18 1

Winter 2007

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Good day from the hallowed halls of ADDL. Today is a cold one for us but all things considered, we have had a very mild winter.

Some of the activities of the ADDL recently include completion of the hunter killed deer Chronic Wasting Disease survey. We tested 1318 samples collected by the Indiana DNR during the '06 hunting season. All samples were found by the immunohistochemistry test method to be "No resistant prions detected". This is the sixth year of has surveying Indiana deer for CWD; so far no evidence of this disease been found in our deer population.

We recently finished the ADDL Annual Report for FY '06. Areas of significant activity change in the laboratories comparing FY -06 to FY ;05 included increase in histopathology accessions by 20% to 48,438; increase in molecular diagnostics requests of 23% to 2,374; increase of toxicology accessions by 184% to 3,973; increase of virology tests of 46% to 92,713; and increase of Heeke ADDL avian accessions of 23% to 3,164. Fiscal year 2006 was a busy and productive year in ADDL. We are blessed to have a dedicated and accomplished faculty and staff to provide these services to the animal owners of Indiana and to owners in other states.

We wish you well for the rest of the winter; if it stays like today, that groundhog will for sure see his shadow in a couple of weeks.

Final diagnosis

Granulomatous meningoencephalomyelitis

In each issue, we featur case submitted to ADDL that we hope will be of Interest to you.

History: A reportedly 3-year-old, spayed female rat terrier dog was submitted to the ADDL for necropsy. This dog originally presented to the referring veterinarian with a history of ataxia and pelvic limb paraparesis. Clinical signs progressed over the next two weeks with the development of tetraparesis, hyperesthesia, loss of anal tone, and hemorrhagic diarrhea. Lower motor neuron reflexes were present in the pelvic and thoracic limbs. Following treatment, clinical signs improved slightly over the next five days with the recurrence of anal tone, increased alertness, and increased mobility in the forelimbs; however, clinical signs subsequently worsened the following week-end resulting in death.

Gross findings: On gross examination, the spinal cord was soft and swollen, filling the vertebral canal. No other significant lesions were observed in the brain or other body systems.

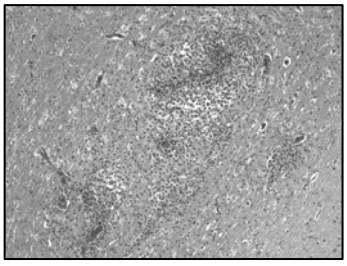
Histologic findings: Along the entire length of the spinal cord, the meninges were markedly expanded by a pleomorphic inflammatory infiltrate, predominantly consisting of macrophages and lymphocytes with fewer plasma cells and neutrophils. Epitheloid macrophages were often arranged in concentric layers forming granulomas which were often oriented around small vessels. Inflammatory cells extended from the meninges into the white, and sometimes, grey, matter, effacing and rarefying the neuropil. There was marked lymphocytic perivascular inflammation which was predominantly in the white matter.

In the cerebrum, macrophages, lymphocytes, and plasma cells expanded the meninges, occasionally extending into and effacing adjacent neuropil. Foci of predominantly perivascular necrosis and granulomatous inflammation, consisting of epitheloid macrophages, lymphocytes, and fewer neutrophils and plasma cells, were scattered throughout the cerebrum, primarily in the white matter. Similar foci of necrosis and inflammation were present in the pons and ventral cerebellum. Special stains, including Gram, periodic acid-Schiff (PAS), and Giemsa stains were performed on sections of spinal cord in order to identify potential infectious agents; however, no organisms were identified.

Discussion: Granulomatous meningo-encephalomyelitis (GME) is an inflammatory disease of unknown etiology, characterized by predominantly perivascular, granulomatous inflammation in the meninges and white matter of the central nervous system. Lesions may be focal, multifocal, or disseminated, affecting the cerebrum, cerebellum, brainstem, spinal cord, or optic tracts. Granulomatous meningoencephalomyelitis occurs in many breeds of dog and can occur both in males and females; however, small breed dogs, poodles, terriers, and female dogs have been shown to have an increased incidence of disease. The age of onset is usually between one and nine years; however, animals may be affected at any age. Affected animals usually present with an acute onset of disease, which progresses over days to months. The clinical presentation is variable, reflecting the distribution of lesions, but may include intermittent fever, lethargy, depression, convulsions, head tilt, circling, cervical pain, hyperesthesia, conscious proprioceptive deficits, paresis, or paralysis.

