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Summer 2005

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

The "Good Ole Summer Time". Thank goodness for the changes of seasons; it is good to welcome summer with fairs, crops laid by, vacations, hot days and cooler nights, and garden produce including home grown tomatoes. With the changes of requirements for exhibition of swine, the testing requests for fairs has been markedly reduced this year. In those long years back when we started on the road to eliminating pseudorabies from Indiana swine, it appeared to be a daunting aspiration but, with the efforts and determination of a lot of people, it has been accomplished. I'm sure that there are a lot of practitioners in the state who are glad to have the late spring/early summer requests to bleed 4-H pigs for the fair to be a thing of the past.

This time of the year has brought some changes to our laboratory personnel. At the end of May, we were saddened to see Dr. Dan Harrington retire. Saddened to see him go, but glad for him to have the opportunity to spend many pleasurable hours on his sailboat; he and his wife, Pat, have a trip planned to sail from the lower end of Lake Michigan up around Mackinac Island and on out into the Atlantic and down the coast to the Caribbean. We wish them well; it has been a pleasure to have Dan here and to have worked closely with him for those many years. On June 1, we welcomed our new diagnostic virologist, Dr. Roman Pogranichniy. Dr. Roman comes to us fresh off his PhD at Iowa State. He did his veterinary degree in the Ukraine and has extensive experience in diagnostic virology at the Iowa State Veterinary Diagnostic Lab. Dr. Roman and his wife, Sherry, have two children; it is a pleasure to welcome them to ADDL and to have Dr. Roman's expertise as an experienced veterinary diagnostic virologist on our faculty.

Included in this missive is a questionnaire requesting some of your opinions of the services provided by ADDL. We would appreciate you taking a few minutes to fill out this questionnaire and drop it in the mail. There is no need to put postage on it, the postage will be taken care of from this end. We would like very much to hear from you via this avenue; it is our intent to provide you with the best veterinary diagnostic services that we can.

We wish you a most enjoyable and safe summer. Stay cool.

FINAL DIAGNOSIS:
Bovine Anaplasmosis

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

Case #1

History: In October, 2004, two mature crossbred beef cows, from a herd of 30 cows, were submitted for diagnostic necropsy. The first cow was a 1000 lb white Charolais-cross which was reportedly 14 years old, and had a history of "suddenly going crazy". Because of her unpredictably aggressive behavior, the farmer used his tractor to separate her from the herd, but the cow charged the tractor head first, fell over, and died shortly after the impact. The second cow was a 950 lb red-and-white Hereford cross which was reportedly 10 years old. This cow had been vaguely lethargic for about 24 hours, followed by sudden death. No clinical symptoms had been observed in the remaining 28 cows.

Gross findings: The gross lesions were similar in both cows, though the carcass of the second cow was markedly autolytic at the time of submission. Both were in good flesh and had adequate to abundant body fat. The serosal surfaces of the abdominal organs had a slight yellow discoloration (mild icterus) and the spleens were markedly enlarged (splenomegaly), dark red in color, and bulged from the cut surface (congestion). The spleen of the first cow weighed 2.1 kg (0.46% of her body weight) and the spleen of the second cow weighed 2.5 kg (0.6% of her body weight.) Normal splenic weight for a cow is 0.17% of body weight. In addition to the mild icterus and marked splenomegaly, the first cow had subcutaneous hemorrhage (bruising) on the forehead, while the second cow had a gravid uterus containing a 21cm male fetus. Other than the subcutaneous hemorrhage, there were no fractures or other traumatic lesions that would account for the death of the first cow.

Histopathologic findings: Because of the aberrant neurological behavior, the brains of both cows were examined histologically. No lesions were found. Other tissues were not examined, but expected histologic lesions would be mild. The liver would be expected to have centrilobular degeneration and necrosis secondary to anemic hypoxia. The spleen would have been congested.

Other laboratory findings: Postmortem pericardial fluid from the first cow was positive for Anaplasma antibodies by the ELISA test. Blood smears were not available for examination. Tests for rabies virus and BSE were negative. No bacteria were isolated from spleen, and no viruses were isolated from spleen and lung.

Final diagnosis: Anaplasmosis was diagnosed on the basis of history, compatible gross lesions, and serology.

