



# Utah State University Summer 2015

## UVDL Quarterly Newsletter

### Welcome to Summer!

Hope everyone is surviving the blistering hot temperatures in Utah. This newsletter is "pig-centric" with a focus on porcine diseases. With the PEDv (Porcine Epidemic Diarrhea virus) that has been sweeping across the country since spring 2013, I thought it was important that we give some focus to problems in pigs. We do not see a lot of pig cases at UVDL, but it seems that we have had increasing numbers of interesting disease recently. Included in this edition are a summary of some of the recent cases, a nice overview of piglet enteric disease by our pathology resident Dr. Chad Clancy, some information on PEDv composed by one of the USU veterinary students, some information on clinical pathology services and other diagnostic services available at UVDL for pig producers and veterinarians, and a very brief update on the high path Avian Influenza situation in Utah. I learnt a lot putting this newsletter together and I hope you do as well. As always, contact me (Dr. E. Jane Kelly, 435-623-1402, [jane.kelly@usu.edu](mailto:jane.kelly@usu.edu)) with feedback, comments, suggestions, and requests for future editions.

### *Pig Diseases Seen Recently at UVDL*

by Dr. E. Jane Kelly

#### **1. Polyserositis, bronchopneumonia, colitis, exudative dermatitis and cobalt deficiency in a piglet that ingested coal**

Mixed infections involving 2 pathogens are not uncommon in domestic animal species. However, it is unusual to diagnose 4 infections caused by 4 different bacterial species in one animal, especially when it is on medicated feed. This type of finding could be suggestive of immune compromise and there are many areas in Utah in which livestock copper and/or selenium deficiencies exist. However, our detection of a cobalt deficiency in an animal is much less frequent.

An 8.2 kg, 8 week-old mixed-breed female piglet from a small commercial swine operation was submitted to the Central Utah Branch of the Utah Veterinary Diagnostic Lab (UVDL) for necropsy in the fall of 2014 with a 2 week history of weight loss. Gross lesions included widespread erythema, alopecia, and crusting of the skin, fibrin

deposition on the serosal surfaces of most abdominal organs, consolidation of the right and left cranial lung lobes, and reddening of the intestinal mucosa. Samples of the lung, spleen, liver, colon, and skin were cultured aerobically.

Histologic lesions included purulent bronchopneumonia, fibrinous peritonitis and epicarditis, necrotizing colitis, and purulent epidermitis/dermatitis. Four different infections were diagnosed in the pig. Greasy pig disease cause by *Staphylococcus hyicus* (skin) was one of the infections (see Figure 1). It usually affects piglets up to 2 months of age and mortality may be up to 90% especially if there are other infections present in the animals. Skin abrasions caused by fighting or rough surfaces predispose the animals to infection. The affected skin is red, thickened and is often covered with a greasy, crusty exudate. Systemic antibiotics and topical antiseptics are recommended for treatment and infection may be prevented by minimizing skin abrasions in pigs (for example, soft bedding, and clipping the needle teeth of newborn

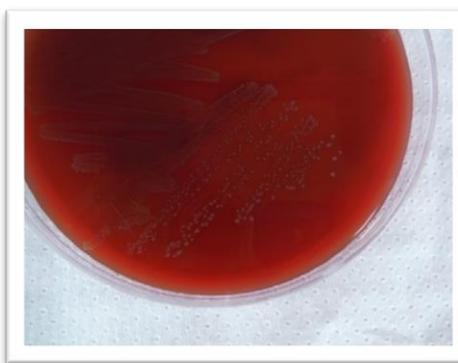
pigs). Another infection present in the pig was polyserositis caused by *Streptococcus suis* which was isolated from the liver and spleen (see Figure 2, bacteria on 5% sheep blood agar plate). *Streptococcus suis* is a commensal organism in the tonsils and digestive tract of pigs, yet is an important pathogen of young pigs when there are other predisposing conditions such as concurrent disease. Clinical signs in affected pigs include fever, anorexia, depression, gait abnormalities, and neurologic disease.



