lafayette facility in 1991.

We look forward to many more years of his expertise, his humor, his dedication, and his commitment to ADDL staff and clients.





FINAL DIAGNOSIS:
EHD virus infection in a Southern Indiana cow

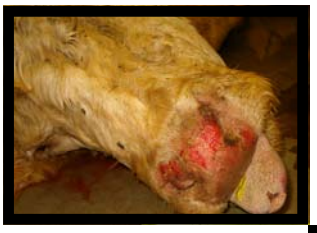
In August 2007, Heeke ADDL was presented with a 9-year-old Charolais cow with a 7 day clinical history of lethargy, standing in water (suspected fever), poor appetite and weight loss. A total of 3 cows in this herd of 42 were exhibiting similar clinical signs, though this was the only cow that had died.

Grossly, the cow had ulcerative and necrotizing rhinitis, stomatitis, reticulitis, and teat dermatitis, along with pulmonary congestion and edema and fetal mummification. The epithelium of the nares was markedly reddened and necrotic and had extensive areas where the epithelium was sloughed. The nasal turbinate mucosa was diffusely reddened but otherwise grossly unremarkable. The dental pad and the epithelial ridges of the palate were severely sloughed and ulcerated. The buccal mucosa had multiple 1-2 mm red shallow erosions, often surrounding the buccal papillae. The gingival mucosa surrounding the lower incisors was yellow-gray and necrotic, with hyperemic margins. The tip of the tongue was dark, dry, and sunken (necrotic), and the epithelium on the ventral midline of the tongue was white, raised (possible remnant of a vesicle), and focally sloughed. The epithelium of the lower lip was yellow-gray, dry, cracked and necrotic. A 3-cm area of the mucosa of the reticulum was gray and dry (necrotic) with hyperemic margins. Esophagus, rumen, abomasum and intestine were grossly unremarkable. The skin of the teats had multiple 1-3 mm red erosions and shallow ulcers. The uterus contained a 12x14x30 cm mass of fetal bones (severely mummified fetus).

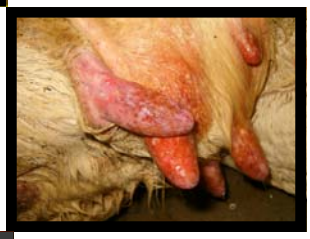
Histologically, the cow had erosive and ulcerative stomatitis, nasal dermatitis, rhinitis, and reticulitis. The oral mucosa had multifocal full-thickness necrosis and ulceration with numerous superficial bacilli, marked congestion of the superficial submucosal blood vessels, and mild perivascular lymphocytic infiltrates. The tongue had a large focal ulcer, with coagulation necrosis of the underlying vessels. Some blood vessels of the tongue contained fibrin thrombi, and colonies of thin filamentous bacilli were present in some of the deeper areas of necrosis. The nasal tip epidermis had necrosis and separation (sloughing) of the epidermis, marked necrosis of the superficial dermis, infiltration of degenerating neutrophils into the superficial dermal papillae, and marked congestion of the superficial dermal capillaries. The nasal turbinate submucosa was congested and edematous, and heavily infiltrated with degenerating neutrophils.

Based upon the time of year, the lesions (especially the characteristic dental pad necrosis and teat lesions), and the presence of an epizootic hemorrhagic disease (EHD) outbreak in the local whitetail deer population, infection with EHD virus was strongly suspected. The differential diagnoses included BVD, IBR, Malignant catarrhal fever, foot-and-mouth-disease and rinderpest. Tissues were submitted to the Foreign Animal Disease Laboratory at Plum Island, New York, and no foreign animal diseases were detected. Lip, oral mucosa, tongue, reticulum, teat, lung, spleen, and lymph node were submitted for fluorescent antibody (FA) testing and virus isolation. FA tests did not detect BVD, IBR, EHD virus in any of the tissues, but EHD virus was isolated from the lung, spleen, lymph node, and oral mucosa. Post-mortem pericardial fluid was submitted for serologic testing, and was found to be positive for EHD by agar gel immunodiffusion (AGID). ELISA testing for Bluetongue virus was negative. A tissue pool was submitted to the National Veterinary Services Laboratory in Ames, Iowa, and PCR tests were negative for alcephaline herpesvirus-1 and ovine herpesvirus-2 (the causes of malignant catarrhal fever). EHD viral RNA was detected in the sample tested by PCR. EHD virus was then isolated from the pooled tissues by inoculation onto BHK-21 cells and cattle pulmonary artery endothelium cells, and was determined to be EHD type-2 by virus neutralization testing. No bacterial pathogens were isolated from the lung and liver. Based upon the characteristic clinical signs and lesions, and the isolation of EHD virus from the affected tissues, bovine EHD was diagnosed in this case.

