

**Fall 2001**



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

As this is written, the events of the World Trade Center, the Pentagon and in a field in eastern Pennsylvania are yet unfolding. Our understanding of the thoughts and feelings of individuals that led to these disasters are largely unavailable. Our thoughts and prayers go out to those who have lost friends and loved ones in the disasters. We pray that our administrators and individuals in significant positions who must make hard and firm decisions in the coming days to address and direct our national responses make

correct and well thought judgements.

In relation to the enormity and consequences of war-like events, any words included here will pale by comparison, but as others have requested and suggested, our operations will continue and the importance of our efforts to the users of our services will not be diminished. We have been reminded to be especially alert to diagnosing unusual or foreign diseases in our animal populations as the potential for terrorism to include bioterrorism tactics affecting our animals and food supplies has also not been diminished.

The identification of West Nile virus in Indiana has had a definite effect on the way we perform necropsies on horses. Even though the viremia of horses has been shown to have low levels of virus, we have initiated procedures to reduce and, if possible, eliminate exposure to the virus during suspect horses' necropsies. We should all remain aware that birds dying from WNV infection usually have high levels of virus in tissues as well as in body fluids and feces. If we are asked to handle WNV birds, we should protect ourselves accordingly.

We are excited about the recent availability of ADDL laboratory results on the web. Our computer people have done an excellent job in installing, adjusting and realigning our new computer system. Steve Vollmer, Tim Kechkaylo and Jennifer Hewitt, as well as Alan Bunning (who moved to a teaching position at Lafayette Ivy Tech a few months ago) are the individuals responsible for the success of our present computer system.

We are also very pleased to have on board our newest faculty member, Dr. Ramesh Vemulapalli, as molecular immunologist on our faculty. Dr. Vemulapalli will be responsible for development and evaluation of new tests to be offered by ADDL. We look forward to his contributions to our test offerings and to serving Indiana animal owners. We also look forward to welcoming Dr. Zheko Kounev to our faculty on October 15 of this year. Dr. Kounev will serve as avian diagnostician and food safety specialist on our faculty. He comes to us with impeccable past experience in both areas in industry and academic appointments.

Hope you have a great fall, stop in to see us if you are in our vicinity.

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## FINAL DIAGNOSIS

### Pulmonary paragonimiasis

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

**History:** The right middle lung lobe from a 3-year-old domestic shorthair cat was submitted for histopathology. The cat had presented to the referring veterinarian with the primary complaint of a persistent cough. The cat was wheezing, but no other abnormalities were noted. A heartworm test was negative. No parasitic ova were detected on fecal flotation. No abnormalities were noted on a CBC and chemistry panel. Thoracic radiographs were sent to a radiologist for interpretation. Approximately one week after the first visit, the cat presented in respiratory distress. Interpretation of the radiographs from the first visit revealed collapse of the right middle lung lobe with prominent bronchial markings in the remainder of the lung. The right middle lung lobe was excised and submitted for histopathology.

**Gross Findings:** A few, 1.0-2.0 cm in diameter, red-brown, poorly delineated, firm nodules bulged from the right middle and right caudal lung lobes.

**Histopathology:** Alterations in sections of the right middle lung lobe included atelectasis, mild multifocal granulomatous pneumonia with intralesional trematode eggs, and hyperplasia of the bronchiolar epithelium and peribronchiolar glands. The trematode eggs were oval, yellow-brown, and approximately 100 microns in length by 55 microns in diameter with a single, flattened operculum, morphologically typical of *Paragonimus kellicotti*. Adult flukes were not observed.

**Discussion:** *Paragonimus kellicotti* is a trematode parasite of cats, dogs, mink, muskrats, raccoons, skunks, coyotes, foxes, goats, pigs and opossums in North America. It is found in the North Central, Midwest, and Southeast regions of the United States.

