



**FROM THE DIRECTOR**  
 H. Leon Thacker, DVM, PhD

In the "Good ole summer'time". Time flies when you're busy and having fun; it seems that it has only been a few days since writing a few paragraphs for the spring issue of the Diagnostic Forum. I send thanks especially to those who have contacted us with words of appreciation and encouragement for the contents of our newsletter. If there are subjects that you would like to see addressed in future issues, please do not hesitate to make request of it. Some items of note since the last issue include the results of immunohistochemistry tests for chronic wasting disease that were run on hunter killed deer from Indiana. Two thousand one hundred seventeen samples were run from the 2002 season and 2181 samples from the 2003 season; all samples were found to be "no resistant prions detected" or, in other words, no evidence of CWD was found in any of the samples run. We expect to receive samples from approximately 2000 deer from the 2004 season from DNR to check for CWD. The bacteriology and serology sections of the ADDL are very busy at this time processing and testing fecal cultures for identification of the causative *Mycobacterium* and serum samples by ELISA for antibodies to the organism in support of the State/Federal Johne's Disease Surveillance Program. It was recently announced that the State program would be continued for at least another year; it is expected that the heavy case load for Johne's testing will continue. We are installing a new atomic absorption spectrophotometer to replace the aged and worn-out AAS in our toxicology lab; one change that this will affect will be the need to receive blood specimens for blood lead determination in anticoagulant tubes rather than clot tubes as required previously because of the new analytical procedure of the new machine. Federal funding has been assigned to place an alkaline tissue digester at the Heeke Southern Indiana ADDL. A means of tissue/carcass disposal at the laboratory has been sorely needed for many years. The pressurized alkaline tissue digester process has been shown to inactivate the prions which are the causative agents of bovine spongiform encephalopathy, chronic wasting disease and scrapie. It is also economical and noted to be much less expensive to operate than incineration for carcass disposal.

A new ADDL fee schedule will go into effect July 1, 2004. This will mainly affect fees for necropsy, bacteriology and histopathology testing; newly available tests in the lab will be listed. A copy of the new fee schedule is included with this mailing. Again, if you have suggestions for improvement of the diagnostic services of our laboratory, please do not hesitate to inform us. Hope you have a great summer.

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## Final Diagnosis

### Bovine Leukosis in a neonatal calf

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

**History:** A reportedly 1-day-old crossbred female calf was submitted dead for necropsy with a history of hemorrhage from multiple organs

and death. Per history, the calf was found cold and weak with blood oozing from the anus and vulva. Petechial hemorrhages were observed in the oral cavity, gingiva and sclera.

**Gross findings:** Petechial hemorrhages were observed throughout the serosal surface of the small intestine. The rectum was edematous and hemorrhagic on the serosal surface; however, on cut section, the mucosal surface did not show evidence of hemorrhage. Ecchymotic hemorrhages were present in the mesentery and paintbrush hemorrhages were visible on the serosal surface of the abomasum. The abomasum and forestomachs were almost empty but contained a small quantity of translucent mucoid material (amniotic fluid). Terminal parts of the colon and rectum contained formed feces (meconium), which had a pasty consistency. The urachus was hemorrhagic and edematous. The liver was pale tan and slightly firm in consistency, granular in appearance and had a thickened capsule. Multifocal to diffuse hemorrhages were also observed in the skeletal muscles, epicardium and endocardium. Lymph nodes were dramatically enlarged throughout the body. The mesenteric and tracheobronchial lymph nodes were markedly enlarged (up to 6x3x2 cm).

**Histologic findings:** Liver: Multifocal to coalescing nodules and random aggregates of discrete round cells with distinct borders were effacing and often compressing the hepatic parenchyma. Most of these nodular aggregates were centered in the portal areas and had a

pattern of spreading out circumferentially into the adjacent hepatic parenchyma from the portal tracts. Random aggregates of such cells were also present multifocally in the hepatic sinusoids and were distributed diffusely beneath the hepatic capsule. These cells had irregularly round to oval large nuclei with marginated, clumped to stippled chromatin rimmed by pale, scant cytoplasm. There was moderate anisocytosis and anisokaryosis. The hepatic arteries were multifocally ectatic and often contained the cells described above. Mitoses were 1-2 per HPF.

**Kidneys:** Multifocally, the interstitium was expanded by cellular aggregates of similar nature as seen in the liver and was especially prominent around blood vessels.

**Lymph nodes and bone marrow:** Neoplastic round cells, similar to those present in the liver and kidneys, diffusely effaced the normal cortical and medullary architecture of the lymph nodes. The bone marrow was hypercellular, with most of the cell population comprised of neoplastic round cells.

**Heart:** Major changes included multifocal areas of hemorrhage, swelling and cytoplasmic vacuolation of cardiomyocytes, and multifocal mild to moderate lymphocytic and histiocytic inflammation and diffuse areas of hemorrhage in the epicardium. Lymphocytes may be neoplastic.

**Skeletal muscle:** Hypereosinophilia of several muscle fibers, hemorrhage, edema, cellular infiltration consisting of macrophages and lymphocytes (possibly neoplastic) often centered on blood vessels, were the main alterations.

**Lungs:** Hemorrhage, lympho-histiocytic inflammation in the interlobular septae and multifocal hemorrhages in alveolar spaces were present. Multifocally, alveoli were collapsed with slit-like alveolar spaces. Mild expansion of interstitium with mononuclear cell

infiltration (possibly neoplastic lymphocytes) and fibrin and perivascular hemorrhages were prominent changes.

**Discussion:** A diagnosis of Bovine Leukosis (congenital) was made in this case as per histological observations. Perinatal infection with BLV is the most likely cause of lymphosarcoma in this calf. Malignant lymphoma was found to have the greatest incidence among neoplastic diseases in calves according to various studies. The congenital form of this disease is most commonly multicentric. Diagnosis of disease prevalence in the herd can be made by ELISA or AGID tests on serum samples although calves to 6-7 months of age having received colostrum with antibodies might return false positive results. Also, animals in the early stages of infection, animals with poor immune response, and cows immediately prior to and after calving, do not make good candidates for such serological testing. Peroxidase linked assay (PLA) and nested-PCR have recently been reported to have high efficiency in making an accurate diagnosis of Bovine Leukosis.