-by Dr. Duane Murphy, Heeke ADDL



Erosion of nasal mucosa



Epithelial erosions of the teats

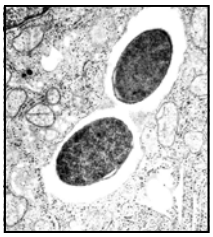
Erosion of dental pad and hard palate

ADDL Schedule

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

September 1……Labor Day
November 27-28……Thanksgiving
December 25-26……Christmas

Clostridium perfringens in Neonatal Pig Diarrhea



Clostridium perfringens is a gram-positive, endospore forming, anaerobic bacilli that is an important pathogen to both humans and animals. It can normally be found in the gastrointestinal tracts of domestic animals as part of the normal flora and also in the soil. There are five different types: A,B,C,D, and E, determined by which of the four major toxins the bacteria produces. The four traditional major toxins are alpha, beta, epsilon, and iota, although *C. perfringens* can produce about 15 different toxins altogether, including enterotoxin, perfringolysin, collagenase, lambda toxin, hyaluronidase, DNase, neuraminidases, and urease. Enterotoxin is implicated in cases of human food-borne illness. The more recently identified cpb2, a beta 2 toxin, is believed to be a major contributor to disease in piglets.

The two most common types of *C. perfringens* that cause diarrhea in pigs are types A and C. Traditionally, type C was the most commonly implicated bacteria in Clostridial diarrhea. Type C is found in extremely low numbers in the normal GI tract and is typically found in very high numbers in pigs with disease. It produces alpha and beta toxins. Beta toxin is important in causing a necrotizing, hemorrhagic disease. *Clostridium perfringens* type A is a normal inhabitant of the colon in pigs and the pathogenesis of disease is not well understood. In a study from the Netherlands, type A was the most commonly isolated clostridial type from pigs with diarrhea. Type A produces the alpha toxin, as do all types of *C. perfringens*. However, in experimental studies, the alpha toxin alone did not demonstrate disease. Both type A and C can produce the cpb2 toxin. In a study by Klaasen, et al., a large percentage of the diarrheic piglets had *C. perfringens* isolates that produced the cpb2 toxin. It is not believed that the cpb2 toxin plays a role in causing enteritis in piglets with type C and, more importantly, in causing disease in type A infections.

Clostridium perfringens is a primary pathogen, but it can colonize the intestines after other diseases, such as transmissible gastroenteritis, coccidiosis, rotaviral enteritis, and porcine epidemic diarrhea. The organism is transferred by direct contact between infected piglets and, most importantly, from the sow. The spores can also persist in the environment and are resistant to heat, disinfectants, and ultraviolet light. The disease is most common in 3-day-old piglets, but can affect piglets from 12 hours to 7 days old. Risk factors for young animals include an immature GI tract, immature intestinal flora, and the relative lack of trypsin, which can inactivate the beta toxin found in *C. perfringens* type C.

Clostridium perfringens multiplies to large numbers in a matter of hours, then attaches to jejunal epithelial cells at villus apices. The toxins produced are cytotoxic and affect tight junctions and ion transport systems leading to loss of fluid, electrolytes, and necrosis. Desquamation occurs and the organism proliferates along the basement membrane. In type C infection, necrosis of the villous lamina propria with hemorrhage is extensive and the necrotic zone advances to involve crypts, muscularis mucosa, submucosa, and muscular layers. In type A infection, attachment and invasion are not as common and induces a more secretory diarrhea by affecting the tight junctions. The jejunum and ileum are mainly affected, but both infections can spread to involve parts of the colon.