The distribution of *Paragonimus kellicotti* is restricted to the area inhabited by its first intermediate host, *Pomatiopsis lapidaria*, an aquatic snail. The second intermediate hosts are crayfish of the genera *Cambarus*, *Procambarus*, or *Orconectes*. Definitive hosts become infected when they ingest crayfish with encysted metacercariae. Metacercariae exist in the small intestine of the definitive host, and the immature flukes penetrate the intestinal wall, migrate in the peritoneal cavity, penetrate the

diaphragm, migrate in the pleural cavity, and penetrate the lung where they mature into adult flukes. Adult flukes live in pairs within cystic cavities connected to bronchioles. Eggs from adult flukes are carried up the mucociliary escalator, swallowed, and excreted with the feces.

The severity of clinical signs caused by *Paragonimus kellicotti* relates to the number of infecting flukes. Migration of immature flukes can cause lethargy, pyrexia, anorexia, and weight loss. Adult flukes and their eggs can cause chronic coughing, dyspnea, salivation and hemoptysis.

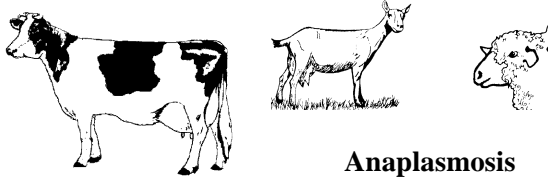
Lesions caused by migration of the immature flukes include multifocal eosinophilic peritonitis, hepatitis, and diaphragmatic myositis. Mediastinal and pulmonary pleural adhesions to the diaphragm and costal pleura can also occur. Cysts containing adult flukes can be observed as 1.0-2.0 cm in diameter, red-brown, firm, spherical pulmonary nodules. Adult flukes are red brown and approximately 1.1 x 0.6 x 0.5 cm. Microscopic lesions include multifocal eosinophilic and granulomatous pneumonia with intralesional trematode ova, hyperplasia of bronchiolar epithelium and peribronchiolar glands, and peribronchiolar infiltration of lymphocytes and plasma cells. Extrapulmonary granulomatous lesions have been reported.

Diagnosis of paragonimiasis can be made with detection of eggs on fecal sedimentation or transtracheal wash. Pulmonary cysts can be observed on radiographs. Treatment of paragonimiasis with an appropriate anthelmintic is usually rewarding. The anthelmintic kills adult flukes, and the cystic lesions resolve. Surgery is typically not necessary.

The incidence of paragonimiasis is reportedly increasing around the Great Lakes due to increased numbers of a second intermediate host, *Orconectes rusticus*, the rusty crayfish, which is thought to have been inadvertently introduced to the Great Lakes by fishermen. Paragonimiasis should be considered in outdoor pets that present with a chronic cough.

-by Matt Renninger, DVM, ADDL Graduate Student





**Anaplasmosis**

Anaplasmosis is a hemoparasitic, infectious, and transmissible disease characterized by progressive anemia with intraerythrocytic *Anaplasma* bodies. *Anaplasma marginale* infects cattle, while *Anaplasma ovis* infects sheep and goats.

The disease can be divided into four stages. The first stage, or incubation stage, ranges from the time of introduction of the organism into the susceptible animal until the time that 1% of the erythrocytes are infected. The first stage typically varies from one to three months in length. No clinical signs are seen during this time. The second stage, or developmental stage, is the stage during which anemia develops and lasts until reticulocytes appear in the peripheral circulation. The second stage typically varies from four to nine days in length. Most of the clinical signs characteristic of anaplasmosis appear during this stage. The third stage, or convalescent stage, marks the resolution of the anemia. The duration of this stage varies greatly. During the fourth stage, or carrier stage, the animal remains infected, but *Anaplasma* bodies cannot be detected in the peripheral blood. The fourth stage can last indefinitely.

### Transmission

Anaplasmosis is typically transmitted by ticks or biting flies. Iatrogenic transmission can occur when instruments are re-used without proper sanitation, including instruments used for dehorning, ear tagging, castrating, and vaccinating. *In utero* transmission has been reported.

### Clinical Signs

Clinical signs increase in severity as the animal ages. The first clinical sign is typically fever, ranging from 103° F to 106° F and lasting 12-24 hours. Most other clinical signs are manifestations of acute anemia, including mucosal pallor, muscle weakness, tachycardia, tachypnea, exercise intolerance, and behavioral

changes. Additional signs that may be present include depression, anorexia, ptyalism, dehydration, constipation, and frequent urination with dark yellow urine. Hemoglobinuria does not occur because the anemia results from the destruction of parasitized erythrocytes in the spleen, not from intravascular hemolysis. Jaundice and weight loss may occur later in the disease. Milk production declines rapidly in dairy cows. *A. ovis* infection in sheep and goats is typically asymptomatic.