(Figure 1)

Lesions seen include serositis, arthritis, endocarditis, and meningitis. Early treatment with parenteral antibiotics may be effective and florfenicol is approved in pigs for treatment of *S. suis* and may be administered in the drinking water. ***Streptococcus suis* is a zoonotic risk for those working with affected pigs.**

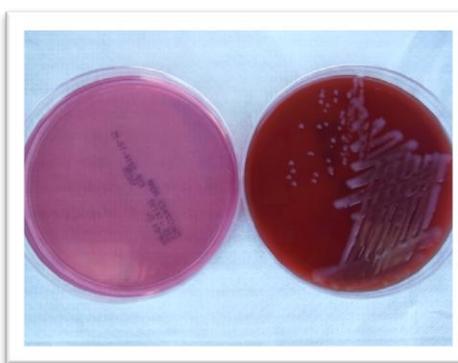
Transmission to humans is via contact of infective fluid to skin wounds and infection may cause severe infection in humans including septicemia and meningitis. There are other causes of polyserositis in pigs including *Haemophilus parasuis*, the cause of classic Glasser's Disease, as well as *E. coli*, *Erysipelothrix rhusiopathiae*, and *Salmonella* spp. *Pasturella multocida* caused purulent bronchopneumonia in this animal. In many cases (though not in this piglet), *P. multocida*, is a secondary complication to a mycoplasmal pneumonia and control of infection is based on control of the primary infection. As with other animals, judicious antibiotic treatment, good ventilation, and minimizing stress are methods of controlling pasteurellosis. See Figure 3 for mucoid isolate on *P. multocida* on blood agar and lack of growth on MacConkey agar.



(Figure 2)

The final infection detected in this pig was necrotizing colitis caused by *Salmonella* Anatum. *Salmonella* spp. are important causes of intestinal disease and septicemia in most species and are, of course, **of zoonotic concern** particularly in immunosuppressed individuals.

In an effort to find an underlying cause of immunosuppression in this animal, a liver mineral analysis was performed. The only abnormality was a low cobalt, which can be indicative of cobalamin deficiency. The cobalt may have also impaired absorption of other water soluble vitamins or the antibiotics that were in the feed resulting in increased susceptibility to infection.



(Figure 3)

## 2. Myocardial degeneration due to multiple nutritional deficiencies and secondary *Streptococcus suis* type II pneumonia in 3 piglets

A breeder of a Heritage Swine breed was having large death losses in piglets and submitted 2 males and a female piglet to the UVDL for necropsy early in 2015. The pigs ranged in size from 3.6 kg to 7.7 kg. and had gross lesions of heart failure (enlarged thin-walled hearts, subcutaneous edema, ascites, and an enhanced reticular pattern in the liver due to centrilobular necrosis). In addition, all

3 pigs also had degeneration and mineralization of skeletal muscle. One pig also had *S. suis* infection, likely secondary to the other problems.

Multiple nutritional problems were suspected based on the lesions. Liver mineral analyses were performed on the two male pigs and a cobalt deficiency was detected. The selenium levels were normal so a vitamin E deficiency was a suspected cause of the lesions in the skeletal muscle. Protein deficiency in the feed was also a possible cause of the hepatosis dietetica (myocardial degeneration and hepatic necrosis) seen in the pigs. Hepatosis dietetica is a disease of young rapidly growing pigs with inadequate dietary protein, vitamin E and/or selenium.

The owner was very concerned with improving the health of his piglets because of the value of the breed. Before breeding the sow again, she was supplemented with minerals and vitamins including vitamin B12, vitamin E, and selenium. She had a litter of 17 piglets, 4 were stillborn, and as of the writing of this article, 9 pigs are thriving.

## 3. Porcine dermatitis and nephropathy syndrome (PDNS) in a gilt

A 65kg gilt in good body and postmortem condition was necropsied at the UVDL in early 2015. There were numerous purple to red slightly raised, irregularly shaped skin lesions of varying sizes on the ears, dorsum, and legs. The kidneys were greatly enlarged and deep red in color. There were also some other lesions in the liver, lung, lymph nodes, and intestine. Many lesions were seen histologically but the primary lesion was vasculitis. In the kidney glomerulonephritis was identified with interstitial fibrosis indicating chronicity.