Follow-up analysis: The referring veterinarian submitted blood samples from nine other cows in this herd; seven of the nine were serologically positive for anaplasmosis by the ELISA test.

Case #2

History: At approximately the same time, a 900 lb, reported nine-year-old Angus cow from a different farm in a different county was submitted for diagnostic necropsy with a one-two day history of vague lethargy and anorexia terminating in sudden death. This cow reportedly came from a herd of about 30 cows from which 6 had died in the past week with similar clinical signs.

Gross findings: The submitted cow was in fair to thin body condition, with very little body fat. Mucous membranes and serosal surfaces had a slight yellow discoloration (slight icterus). The spleen was markedly enlarged (1.7 kg, 0.42% of body weight) and congested. Feces were dry and hard. No other remarkable gross lesions were present.

Other laboratory findings: A sample of postmortem body fluid was serologically positive for Anaplasma by the ELISA test. No viruses were isolated from lung or spleen.

Final diagnosis: Anaplasmosis was diagnosed on the basis of history, compatible gross lesions, and serology.

Follow-up analysis: The referring veterinarian went to observe the herd and reported that the outbreak looked like anaplasmosis, with several cows in the herd exhibiting weakness, anemia, and jaundice. Serum was collected from one of these cows and submitted for serology; it was found to be positive for anaplasmosis by the ELISA test.

Retrospective study: Purdue ADDL records were searched for all cases to which a pathologist had assigned the diagnosis of

'anaplasmosis'. Records searched were from a five year period extending from July 2000 to June, 2005. In that time period, anaplasmosis was diagnosed in 13 different submissions representing 11 different outbreaks (average of 2.6 submissions/year). All submissions were from the southern third of Indiana, with 4 from Jefferson County, 2 from Harrison County, 2 from Lawrence County, and one each from Orange, Perry, Clark, Switzerland and Franklin Counties. All cases were submitted during the months of August (1 case), September (5 cases), or October (7 cases) The age of the affected cattle ranged from 3-14 years, with a median age of 7.5 years. The most common clinical history was sudden death (7 cases), but several had a history of sudden aggression progressing to death in 1-2 days (3 cases) or vague lethargy progressing to death in 1-2 days (3 cases).

Discussion: Although anaplasmosis is not a common disease, it does occur in southern Indiana and is diagnosed on a fairly regular basis. Based upon conversations with experienced practitioners, anaplasmosis has been present in southern Indiana for as long as can be recalled. Because the signs and lesions of anaplasmosis can be subtle, its diagnosis is often missed if the practitioner is not already familiar with the disease. One practitioner commented that he never "saw" anaplasmosis until ADDL had diagnosed a case for him. He then realized he had been seeing it for years without recognizing it. After that, he diagnosed it much more frequently.

The cause of anaplasmosis is *Anaplasma marginale*, a rickettsia-like organism that is an obligate parasite of bovine red blood cells. The same organism can cause subclinical infections in sheep, goats, whitetail deer, and other ruminants, but the role that these other species might play in maintaining and spreading the disease is unknown. A related organism, *Anaplasma ovis*, causes clinical anaplasmosis in sheep and goats. Anaplasmosis is spread by the transfer of infected blood via ticks, biting flies and gnats, castration tools, hypodermic needles, etc. Ticks are the most important vector, as some species are biological vectors, maintaining the infection for long periods of time. Species of ticks believed to serve as vectors for anaplasmosis include *Dermacenter andersoni, Dermacenter variabilis, Boophilus annalatus, Boophilus microplus,* and *Argas persicus. Dermacenter variabilis* is common in Indiana and may be the vector in southern Indiana. The incubation period after an infective tick bite is believed to be about 1-3 months.

Morbidity and mortality can vary greatly. In my experience, anaplasmosis usually presents as sporadic death loss, affecting only one or a few animals in a herd. But occasionally, as in the second case presented above, it can affect a large number of animals. This may depend upon the herd's prior exposure to the organism. Recovered or latently infected animals are believed to be fairly resistant to the clinical disease, whereas naïve animals are believed to be highly susceptible. Animals that survive infection or that are subclinically infected are believed to maintain a low level of infection throughout life and can serve as a source of infection to other animals. Interestingly, young animals seem to be quite resistant to the disease, and I have never seen a clinical case in an animal less than three years of age. Young animals, however, do easily acquire subclinical latent infections. There is some thought that latently infected animals may become clinically diseased later in life if they experience some kind of physiologic stress such as poor nutrition or other disease.