-by Dr. Gopakumar Gopalakrishnan, ADDL graduate student



### **Porcine Proliferative Enteropathy**

**Introduction:** Porcine proliferative enteropathy (PPE), commonly referred to as ileitis, is caused by the obligate, gram-negative, intracellular bacterium *Lawsonia intracellularis*. Ileitis is an economically important disease that affects swine populations throughout the world. Incidence ranges from 32.7% of farms with less than 2,000 head total inventory, 53.7% in medium sized farms

(2,000-9,999), and 75% in large farms (10,000+).

**Pathophysiology:** The pathogenesis of PPE develops as progressive proliferation of immature intestinal epithelial cells in the terminal ileum, and less commonly, the large intestine. *L. intracellularis* organisms enter dividing crypt cells via an entry vacuole. The vacuole quickly breaks down, releasing bacteria into the cytoplasm where they rapidly multiply. The mechanism by which infected crypt cells continue to divide, undergo mitosis, yet fail to mature is unclear.

A less common acute form of the disease, known as proliferative hemorrhagic enteropathy (PHE), is characterized by widespread degeneration and desquamation of enterocytes with severe congestion of mucosal vasculature and hemorrhage from capillary beds. Mucosal thickening may not be as marked as in traditional PPE. The trigger for hemorrhagic crisis is not known.

**Clinical signs:** The majority of PPE cases are subclinical infections characterized by decreased daily gains, poor feed efficiency, and increased days to market. Clinical disease usually occurs in grower-finisher pigs between 2-6 months of age. Affected pigs exhibit signs of anorexia, poor growth, and watery yellow-green diarrhea. Most pigs recover, although some may progressively deteriorate and die. The PHE form of disease usually affects older animals between the ages of 4-12 months. Clinical signs are acute, including severe, dark brown-bloody watery diarrhea and sudden death.

**Gross lesions:** Gross lesions are consistently observed in the terminal ileum, but less commonly occur in the distal jejunum, proximal spiral colon, and cecum. The most characteristic gross lesion is thickening of the mucosa with an irregular nodular or folded appearance. In most severe cases, the mucosa may be eroded with a granular appearance and adherent fibrinonecrotic

debris. Proliferative lesions are common to both forms of the disease, though, in the PHE form, a large amount of undigested blood is also observed within the lumen.

**Histopathology:** Microscopic lesions of PPE are characteristic and often diagnostic. Proliferation of immature crypt epithelial cells replaces the normal mucosal architecture and produces the thickened appearance observed grossly. Crowded, immature epithelial cells populate proliferating crypts, which may become branched and extend to the mucosal surface. Mitotic figures are common and goblet cells are absent from the abnormal epithelium. Additionally, crypts may be dilated and contain necrotic debris and neutrophils. *L. intracellularis* organisms are detected as small, curved, rod-shaped bacteria in the apical portion of immature crypt cells by Warthin-Starry silver staining. In cases of PHE, bacteria may also be found free, in macrophages, and within capillaries and lymphatics. In chronic cases of PPE, however, microscopic lesions are less diagnostic due to production of fibrous connective tissue throughout the mucosa.

**Diagnostic Tests:** Because *L. intracellularis* does not grow in conventional cell-free media, bacterial culture is not employed for diagnostic testing. The most commonly used diagnostic tests, in addition to histopathology, are serology and polymerase chain reaction (PCR) of fecal and tissue samples. Fecal PCR has limited efficacy for determining exposure to *L. intracellularis* and may be more useful to identify animals that are actively shedding the organism. In one study, only 60% of pigs orally infected with *L. intracellularis* had positive fecal PCR results 3 weeks after challenge, which declined to 30% by 6 weeks post-infection. Tissue PCR has nearly 100% specificity and sensitivity. Serial samples collected from a large number of animals of different age groups will yield the best results.

**Treatment:** Treatment of clinical PPE is often successful in the mid to late finisher. Treatment of animals showing clinical signs must be immediate and aggressive. Injectable tylosin and lincomycin are the antibiotics of choice. Other treatments include oral antibiotics in the feed or water. Examples of antibiotics with label claims include: Tylan (tylosin), Denagard (tiamulin), Lincomix (lincomycin), and BMD plus aureomycin. Determining age of exposure by use of the previously outlined diagnostic tests will facilitate proper placement of oral antibiotics and reduce the economic impact of the disease.

**Prevention:** Preventative measures to reduce the incidence of PPE in swine herds involve the implementation of proper sanitation, disinfection, and vaccination protocols. Enterisol Ileitis, an avirulent live vaccine administered orally in the water, is currently available. Although the vaccine has shown success, proper administration is essential to maintain efficacy. For proper protection, the vaccine must be administered several weeks prior to exposure to the organism. For example, if pigs are exposed to *L. intracellularis* in the early finisher, the vaccine should be placed in the middle nursery stage of production. Additionally, no antibiotics should be added to feed or water for several days before and after administering the vaccine.

-by Tony Lahr, Class of 2003

-edited by Dr. Kim Maratea, ADDL  
Graduate Student

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the United States. While great strides have been made throughout the last decade in an effort to understand EPM, many questions remain unanswered concerning its etiology, pathogenesis, occurrence, treatment, and diagnosis.

*Sarcocystis neurona* is by far the most commonly implicated agent in cases of EPM; however, recent investigation implies that the syndrome, in rare cases, can be caused by *Neospora caninum* and/or *Neospora hughesi* infections as well. Unfortunately, infection with these other protozoan species is clinically indistinguishable from infection with *Sarcocystis neurona*. For the purpose of this paper, only infection with *Sarcocystis neurona* as a causative agent of EPM will be described.