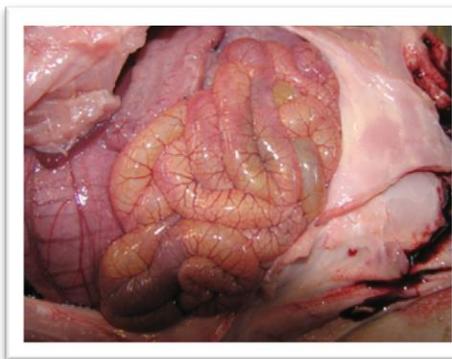
Differential diagnoses included PDNS as the top differential as well as bacterial septicemia, porcine respiratory and reproductive syndrome (PRRS), and Classic Swine Fever (CSF), a foreign animal disease (FAD). Because of the potential importance of a FAD, tissues were submitted to the University of Minnesota for PCR (polymerase chain reaction) diagnostics. Minnesota (and Iowa) do much more porcine diagnostics than we do and we rely on their expertise.

The spleen was positive for Porcine Circovirus Type 2 (PCV2) by PCR. Diseases associated with PCV2 include Postweaning multisystemic wasting syndrome (PMWS) and PDNS. PMWS is a complex disease involving many factors of which infection with PCV2 is one. Age of the pigs, environmental conditions, and genetics are

believed to be involved. Clinically affected swine are usually 8 to 18 weeks old and show clinical signs of decreased growth, dyspnea, anemia, diarrhea, and sometimes death. PCV2 infection in sows may lead to reproductive problems such as abortion and stillborn pigs. PDNS usually affects immature pigs and rarely adults. The prevalence of PDNS in an affected herd is usually low. Some pigs die due to due to kidney failure and surviving pigs often recover from the infection after several days. Vaccines are available and are the best means of controlling porcine circoviral infections. As a point of interest, the first porcine circovirus identified was nonpathogenic. The virus associated with clinical disease (PCV2) was first described in North America in the 1990s.

#### 4. Enteric disease in young piglets

We have had several recent cases of enteric disease in piglets, including TGE (Transmissible gastroenteritis) (see Figure 4 for gross lesions in a case of TGE), *E.coli* and *Salmonella* spp. These are covered in detail in other articles in this newsletter, but I wanted to emphasize the zoonotic potential of *Salmonella* spp. A recent case in some very cute mini pigs reminded me of how easily young children could become infected. After all, who wouldn't want to pick up those cute little pigs and kiss them? Those working with sick pigs should wash their hands frequently and young children and immunosuppressed people should not be around pigs with enteric disease.



(Figure 4)

*Salmonella* spp. Is a common cause of enteritis and septicemia in neonatal animals. The source of the bacteria may be adult animals that are asymptomatic and shedding it, other animals such as rodents and birds, and contaminated feed. The bacteria may also contaminate the environment and may be carried on fomites such as boots, feed troughs, etc. Stressed animals (for example,

animals recently moved) are more likely to break with Salmonellosis. Control of infection relies on strict sanitation and biosecurity, isolation of sick animals, and, in some cases, vaccination.

For those that would like more information on the Heritage Swine please go to The Livestock Conservancy website ([www.livestockconservancy.org](http://www.livestockconservancy.org)).

Heritage Swine are breeds of pig that came to the United States with colonists and were an important part of agriculture. Many of these breeds are now in danger of extinction. The Livestock Conservancy is supporting maintenance of these breeds. Heritage Swine are purebred, are endangered, and have a long history in the U.S. (since 1925). This is similar to heirloom plants.

## Pathology spotlight: Fluid Sampling

by Dr. Johanna Rigas

There are numerous diseases in veterinary species that cause fluid accumulation in joints, thoracic, abdominal, and pericardial cavities. In Pigs, just a few diseases associated with abnormal fluid production include Glasser's disease, *Strep. Suis*, and *Erysipelas*. Premortem evaluation of fluid samples can help to determine a primary disease process, or at least help to determine what the next diagnostic step will be.

There are multiple features of fluids evaluated by the laboratory to completely characterize the sample in an effort to determine the disease of pathology. These include the nucleated cell count, total protein, and possibly viscosity (namely joint fluid) or red blood cell count. In order to maximize the diagnostic potential of the sample, it must be obtained and stored in a specific manner.