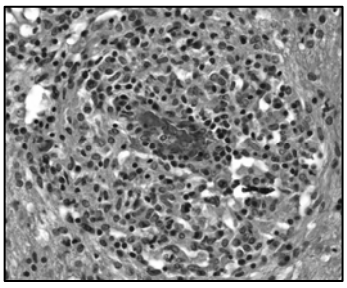
Clinical diagnosis of GME is based on clinical signs and cerebral spinal fluid analysis, which is characterized by pleocytosis, increased protein, and normal to increased pressure in the absence of infectious agents. A definitive diagnosis of GME is based on characteristic histopathologic lesions. Including primarily perivascular granulamatous inflammation in meninges and white matter with no associated infectious agents. Immunosuppressive therapy, primarily consisting of corticosteroids, has been the principle treatment modality for GME and, in some cases, can result in long term improvement of clinical signs; however, GME is a continuously progressive disease, often resulting in recurrence and progression of clinical signs, especially when immunosuppressive therapy is discontinued. Radiation therapy has also been used with some success, particularly in the presence of focal disease. In general, due to its progressive nature, GME is associated with a poor prognosis. However, in one study, animals with focal disease had longer median survival times as compared to those with multifocal disease. Currently, the cause of granulomatous meningoencephalomyelitis is unknown, and GME has not been associated with any infectious agents. Based on the predominance of CD3+ T-cells and MCH II+ macrophages, one study has proposed that GME may be a T-cell mediated autoimmune disorder; however, additional studies have not been performed to support or refute this hypothesis.

-by Dr. Joshua Webster, ADDL Graduate Student



Perivascular granu-lomatous inflammation in cerebral white matter

20X

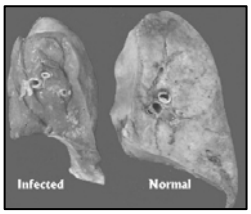


Perivascular granu-lomatous inflammation in cerebral white matter

40X

References:
1. Baumgärtner, KA, Vogl C, Gaedke K, Wellman M: 1998. Immunohistochemical characterization of inflammatory cells in brains of dogs with granulomatous meningoencephalitis. Vet Pathol 35: 43-52.
2. LeCouteur Ra, Grandy JL: 2005. Diseases of the spinal cord In: Textbook of Veterinary Internal Medicine: Diseases of the Dog and Cat, 6th ed. Ettinger SJ, Feldman EC, eds. Elsevier, St. Louis MO. 1: 842-887.
3. Munana KR, Luttgen PJ: 1998. Prognostic factors for dogs with granulomatous meningoencephalo-myelitis: 42 cases (198201996). JAVMA 212: 1902-1906
4. Zachary JF: 2006. Nervous system In: Pathologic Basis of Veterinary Disease, 4th edition. McGavin MD, Zachary JF, eds. Elsevier, St. Louis MO. Pp 833-971.

Ovine Progressive Pleuropneumonia



Ovine Progressive Pleuropneumonia (OPP) is a chronic, debilitating disease of sheep caused by ovine lentivirus (OvLV), a nonocogenic retrovirus that is closely related to caprine arthritis encephalitis virus (CAEV) and in the same virus subfamily as human and animal immuno-deficiency viruses (HIV, SIV, FIV). OvLV, also known as Maedi-Visna virus, causes two distinct clinical syndromes in sheep: maedi and visna. OPP, or Maedi (which means "shortness of breath in Icelandic), is a slowly progressive interstitial pneumonia. Visna (which means "wasting" in Icelandic) is the neurologic form characterized by leukoencephalomyelitis with demyelination. OPP is the most common clinical manifestation of OvLV infection in the United States. Other chronic inflammatory conditions associated with OvLV include polyarthritis and mastitis.