**Pathogenesis:** The definitive host of *Sarcocystis neurona* is the opossum. Infected opossums shed sporocysts in feces which are in turn infective to the intermediate host when ingested. Many intermediate hosts are currently recognized in the life cycle of this protozoan, and the full range of hosts has yet to be identified. Current species implicated include cats, armadillos, skunks, raccoons, and sea otters. Once the intermediate host is infected, it goes on to develop sarcocysts in its skeletal muscle. When this muscle is ingested by the opossum, the life cycle is completed.

The horse is considered an aberrant or dead-end host of *Sarcocystis neurona*. While the horse is presumably infected by ingestion of sporocysts in contaminated feed and water, there are many unanswered questions concerning the pathogenesis of this protozoan once it actually infects the horse. It is suggested that sporozoites released from the ingested sporocysts are able to penetrate the intestinal wall and enter arterial endothelial cells. Schizonts then develop in these cells until they rupture releasing merozoites into the bloodstream. This stage of the life cycle may be repeated several times producing large amounts of merozoites.

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

**July 5, 2004.....Independence Day**  
**September 6, 2004.....Labor Day**  
**November 25-26.....Thanksgiving**



## **Equine Protozoal Myeloencephalitis**



### **Introduction and Etiology:** Equine

protozoal myeloencephalitis (EPM) is a progressive, degenerative neurological disease of the central nervous system that has been described in horses for over 30 years. The disease is one of the most commonly diagnosed neurological disorders of horses in the United States. A recent USDA study revealed an incidence of 14 new cases of EPM diagnosed per 10,000 horses per year in

At this point, the infection can be cleared leading to seropositivity but no clinical signs or the protozoan can progress to the central nervous system. It is unknown how *S. neurona* enters the CNS in horses. It has been suggested that merozoites enter the CNS via infected leukocytes or through the cytoplasm of endothelial cells. Once the merozoite has gained access, schizonts form in one or more areas of the central nervous system including the cerebrum, the cerebellum, the brainstem, cranial nerves, and/or the spinal cord. Schizonts and daughter merozoites in the neural tissue remain uninfected and, therefore, transmission from the infected horse to other animals is not possible.

Recent studies suggest that anywhere between 22-65% of horses in the United States are seropositive with *S. neurona* antibodies, depending on the geographic location. Although several theories have been developed concerning why only some horses develop clinical disease, the reason is unknown. Theoretical contributing factors to the development of this disease include stress and other unrelated health events that occur before the onset of clinical EPM. In addition, little is known concerning the incubation period between exposure to the protozoan and development of clinical disease.

**Clinical Pathology and Necropsy Findings:** No characteristic changes are seen in the hemogram or serum chemistries found in horses affected with equine protozoal myeloencephalitis. Cytological examination of cerebral spinal fluid typically does not reveal significant changes. Gross pathological changes are apparent in the affected portions of the brain and spinal cord, and include multifocal areas of hemorrhage and malacia or both grey and white matter. Gross changes of muscular atrophy may also be seen in the skeletal muscle of affected horses. Histological examination of affected nervous tissue reveals neuronal

necrosis and loss in addition to marked mononuclear perivascular cuffing. Infiltration of monocytes, lymphocytes, some eosinophils, and rare neutrophils can also be observed. Direct visualization of the organism is often not achieved because they are often present in very low numbers. This is especially true if the animal has been previously treated with antiprotozoal medications.

**Diagnosis:** A definitive diagnosis of EPM in a live horse is challenging. Simple seropositivity toward *S. neurona* antigen is inadequate for diagnosis since the protozoan must enter the central nervous system from the systemic blood circulation in order to cause the disease. In other words, a seropositive horse has been exposed to the organism, but may or may not have EPM. While there are several tests available to diagnose EPM, all of them are problematic.

The most recent major advance in diagnosis is the introduction of the immunoblot test for detection of IgG antibodies against *Sarcocystis neurona*. Antibodies detected in the cerebral spinal fluid (CSF) provide support for a diagnosis of EPM. However, if the CSF is accidentally contaminated with blood during the procedure, a false positive can result. For this reason, it is recommended that only CSF samples with less than 50 RBCs/ $\mu$ L be submitted for immunoblot testing. It has also been suggested that limited antibody movement into the CSF can occur without the presence of CNS disease. This could also result in a false positive immunoblot test result.

Although the immunoblot test is reported to have a sensitivity and specificity of 89%, a recent study by Daft et al determined that the specificity of the Western blot test (a type of immunoblot test) is between 44-60%. These results, as well as the risk of a false positive test, suggest that the use of immunoblot analysis is most useful in

ruling out EPM rather than diagnosing the disease.

Other diagnostic tests for EPM include polymerase chain reaction (PCR) testing, the albumin quotient test, and the IgG index test. While PCR testing can detect minute amounts of protozoan DNA, the rapid destruction of DNA in the CSF environment and the possibility that DNA may not be present in the CSF makes the sensitivity of this test questionable. The albumin quotient test was developed to detect contamination of the CSF sample with blood. Unfortunately, the test does not decipher whether the contamination is iatrogenic or a simple "leakage" of protein through the blood-brain barrier. The IgG index test was developed to detect the production of IgG in the CSF. However, subsequent studies have found little difference between index values of EPM-affected horses and normal control horses.

The detection of characteristic lesions on necropsy is considered the gold standard of diagnosis by some. Due to the small number of organisms needed to cause the disease, however, the diagnosis can be missed even with a full neurologic necropsy. In general for the live animal, a clinical diagnosis is best established in horses with neurological disease consistent with EPM and a positive immunoblot test or an uncontaminated CSF sample. Another clue for diagnosis is an improvement of clinical signs in response to treatment of EPM. Overall, it is imperative that the diagnosis be based not only on test results, but in conjunction with a thorough diagnostic examination that rules out other causes of neurological disease.