The primary effect of the infection is to induce extravascular hemolytic anemia, and anemia is presumed to be the cause of death. The clinical signs and lesions of anaplasmosis can be subtle, but if affected animals are examined closely, they will be found to be anemic and may have mild icterus. In the acute phase of infection, animals will be febrile. Aggression and excitability are often reported in affected animals. The cause for this aggression is not known, but it is presumed to be due to the effects of hypoxia on the brain. Postmortem lesions in the dead animal are often mild. In my experience, the most useful postmortem lesions are anemia combined with marked splenic enlargement and congestion. Assessment of splenic size is sometimes difficult for those who do not necropsy many cattle, particularly if the carcass has become autolytic and the spleen has become artifactually inflated with postmortem emphysema. In this case, it is useful to

weigh the spleen and compare it to the body weight of the cow. In the normal cow, the spleen should account for 0.17% of the normal body weight. If the spleen is more than twice this weight, it can be determined with confidence that the spleen is enlarged.

It is difficult to confirm a diagnosis of anaplasmosis from a dead animal, as *Anaplasma marginale* cannot be cultured. If body bluid or serum can be collected, it can be submitted for serology. It is probably better to closely examine the remaining herdmates, looking for an animal that is febrile, anemic, and/or icteric, and collect serum and unclotted blood from that animal. A CBC should have changes typical of regenerative hemolytic anemia and *Anaplasma* organisms may be seen at the margins of red blood cells. However, the organisms are not always easily found and it is often necessary to submit serum for serologic testing to confirm the diagnosis.

Animals affected by anaplasmosis can be treated with tetracycline. Penicillin, cephalosporins, and related antibiotics are not effective. A good clinical reference manual should be consulted for more detailed treatment information.

-by Dr. Duane Murphy, ADDL Pathologist

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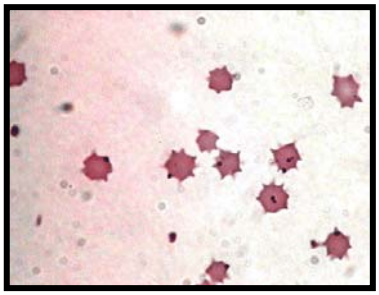

Anaplasma marginale

Pieces of the Puzzle: Johne's Diagnostic Testing

The diagnosis of Johne's Disease is made difficult primarily by the cause of the disease itself, *Mycobacterium paratuberculosis*. An animal can become infected at a very young age, but does not become clinically ill until 2-10 years later, when weight loss and diarrhea occur. According to recent data, it is estimated that 22% of U.S. dairy herds are infected with *M. paratuberculosis*. The expansion of dairy herds across the U.S. has facilitated the spread to uninfected herds and, with a cost ranging from $100-$200+ per cow in herds with high prevalence, producers and veterinarians are looking for detection methods to decrease or eliminate prevalence in their herds.

To understand the testing methods, it is important to understand the general stages of the disease since testing depends on the stage of the animal's disease (in general, the later the stage of disease, the higher the likelihood the test will be positive). The stages include Stage 1, silent infection of calves and young stock; Stage 2, inapparent carrier state of adults; Stage 3, clinical disease state of adult cattle; Stage 4, advanced clinical disease of cattle. For every animal that is in the advanced stage of the disease, there are 10-15 silently infected calves and 6-8 carriers in a herd.

The major types of tests for Johne's disease can be divided into two categories: fecal culture or antibody (ELISA or AGID) tests on serum. When choosing a testing method for one cow or a group of cows it is important to consider the level of the disease status, the sensitivity and specificity of the tests chosen, and the percentage of cows in the herd that are known to have clinical disease. The following table is a summary of the comparison between fecal culture and ELISA tests.

Diagnostic Test	Sample and Detection	Specificity	Sensitivity	Turnaround Time
Culture	Feces-Detects organism	High specificity	Ranges are 40-50%	Weeks to months
ELISA	Serum-Detects antibodies	Lower specificity	Depends on the level of diseases – ranges from 15-75%	Days

Fecal culture is the gold standard for diagnosing true positives. ELISA has been a good screening test for many testing protocols.