Clostridium perfringens type C typically affects 0-7 day old piglets. The fertility rate varies, but is typically over 50%. The disease can present as peracute, acute, or chronic. Peracute cases have signs of intense abdominal pain, depression, weakness, decreased temperature, and bloody diarrhea; death typically occurs within 24 hours. In acute cases, piglets may survive for 1-2 days after clinical signs, have reddish-brown diarrhea with gray shreds of tissue debris, are dehydrated with loss of body condition, and become weak. In chronic cases, piglets tend to remain active, alert and appetent, but become progressively thin and dehydrated with intermittent yellow-gray mucoid diarrhea. In *C. perfringens* type A infection, piglets are affected within their first week of life and develop creamy or pasty diarrhea which may become mucoid and pink. Piglets may recover, but tend to be stunted.

Features identified during necropsy examination of pigs with peracute and acute *C. perfringens* type C include an edematous abdominal wall, intense small intestinal hemorrhage localized to the jejunum and ileum, emphysema in the wall of the intestine, and variable peritonitis with bloodstained abdominal fluid. The intestinal contents are bloody. Mesenteric lymph nodes may be reddened. Deposition of urate crystals in the kidney is common due to severe hydration. Chronic cases typically have adhesions between thickened, well-defined affected areas of small intestine. The mucosa is oftened covered by a tightly adhered necrotic membrane. Pigs with *C. perfringens* type A infection have a flaccid, thin-walled, gas-filled small intestine with mild mucosal inflammation and typically no blood.

Histopathologically, pigs with *C. perfringens* type C enteritis have necrotic jejunal villi carpeted by large gram-positive bacilli with profuse hemorrhage. The necrotic area is homogeneous and eosinophilic with scattered pyknotic or karyorrhectic nuclei and an inflammatory cell infiltrate composed mainly of neutrophils and some mononuclear cells. *Clostridium perfringens* type A induces superficial villous tip necrosis with localized fibrin and gram-positive bacilli.

Clostridium perfringens enteritis can be diagnosed based on clinical signs, pattern of mortality, and nature of gross and microscopic lesions. Microscopic lesions with the carpet of large gram-positive bacilli on necrotic, atrophied villi are pathognomonic. A definitive diagnosis of differentiation of type can be done by bacteriologic culture followed by toxin detection or genotyping. PCR methods can also be used to detect genes for the major toxins. Cultures can be negative in chronic cases and may yield a mixture of type C and type A organisms. Failure to demonstrate other agents also supports a diagnosis of *C. perfringens* infection. If a type A infection is found, it is very likely that the cpb2 toxin can be revealed.

Prophylaxis is preferred to treatment in animals with clinical signs. The best way to control clostridial infections is by vaccination of the sows with a clostridial toxoid. Currently, most herds are vaccinated with type C toxoid at breeding or at midgestation and 2-3 weeks before farrowing. A vaccination program usually eliminates the disease within one farrowing cycle. Ingestion of adequate colostrums also helps to protect piglets. A type A vaccination is not commercially available, but can be made via custom biologics.

-by Dr. Lynn Statler, Class of 2008
-edited by Dr., Pam Mouser, ADDL Graduate Student

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Beginning in October, 2008 the Diagnostic Forum will be available only on our website-www.addl.purdue.edu—unless a hard copy is specifically requested. If you'd like to continue receiving a copy in the mail, please contact me at 765-494-7448
or email: yankovil@purdue.edu

The Forum will be posted in early January, April, July, and October

All six ADDL anatomic pathology graduate students, joined by Dr. Peg Miller and Alicia Burcham,finished the Geist half marathon in Indianapolis May 17, 2008.
Front row, left to right:: Drs. Peg Miller, Grant Burcham, Pam Mouser, Dinesh Bangari, Robert Johnson.
Back row, left to right: Drs. Josh Webster and Ikki Mitsui

ADDL welcomed four new graduate students on July 1, 2008.