### Lesions

There are no pathognomonic gross lesions for anaplasmosis, but lesions can be suggestive of the disease. In acute cases, the blood is thin and watery and fails to readily clot. Gross postmortem findings typically include severe anemia with pallor of tissues and occasionally icterus. The spleen is generally enlarged with reddish-brown pulp and enlarged splenic follicles. The liver may be enlarged with rounded edges, and may be yellow in cases of icterus. The gall bladder is typically distended with bile. Histologic findings include bone marrow hyperplasia and extramedullary hematopoiesis in the spleen and other organs. *Anaplasma* organisms may be found in erythrocytes with smears of the peripheral blood.

### Treatment

Tetracyclines have been shown to reduce the severity of the disease, and blood transfusions have been used as supportive therapy. After recovery, additional treatment with tetracyclines is necessary to prevent animals from becoming asymptomatic carriers.

-by Julie Tucker, Class of 2001

-edited by Dr. Matt Renninger, ADDL  
Graduate Student

References on page 3.

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ADDL introduces its newest faculty member, **Dr. Ramesh Vemulapalli**. In addition to research activities, he will focus on efforts to develop additional molecular

diagnostics tests at Purdue ADDL.

Dr. Vemulapalli completed his BVSc and MVSc degrees at the Andhra Pradesh Agricultural University and the Indian Veterinary Research Institute, respectively, both in India. He earned his PhD in Molecular Microbiology and Immunology in 1996 from the University of Maryland and completed postdoctoral study at the Center for Molecular Medicine and Infectious Diseases at Virginia Tech in 2000.

Please join us in welcoming Dr. Vemulapalli, his wife, Dr. Tracy Vemulapalli and daughter Maya to Purdue.



**ADDL STAFF NEWS**

Dr. **Cindy Fishman** completed her PhD program at Purdue ADDL and Eli Lilly in June, 2001. She has accepted a position as pathologist at Merck and Co. in New Jersey.



Dr. **Matti Kiupel** completed his PhD program in June, 2001. He is a pathologist at Michigan State University Animal Health Diagnostic Laboratory in Lansing, Michigan.



ADDL welcomes its new graduate students Drs. **Theresa Boulineau, Alok Sharma and Sandra Schoeniger**.



Dr. Boulineau



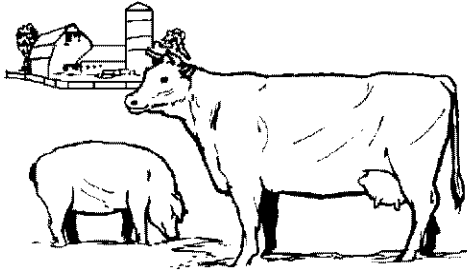
Dr. Sharma



Dr. Schoeniger

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

November 22-23.....Thanksgiving  
 December 24-25.....Christmas  
 December 31- January 1..... New Year's



### **Foot and Mouth Disease: Prevention and Diagnostics**

Foot and mouth disease (FMD) is a highly contagious, rapidly spreading disease that lives in the lymph nodes and bone marrow of cloven-hooved animals. Infected and carrier animals, as well as humans, vehicles, water, semen, equipment, facilities, clothing, footwear and feedstuffs can spread the virus. FMD causes nearly 100% morbidity and usually low mortality. The loss of production during illness and failure of animals to return to previous performance levels (meat and milk production) after infection cause continuous and severe economic losses. FMD is associated with blisters in the mouth, on the teats and on the feet, which rupture leaving erosions. Signs such as excessive salivation, lameness, reluctance to stand, abortion, and decrease in productivity may also indicate FMD. Humans are not severely affected by the disease, but may carry it in their nasal passages and subsequently spread the disease to animals.