Fluid for cytologic review is best collected into an EDTA tube. This helps to suppress fibrin and clot formation which can develop without obvious hemorrhage. Also, if there are bacteria present, excess growth will be inhibited by the EDTA. Once the sample is collected, it should be maintained at cool temperature. Refrigeration is best. In addition, one or multiple direct fluid smear preparations on glass slides are recommended for samples that may be analyzed after approximately 4 hours from sampling. Nucleated cells start to degenerate quickly when out of the body,

and making a direct smear can preserve the cell morphology. Note that once the slide

sample is air dried, fixation is not needed immediately. For sample that appear poorly cellular, consider gently concentrating the cells in a centrifuge as one does for a urinalysis sediment examination. The slide sample should not be refrigerated as condensation during rewarming can harm the sample.

The total protein can be evaluated by refractometry. Once the total cell count and total protein are determined, a cavity effusion can be classified as a transudate, a modified transudate, or an exudate. These classification schemes assist to narrow differential diagnoses.

It is advisable to collect effusion fluid into a red top tube as well. This can be reserved for additional diagnostic testing including bacteriology or fungal culture.

# Porcine Epidemic Diarrhea Virus (PEDv)

By Michael Noyes

USU/WSU DVM Candidate Class of 2016

PEDv is an alphacoronavirus of the Coronaviridae family. It was first discovered in the United Kingdom in 1971, and since that time has caused significant economic loss throughout much of Europe, Asia, the United States and Canada. The virus was first confirmed in the U.S. in Iowa in May of 2013, and from that time, has spread quite rapidly. It was officially identified in Utah in September 2014. A federal order was issued June 5, 2014 making all PEDv cases, presumptive and confirmed, reportable. Producers with reported PEDv cases are required to work with veterinary officials to establish a management plan specific to their facility to contain the virus and prevent it from spreading further.

PEDv is similar to Transmissible Gastroenteritis Virus (TGEV), and cannot be clinically distinguished without the assistance of a diagnostic laboratory. Definitive diagnosis of PEDv requires submission of fecal, oral fluid, or intestinal tissue samples from suspect animals to a diagnostic laboratory for the diagnosis via PCR, IHC, and Histopathology. The severity of signs will depend on the immune status of the herd. The primary clinical signs seen include vomiting and acute watery diarrhea in all age of affected pigs. Naive pigs can have up to 100% morbidity. Suckling pig mortality is generally higher, ranging from 50-100%, while mortality in weaned pigs in the growing or feeder stages ranges from 1-3%. Endemic herds often show persistent watery diarrhea in recently weaned pigs. Incubation time of PEDv can be as low as 12-24 hours, and infected pigs will continue to shed the virus for up to 4 weeks.

Treatment for PEDv is supportive, including providing fluids to maintain hydration, and keeping the pigs warm and comfortable. There are currently two vaccines with conditional approval in the U.S. for use against PEDv. Harris Vaccines and Zoetis were given conditional licenses for their PEDv vaccines in 2014. However, the efficacy of these vaccines is unclear due to the relatively short amount of time the vaccines have been available.

Transmission of PEDv is primarily through oral contact with contaminated feces and oral fluids, although respiratory droplets and other secretions or excretions have also been suggested as possible means of transmission. PEDv can be carried from one animal to another or from one production site to another on any potential fomite. An article in the May 2014 edition of Emerging Infectious Disease stated that transport vehicles were likely a major factor in the rapid spread of PEDv from the location of the initial U.S. case, especially transport vehicles entering or exiting common collection points such as harvest facilities and livestock auction markets. This highlights the importance of strict hygiene and biosecurity practices in controlling PEDv and limiting it from spreading further.

Management of a herd with confirmed PEDv cases is a controversial topic. Some advocate a plan to rapidly infect all individuals on the premises, the reasoning being that due to the highly contagious nature of the virus and the short incubation time, it is best to try to force herd immunity and decrease the overall amount of time any individuals in the herd may continue to shed the virus. Others support a more conservative plan of enforcing strict biosecurity protocol to contain the virus to the area of the confirmed PEDv cases, using an "all-in, all-out" procedure in the hopes of eliminating the virus from the premises as soon as possible. Ultimately, due to factors such as the amount of animals on the premises, financial stability, physical layout of the operation, and the ability and willingness of the owner and workers to comply with management protocol, there is no single, all-encompassing management plan that is suited to fit the needs of every swine operation.

While PEDv is not zoonotic and therefore is not considered a threat to the American food supply, the incredible economic toll it has wreaked upon the Pork industry has served as a wakeup call to producers and others involved in the industry concerning the need for improved biosecurity protocol and

training of individuals at all levels of swine production. Improvements must occur not only at the production sites themselves, but also in harvest facilities and livestock auction markets, and the transport vehicles and their drivers that carry the pigs to and from these common collection points.