Ingestion of infected colostrum is the primary route of OvLV transmission. Horizontal transmission by ingestion or inhalation of aerosolized virus from respiratory secretions or ingestion of contaminated food or water also occurs, albeit less efficiently, and is typically associated with close confinement during winter months. In utero transmission has been reported, but the frequency is unknown. Initial infection is followed by a latent period of months to years, during which virus replication is limited and seroconversion is delayed. An important feature of retrovirus replication is that viral genetic material becomes integrated into the host cell DNA, resulting in lifelong infections despite production of specific antibodies. Approximately 20% of sheep infected with OvLV will eventually develop clinical disease; however, these animals typically do not show clinical signs until approximately 2 years of age.

Tentative diagnosis of OPP is often based on clinical signs, including chronic afebrile pneumonia with progressive respiratory failure and loss of body condition despite a good appetite. Expiratory dyspnea, abdominal breathing, and severe tachypnea (up to 80-12 breaths/min) may be observed. Pregnant ewes often give birth to small or weak lambs. Other clinical signs include chronic polyarthritis and mastitis ("hard bag"). At necropsy, the lungs have a rubbery consistency, fail to collapse, and may be 3-4 times normal weight. The basic microscopic lesion in all affected tissues, including lungs, mammary gland, and central nervous system, is lymphocytic interstitial inflammation accompanied by formation lymphoid nodules with germinal centers. OvLV is tropic for mononuclear phagocytes, and persistent activation of macrophages causes chronic stimulation of the immune system, resulting in the lymphoid hyperplasia and follicle development observed in various tissues.

Serologic and molecular-based diagnostic tests for OvLV are available. Serologic tests such as agar gel immunodiffusion (AGID) and enzyme linked immunosorbent assay (ELISA) demonstrate the presence of virus-specific antibodies in serum. AGID is the most commonly used serologic screening test, but has a lower sensitivity for detection of antibodies than ELISA-based tests. In experimentally infected animals, ELISA tests were able to detect seroconversion earlier than AGID. The PCR test detects proviral DNA in whole blood or tissue samples. The PCR test is able to detect infected animals before they mount an antibody response, but is more costly than the serology-based diagnostic tests.

OPP is a chronic, progressive disease for which no effective treatments or vaccines are available. Control and prevention programs are paramount. Periodic AGID or ELISA screening tests are recommended to identify infected individuals in the flock. Lambs may be removed from infected mothers at birth and raised in separate flocks. Preferably, these lambs should be fed colostrum and milk from certified OPP-free ewes. Colostrum from infected ewes should be heated at 56ºC for 60 minutes and milk should be pasteurized. Alternatively, seropositive ewes may be culled from the flock. Total herd replacement or annual purchase of OPP-free replacements should also be considered in lamb-producing flocks. The most recent National Animal Health Monitoring System (NAHMS) sheep survey in 2001 reported that 24.2% of sheep from the 3,210 operations surveyed nationally were seropositive for OvLV using the ELISA test. These figures were slightly higher in the central region, in which 24.4% of sheep tested positive and 46.6% of the operations surveyed had one or more seropositive animals.

-by Morgan Hennessey, Class of 2006
-edited by Dr. Kim Maratea, ADDL Graduate Student

References

1. Blacklaws BA, Berruita E, Torsteinsdottir S, Watt NJ et al: 2004. Transmission of small ruminant lentiviruses. Vet Microbiol 101: 199-208.
2. Cutlip RC, Lehmkuhl HD, Schmerr MJ, Brogden KA: 1988. Ovine Progressive Pneuropneumonia (Maedi-Visna) in Sheep. Vet Microbiol 17: 237-250.

3. DeAndres D, Klein D, Watt NJ, Berriatua E, et al: 2005. Diagnostic tests for small ruminant lentiviruses. Vet Microbiol 107: 49-62.
4. Pepin M, Vitu C, Russo P, Mornex JF, et al: 1998. Maedi-Visna virus infection in sheep: A review. Vet Res 29: 341-367.
5. Peterhans E, Greenland T, Badiola J, Harkiss G, et al: 2004. Routes of transmission and consequences of small ruminant lentiviruses infection and eradication schemes. Vet Res 35: 257-274.
6. Petursson G, Matthiasdottir S, Svansson V, Andresdottir V, et al: 2005. Mucosal vaccination with an attenuated Maedi virus clone. Vaccine 24: 3223-3228.
7. Petursson G, Hoff-Jorgensen R: 1990. Maedi-Visna and related diseases. Kluwer Academic Publishers, Boston, MA.
8. Preziuso S, REnzoni G, Allen TE, Taccini E, et al: 2004. Colostral transmission of maedi visua virus: sites of viral entry in lambs born from experimentally infected ewes. Vet Microbiol 104: 156-164.
9. USDA: APHIS: VS: National Animal Health Monitoring System: Sheep 2001, April 2003.