Dr. Abby Durkes, PUSVM Graduate

Dr. Ryan Jennings, Michigan State CVM Graduate

Dr. Chad Frank, Michigan State CVM Graduate

Dr. Tiffany Reed, University of Georgia College of Veterinary Medicine

THE START

THE FINISH

On the Road

Drs. **Ching Ching Wu** and **Tsang Long Lin** were members of the Indiana delegation to the National Poultry Improvement Plan meeting in Portland, Maine, June, 2008.

Dr. **Roman Pogranichniy** and graduate students Drs. **Huiling Wei** and **Ingeborg Langohr** attended the North Central Conference for Veterinary Laboratory Diagnosticians in Madison, Wisconsin, June, 2008.

Gastroenteritis in the Ferret (Mustela putorius furo)

With the burgeoning interest in ferrets as household pets, veterinarians and diagnosticians should be aware of the common diseases and unique syndromes of ferrets. Gastroenteritis is a common clinical finding in ferrets. While most cases of gastroenteritis are due to foreign bodies, specific disease entities, such as eosinophilic gastroenteritis, *Helicobacter mustelae*– associated gastritis, and epizootic catarrhal enteritis may be involved. It is important to know the clinical picture, diagnostic findings, and pathogeneses unique to these syndromes in order to correctly identify the cause of gastroenteritis.

Eosinophilic gastroenteritis: Eosinophilic gastroenteritis (EGE) is a relatively uncommon alimentary disease in ferrets characterized by eosinophilic infiltrates in the gastrointestinal tract with clinical signs of chronic gastroenteritis. Eosinophilic gastroenteritis has been described in other species such as the horse, dog, cat and humans, and has been associated with Hodgkin's-like lymphoma in the ferret. The cause of EGE in ferrets is unknown.

Clinical signs of EGE include chronic weight loss, diarrhea, vomiting, lethargy, and inappetance. Intestinal loops can be thickened with cystic, dilated serosal lymphatics. One of the most suggestive diagnostic findings of EGE is a peripheral eosinophilia; however, cases have been described in which no eosinophilia was observed throughout the course of the clinical assessment. A definitive diagnosis is based on histopathology. Histologic lesions of EGE are characterized by focal diffuse inflammatory infiltrates in the stomach or intestinal mucosa with significant proportions of eosinophils. Eosinophilic infiltrates are often present in other tissues including the liver, pancreas, and lymph nodes. The Splendore-Hoeppli phemomenon may also be seen in mesenteric lymph nodes. Treatment is based on immunosuppression with corticosteroids which might be life-long.

***Helicobacter mustelae*–associated gastritis:** *Helicobacter mustelae i*s a gram negative comma-shaped (*Campylobacter*-like) bacterium which has been associated with atrophic gastritis, peptic ulcers, gastric adenocarcinomas and gastric mucosa-associated lymphoid tissue (MALT) lymphoma in ferrets. Chronic atrophic gastritis caused by *H. mustelae* is associated with bacterial stimulation of a lymphoplasmacytic inflammatory response and increases in gastric pH. Peptic ulcers may be related to elevated blood gastrin levels. Ferret colonies often have up to 100% prevalence of *Helicobacter mustelae* despite clinical disease being quite uncommon. The inciting cause of the transition from benign gastric inhabitant to relevant pathogen is unknown; however, similarities between *H. mustelae* infections in ferrets and *Helicobacter pylori* infections in humans has led to the use of *H. mustelae* as a model for *Helicobacter pylori*-related human gastric disease and neoplasia.

Clinical signs of *H. mustelae*-associated gastritis include lethargy, anorexia, vomiting, emaciation, dehydration and anemia. Stress appears to be a factor in disease severity, and might be related to inadequate diets or changes in feed. Gross lesions are infrequent in mild forms of the disease; however, in severe cases, the gastric mucosa contains small ulcers and is covered with digested blood.

Histopathology is needed for a definitive diagnosis and the pylorus is the preferred biopsy site. Histologic lesions consist of lymphoplasmacytic gastritis with loss of glandular epithelium. Bacteria can be demonstrated with a Warthin-Starry silver stain..Treatment involves antimicrobials and gastric protectants.