The key to preventing an FMD outbreak is an excellent biosecurity program. Visitors that are from or have recently visited countries where FMD is known to occur should not be allowed contact with susceptible animals for five days after return to the United States. Clean boots and coveralls should also be provided to these guests before entering a farm or other premises where susceptible animals are kept. The importation of live ruminants and swine and their by-products from countries with FMD has been prohibited by the USDA. If traveling to an FMD-affected country, one should avoid farms, sale barns, stockyards, fairs, zoos, etc. Proper disinfection and cleaning of clothing and footwear should be done immediately upon return to the U.S. Again, these persons should

wait at least five days before visiting areas with susceptible livestock. Swill feeding has also come under fire as a possible source of FMD. Swill should be cooked at appropriate temperatures to denature the FMD virus proteins before being fed to hogs. The practice of swill feeding may soon be prohibited in the United States. Sheep may not always show signs of the disease while they are infected; therefore, they should be kept away from other susceptible animals (such as cattle) on the farm. Animals should be inspected daily for signs of FMD and any abnormalities should be further inspected by a veterinarian.

In the face of a Foot and Mouth Disease outbreak, vaccination is an option to decrease the spread of the disease. The U.S. does not currently vaccinate because it is currently in FMD-free status. Countries that vaccinate for FMD lose their FMD-free status for at least three months to a year after the slaughter of the last vaccinated animals. The economic impact due to restrictions on livestock exports is tremendous. Cattle that have been vaccinated can become persistently infected without ever showing clinical signs. Vaccinated cattle which are persistently infected cannot be distinguished from animals that have merely been vaccinated. Some benefits of vaccination to prevent the spread of FMD are the small number of people needed to carry it out, limited traumatic experiences to the farmers (other than on outbreak farms) and minimal social or economic damage to rural societies. Sanitary rules for containment must also be carried out in compliance with the vaccination protocol for success in the prevention of FMD.

Demonstration of FMD viral antigen alone in samples of tissue or fluid is sufficient for a positive diagnosis. The preferred tissue for diagnosis is epithelium from unruptured or freshly ruptured vesicles. When this is not possible, blood and/or esophageal-pharyngeal fluid samples taken by a probing cup in ruminants or throat swabs in pigs are acceptable. Myocardial tissue or blood can also be submitted from fatal cases. The enzyme-linked immunosorbent assay (ELISA) is an extremely sensitive and specific test used to identify the FMD antigen. The ELISA test has a high sensitivity for detection of acute infection,

subclinical infection and late stages of persistent infection in affected animals. The ELISA test is rapid, safe and economical as well as being sensitive and specific.

Diagnosis by detection of a specific humoral antibody can also be used, but this requires a history of no prior vaccination. These serological tests may be performed in mild cases or where epithelial tissue cannot be collected. The liquid-phase blocking ELISA test and virus neutralization tests are used as serotype-specific serological tests. The ELISA test is much quicker to perform and does not rely on cell cultures, whereas the virus neutralization test does.

The reverse transcriptase polymerase chain reaction (RT-PCR) is an extremely sensitive method of detecting the FMD viral genome. The advantage of this test is that viable virus or intact viral antigen are not necessary. However, there are several disadvantages to this test. The high sensitivity comes with the possibility of false positive results. The test also requires very stringent working practices and a sterile environment in order to prevent contamination of the sample. RT-PCR is rarely used as the primary test for identifying FMD, but may be used as an additional confirmatory test. The test does, however, provide an efficient distinction between FMD and other viruses that cause clinically indistinguishable vesicular lesions.

Virus isolation may be performed on tissue culture where there is not sufficient antigen in the sample to give a positive diagnosis of FMD by other tests. Live virus in the tissue samples is amplified by culture. The presence of FMD virus is determined by the observation of virus-specific cytopathic effect after inoculation.

Foot and mouth disease is a rapidly-spreading, devastating disease. Please educate your producers about symptoms and precautions to help prevent the spread of the disease into the United States.