**Treatment and Prognosis:** Treatment of equine protozoal myeloencephalitis is expensive, and even mildly affected horses can require prolonged therapy. The standard treatment for many years has been combinations of antifolate drugs including sulfadiazine and pyrimethamine with or without

trimethoprim. The use of folic acid supplements in conjunction with this therapy has been recommended by some in an effort to reduce the risk of folic acid deficiency. A recent case report showed that supplementation failed to prevent the development of folic acid deficiency, however. The use of nonsteroidal anti-inflammatory medications in conjunction with traditional therapy has been routinely used for many years. Supplementation with various vitamins has been recommended by some as well as the use of acupuncture in an effort to treat EPM; however, the efficacies of these practices have not been proven in clinical trials.

The most recent breakthrough in the treatment of EPM is the development of triazine-derivative drugs. These medications were initially developed as herbicides and have historically been used as coccidiostats in poultry and swine. Ponazuril (Marquis™) is a member of this family, and is the first approved medication for the treatment of EPM. Other drugs in this class include diclazuril and toltrazuril. Ponazuril is a primary metabolite of toltrazuril, and has shown anticoccidial activity against several parasites, including *Sarcocystis neurona*. Treatment regimen requires once a day dosing for 28-56 days. While studies show ponazuril can effectively rid horses of *S. neurona*, it does not improve the CNS damage that occurs before treatment begins.

Studies estimate that clinical improvement can be seen in 70% of treated horses, but fewer than 25% return to original function. Relapse of disease occurs in approximately 5-28% of horses when treatment is discontinued. The mechanism of this relapse is unknown, but reemergence of a latent stage parasite, persistence of a small focus of infection despite treatment, and reexposure to *S. neurona* have all been proposed.



**Prevention and Control:** Due to the lifestyle and eating habits of the definitive host of *Sarcocystis neurona* (the opossum), prevention and control of EPM are potentially problematic. Current recommendations include preventing access of opossums to hay, grain, pasture, and water sources. This may be difficult, especially if food and water are in short supply for the opossum. The most reasonable and simple precaution for horse owners to take is to deny access of stored hay and grain bins to the opossum.

The opossum is considered a scavenger and will consume whatever is available to it, including road-kill. Recommendations to prevent EPM commonly include picking up road-kill in the immediate area. This suggestion may be somewhat ineffective, however, since there are likely many other unknown intermediate hosts that are perpetuating the lifecycle.

A killed vaccine against *Sarcocystis neurona* has been developed using merozoites. No clinical evidence supports the efficacy of the vaccine and little research has been completed to date, however. Overall, there are few suggestions to aid in the prevention of *Sarcocystis neurona* exposure.

-by Katherine Gilmore, Class of 2004

-edited by Dr. Theresa Boulineau, ADDL Graduate student

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## Granulomatous Meningoencephalomyelitis

Granulomatous meningoencephalomyelitis (GME) is a sporadic, idiopathic inflammatory disease of the CNS of dogs and, rarely, cats. This disease appears to have a worldwide distribution, with recent reports coming from the USA, Australia, New Zealand, and Europe.

**Signalment and Clinical Signs:** Most cases of GME occur in small breed dogs, especially in Pug dogs, although any breed may be affected. The majority of confirmed cases occur in young to middle-age dogs, with a mean age around five years. GME occurs in both sexes; however, there appears to be a higher prevalence in females. The onset of disseminated GME is acute, with a progressive course over a 1-8 week period, and dogs with focal GME tend to have a longer clinical course. Common clinical signs include incoordination, ataxia and falling, cervical hyperesthesia, head tilt, nystagmus, facial and/or trigeminal nerve paralysis, circling, visual deficits, seizures, depression and titanic spasms. An infrequently reported ocular form of GME appears to be related to lesions localized in optic nerves and optic chiasm resulting in visual impairment, uveitis, and abnormal papillary reflexes. A hyperemic and edematous optic disk may be seen on ophthalmological examination, vessels may be dilated, and focal hemorrhage may be present. Occasionally, ocular and neurological signs may be seen concurrently in the affected animals.

**Etiopathogenesis:** The cause of GME is unknown. Immunohistochemical studies have indicated that many lymphocytic/lymphoblastic cells are immunoglobulin bearing. The recent report of IgE positive cells in perivascular cuffs in two dogs with GME lends credence to a possible underlying immunopathological perturbation.

Results of an immunomorphological study further suggested a T cell-mediated delayed-type hypersensitivity of an organ-specific autoimmune disease as a possible pathogenic mechanism for this unique lesion. It is also possible that GME represents an altered host response to an infectious agent.

**Diagnosis:** A tentative diagnosis of GME may be suggested by signalment data, clinical course of the disease, and clinical signs. Hematology, serum chemistry, and urinalysis are usually normal. GME must be distinguished from other granulomatous lesions in the brain that are associated with known infectious agents as well as from other CNS disorders including sporadic necrotizing meningoencephalitis and granulomatous leptomeningitis in beagles associated with *E. coli*. The most useful diagnostic aid is cerebrospinal fluid (CSF) analysis. In most GME cases, CSF is abnormal with mild to pronounced pleocytosis, ranging from 50-900/ul. Cells are predominantly mononuclear, including lymphocytes (60-90%), monocytes (10-20%) and variable numbers of large anaplastic mononuclear cells with abundant lacy cytoplasm. While neutrophils typically comprise 1-20% of the cell type differential, they may be the predominant cell type on rare occasions. Protein in CSF is usually mildly to moderately elevated, ranging from 40-400 mg/dl. CSF pressure may be normal or increased. A combination of CSF and MRI findings may also be useful, the latter being characterized by isointense lesions in T1-weighted images. Although infrequently performed, a brain biopsy can be a very useful diagnostic test in animals with focal lesions.