On the Road

Drs. Leon Thacker, Steve Hooser, Peg Miller, Jose Ramos-Vara, Roman Pogranichniy, Duane Murphy, Ramesh Vemulapalli, Ching Ching Wu, Robert Everson, Vimala Vemireddi, Dinesh Singh, Ikki Mitsui, Pam Mouser, Kent Fenton and **Linda Hendrickson, Buffie Mink** and **Steve Vollmer** attended the annual meeting of the American Association of Veterinary Laboratory Diagnosticians in Minneapolis, MN, October 2006.

Dr. Roman Pogranichniy made a scientific presentation at the Conference for Research Workers in Animal Diseases in Chicago, IL, November, 2006

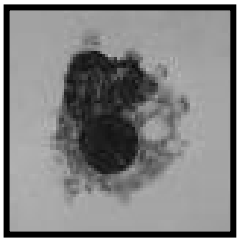
Drs. Peg Miller, Steve Lenz, ADDL pathologists, and **Drs. Michael Owston, Vimala Vemireddi**, **Pam Mouser, Gopakumar Gopalakrishnan**, and **Ingeborg Langohr**, ADDL graduate students, attended the annual American College of Veterinary pathologists meeting in Tucson, AZ, December, 2006.

Dr. Steve Lenz attended a Foreign Animal Disease Training Course at the USDA Plum Island facility in November, 2006.

Drs. Roman Pogranichniy and **Greg Stevenson** attended the Indiana Swine Group meeting, Indianapolis, IN, December, 2006.

Potomac Horse Fever

Potomac Horse Fever (PHF), also known as equine monocytic ehrlichiosis, is a febrile gastrointestinal disease affecting horses of all ages. PHF was first recognized in 1979 as a distinct clinical entity in Montgomery County, Maryland (the Potomac region). To date, PHF has appeared as a sporadic disease observed mostly in the summer months throughout many states in the United States and has also been confirmed in Canada and Europe. Although a clear seasonal pattern exists with cases first reported in late May or June and ending in October or November, the peak prevalence of PHF occurs in July, August, and September.



PHF is caused by *Ehrlichia risticii (*now called *Neorickettsia risticii),* a member of the family Rickettsiaceae. *E. risticii* is found in association with trema-todes in freshwater snails. Although rickettsia are often transmitted by arthropods and the disease is seasonal, an arthropod host has not been identified. *E. risticii* is a gram-negative, obligate intracellular bacterium that infects monocytes. These infected cells have a predilection for the intestine, especially the cecum and colon. The organism then multiplies in macrophages and mast cells, in the lamina propria and submucosa, and in the cytoplasm of glandular epithelial cells causing cell exfoliation.

Horses affected with PHF usually develop acute depression, fever (102-107ºF) and anorexia. After 24-48 hours, the horse often develops profuse, watery diarrhea without blood, characteristic odor, or leukocyte excretion. The diarrhea may be present for up to 10 days but, in most cases, only lasts 1-5 days. However, not all horses have diarrhea or diarrhea may be transient in nature. Laminitis, a frequent sequel to PHF, occurs most commonly within three days of the onset of diarrhea. Other clinical signs include colic and subcutaneous edema of the legs and ventral abdomen. Borborygmal sounds are typically decreased or absent in the early stages of the disease. Although horses show different hematologic signs, most horses with PHF have leukopenia characterized by neutropenia, lymphopenia, and eosinopenia. In addition, some horses may develop thrombocytopenia and, rarely, petechiation. Acid base and electrolyte values are typical of most horses with acute diarrhea, namely metabolic acidosis, hyponatremia, hypokalemia, and renal azotemia.

Since the clinical signs closely resemble those of salmonellosis, multiple samples of fecal matter should be submitted to a laboratory for culture.