-by Sara Wesdorp, Class of 2002

-edited by H.L. Thacker, DVM, PhD,

ADDL Director

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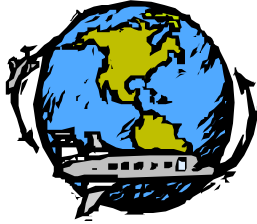
[www.aphis.usda.gov](http://www.aphis.usda.gov)

[www.maff.gov.uk](http://www.maff.gov.uk)

[www.aleffgroup.com/avisfmd](http://www.aleffgroup.com/avisfmd)

[www.guardian.co.uk/footandmouth](http://www.guardian.co.uk/footandmouth)





### ON THE ROAD

Drs. Ching Ching Wu and Tsang Long Lin were invited speakers at the 2<sup>nd</sup> International Symposium on Infectious Bursal Disease Virus and Chicken Anemia Virus in Rauschholzhausen, Germany, June 2001.

Drs. Leon Thacker, Ching Ching Wu, and Tsang Long Lin attended the American Veterinary Association annual meeting in Boston, Massachusetts, July 2001.

Dr. Leon Thacker was invited to speak on international export/import regulations by the Department of Agriculture, Beijing, China, August, 2001

Christina Wilson and Regina Bedel presented papers at the American Association of Analytical Chemists meeting in Peoria, Illinois, June 2001.

Drs. Kaori Sakamoto and Leon Thacker spoke to veterinary students at North Carolina State University on pathology as a career choice in September, 2001.

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### Case Study: Enteroviral Polioencephalomyelitis in Finishing-age Pigs

#### History

On a 300 sow, farrow-to-finish pork production facility in Indiana, 2 out of approximately 2500 (0.08%) 12 to 15-week old finishing pigs were getting progressively ataxic and uncoordinated over a period of one day. No other pigs in the finishing barn were showing clinical signs and mortality in the nursery and finishing barn for the past 6-9 months had averaged 1%. The owner wanted to know what the problem was before a potentially large outbreak occurred. What differentials should be considered?

#### Relatively Common Causes of CNS Disease in Finishing-age Pig

1. Bacterial otitis media – usually few pigs are affected with head-tilt, a droopy ear, ataxia and/or recumbency.
2. Bacterial meningitis caused by *S. suis* or *H. parasuis* – although less common in finishing pigs than in nursery-age pigs, limited sporadic outbreaks occur in finishing pigs.
3. Pseudorabies virus – although it is tempting to eliminate this pathogen from consideration following eradication efforts, it is better to be safe than sorry! Clinical symptoms in finishing pigs include flu-like pneumonia with coughing and dyspnea, tremors, circling, head tilt, lateral recumbency, opisthotonus, nystagmus and convulsions.
4. Water deprivation/salt toxicity – commonly caused by human error, but may be due to faulty or poorly adjusted equipment. Symptoms include irritability, convulsions and lateral recumbency

#### Less Common Causes of CNS Disease in Finishing-age Pigs

1. Enteroviruses – cause sporadic encephalomyelitis manifested as tremors,

convulsions, recumbency, ataxia, posterior paresis and/or posterior paralysis.

2. PRRS virus – although uncommon, some highly virulent strains of PRRSV can cause cerebral vasculitis and encephalitis that is fatal.
3. Rabies virus – very uncommon in pigs due to husbandry practices, but should be considered due to fatal zoonotic implications.
4. Edema disease – more common in nursery-age pigs, but can occur sporadically in finishing pigs (known as cerebrospinal angiopathy). Clinical symptoms include ataxia and convulsions.
5. Bacterial encephalitis caused by *Salmonella choleraesuis*. Some strains of *S. choleraesuis* can cause cerebral vasculitis and encephalitis as a component of septicemic salmonellosis. In these cases, the more common purple discoloration of skin, ischemic necrosis of skin on the tips of the ears, diarrhea and other well-known gross lesions are usually present and help with a strong presumptive diagnosis.
6. Intoxications – chlorinated hydrocarbon or organophosphate pesticides are the most common toxic causes of tremors or convulsions. Selenium intoxication due to feed mixing errors causes ataxia and posterior paresis/paralysis. Inorganic arsenicals cause paralysis.

### **On-Farm Examination**

**Clinical Examinations:** On arrival, only the 2 reported finishing pigs were affected. They were both in good flesh and had normal rectal temperatures. Pig 1 was alert, but severely ataxic. Pig 2 was mentally dull and laying down. Attempts to get the pig up resulted in a sitting position, with the pig soon opting to lay down.