**Pathology:** Lesions associated with GME are restricted to the CNS. In brain and/or spinal cord, soft, grey, oval lesions with irregular or well-defined margins occasionally can be discerned on gross sectioning, especially with

large focal lesions. Sometimes, the cut section of the CNS has a granular, mottled appearance with finger-like projections. Meninges may appear thickened and cloudy, and occasionally optic nerves are grossly enlarged. Internal hydrocephalus may be present in some dogs. Microscopic lesions are usually widely distributed through the CNS, but primarily in the white matter of the cerebrum, caudal brain stem, cerebellum, and cervical spinal cord. The lesions are characterized by a dense aggregation of mesenchymal cells arranged in a whirling perivascular pattern. These perivascular cuffs are composed of histiocytes and varying numbers of predominantly CD3 antigen-positive lymphocytes, monocytes, and plasma cells set in nets of reticulin fibers. Aggregates of histiocytic cells (granulomatous nodules), sometimes with an apparent epitheloid differentiation, appear to develop eccentrically from a previously formed lymphocytic cuff and may also be seen at the center of the most severe lesions. Granulomatous lesions may compress and invade adjacent CNS parenchyma, resulting in necrosis, glial cell reaction, and edema. Coalescence of granulomatous lesions from a large number of adjacent blood vessels may produce a true space occupying mass. Focal lesions most commonly occur in the brain stem, especially in the pontomedullary region, and cerebral white matter. Large, focal lesions usually produce signs suggestive of a single, space-occupying mass, with signs varying according to the location of the lesions. These lesions can usually be detected using CT or MRI imaging techniques.

**Treatment and prognosis:** Prognosis for permanent recovery is poor. Shortest survival periods, ranging from several days to weeks, are seen with the disseminated and ocular forms. Longer survival periods of from 3-6 months are more suggestive of a focal lesion. Some dogs die from aspiration

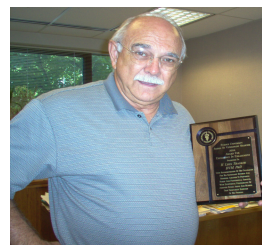
pneumonia secondary to megaesophagus. Long-term therapy is generally unsatisfactory, although temporary remission of signs is often achieved with corticosteroid administration, such as oral prednisone. Most dogs will require continued therapy to prevent recurrence of signs. Improvement may last for several days, week, or months, although most eventually succumb to the disease. Part of the temporary improvement may be related to a reduction in mast cell function in dogs receiving glucocorticoid medication. Cessation of glucocorticoid therapy is invariably associated with rapid and dramatic clinical deterioration. The ocular form of GME may be treated initially with repositol retrobulbar glucocorticoid in conjunction with oral prednisone therapy.

-by Domenico Bianco, ECFVG Student  
-edited by Dr. Angela Smith, ADDL Graduate student



## ADDL NEWS

**Dr. Leon Thacker**, ADDL Director, was awarded the Purdue University School of Veterinary Medicine 2004 Award for Excellence in Engagement at the annual SVM awards banquet in May, 2004.



## ***Salmonella newport*** An emerging disease in dairy cattle



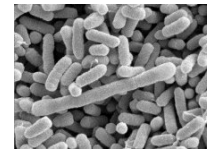
**Overview:** *Salmonella enterica* serovar *newport* has recently been named an emerging disease by the American Association of Veterinary Laboratory Diagnosticians (AAVLD). Salmonellosis, due to any of 2200 *Salmonella* serotypes (serovars) is one of the few diseases that is increasing in prevalence. While most serotypes are potential human and animal pathogens, only 10 serotypes are responsible for most disease in cattle. The National Veterinary Services Laboratories (NVSL) listed *S. newport* as one of the top ten most frequently identified *Salmonella* serotypes from U.S. cattle from July 1998 through June 1999. Nontyphoidal salmonellosis is an infection estimated to cause over 1 million cases of illness and 500 deaths in humans annually in the United States. Cull (market) dairy cows account for a large amount of beef, especially ground beef. Of 58 serotypes isolated by culture from culled dairy cows in five regional market cow establishments in the U.S., *S. newport* was among the 30 most prevalent serotypes.

*Salmonella newport* causes significant clinical disease in livestock, particularly cattle, in humans, and in other animal species. Multiple antimicrobial resistant strains of *S. newport* have been recorded in the U.S. and Canada. All of these strains are resistant to ampicillin, chloramphenicol, streptomycin, sulphonamides and tetracycline (ACSSuT). In addition, many of these strains show intermediate or full

resistance to third-generation cephalosporins, kanamycin, potentiated sulphonamides and gentamicin.

**Clinical signs:** As with most serotypes, *Salmonella spp.* Infections can cause a variety of clinical signs, and infections in cattle are most commonly subclinical. *Salmonella newport* has been implicated most commonly with adult dairy cow diarrhea and chronic weight loss or poor production. When clinical signs are present, the most common are fever and diarrhea, although weakness, dyspnea and sudden death would also be consistent with a diagnosis of suspected salmonellosis. Diarrhea may vary from watery to mucoid with fibrin and blood. Bacteremia may occur rapidly, especially in calves under two months of age. In adult dairy cattle, septicemia, diarrhea or abortion may occur. Outbreaks in older animals must be differentiated from bovine viral diarrhea, winter dysentery, and feed-induced indigestion.

**Etiology:** *Salmonella* is a gram-negative, facultative intracellular bacterium which may penetrate ocular, nasal, oral or intestinal mucous membranes. Infection is most often transmitted by fecal-oral contamination from livestock or rodents or by feeding contaminated feed. Approximately 40% of animal by-products in the U.S., such as fish meal, meat meal, bone meal, or feather meal, are contaminated. Forages or plant proteins such as soybean or cottonseed have also been sources of an outbreak.



Infection in a disease may occur as a cyclic endemic disease. Bacteria are rapidly spread among livestock and into the environment, potentially causing prolonged illness within the herd. Disease is maintained by carrier animals, infected animals, rodents, and environmental contamination, making it difficult to eliminate. It is estimated that >31% of dairy herds in Ohio have at least one infected cow, and that approximately 6% of cows are shedding

*Salmonella* spp, of one or more serotypes, at a point in time.