A former disadvantage of the fecal culture is the length of time it takes to complete culture growth. However, new breakthroughs in technology, such as new molecular techniques in combination with new automated technology at ADDL, can identify highly infected animals within 2-3 weeks and animals with low levels of infection in 4-8 weeks compared with months that were usually needed. Other types of tests to evaluate cell mediated immunity are also being evaluated. The Johne's skin test and gamma interferon assay may be used more frequently in the United States in the near future.

In summary, there is no definitive answer for the "best test" for Johne's disease. A great deal of thought and selection should go into the testing process when a producer and veterinarian choose the diagnostics for an animal or herd.

-by Sarah Stewart, Class of 2005
-edited by Dr. Leon Thacker

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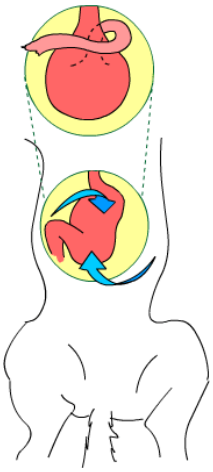
Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2005.

Labor Day……..……Sep 5
Thanksgiving…….....Nov 24-25
Christmas……..Dec 23,26
New Year……....Dec 30, Jan 2

Acute Gastric Dilatation-Volvulus in Dogs



Introduction: Canine acute gastric dilatation-volvulus (GDV) is a medical and surgical emergency that is seen most commonly in large and giant breed dogs. The syndrome is characterized by accumulation of gas in the stomach and malpositioning of the stomach with obstruction of eructation and pyloric outflow. Subsequent systemic effects of GDV including hypovolemic shock, endotoxemic shock, respiratory compromise, concurrent metabolic acidosis and alkalosis, and reperfusion injury are all implicated in the resultant death of affected dogs. Though this disease has been studied for years, the exact etiology and pathogenesis is still not clear. However, several risk factors for development of the disease have been found, as well as preventative measures for dogs at increased risk.

Signalment and risk factors: Most dogs with GDV are large and giant breed dogs.

Dogs with narrow, deep chests are at an increased risk for GDV, as are those dogs with history of GDV in a first degree relative. Eating meals from raised bowls, large meals fed once daily, and high speed of eating are found to be risk factors as well. Temperament plays a role also, since aggressive dogs are at a higher risk than dogs who are submissive to people and other dogs. It is also possible that stressful events such as boarding, trips to the veterinarian, and rides in the car can precede an episode of GDV.

Pathophysiology: It is uncertain whether dilatation or volvulus occurs first in the pathogenesis of the disease. It is generally accepted that since the majority of the gas in the stomach during an episode of GDV from aerophagia that dilatation happens first, followed by volvulus. However, it is also possible that volvulus occurs first which causes an inability to eructate and an impairment of gastric outflow. This pathogenesis is also supported by the prevention of GDV in dogs that have undergone gastropexy. The pathophysiology of GDV has grievous systemic effects that ultimately end in death of the dog. Cardiovascular effects include decreased venous return to the heart via mechanical compression of intra-abdominal veins by the grossly distended stomach, decreased cardiac output due to metabolic acidosis and cardiodepressant factors released by the pancreas, all resulting in hypovolemic shock, Gastric distention also causes a mechanical impediment to movement of the diaphragm, ultimately resulting in hypoxia and hypercapnia. Decreased respiratory function and increased anaerobic metabolism cause respiratory and metabolic alkalosis. Translocation of bacteria and endotoxins from the gastrointestinal tract as a result of ischemia can result in endotoxemia. A more recently recognized factor in the pathophysiology of GDV is reperfusion injury as a result of circulation of oxygen free radicals released from ischemic tissues.

Clinical signs and diagnosis: Diagnosis of GDV in dogs is most often made on the basis of signalment, history, and clinical signs. Radiographs can aid in determining the nature of the volvulus but are often not necessary to diagnose GDV. Clinical signs of the disease include a distended, tympanic abdomen, retching, unproductive vomiting and hypersalivation. Affected dogs will present with signs of tachypnea, dyspnea, tachycardia, pale, dry, or muddy mucous membranes, lethargy and possibly coma.