**Necropsy Examinations:** In pig 1 the lungs were diffusely gray and rubbery with petechia on the pleural surface. Frothy white fluid oozed from the cut surface of the lung. Interstitial

pneumonia was suspected. In pig 2 there were no observed gross lesions.

### **Submission to a Diagnostic Laboratory**

Tissues should be collected in 10% neutral buffered formalin for histopathology and fresh/refrigerated for bacteriology, virology, and toxicology.

- Meningeal swabs collected aseptically for bacterial culture. Disarticulate the skull from the atlas, flame the foramen magnum with a portable torch, carefully insert a culturette™-type swab into the calvarium through the foramen magnum, swab around the brain stem and cerebellum, withdraw and insert into the plastic sheath-release the fluid to keep the swab moist and refrigerate.
- Brain
- Spinal cord. **Due to the history of ataxia and paresis, it is imperative to remove the spinal cord for testing.** It may be easier to anticipate the difficulty and time required and, instead, submit the entire pig for necropsy.
- Ileum or ileal mucosal swab for bacterial culture (*E. coli* strains associated with edema disease; positive for F18 or K88 fimbria and contain the gene for edema disease toxin – synonyms include SLTII<sub>variant</sub>, STX toxin and shigatoxin E).
- Tonsil
- Spleen
- Lung
- Serum

### **Results of Laboratory Testing**

#### ***Histopathology:***

Brainstem: nonsuppurative encephalitis, multifocal, moderate  
Spinal cord: nonsuppurative poliomyelitis, multifocal, severe  
Lung: interstitial pneumonia, histiocytic, multifocal, moderate

#### ***Bacteriology:***

No significant or consistent bacterial isolates



## ***Virology***

Serum: PRRSV positive by virus isolation  
Tonsil and brain; PRV negative by  
fluorescent antibody test

**Brain and spinal cord: Enterovirus,  
positive by virus isolation test**

## ***Final Diagnosis***

PRRS  
Entoviral polioencephalomyelitis

## **Additional History and Comments**

Five years ago this farm suffered high morbidity and financial losses due to enteroviral poliomyelitis in finishing-age pigs. The offending enterovirus was isolated and serotyped as a type 6. As many as 50% of pigs in finishing barns became ataxic and developed posterior paralysis necessitating euthanasia or premature sale. For this reason, and due to concurrent problems with PRRS, the farm was depopulated for one month, the pits were emptied, all buildings including pits were cleaned and disinfected and the farm was repopulated.

In the Purdue Animal Disease Diagnostic Laboratory, we typically diagnose 3-5 cases of polioencephalomyelitis due to porcine enteroviruses per year. Most cases involve either nursery- or finishing-age pigs. Usually, few animals are affected and the disease is quickly self-limiting. However, in the past 5 years, we have seen 2 outbreaks on unrelated geographically distant farms that were very severe and that continued despite various attempts to prevent the disease through management changes. It is unclear whether these severe outbreaks are due to unusually virulent viral strains, introduction of new viral strains onto farms with immunologically naïve pigs, changes in modern swine rearing practices that create subpopulations of pigs that are more susceptible to endemic enteroviruses or other factors.

## **Porcine Enteroviruses and Disease**

***Etiology:*** Family Picornaviridae, virions are spherical, 25-31nm, nonenveloped, single stranded RNA virus. They are stable when treated with lipid solvents and heat. The virions are resistant to pH values from 2 to 9 and are resistant to various disinfectants. Only sodium

hypochlorite and 70% ethanol inactivate enteroviruses. The virus survives in the environment for more than 168 days at 15°C and can also survive in liquid manure for long periods of time. Manure aeration, ionizing radiation of liquid manure and anaerobic digestion can inactivate the virus.