The group C *Salmonella* sp. , particularly *S. newport* and *S. montivideo* tend to become endemic for years on a dairy farm following a prolonged course of herd illness, as was demonstrated by several dairies in California recently. Cultures taken from calves brought in for necropsy showed that the percentage of dairies in the Tulare area from which *S. newport* could be isolated increased from 10% in 1985-1986 to 36% in 1987-1988. During both periods, *S. newport* was second only to *S. dublin*, which was isolated from 78% of farms in 1985-1986 and 53% in 1987-1988. Thus, the incidence of *Salmonella* serotypes on a farm can quickly change with the introduction and persistence of new serotypes.

**Diagnosis:** As gross lesions may vary greatly depending on the course and extent of infection, definitive diagnosis requires culture of the organism from feces, blood, or tissues. Local and regional laboratories can often identify an isolate to its group (e.g. B, C1, C2, C3), but final confirmation of serotypes comes from the National Veterinary Services Laboratory in Ames, Iowa. Confirmation of the development of anti-*Salmonella* antibodies by serology is useful, but not commonly performed and is not available in most laboratories.

**Control:** *Salmonella* has traditionally been a difficult disease to treat and control, and requires an integrated herd approach. Herd serologic profiling using ELISA can be used for all serotypes except *S. dublin*. Suspect animals should be culled or tested with serology or by multiple fecal or milk cultures. Five samples at weekly intervals is the current recommendation. Positive animals should be culled. Sanitation and disinfection of the environment may be monitored by frequent swab cultures of the pens. Preventing the introduction of exotic serotypes by culturing feeds before use on a farm is currently the

only means of protection from an outbreak.

Prompt treatment and isolation or separation of affected calves is also essential for control. Historically, *Salmonella* spp. were sensitive to florphenicol, 3<sup>rd</sup> generation cephalosporins, trimethoprim sulfa, gentamicin, amikacin, and fluoroquinolones. However, as stated previously, the presence of multiple resistant strains of *S. newport* makes antimicrobial sensitivity testing increasingly important.

-by Sara Clark, Class of 2004

-edited by Dr. Leon Thacker, ADDL  
Director

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Percent of Micro-organisms that are Resistant to Selected Antibiotics from Jul.- Dec. 2003 and Jan.-June 2004-Companion animal