Epizootic Catarrhal Enteritis: Epizootic catarrhal enteritis (ECE) is a high morbidity, low mortality, self-limiting diarrheal disease in ferrets caused by a ferret coronavirus. Clinically, ECE presents with the sudden onset of profuse bright green diarrhea in older ferrets, which rapidly distributes throughout the ferret colony. The incubation period is 48-72 hours and primarily clinical signs last approximately 5-7 days. Affected ferrets may be lethargic, anorexic and dehydrated. Mortality is low with few animals dying from dehydration or concurrent illnesses. Treatment is aimed at correcting and preventing dehydration and secondary infections.

The diagnosis of ECE is based on clinical signs, epidemiological characteristics and histopathology. Biopsies acquired from the jejunum of affected ferrets show vacuolar degeneration and necrosis of apical enterocytes with significant villus atrophy and fusion, and lymphocytic proprial infiltrates. Coronavirus particles can be seen in feces via electron microscopy during the acute stages of the disease, although lack of viral particles does not rule out coronaviral disease.

-by Dr. Ryan Jennings, Michigan State Extern
-edited by Dr. Robert Johnson, 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 July-Dec. 2007 and Jan.-June 2008)

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	47	39	0	0	50	0	0	33	60	76	0	0	33	100	nt	nt	62	57	0	13	76	41	61	52	3	2
Ceftiofur	20	15	0	0	0	0	0	33	32	46	100	0	0	0	nt	0	46	52	0	0	34	36	44	19	4	2
Chlortetracycline	86	78	0	0	0	0	0	33	83	94	0	0	100	100	nt	nt	65	52	10	19	95	94	74	77	92	93
Clindamycin	100	100	100	100	100	100	100	100	100	100	0	100	20	100	nt	nt	100	100	10	13	100	100	100	100	90	85
Enrofloxacin	14	12	0	0	0	0	0	33	19	23	0	0	100	0	nt	nt	4	4	5	0	1	0	0	0	1	5
Florphenicol	57	61	0	0	0	0	0	33	62	73	0	0	0	0	nt	nt	58	57	0	0	63	59	57	52	0	0
Gentamicin	21	17	0	0	0	0	0	33	46	58	0	0	20	0	nt	nt	15	4	15	63	13	21	9	7	6	17
Neomycin	29	22	0	0	50	0	0	33	75	89	0	67	100	100	nt	nt	54	35	45	56	52	55	4	29	31	27
Oxytetracycline	86	81	100	0	50	33	0	33	85	97	0	33	80	100	nt	nt	69	57	35	50	97	95	83	81	94	97
Penicillin	100	100	0	nt	100	0	100	100	100	100	0	nt	100	100	100	nt	100	100	100	100	100	100	100	100	24	7
Spectinomycin	36	34	0	0	0	0	25	67	73	79	0	33	40	100	nt	nt	42	57	30	33	54	57	78	68	13	17
Sulphadimethoxine	60	68	0	33	100	0	75	100	59	78	0	50	67	100	100	0	92	83	20	13	80	81	83	94	73	70
Tiamulin	93	95	0	0	0	0	100	100	96	97	0	0	20	0	nt	nt	100	100	0	0	97	100	100	100	23	10
Tilmicosin	100	100	0	0	50	0	100	100	100	100	0	0	80	0	nt	nt	100	100	5	6	100	100	100	100	84	81
Triple Sulfa	36	22	0	0	0	0	0	33	65	77	0	0	20	100	nt	nt	31	13	5	0	21	26	9	10	1	7
Tylosin	nt	nt	nt	nt	nt	100	nt	nt	nt	nt	100	nt	nt	100	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt	nt
number of isolates	11	41	1	3	2	3	4	3	52	62	1	4	6	10	8	9	26	23	20	16	101	106	23	31	71	59

nt - not tested

Percent resistance to antimicrobials from selected animal pathogens --data supplied by Bacteriology Section, ADDL, Purdue University

(periods of July-Dec. 2007 and Jan.-June 2008)