***Epidemiology:*** Transmission of porcine enterovirus is by the fecal-oral route and transmission by mechanical vectors is likely. There are at least 11 serotypes of enteroviruses in swine, based on virus neutralization tests. The degree of cross-protection between serotypes is unknown. Teschen disease, a severe encephalomyelitic disease, is caused by highly virulent serotype 1 strains not found in North America. Less virulent serotype 1 strains cause a milder form of polioencephalomyelitis known as Talfan disease or enzootic paresis. These Talfan strains, as well as strains of all other serotypes, are found in North America. Endemic infection with porcine enteroviruses is common. In fact, nearly all swine herds are endemically infected with multiple serotypes of porcine enterovirus. Infection is likely maintained in susceptible weaned pigs following decay of maternal antibody. Factors that cause periodic limited or less common severe outbreaks of disease are poorly understood.

***Clinical signs:*** Most infections with porcine enteroviruses are asymptomatic; i.e., endemic infection of most swine herds is completely without clinical disease. The best characterized and most financially significant clinical syndromes caused by porcine enteroviruses include nervous disease (polioencephalomyelitis) and reproductive failure (SMEDI syndrome). Enteroviruses have also been suggested as possible causes of mild diarrhea, pneumonia, pericarditis and myocarditis.

**Polioencephalomyelitis** – Teschen disease, caused by highly virulent serotype 1 strains, is characterized by high morbidity and mortality in all ages of pigs. Polioencephalomyelitis manifests clinically first with fever, anorexia and ataxia and later with opisthotonus, convulsions, nystagmus, paralysis, coma and death within 3-4 days. Talfan disease is less severe with lower morbidity and mortality and usually occurs in nursery- or finishing-age pigs.

Reproductive failure – The constellation of symptoms associated with enteroviral reproductive failure is known by the acronym “SMEDI” which stands for stillbirth, mummified fetuses, embryonic death and infertility. Pregnant females show no clinical symptoms and overt abortions are not common so disease is manifested as an increase in “not-in-pig” or NIP sows that once tested positive for pregnancy as well as an increase in stillborn and mummified pigs in newborn litters.

**Diagnosis:** Enteroviral disease is confirmed by demonstrating appropriate clinical symptoms, gross lesions (reproductive disease) or microscopic lesions (polioencephalomyelitis) and the presence of enterovirus in fetuses (reproductive disease) or central nervous tissue (polio-encephalomyelitis). Because of the ubiquitous nature of porcine enteroviruses, demonstration of virus in tissues other than nervous tissue in pigs with nervous disease does not constitute a diagnosis. Virus isolation is the method commonly employed to detect enteroviruses; however, the test has only moderate sensitivity in nervous tissues from pigs with nervous disease and very low sensitivity in degenerate tissues from stillborn and mummified fetuses. Recently, an nRt-PCR test was described, but the test is not generally available. The veterinary diagnostic laboratory at Iowa State University has just recently developed an Rt-PCR test for porcine enteroviruses. The sensitivity is not well characterized, but should exceed that of virus isolation.

**Treatment:** None

**Prevention:** Modified live and inactivated vaccines are described in the literature and have been used for Teschen disease in Europe; however, there are no commercial vaccines available in the United States. Vaccines would likely be serotype-specific with little cross protection between serotypes. Due to the large number of serotypes in the U.S. and the infrequency of enteroviral disease in swine, development of a commercial vaccine is unlikely. Other methods of prevention or control of disease are not described.

-by Roberta Alvarez, DVM, Production Medicine Resident

-edited by Greg Stevenson, DVM, PhD, ADDL Pathologist

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## WEB ACCESS TO ADDL REPORTS



ADDL is pleased to announce a new method of obtaining laboratory reports. Any licensed veterinarian or clinician can access completed reports that have been generated from our VisuaLab Information System from any web browser through ADDL’s web page. In order to have access, we must set up an account for you. Log onto our website at:

[www.addl.purdue.edu/onlineaccess/contact.asp](http://www.addl.purdue.edu/onlineaccess/contact.asp)

From there you can fill out an online form or download a form to fill out and fax back to us.

In order to protect the privacy of your patients, we are using a security model that grants access based on clinic location. You will be able to see all the reports from cases submitted by your current clinic location and nowhere else.

We hope you find this service valuable.

In the 4<sup>th</sup> quarter of this year, we will be implementing a system whereby reports can be automatically emailed to veterinarians and clinicians. If you are interested using this service, please send me a note at

<http://www.addl.purdue.edu>

-by Steve Vollmer, Systems Manager