1. Lowe J, Gauger P, Harmon K, et al. Role of transportation in spread of porcine epidemic diarrhea virus infection, United States. *Emerging Infectious Diseases*. 2014;20(5):872-4

## Brief update on Highly Pathogenic Avian Influenza (HPAI) in Utah

By Dr. E. Jane Kelly

Since December of 2014 over 200 cases of HPAI H5 have been reported in U.S. commercial poultry and wild birds. You are all probably aware that it was detected in a wild duck (American wigeon) in Utah at the beginning of the year. You are probably also aware that many states have been hit hard with cases of HPAI in commercial birds. Some of these states include Iowa, Minnesota, Nebraska, South Dakota, and Wisconsin. As of the writing of this newsletter, there have been no more reports of HPAI H5 in Utah. To our knowledge, this Avian Influenza is not pathogenic for humans. Are you aware that people can order chicks and eggs on the internet (e.g. from e-bay) and have them shipped directly to their residence from all over the country? I did not realize this until recently. These types of transactions are very hard to monitor for those involved in AI surveillance and tasked with keeping it out of our commercial flocks in Utah.

## Enteric Diseases of Post-Weaning Piglets

By Dr. Chad Clancy, UVDL pathology resident

The stereotypical commercial swine operation functions as a biosecure unit, tightly controlling disease exposure through limiting human contact and ensuring a single herd source with extensive vaccination protocols in place. Commercial swine are frequently housed in fully-enclosed buildings with cement slab flooring and may even have HEPA filtered ventilation to reduce the risk or aerosolized exposure of respiratory pathogen (more prevalent in the Midwest after the PRRS outbreak began). In stark contrast, the majority of swine owners in Utah are small herd farmers or 4H/FFA show swine owners whose herds are frequently of mixed origin, housed in a pasture or paddock with dirt flooring and have minimal (if any) vaccination history. A significant number of show swine are brought in from out-of-state sources, providing a particularly difficult diagnostic challenge to the unfortunate practitioner called out to evaluate a recently acquired piglet with diarrhea.

Etiologic agents involved in post-weaning diarrhea in piglets can generally be grouped into two sources: viral and bacterial. While parasitic disease is common in swine housed on dirt, the most common clinical signs of parasitism in young swine is poor weight gain and coughing due to larval migration through the lungs. *Trichuris suis* has been known to cause diarrhea in swine, but more frequently in near-market age swine.

Bacterial agents frequently implicated in nursery-age piglet diarrhea include *E. coli*, *Salmonella*, *Brachyspira hyodysenteriae* (swine dysentery) and *Lawsonia intercellularis* (ileitis/proliferative enteritis). While cases of colibacillosis and salmonellosis are generally sporadic, both swine dysentery and proliferative enteritis are endemic herd problems with high morbidity rates. *Brachyspiral* organisms are environmentally stable and can persist in rodent populations. Control of swine dysentery is focused on sanitation and appropriate manure removal from the housing facility. However, complete depopulation of the herd may be necessary to achieve adequate results as clinically normal animals may serve as carriers. *Lawsonia intracellularis* is a prevalent disease within the industry that causes a variety of enteric diseases ranging from mild, chronic, enteritis to severe, acute necro-hemorrhagic enteritis. The more

severe disease is generally associated with late finishing animals. Due to the prevalence of *Lawsonia* within the swine industry, the organism is largely ubiquitous. Moreover, it is persistent through antibacterial treatment and can cause intermittent diarrhea with poor weight gain in market weight animals. Reliable and efficacious oral vaccinations exist for *Lawsonia* to prevent persistence of the most severe disease state within a herd.

Viral enteritis in nursery age piglets is largely limited to rotavirus and coronavirus. The clinical presentation of both infections is similar with coronaviral infection (transmissible gastro-enteritis, TGE) having a higher prevalence of vomiting. Clinical signs are most severe in young animals and are frequently limited to diarrhea and dehydration in nursery age and older swine. Recovery occurs within a week to ten day in mild to moderately affected animals. Supportive therapy with fluids and electrolytes may be beneficial in more severely affect animals. Molecular diagnostics (PCR) for viral and bacterial enteric pathogens are readily available through numerous diagnostic laboratories in the United States with a minimum requirement of 1 gram of feces from and actively affected animal.