Treatment and Prognosis: GDV should be considered as a disease requiring emergency management. Resolution of hypovolemia is the primary concern followed by decompression of the stomach, surgical correction of volvulus, and adequate postoperative care. Fluid therapy should consist of crystalloid and colloid administration in order to restore circulatory function. Once fluid support has been initiated, gastric decompression can be attempted via passage of an orogastric tube. If passage of a tube is impossible or ineffective 14 gauge needles can be used to trocharize the stomach and relieve pressure. Once the dog has been adequately stabilized, the volvulus should be surgically reduced via cranioventral midline laparotomy. Care must be taken to evaluate the stomach, intestines, and spleen for ischemic damage and necrosis, and appropriate measures performed if this is found. Gastropexy should be performed at this time to prevent recurrence of volvulus. Postoperative care consists of fluid therapy, arrhythmia management, administration of antioxidants to prevent reperfusion injury and opioid pain control.

The mortality rates for GDV are reported to be as high as 15-25%. Prognostic indicators such as recumbency, depression or coma on presentation, gastric necrosis, and arrhythmia all increase mortality; thus, the most effective means of reducing mortality is expedient treatment of GDV.

Prevention: The most effective prevention of GDV in at-risk dogs is thorough understanding of risk factors and subsequent management of those factors. Several small meals fed throughout the day, coupled with feeding from a bowl at the dog's feet, will help minimize occurrence of GDV as well as minimizing stressful situations that could precipitate an episode. Avoid breeding affected animals and those animals that have a first degree relative with a history of GDV. Finally, prophylactic gastropexy is very useful in preventing GDV in high-risk breeds.

-by David Lee, Class of 2005

-edited by Dr. Leon Thacker, ADDL Director

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On the Road

Paula Brost and **Deidre DuSold** attended the Indiana Society of Histotechnology annual meeting and workshops in Indianapolis, IN, March, 2005.

Leon Thacker chaired an American Association of Veterinary Laboratory Diagnosticians Accreditation Committee meeting and attended a Laboratory Quality Assessors training workshop in Las Vegas, NV, February, 2005.

Drs. **Leon Thacker** and **Bill VanAlstine** attended the North Central Conference of Veterinary Laboratory Diagnosticians in Fargo, ND, May, 2005.

Mary Woodruff attended the Association of Veterinary Microbiologists meeting in Fargo, North Dakota, May, 2005.

Dr. **Tsang Long Lin** made a presentation at the Conference on International Conference on Immunopotentiators in Modern Vaccines, Malaga, Spain, May, 2005.

Dr. **Ramesh Vemulapalli** attended the "Train the Trainer" session on real-time PCR detection of foreign animal diseases, organized by the National Animal Health Laboratory Network, USDA, Madison, Wisconsin, April, 2005.

ADDL News

ADDL says goodbye to **Dr. Dan Harrington** who retired from Purdue on May 31, 2005.

Dr. Harrington began his career at Purdue in 1973 in the Veterinary Pathobiology department and the ADDL. He and wife Pat enjoy spending time with their 8 grandchildren (which includes triplets) and sailing the Weal Sea, their 35-foot sailboat. They are in the process of planning their sail this summer from Chicago to the Bahamas.

We welcome four new graduate students. Drs. **Ingrid Pardo, Ikki Mitsui, Dinesh Singh**, and **Pam Mouser** who joined ADDL in summer 2005.

Drs. Ingeborg Langohr and **Phaedra Cole** have successfully completed their residencies at ADDL. Both will pursue PhD degrees in the School of Veterinary Medicine.

Dr. Ching Ching Wu, Head of Microbiology Services/Avian Laboratory Services was selected as the recipient of the 2005 Pfizer Award for Research Excellence. In addition to this award, Dr. Wu was presented the Purdue Agriculture 2005 Team Award as part of a team headed by Dr. Al Heber of Agricultural Engineering.

Bovine Spongiform Encephalopathy

Introduction: Bovine Spongiform Encephalopathy (BSE), commonly known as Mad Cow Disease, has become one of the most important issues facing the U.S. cattle industry. After BSE was found in a Canadian herd, Canada's beef industry lost 5,000 jobs and $11 million per day. From May-August, 2003, there was a total ban on Canada's exported cattle market. Subsequently, a single case of BSE was found in the United States. It was found that the cow had been imported from Canada and did not represent a true "native" infection. Nevertheless, U.S. trading partners are now demanding proof that we have no other cases of BSE and U.S. beef exports remain at risk.