In addition, colitis X, antibiotic-associated diarrhea, endotoxic shock, and peritonitis should be ruled out as differential diagnoses for horses with acute diarrhea. The characteristic epidemiologic features including the seasonal nature, the sporadic occurrence on farms located near rivers, and lack of *Salmonella* isolation renders PHF a more likely diagnosis.

Currently, the most convenient way to diagnose PHF is by indirect fluorescent antibody test (IFA). The IFA detects antibodies in sera to the causative agent *E. risticii*. Most horses have detectable antibody titers at the onset of clinical signs although a rapid rise in antibody titer after the first few days of infection with *E. risticii* merits paired serum samples. Though significant changes in titer of affected horses can occur in 2-3 days, paired serum samples should be taken at least two weeks apart. Nonetheless, the presence of antibodies of the IFA does not indicate that the horse has clinical PHF, only that the horse was exposed to *E. risticii* sometime in the past. At least a four-fold increase in antibody titer has been suggested as a criterion for serologic diagnosis.

A definitive diagnosis of PHF is made by isolation of *E. risticii* from the peripheral blood of a clinically affected horse. The organism is best isolated in macrophages in tissue culture media without antibiotics. In addition, electron microscopic examination of peripheral blood monocytes can be done as a definitive diagnosis by using Giemsa stain to demonstrate the organism in dried blood or buffy coat smears.

The gross lesions at necropsy may be minimal affecting primarily the cecum and colon and, to a lesser degree, the small intestines. Affected segments are distended with fluid mixed with ingesta and the fluid is pale brown and fetid. Very little hemorrhage or damage of the mucosal surface is evident, except for some patchy hyperemia (approximately 4-8 cm of the small intestine affected and approximately 5-20 cm of the cecum or colon.) In contrast to large areas of necrosis and ulceration with some cases of Salmonellosis, the scattered mucosal erosions and ulcers present in the mucosa
from horses with PHF are less frequent. In addition, splenomegaly and ventral edema may be present at necropsy.

Microscopically, the mucosa of the cecum and colon is reduced in thickness due to the loss of surface epithelium, a marked decrease in the number of intestinal crypts, and collapse of the lamina propria. The denuded surface epithelium may be covered by a pseudomembrane focally, which consists of necrotic cells, fibrin, and bacteria. Capillaries and veins of the mucosa are engorged with blood. The remaining intestinal crypt cells have intact epithelium. The lamina propria is hypercellular, especially at the base of the crypts. The submucosa is edematous. Both lamina propria and submucosa contain macrophages with clusters of the infectious agent. These clusters of *E. risticii* are demonstrated using a modified Steiner's or Dieterle's silver stain. The germinal centers of the lymphoid follicles are depleted of lymphocytes and contain karyorrhectic nuclei.

Treatment of horses with PHF includes antibiotics, fluid and electrolyte replacement therapy for animals exhibiting diarrhea, and nonsteroidal anti-inflammatory drugs for the relief of pain in cases of colic and/or laminitis.

Vaccines are available for protection against PHF. However, protection may be short-lived and incomplete because, in many cases, vaccination has been shown to reduce clinical signs, rather than provide complete protection. Nonetheless, vaccination may be necessary for horses residing in or traveling to endemic areas.

The prognosis for survival of PHF has increased to over 85% in recent years with the heightened awareness of the disease among veterinarians and horse owners. The greatest percentage of mortality is attributed to complications of laminitis. Outbreaks of PHF will decline in the future with vaccination, increased awareness, and initiating diagnosis and treatment at the early stages of the disease.

-by Ashley Armstrong, Class of 2006
-edited by Dr. Dinesh Singh, ADDL Graduate student