| 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         | 47               | 69        | 0               | 7         | 0             | 0         | 0                  | 0         | 0         | 11        | 100            | 0         | 0             | 0         | 7                  | 0         | 100         | 100       | 92                   | 100       | 0         | 0         | 75               | 50        | 0               | 0         | 0             | 0         |
| Amoxycillin/Clauvulinic acid | 24        | 22        | 42               | 23        | 100             | 86        | 55            | 25        | 16                 | 26        | 5         | 17        | 100            | 0         | 43            | 25        | 43                 | 16        | 0           | 0         | 0                    | 0         | 11        | 17        | 25               | 0         | 100             | 100       | 60            | 100       |
| Ampicillin                   | 61        | 53        | 42               | 23        | 100             | 87        | 91            | 75        | 48                 | 65        | 44        | 41        | 100            | 50        | 100           | 50        | 64                 | 16        | 0           | 0         | 0                    | 0         | 36        | 40        | 25               | 0         | 100             | 100       | 100           | 100       |
| Cefazolin                    | 28        | 23        | 79               | 85        | 100             | 93        | 36            | 8         | 0                  | 4         | 7         | 17        | 100            | 25        | 14            | 0         | 29                 | 16        | 0           | 0         | 0                    | 0         | 7         | 17        | 100              | 50        | 100             | 100       | 0             | 33        |
| Cefotaxime                   | 11        | 5         | 56               | 31        | 8               | 64        | 36            | 8         | 0                  | 4         | 0         | 0         | 100            | 0         | 17            | 0         | 33                 | 16        | 0           | 0         | 0                    | 0         | 4         | 4         | 25               | 50        | 30              | 0         | 0             | 33        |
| Cefoxitin                    | 23        | 22        | 89               | 85        | 100             | 93        | 36            | 8         | 0                  | 4         | 7         | 13        | 100            | 0         | 14            | 0         | 43                 | 16        | 0           | 0         | 0                    | 0         | 14        | 17        | 75               | 100       | 100             | 100       | 0             | 33        |
| Ceftiofur                    | 23        | 17        | 84               | 92        | 100             | 87        | 36            | 8         | 0                  | 4         | 7         | 13        | 100            | 25        | 14            | 0         | 29                 | 11        | 0           | 0         | 0                    | 0         | 4         | 16        | 75               | 50        | 92              | 75        | 0             | 33        |
| Cephalothin                  | 39        | 37        | 79               | 77        | 100             | 93        | 36            | 8         | 0                  | 4         | 9         | 24        | 100            | 25        | 14            | 0         | 36                 | 16        | 0           | 0         | 0                    | 0         | 11        | 17        | 50               | 50        | 100             | 100       | 0             | 33        |
| Chloramphenicol              | 24        | 17        | 0                | 8         | 93              | 79        | 0             | 0         | 0                  | 4         | 21        | 33        | 100            | 50        | 0             | 0         | 14                 | 0         | 0           | 0         | 8                    | 0         | 11        | 13        | 0                | 0         | 100             | 100       | 0             | 0         |
| Ciprofloxacin                | 25        | 20        | 44               | 0         | 0               | 14        | 36            | 17        | 0                  | 4         | 0         | 4         | 100            | 0         | 0             | 0         | 17                 | 0         | 0           | 0         | 0                    | 0         | 4         | 17        | 25               | 50        | 0               | 0         | 0             | 33        |
| Clindamycin                  | 100       | 99        | 83               | 69        | 100             | 93        | 27            | 8         | 13                 | 13        | 100       | 98        | 0              | 100       | 0             | 0         | 29                 | 11        | 0           | 0         | 0                    | 5         | 100       | 100       | 50               | 100       | 100             | 100       | 20            | 33        |
| Enrofloxacin                 | 24        | 20        | 58               | 23        | 50              | 33        | 36            | 17        | 0                  | 4         | 2         | 4         | 100            | 0         | 0             | 0         | 14                 | 0         | 0           | 0         | 0                    | 0         | 4         | 16        | 25               | 50        | 42              | 0         | 20            | 33        |
| Erythromycin                 | 100       | 98        | 37               | 31        | 100             | 93        | 18            | 8         | 16                 | 17        | 100       | 98        | 0              | 100       | 14            | 0         | 21                 | 21        | 50          | 50        | 23                   | 62        | 100       | 100       | 25               | 50        | 100             | 100       | 20            | 33        |
| Gentamicin 500 microgm/ml    | 0         | 2         | 6                | 31        | 0               | 0         | 0             | 0         | 0                  | 0         | 0         | 11        | 100            | 0         | 0             | 0         | 0                  | 0         | 0           | 0         | 0                    | 0         | 4         | 0         | 25               | 50        | 0               | 0         | 0             | 0         |
| Gentamicin                   | 17        | 11        | 42               | 31        | 0               | 13        | 0             | 0         | 10                 | 0         | 28        | 30        | 100            | 13        | 0             | 50        | 14                 | 5         | 0           | 0         | 69                   | 81        | 4         | 4         | 25               | 50        | 0               | 0         | 0             | 0         |
| Sulphadimethoxine/Ormetoprim | 41        | 33        | 6                | 8         | 100             | 93        | 9             | 0         | 6                  | 0         | 100       | 54        | 100            | 25        | 17            | 0         | 17                 | 11        | 0           | 0         | 0                    | 0         | 12        | 13        | 0                | 0         | 100             | 100       | 0             | 0         |
| Oxacillin + 2% NaCl          | 99        | 99        | 83               | 92        | 100             | 86        | 36            | 8         | 0                  | 4         | 100       | 98        | 0              | 100       | 14            | 0         | 29                 | 11        | 0           | 0         | 0                    | 0         | 100       | 100       | 100              | 100       | 92              | 100       | 0             | 33        |
| Penicillin 003               | np        | 99        | np               | 92        | np              | 93        | np            | 58        | np                 | 52        | np        | 98        | np             | 100       | np            | 25        | np                 | 26        | nt          | 0         | np                   | 0         | np        | 100       | np               | 100       | np              | 100       | np            | 67        |
| Penicillin                   | 100       | 99        | 47               | 23        | 100             | 93        | 91            | 67        | 35                 | 57        | 100       | 98        | 0              | 100       | 71            | 50        | 57                 | 16        | 0           | 0         | 0                    | 0         | 100       | 100       | 25               | 0         | 100             | 100       | 60            | 100       |
| Rifampin                     | 94        | 99        | 5                | 8         | 100             | 93        | 0             | 0         | 0                  | 0         | 95        | 98        | 0              | 100       | 0             | 0         | 14                 | 0         | 0           | 0         | 0                    | 0         | 82        | 78        | 25               | 0         | 100             | 100       | 0             | 0         |
| Tetracycline                 | 46        | 47        | 58               | 46        | 33              | 36        | 0             | 17        | 26                 | 35        | 49        | 41        | 100            | 50        | 14            | 25        | 21                 | 16        | 0           | 0         | 38                   | 43        | 7         | 26        | 50               | 50        | 58              | 0         | 20            | 0         |
| Ticarcillin                  | 55        | 43        | 47               | 23        | 20              | 0         | 91            | 67        | 35                 | 57        | 44        | 39        | 71             | 50        | 71            | 50        | 64                 | 21        | 0           | 0         | 0                    | 0         | 36        | 39        | 25               | 0         | 17              | 0         | 60            | 100       |
| Tribrissen                   | 41        | 32        | 6                | 8         | 58              | 29        | 18            | 0         | 23                 | 9         | 39        | 54        | 100            | 25        | 33            | 25        | 17                 | 16        | 0           | 0         | 0                    | 0         | 12        | 13        | 0                | 50        | 40              | 25        | 25            | 0         |
| Vancomycin                   | 100       | 99        | 0                | 0         | 100             | 93        | 0             | 0         | 0                  | 0         | 100       | 98        | 100            | 100       | 0             | 0         | 0                  | 5         | 0           | 0         | 0                    | 0         | 100       | 100       | 0                | 0         | 100             | 100       | 0             | 0         |
| number of isolates           | 83        | 81        | 19               | 13        | 16              | 15        | 11            | 12        | 31                 | 23        | 43        | 46        | 7              | 8         | 7             | 4         | 14                 | 19        | 2           | 4         | 13                   | 21        | 28        | 23        | 4                | 2         | 12              | 4         | 5             | 3         |

np - not performed

Percent of Micro-organisms that are Resistant to Selected Antibiotics from Jul.- Dec. 2003 and Jan.-June 2004-Food animals