Antibiotic	Canine										Equine												Feline							
	E. Coli		Enterococcus sp.		Psc. 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	3	3	74	64	5	0	12	9	0	0	3	7	0	0	50	47	7	18	40	0	11	7	0	0	57	0	0	0	67	0
Amoxycillin/Clauvulinic acid	23	22	28	29	100	100	16	13	25	0	3	19	0	0	0	40	0	36	0	0	0	0	22	13	57	100	100	100	67	0
Ampicillin	52	43	37	36	100	100	64	79	50	67	35	39	0	57	100	100	33	55	40	0	0	7	44	25	57	100	100	100	100	100
Cefazolin	33	24	78	82	100	100	16	13	25	0	3	16	0	57	0	40	0	32	20	0	0	0	22	19	0	100	100	100	67	0
Cefoxitin	29	21	78	89	100	100	16	13	25	0	3	19	0	0	0	40	7	32	20	0	0	0	22	13	100	100	100	100	67	0
Cefpodoxime	30	24	89	93	100	100	17	15	25	0	3	13	0	0	13	40	13	46	20	0	0	7	22	13	100	100	100	100	67	0
Ceftiofur	26	22	80	89	100	100	16	14	25	0	3	13	0	0	0	40	4	41	40	0	6	21	22	6	86	100	100	100	67	0
Cephalothin	36	31	76	78	100	100	16	13	25	0	3	19	0	57	0	40	0	23	0	0	0	7	22	19	100	100	100	100	67	0
Chloramphenicol	18	16	7	21	78	78	7	3	0	0	17	16	0	0	0	0	0	14	0	0	0	0	11	13	14	0	100	0	0	0
Clindamycin	100	100	94	93	100	100	48	42	25	0	100	100	100	100	38	53	33	67	60	0	6	8	100	100	100	100	100	100	100	50
Enrofloxacin	27	24	52	57	20	17	16	24	0	0	10	7	0	0	0	0	13	41	60	0	0	7	22	6	57	100	50	0	67	0
Erythromycin	100	100	48	54	100	100	48	41	25	0	100	100	100	100	38	53	27	59	20	0	17	36	100	100	86	100	100	100	100	50
Gentamicin	20	18	37	43	7	6	12	8	0	0	14	26	0	0	50	47	7	18	0	0	6	0	22	13	29	0	0	0	67	0
Imipenem	0	0	37	39	5	0	73	72	25	0	0	0	0	0	63	80	13	41	0	0	0	0	0	0	57	100	0	0	100	100
Marbofloxacin	28	23	nt	nt	nt	nt	16	26	0	0	10	7	0	0	0	0	13	41	20	0	0	7	22	6	nt	nt	nt	nt	67	0
Orbifloxacin	28	24	39	46	25	28	16	26	0	0	nt	nt	nt	nt	nt	nt	nt	nt	nt	0	nt	nt	22	6	57	100	50	0	67	0
Oxacillin + 2% NaCl	99	100	91	89	100	100	16	13	25	0	100	100	100	100	0	40	0	23	0	0	0	7	100	100	100	100	100	100	67	0
Penicillin	100	100	33	36	100	100	100	100	75	67	100	100	100	100	100	100	40	55	20	0	0	7	100	100	57	100	100	100	100	100
Rifampin	93	98	33	18	100	100	0	0	0	0	90	100	100	100	0	0	0	5	0	0	0	7	83	100	57	100	100	100	0	0
Tetracycline	41	32	72	65	100	100	36	42	0	33	38	36	0	57	25	47	40	32	60	0	17	43	33	31	100	100	100	100	0	0
Ticarcillin	47	39	30	32	8	17	22	18	25	0	35	29	0	57	0	50	0	23	0	0	0	7	44	25	57	100	50	0	67	0
Ticarcillin/Clavulanic Acid	14	14	30	36	5	6	16	13	25	0	3	0	0	57	0	40	0	23	0	0	0	0	22	6	57	100	50	0	67	0
Trimethoprim/Sulphamethoxazole	31	26	28	18	81	89	9	19	25	0	52	42	0	0	25	40	20	33	0	0	6	0	28	7	57	100	100	100	0	0
number of isolates	160	110	46	28	40	18	81	100	4	3	29	31	8	7	8	15	15	22	5	0	18	14	18	16	7	1	2	1	3	4

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, West Lafayette, IN 47907

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