References

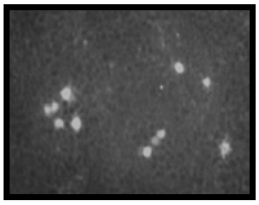
1. Hillyer M: 2004. A practical approach to diarrhoea in the adult horse. In Practice. Pp 2-11.
2. Madigon JE, Joon-seok C, Pusteria N: 1999. Potomac Horse Fever: Identification and Transmission of the Causative Agent via Trematodes of Freshwater Snails. AAEP Proceedings 45:45-47.
3. McGavin MD et al: 2001. Thompson's Special Veterinary Pathology, 3rd ed. Mosby.
4. Phipps LP: 1994. Potomac Horse Fever. Equine Vet Edu 6:321-322.
5. Reed S et al: 1995. Potomac horse fever. Equine Practice 17:12-14.
6. Rikihisa Y et al: 1992. Loss of absorptive capacity for sodium and chloride in the colon causes diarrhea in Potomac horse fever. Res in Vet Sci 52: 353-362.
7. Smith BP: 2002. Large Animal Internal Medicine, 3rd ed. Elsevier USA.
8. Whitlock RH: 1986. Colitis: Differential diagnosis and treatment. Equine Vet J: 18(4): 278-283.
9. Whitlock RH, Palmer J: 1986. Potomac horse fever: Clinical signs, diagnosis and treatment. AAEP Proceedings 32: 405-413.

Diagnosis of Porcine Circovirus Associated Diseases (PCVAD) in ADDL from infected pigs.

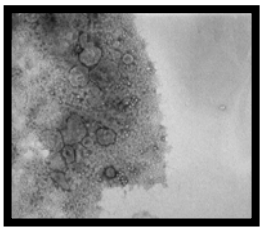
Porcine circovirus (type 1) was discovered in 1972. The virus had been studied and it was concluded that it is not pathogenic based on animal inoculation studies and high prevalence in the swine population. Type 2 PCV (PCV2) drew swine veterinarians' attention after the description of Post-weaning Multisystemic Wasting Syndrome (PMWS) in 1997 or earlier. PCV2 is thought to be the only consistent essential infectious cause of PMWS since the level of virus in swine tissues or serum is directly proportional to severity of clinical disease and lesions. Even though the prevalence of PCV2 infection is very high and can be 95-100% of the pig population, the incidence of PMWS is typically low, ranging from 5-15% of pigs on affected farms. Although PCV2 is thought to be the only essential infectious agent, PMWS has been difficult to reproduce in pigs inoculated only with PCV2. In a majority of studies, PCV2 alone as inoculum failed to reproduce any pathological changes and/or clinical signs similar to those observed in field cases of PMWS, even though the virus or viral antigens were detected in various tissues. In a few studies, a low proportion of pigs developed some lesions or clinical signs of PMWS, but few developed **all** clinical signs and histologic lesions of PMWS, although the virus or viral antigens were detected in various tissues. Recently, new terminology was introduced and approved by the American Association of Swine Veterinarians (AASV) – Porcine Circovirus ASSOCIATED disease (PCVAD)- used to denote the entire spectrum of diseases that are associated with PCV2 including PMWS, respiratory disease, reproductive failure, diarrheal disease, Porcine Dermatitis Nephritis Syndrome (PDNS), and high mortality in pigs. In many cases, different co-factors (infectious and non-infectious) are required, along with PCV2, to reproduce clinical signs and lesions in these pigs. The following pathogens should be considered as differentials when diagnosing PCVAD in a swine population: Porcine Respiratory and Reproductive Syndrome (PRRS), Swine influenza virus (SIV), Transmissible Gastroenteritis/Porcine Respiratory Corona-virus (TGE/PRCV), Porcine Parvovirus (PPV), pestiviruses, *M. hyopneumonia,* and *Salmonella*. Also, one should take into consideration the history of the animals' on- farm management, vaccination, breed, stress, etc. PCV2 is infectious to swine, can replicate at a high level in sick animals, is associated with unique lesions and has a significant impact on the swine industry.

Detection of porcine circovirus types1 and 2, as well as detecting antibodies to PCV, can be accomplished using several testing methods.

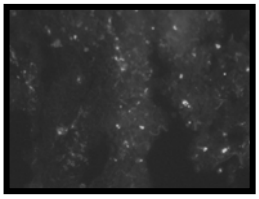
Virology Section: Fluorescent antibody on tissue sections (FATS), electron microscopy (EM) and isolation of the virus from infected tissues (VI). The best samples for virology assays are fresh tissues (lung, tonsil, enlarged lymph nodes, kidney, spleen) submitted on ice packs.