| Antibiotic             | Beef      |           |                   |           |                 |           |                |           | Dairy     |           |                   |           |                 |           |               |                | Swine     |           |           |           |           |                |           |             |           |           |
|------------------------|-----------|-----------|-------------------|-----------|-----------------|-----------|----------------|-----------|-----------|-----------|-------------------|-----------|-----------------|-----------|---------------|----------------|-----------|-----------|-----------|-----------|-----------|----------------|-----------|-------------|-----------|-----------|
|                        | E. coli   |           | Past. Haemolitica |           | Past. Multocida |           | Salmonella sp. |           | E. coli   |           | Past. Haemolitica |           | Past. Multocida |           | Staph. aureus | Salmonella sp. |           | APP       |           | 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.      | Jan.-June | July-Dec. | Jan.-June | July-Dec. | Jan.-June | July-Dec.      | Jan.-June | July-Dec.   | Jan.-June | July-Dec. |
| Ampicillin             | 28        | 33        | 14                | 25        | 8               | 0         | 50             | 75        | 59        | 55        | 50                | 40        | 0               | 0         | 32            | 38             | 54        | 75        | 18        | 16        | 59        | 61             | 63        | 35          | 4         | 1         |
| Apramycin              | 3         | 4         | 71                | 0         | 100             | 25        | 0              | 3         | 22        | 26        | 0                 | 0         | 70              | 75        | na            | na             | 0         | 3         | 5         | 2         | 17        | 21             | 8         | 3           | 29        | 28.3      |
| Ceftiofur              | 3         | 8         | 0                 | 0         | 8               | 0         | 50             | 69        | 20        | 25        | 0                 | 0         | 0               | 0         | 5             | 0              | 50        | 69        | 0         | 2         | 13        | 17             | 30        | 15          | 9         | 3         |
| Chlortetracycline      | 52        | 57        | 14                | 0         | 8               | 0         | 50             | 78        | 78        | 82        | 50                | 40        | 0               | 13        | np            | np             | 53        | 78        | 5         | 5         | 92        | 95             | 83        | 85          | 85        | 92        |
| Clindamycin            | 100       | 100       | 100               | 100       | 100             | 100       | 100            | 100       | 96        | 96        | 50                | 100       | 90              | 100       | np            | np             | 100       | 100       | 27        | 5         | 100       | 100            | 100       | 100         | 95        | 90        |
| Enrofloxacin           | 14        | 6         | 14                | 50        | 8               | 0         | 0              | 0         | 15        | 16        | 0                 | 40        | 0               | 0         | np            | np             | 0         | 0         | 0         | 0         | 0         | 0              | 0         | 0           | 1         | 4         |
| Erythromycin           | 100       | 100       | 14                | 25        | 38              | 0         | 100            | 100       | 98        | 97        | 0                 | 20        | 8               | 38        | 5             | 6              | 98        | 100       | 5         | 14        | 100       | 100            | 100       | 100         | 95        | 97        |
| Florphenicol           | 100       | 100       | 14                | 25        | 25              | 0         | 100            | 100       | 98        | 96        | 0                 | 20        | 10              | 38        | np            | np             | 100       | 100       | 0         | 2         | 99        | 100            | 100       | 100         | 58        | 60        |
| Gentamicin             | 17        | 12        | 0                 | 0         | 25              | 0         | 25             | 0         | 40        | 45        | 0                 | 20        | 0               | 13        | np            | np             | 11        | 0         | 0         | 0         | 21        | 23             | 5         | 0           | 7         | 4         |
| Neomycin               | 21        | 24        | 43                | 25        | 75              | 0         | 50             | 25        | 67        | 70        | 50                | 80        | 20              | 63        | np            | np             | 25        | 25        | 5         | 18        | 40        | 51             | 28        | 9           | 49.3      | 50.3      |
| Oxytetracycline        | 62        | 61        | 29                | 50        | 63              | 0         | 50             | 78        | 82        | 82        | 50                | 80        | 20              | 63        | np            | np             | 55        | 78        | 36        | 66        | 96        | 97             | 85        | 85          | 89.2      | 95.1      |
| Penicillin             | 100       | 100       | 57                | 25        | 25              | 25        | 100            | 100       | 98        | 97        | 100               | 80        | 17              | 25        | 32            | 38             | 100       | 100       | 68        | 70        | 100       | 100            | 100       | 100         | 8         | 5         |
| Sulphadimethoxine      | 48        | 39        | 43                | 50        | 75              | 0         | 50             | 83        | 57        | 65        | 50                | 80        | 42              | 63        | 100           | 88             | 61        | 83        | 18        | 25        | 69        | 58             | 65        | 74          | 60.4      | 59.1      |
| Spectinomycin          | 41        | 33        | 71                | 100       | 75              | 50        | 100            | 100       | 64        | 63        | 50                | 80        | 20              | 75        | np            | np             | 93        | 100       | 32        | 64        | 62        | 74             | 95        | 97          | 35.8      | 21.5      |
| Sulphachloropyridazine | 48        | 39        | 43                | 50        | 75              | 25        | 50             | 78        | 80        | 82        | 0                 | 40        | 80              | 88        | np            | np             | 55        | 78        | 18        | 14        | 69        | 86             | 63        | 74          | 59.3      | 59.1      |
| Sulphathiazole         | 48        | 39        | 57                | 50        | 75              | 50        | 50             | 78        | 78        | 82        | 50                | 80        | 60              | 88        | np            | np             | 55        | 78        | 14        | 50        | 68        | 86             | 63        | 74          | 61.3      | 63.4      |
| Tiamulin               | 100       | 100       | 71                | 50        | 88              | 75        | 100            | 100       | 96        | 96        | 50                | 40        | 90              | 100       | np            | np             | 100       | 100       | 9         | 5         | 100       | 99             | 100       | 100         | 24.4      | 18.6      |
| Tilmicosin             | 93        | 96        | 14                | 25        | 63              | 0         | 100            | 100       | 95        | 96        | 0                 | 0         | 10              | 50        | np            | np             | 100       | 100       | 9         | 7         | 98        | 100            | 100       | 100         | 85.5      | 85.6      |
| Triple Sulfa           | 24        | 18        | 14                | 0         | 0               | 0         | 25             | 8         | 56        | 55        | 0                 | 40        | 0               | 13        | np            | np             | 4         | 8         | 0         | 2         | 14        | 28             | 18        | 9           | 2         | 1         |
| Tylosin                | 100       | 100       | 100               | 100       | 75              | 100       | 100            | 100       | 96        | 96        | 100               | 100       | 90              | 100       | np            | np             | 100       | 100       | np        | np        | 100       | 100            | 100       | 100         | np        | np        |
| number of isolates     | 29        | 51        | 7                 | 4         | 8               | 4         | 4              | 36        | 55        | 76        | 2                 | 5         | 10              | 8         | 22            | 15             | 55        | 36        | 22        | 44        | 121       | 159            | 40        | 34          | 105       | 103       |

np - not performed