BSE is a progressively fatal disease affecting the central nervous system of adult cattle (20 months+). The exact origin of BSE remains unknown, but it is believed that the cattle were originally infected by eating feed containing sheep meat-and-bone meal that was contaminated with scrapie, a spongiform encephalopathy affecting sheep and goats. Once BSE became established in cattle, it spread from cow to cow by the feeding of infected cattle origin meat-and-bone meal to other cows and calves. The first case of BSE was reported in 1986 in the United Kingdom. Since then, there have been BSE cases reported in 20 different European countries, Japan, Israel, Canada and the United States. BSE has become an important disease to humans because infection can spread from cattle to humans via ingestion. This can cause variant Creutzfeldt-Jakob disease (vCJD), which is the human form of BSE. Although BSE can spread from cattle to humans, the chance of acquiring the disease is low. The species barrier provides substantial, yet still incomplete, protection against the infection in humans. However, BSE is potentially fatal to humans and is therefore a major issue in human public health and the cattle industry.

Pathogenesis: The pathogenesis of this disease is not completely known, though there is an emerging concensus among scientists. The most widely accepted theory suggests that the agent is a transmissible "prion", an abnormal variant of a normal protein. The new prion is a protein abnormally folded to form a different shape. When transmissible prions are ingested through infected food, they are able to convert the body's normal proteins into more abnormal transmissible prions. The transmissible prions accumulate in the intestines (duodenum to rectum), tonsils, and central nervous tissues such as brain and spinal cord. As these defective proteins accumulate in the brain, they interfere with normal brain functions, resulting in the neurological symptoms typical of the disease. Affected animals eventually die of starvation or accidental trauma caused by nervous dysfunction. Although this prion theory is most commonly accepted, a few researchers still think BSE is caused by a virus-like organism, though no viruses have ever been linked to BSE.

Clinical signs: There are no clinical signs in the beginning phases of this disease. The incubation period can last for 20 months to 15 years. Once it becomes clinical, BSE is distinguished by a variety of neurological clinical signs; the most commonly reported is change in behavior or temperament, usually pertaining to increased nervousness or apprehension. Hypersensitivity to sound and touch are frequently observed, along with head shyness, kicking, excessive ear movement, and nose licking. Weight loss is also common, even with a continuation in normal eating patterns. In late stages of this disease, cattle may develop incoordination progressing to paresis or paralysis, recumbency, and death.

Gross lesions: There are no gross lesions associated with BSE. Abrasions, lacerations, and contusions may result secondarily from injuries due to incoordination.

Diagnosis: There is no diagnostic test to confirm BSE in a live animal. Animals suspected for BSE should be euthanized and submitted for postmortem examination. Histologic examination of the brain stem is the widely accepted way to confirm the presence of BSE, the typical lesion being vacuoles (spongy holes) in the cytoplasm of neurons. Autolyzed brain tissue can be extracted and examined for scrapie-associated fibrils by electron microscopy. Recently, immunohistochemistry has been used for widespread testing of U.S. cattle. These are enzyme-based tests which utilize

antibodies specific for prion proteins. ELISA testing is also becoming available.

Treatment: Treatment is ineffective. Infection is best controlled by prevention.

Prevention and control: Prevention and control of BSE in cattle is difficult since BSE is not a traditional infectious agent (bacteria or virus); therefore, vaccination is not effective. Because BSE is spread by ingestion, the key to control is eliminating infected material from feed. Sheep and goat meat-and-bone meal is prohibited in all animal feeds and bovine meat-and-bone meal is prohibited in all ruminant feed. Also, no cattle may be imported from countries with a history of BSE, although Canada may be the first exception. Because the BSE agent is a protein and not a living organism, cooking with heat and other traditional food disinfection methods (heat, freeze, UV light, chemical disinfectants, etc) are ineffective. The key to prevention of BSE in humans (vCJD) is to eliminate infected material from the human food supply. To accomplish this, the USDA has put stipulations on the U.S. food supply. No sick or "downer" cows are allowed for human consumption. Specific risk materials (head, intestines, central nervous tissues, and spinal cord) from all cows and mechanically separated beef are prohibited in the human food supply. Finally, the USDA has begun comprehensive surveillance of the U.S. bovine population to ensure there is no significant threat of BSE within our domestic beef supply.