Virus isolation of PCV

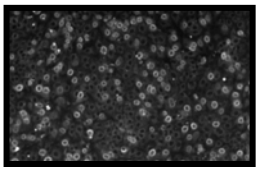


Detection of PCV using Electron Microscopy



Detection of PCV by FATS

Serology Section: Antibodies to porcine circovirus can be detected in the Serology Section by IFA. Submit clear serum.

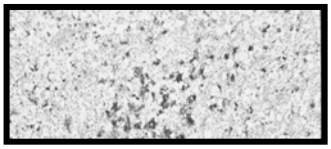


IFA assay using serum samples with antibody to PCV

Molecular Diagnostics: Porcine circovirus can be detected and typed in infected tissues or serum samples by PCR.

Detection and typing of PCV1 and PCV2 virus by PCR in serum and tissues.

Histopathology section: PCV virus can be detected by Immunohistochemistry (IHC) in infected tissues.



Detection of the PCV virus in tissues by IHC

Detection of other pathogens is also possible in ADDL upon request. Cost of tests and submission information can be obtained on ADDL's website – www.addl.purdue.edu – or by calling (765) 494-7440.

Confirmation of a diagnosis of PCVAD requires the demonstration of PCV2 in high concentrations within typical lesions.

For necropsy examination at ADDL, submit affected live pigs or pigs that have recently died.

If necropsy is performed onsite, collect serum and the following tissues: several enlarged lymph nodes, tonsil, spleen, kidney, liver, lung (several locations that are representative of all gross lesions observed) and ileum. Fix one set of tissues in 10% neutral buffered formalin. Send additional sets of fresh, chilled tissues (1 set each for virology and bacteriology if desired) placed in separate sterile, sealable bags. When TGE/coronavirus is suspected as a co-factor, also include jejunum and a fecal sample.

For fixation, parenchymal tissues should be no thicker than 0.5 inches and gut segments approximately 1 inch long and rinsed free of contents prior to placing the tissues in 10% neutral buffered formalin.

Do not mix lung samples or gut samples with any other tissue in a specimen bag if submitting for viral or bacterial testing.

Containers should be clearly labeled and all should be shipped chilled to ADDL by 24 hour couriers. Please do not ship on Friday to avoid degeneration of tissues in a shipping warehouse over the week-end.

-by Dr. Roman Pogranichniy, Head of Virology/Serology and Dr. Greg Stevenson, Head of Pathology

References:

1. Allan, GM, Kennedy, S, McNeilly F, Foster JC, Ellis JA, Krakowka, SJ, Meehan BM and Adair, BM: 1999. Experimental reproduction of severe wasting disease by coinfection of pigs with porcine circovirus and porcine parvovirus. J Comp Pathol 121:1-11.
2. Allan GM, McNeilly F, Kennedy S, Daft B, Clarke EG, Ellis JA, Haines DM, Meehan BM, Adair, BM: 1998. Isolation of porcine circovirus-like viruses from pigs with a wasting disease in the USA and Europe. J Vet Diagn Invest 1;: 3-10.
3. Clark EG: 1997. Post-weaning multisystemic wasting syndrome. Proc AASP Annual Meeting. Pp 499-501.
4. Ellis J, Hassard L, Clark E, Harding J, Allan G, Willson P, Strokappe J, Martin K, McNeilly F, Meehan B, Todd D, Haines D: 1998. Isolation of circovirus from lesions of pigs with postweaning multisystemic wasting syndrome. Can Vet J39: 44-51
5. Harding J: 1997. Post-weaning multisystemic wasting syndrome (PMWS): Preliminary epidemiology and clinical presentation. Proc AASP Ann Meet p. 503.
6. Harding JS, Clark EG: 1997. Recognition and diagnosing postweaning multisystemic wasting syndrome (PMWS). Swine Health and Production 5:201-203.
7. Harms PA, Sorden SD, Halibur PG, Bolin SR, Lager KM, Morozov I, Paul PS: Experimental reproduction of severe disease in CD/CD pigs concurrently infected with type 2 porcine circovirus and porcine reproductive and respiratory syndrome virus. Vet Pathol 38: 528-539.
8. Kennedy S, Moffett D, McNeilly F, Meehan B, Ellis J, Krakowka S and Allan GM: 2000. Reproduction of lesions of postweaning multisystemic wasting syndrome by infection of conventional pigs with porcine circovirus type 2 alone or in combination with porcine parvovirus. J Comp Pathol 122:9-24.
9. Krakowka S, Ellis JA, Meehan B, Kennedy S, McNeilly F, Allan G: 2000. Viral wasting syndrome of swine: experimental reproduction of postweaning multisystemic wasting syndrome in gnotobiotic swine by coinfection with porcine circovirus 2 and porcine parvovirus. Vet Pathol 37: 254-263.
10. Opriessnig T, Thacker EL, Yu S, Fenaux M, Meng XJ, Halbur PG: 2004. Experimental reproduction of postweaning multisystemic wasting syndrome in pigs by dual infection with *Mycoplasma hyopneumoniae* and porcine circovirus type 2. Vet Pathol 41: 624-640.
11. Pogranichniy RM, Yoon K-J, Harms PA, Sorden S, Daniels J: 2002. Case-control study on the association of porcine circovirus type 2 and other swine viral pathogens with postweaning multisystemic wasting syndrome. 14(6):449-456.
12. Pogranichniy RM, Yoon K-J, harms PA, Swenson SL, Zimmerman JJ, Sorden SD: 2000. Characterization of immune response of young pigs to porcine circovirus type 2 infection. Viral Immunol 13: 143-153.
13. Sorden SD: 2000. Update on porcine circovirus and postweaning multisystemic wasting syndrome (PMWS). Swine Health and Production 8:133-136.
14. Tischer K, Bode L, Peters D, Pociuli S, Germann B: 1995. Distribution of antibodies to porcine circovirus in swine populations of different breeding farms. Arch Virol 140:737-743.
15. Tischer I, Gelderblom H, Vettermann W, Koch MA: 1982. A very small porcine virus with circular single-stranded DNA. Nature 295:64-66.
16. Rischer I, Mields W, Wolff D, Vagt M, Griem W: 1986. Studies on epidemiology and pathogenicity of porcine circovirus. Arch Virol 91: 271-276.
17. Rischer I, Rasch R, Tochtermann G: 1974. Characterization of papovavirus-and picorna-virus-like particles in permanent pig kidney cell lines. Zentralbl Bakteriol (Origin A) 226:153-167.

ADDL NEWS

ADDL pathology graduate students won several awards at recent American Association of Veterinary Laboratory Diagnostician (Minneapolis, October 2006) and American College of Veterinary Pathologists (Tucson, December, 2006) meetings.

American Association of Veterinary Laboratory Diagnosticians

Dr. Ikki Mitsui
Best Graduate Student Poster Award

Dr. Pam Mouser
Travel Award

Dr. Dinesh Singh
Travel Award

American College of Veterinary Pathologists

Dr. Ingeborg Langohr
-Young Investigator Award in the Diagnostic Pathology Specialty Group, 2nd place
- Travel Award

Dr. Kim Maratea
Young Investigator Award in the Diagnostic Pathology Specialty Group, 3rd place

Dr. Ingrid Pardo
Natural Disease Specialty Group, 2nd place

Dr. Shawn Clark
- Toxicologic Pathology Specialty Group, 2nd place
- Student speaker award

Dr. Julia Lucas, (Clinical Pathology, Department of Comparative Pathobiology)
Toxicologic Pathologic Pathology Specialty Group, 1st place

Our congratulations to all

From Dr. Tom Bryan, Avian Diagnostician, Heeke ADDL

-Update to an article published in the Spring 1996 Diagnostic Forum entitled “Ascarid Migration in Turkey Livers”

With the advent of fenbendazole use in turkey feed (14.5 gallons/ton feed for sic consecutive days) ascarid migration and liver condemnations have been greatly reduced. Cresylic acid is no longer available as an approved disinfectant for poultry.

ADDL Schedule

Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2007.

January 15.....Martin Luther King Day
May 28.....Memorial Day
July 4.....Independence Day
November 22-23.....Thanksgiving
December 24-25.....Christmas

ADDL test results are available on the Internet. Call 765-494-7440 to set up an account or visit our web page at www.addl.purdue.edu and follow instructions after clicking on Online Reports tab.