### Porcine Diagnostic Services Available at UVDL

By Dr. E. Jane Kelly

We offer a variety of services for pig owners and veterinarians including full necropsies on all sizes of pigs, biopsy histopathology, incineration, and private cremations. The necropsy fee includes histopathology. Bacterial (aerobe and anaerobe) and fungal cultures and antibiotic susceptibility profiles are available as well as cytology, complete blood counts, and chemistry profiles. We also run brucellosis assays (serology). Please refer to our website ([www.usu.edu/uvdl](http://www.usu.edu/uvdl)) for the current pricing. Note: please call me with any questions about PCR (polymerase chain reaction) diagnostics for viruses: Iowa and Minnesota veterinary diagnostic labs offer many of these tests.

Disease (and Agent)	Groups of Pigs Affected	Diagnostic Features
Enteric colibacillosis ( <i>Escherichia coli</i> )	Neonates one day old to pigs up to 2-4 weeks post weaning.	Watery diarrhea, possibly vomiting. Minimal lesions; jejunum and ileum may have mild villous atrophy. Many Gram-negative rods on mucosa. Culture uniform population of <i>E. coli</i> from small intestine. Identify enterotoxigenic <i>E. coli</i> enterotoxins and/or pili, usually by PCR. <i>E. coli</i> may also cause fibrinous polyserositis.
Rotaviral enteritis (Rotavirus)	Usually 1-6 week old piglets. Also common about one week after weaning.	Diarrhea, occasional vomiting. Usually nonfatal. Variable enteritis with moderate villous atrophy. Identify rotavirus in feces of early cases by EM or ELISA or in small intestinal epithelium by FAT or IHC.
Transmissible Gastric Enteritis-TGE (Coronavirus)	All age groups susceptible if previously unexposed. Most severe in piglets < 4 weeks old.	Acute form: vomiting and very high mortality in piglets <3 weeks old. Bright yellow feces often seen in older piglets. Marked diarrhea in feeder age pigs. Acutely infected sows may vomit, are depressed and refuse to nurse piglets. Endemic form: similar signs but much less severe and with reduced mortality. FAT or IHC on intestine of acutely affected pigs of PCR on feces from acutely affected pigs.
Salmonellosis ( <i>Salmonella</i> serotypes Choleraesuis, Typhimurium or Heidelberg)	More common in weaned and growing pigs. All ages susceptible if previously unexposed.	Intestinal form: necrotizing enteritis of large and small intestine. Enlarged mesenteric lymph nodes. Congested lungs. Chronic cases may have rectal strictures or “button ulcers” in large intestine. Septicemic form: red/purple skin lesions on ears, tail, snout, feet, abdomen. Congested lungs, splenomegaly and hepatomegaly. Perhaps gray foci in the liver. Diagnosis: severe septicemia or necrotizing enterocolitis. Culture <i>Salmonella</i> from lymph nodes, liver, lungs, spleen, kidneys and perhaps brain if CNS signs. Histology: paratyphoid nodules in liver.
Swine dysentery ( <i>Brachyspira hyodysenteriae</i> )	From 3 weeks to adults if previously unexposed.	Mucohemorrhagic diarrhea (fresh mucus and fresh blood) is very suggestive. Mucohemorrhagic to fibrinonecrotic typhlitis and colitis but no lesions in small intestine. Histology: lesions largely restricted to mucosa with organisms in epithelium and crypts. Culture <i>Brachyspira hyodysenteriae</i> from mucosa or feces. <i>Brachyspira pilosicoli</i> is a sporadic cause of colitis.
Proliferative enteritis ( <i>Lawsonia interacellularis</i> )	Weaned pigs and all older swine.	Signs: Subclinical disease with weight loss most consistent sign. Clinically affected have either an acute form with hemorrhagic diarrhea or chronic form with diarrhea and wasting. Lesions: thickening of mucosa of jejunum and/or ileum and/or colon. Curved bacilli in affected enterocytes. PCR (feces) and IHC (lesions) confirm organism and disease.

Table adapted from *Swine Disease Manual Fourth Edition*. Neuman, Ramirez and Schwartz, 2009.