-by Andy Reynolds, Center College Student, Heeke ADDL Intern

-edited by Dr. Duane Murphy, ADDL Pathologist

Normal prion Abnormal prion

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ADDL is pleased to introduce our new Head of Virology/Serology Services, **Dr. Roman Pogranichniy,** who joined our faculty on June 1, 2005. Dr. Pogranichniy received his DVM from the Ukrainian State Agricultural University in 1993 and his MS in 2000 from Iowa State University where his research involved the role of porcine circovirus in postweaning multisystemic wasting syndrome. He completed his PhD, "Search for etiology of porcine reproductive and neurological syndrome: identification and characterization of a novel swine pestivirus", in 2005.

Dr. Pogranichniy and wife, Sherry, have two children, Sophia and Nicholas.

Dr. Pogranichniy welcomes your input. You can reach him at 765-494-7440.

Percent of Micro-organisms that are Resistence to Selected Antibiotics from Jul.- Dec. 2004 and Jan.-June 2005.

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	1	19	56	4	0	0	0	0	0	0	11	0	0	0	0	0	0	67	50	83	15	0	0	30	0	0	0	14	0
Amoxycillin/Clauvulinic acid	23	19	15	17	96	95	18	11	32	0	6	11	57	17	20	0	38	23	0	0	0	0	0	22	20	0	100	100	86	50
Ampicillin	40	43	15	17	96	100	55	68	58	53	35	25	64	33	40	50	38	50	0	0	0	0	24	35	20	0	100	100	100	0
Cefazolin	22	23	63	89	96	100	0	11	11	0	6	14	57	17	20	0	23	18	0	0	0	0	3	22	50	0	100	100	41	0
Cefotaxime	50	21	0	100	0	100	0	11	0	0	0	14	0	17	nt	0	nt	18	nt	0	0	0	nt	22	nt	0	nt	100	nt	0
Cefpodoxime	nt	20	nt	100	nt	100	nt	0	nt	0	nt	15	nt	33	nt	0	nt	29	nt	0	nt	0	nt	22	nt	0	nt	100	nt	0
Ceftiofur	18	16	74	89	88	100	0	11	13	0	6	11	57	17	20	0	23	18	0	0	0	0	0	22	90	0	100	100	41	0
Cephalothin	24	27	52	89	96	100	0	11	13	0	13	17	57	17	20	0	15	18	0	0	0	0	3	17	50	0	100	100	41	0
Chloramphenicol	11	18	4	6	91	90	0	0	0	0	16	17	64	33	0	0	0	9	0	0	0	0	0	17	0	0	100	100	0	0
Clindamycin	99	100	81	83	96	100	0	11	13	4	100	100	100	100	20	0	8	14	0	0	0	0	100	100	80	0	100	100	57	0
Enrofloxacin	20	19	48	39	54	43	27	11	21	4	13	0	0	0	0	0	0	9	67	0	38	6	0	13	50	0	0	0	57	50
Erythromycin	99	99	33	28	100	100	0	26	18	9	100	100	100	100	20	0	38	23	0	0	8	0	100	96	20	0	100	100	71	0
Gentamicin	10	18	11	11	8	5	0	0	5	4	23	17	43	33	40	25	8	9	33	50	71	0	3	17	40	0	0	0	14	0
Imipenem	1	1	15	17	0	5	0	11	14	0	0	0	0	0	20	0	15	18	0	0	0	0	0	0	20	0	0	0	71	0
Marbofloxacin	nt	1	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	0	nt	nt	nt	0	nt	nt
Orbifloxacin	24	19	31	28	39	38	0	11	19	4	13	0	0	0	0	0	0	9	0	0	0	0	0	13	10	0	0	0	57	0
Oxacillin + 2% NaCl	99	100	27	100	87	100	0	11	13	55	100	100	100	100	20	0	15	18	0	0	0	100	100	96	60	0	100	100	71	0
Penicillin	99	100	26	22	96	100	55	68	59	0	100	100	100	100	40	50	31	45	0	0	0	0	100	100	20	0	100	100	100	0
Rifampin	94	87	26	33	100	100	0	0	0	0	100	97	100	100	0	0	0	9	0	0	0	0	82	70	10	0	100	100	14	0
Tetracycline	26	29	41	72	75	81	27	100	21	15	39	33	71	33	40	13	8	14	0	0	54	18	21	17	80	0	43	50	14	0
Ticarcillin	34	41	22	17	25	5	55	21	62	55	35	22	64	17	40	50	38	50	0	0	0	0	24	26	20	0	14	0	100	0
Ticarcillin/Clavulanic Acid	nt	15	nt	22	nt	5	nt	68	nt	0	nt	3	nt	17	nt	0	nt	23	nt	0	nt	0	nt	9	nt	0	nt	0	nt	0
Trimethoprim/Sulphamethoxazole	nt	27	nt	11	nt	90	nt	0	nt	0	nt	36	nt	17	nt	13	nt	9	nt	0	nt	3	nt	9	nt	0	nt	100	nt	0
number of isolates	128	150	27	18	23	21	11	19	39	47	31	36	14	6	5	8	13	22	3	2	24	33	34	23	10	0	7	2	7	2

nt - not tested

Percent of Micro-organisms that are Resistence to Selected Antibiotics from Jul.- Dec. 2004 and Jan.-June 2005.

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	35	29	0	0	0	0	67	70	52	61	13	63	0	8	89	47	66	52	13	20	69	71	48	68	3	3
Apramycin	8	0	33	100	100	100	0	0	19	23	50	0	71	71	nt	nt	0	0	4	9	20	20	0	13	43	25
Ceftiofur	19	3	0	0	0	0	0	0	27	28	0	0	0	0	0	0	63	52	0	4	25	28	10	28	12	11
Chlortetracycline	65	63	0	0	0	0	0	63	84	85	13	38	0	0	nt	nt	69	56	0	2	98	95	81	85	92	92
Clindamycin	96	100	100	100	100	100	100	100	95	99	100	100	100	100	nt	nt	100	100	13	20	100	99	100	100	82	88
Enrofloxacin	15	3	0	0	1	0	0	0	19	15	25	25	0	0	nt	nt	0	0	0	2	0	1	0	0	0	6
Erythromycin	96	99	0	0	50	0	100	100	96	99	13	50	25	17	0	0	100	96	8	6	100	99	95	98	81	86
Florphenicol	96	100	0	0	33	0	100	88	95	99	0	25	14	10	nt	nt	100	96	0	0	100	99	95	100	69	66
Gentamicin	23	9	0	0	17	0	0	0	43	44	0	25	0	0	nt	nt	13	4	0	0	20	21	10	15	5	8
Neomycin	35	23	33	0	83	50	67	63	69	74	25	63	57	80	nt	nt	53	44	13	29	53	49	14	30	64	38
Oxytetracycline	69	69	33	0	83	0	67	63	86	86	25	75	57	50	nt	nt	69	56	63	24	99	98	81	90	94	96
Penicillin	96	100	33	0	67	0	100	100	96	99	75	75	25	25	89	47	100	100	50	55	100	100	95	100	16	17
Sulphadimethoxine	50	60	33	0	83	25	67	80	62	74	25	63	50	58	100	87	69	68	17	22	77	84	86	90	62	72
Spectinomycin	54	33	100	50	50	0	100	100	73	72	100	75	86	40	nt	nt	100	76	67	39	53	66	95	98	29	20
Sulphachloropyridazine	50	61	33	0	83	100	67	88	80	88	0	63	86	80	nt	nt	69	76	13	29	77	84	81	88	62	76
Sulphathiazole	50	60	33	50	83	50	67	75	80	86	25	88	71	70	nt	nt	72	68	50	45	77	82	81	85	60	80
Tiamulin	96	100	67	50	83	50	100	100	95	99	75	63	100	80	nt	nt	100	100	8	16	100	99	100	100	22	26
Tilmicosin	96	94	0	0	50	0	100	100	94	99	0	38	29	20	nt	nt	100	100	9	2	100	99	95	100	81	87
Triple Sulfa	31	19	0	0	33	0	0	10	56	62	13	50	14	20	nt	nt	34	28	0	0	32	25	0	15	0	6
Tylosin	96	100	100	100	83	75	100	100	95	99	100	100	100	70	nt	nt	100	100	nt	nt	100	100	100	100	nt	nt
number of isolates	26	70	3	2	6	4	3	10	81	81	8	8	7	10	9	15	32	25	24	49	101	170	21	40	86	104

nt - not tested