UNIVERSITY of GUELPH LABORATORY SERVICES

Animal Health Laboratory

# AHL Newsletter

Volume 9, Number 1, page 1 March, 2005 ISSN 1481-7179

## In this issue:



## Unusual clinical signs in cattle associated with viral infection

*Lloyd Wieringa, Tony van Dreumel, Ann Godkin, Susy Carman*

Some baffling clinical signs were noted recently among the cows in a 90-head well-managed cross-bred cow-calf herd. The herd had been vaccinated pre-breeding with a 10-way MLV and bacterin (lepto) vaccine.

On Oct 19, the herd veterinarian did the annual herd pregnancy check. All animals were healthy; and all were in a single group. On Nov 8, calves were weaned and the herd was separated into two groups – dry cows and bred heifers, and cows that had newly weaned a calf. On Nov 11, the owner noticed that some of the cows in the weaned group of 16 had mild nasal discharge and prominent submandibular swelling.

A veterinarian was called on Nov 13, when 16 cows (predominantly $\leq$ 4 yrs old) from both groups were affected. The cows had rectal temperatures of 104-106 $^{o}$F, but were still bright and eating. Some cows had profuse mucopurulent nasal discharge; some were drooling. No abnormal lung sounds were noted. In 2 cows, the edema was extensive and reached from eye to eye involving the cheeks and below the mandible, but not the neck area (Fig. 1). All cows were treated with long-acting tetracycline, and about a third received anti-inflammatories as well. By Nov 15, the severe edema in the 2 cows had reduced, but temperatures were still elevated. Ten days later (Nov 21), nose and lip erosions developed in 4 to 5 of the original 16 animals (Fig. 2). Animals gradually recovered over the next few weeks and now appear normal.

The practitioner submitted sera to the AHL. Acute and convalescent sera were collected from 6 animals allowing a short (10 d) and a longer (20 d) interval to elapse. On all animals, across both time intervals, the pairs of serum had stable titers to *Bovine adenovirus 2*, *Bovine respiratory syncytial virus*, *Bovine herpesvirus 1* (IBRV), and *Bovine viral diarrhea virus*. Five of 6 animals had significant 4-fold or greater increases in antibody titer to ***Bovine parainfluenza virus 3*** during the short duration assessment, and confirmed at the longer interval. Three of 6 animals also had significant 4-fold increases in antibody to ***Bovine coronavirus***. CFIA tests of these animals were negative for vesicular stomatitis, foot-and-mouth disease, epizootic hemorrhagic disease, and bluetongue. *AHL*

Figure 1. Beef cow with submandibular edema, Nov 16.

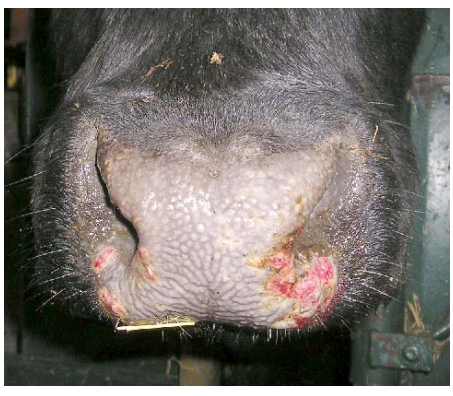

Figure 2. Beef cow with nasal erosions, Nov 24.

# Pulmonary aspergillosis in 3 farmed elk: incidental lesions resembling tuberculosis

*Alexandre Loretti, Gary Halbert, Rob Foster, Gary Thomson, Jeff Caswell, Ann Hewson, Rob Swackhammer*

During late Nov-Dec 2004, 3 farmed elk calves <1 y of age from 2 farms in Ontario were submitted to the Department of Pathobiology, OVC/AHL for necropsy. On 1 of the premises, 12 of 45 calves had died since Aug 2004. Clinical signs included listlessness and depression or acute bloat. The clinical course of this fatal disease varied from 1-4 d. Clostridial enterotoxemia, coccidiosis and intestinal parasitism were suspected clinically.

At necropsy, carcasses were emaciated, and fat deposits and muscle mass decreased. **A few to numerous, well-demarcated, firm, white or gray nodules 0.2-0.7 cm in diameter were scattered throughout the pulmonary parenchyma or on the pleural surface.** On cut surface, homogeneous, yellow, thick, caseous material was surrounded by a thin, white, fibrous capsule. Other findings included limb fractures and heavy parasitism by whipworms (*Trichuris* sp.). No gross lesions were found in lymph nodes, tonsils, or other tissues. The pulmonary lesions were interpreted as abscesses or granulomas. Lung nodules were submitted for microbiological culture in order to rule out tuberculosis and determine the etiology.

Microscopically, **large numbers of hyphae morphologically consistent with *Aspergillus* spp. were observed in wet mounts and in histological sections within foci of caseous necrosis.** *Aspergillus fumigatus* was cultured in one of the cases. No acid-fast bacilli were seen in smears or histological slides. From 1 case, a non-tuberculous, rapid-growing, *Mycobacterium* was cultured by the Central Public Health Laboratory, Toronto, ON, and was considered to be an incidental, non-significant finding. No BVDV or other viruses were cultured, and ELISA for *Clostridium perfringens* enterotoxin was negative. The cause of death could not be determined, and the pulmonary lesions were considered to be incidental.

Pulmonary mycoses in domestic and wild ruminants can be caused by ubiquitous saprophytic molds of the genus *Aspergillus*. Infection can be initiated by inhalation of fungal spores from moldy litter and feed, or by hematogenous spread from a GI lesion. It appears to be a complication of other debilitating disease; the cause of emaciation in the elk calves was not identified. There are a few published cases of pulmonary aspergillosis in deer, with some submitted by veterinary practitioners to diagnostic labs because of macroscopic lesions suggestive of tuberculosis. The disease has not been described in elk.

**Gross lesions of *Aspergillus* spp. in the lungs of farmed cervids should be differentiated from those induced by other pathogens that can cause similar multifocal, nodular, firm lesions with central caseation, including *Mycobacterium bovis, Arcanobacterium pyogenes, Yersinia pseudotuberculosis,* and lungworms (*Dictyocaulus viviparus*).** *AHL*

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*Its mission is to inform AHL clients and partners about AHL current activities, and lab-based animal disease events and disease trends.* 

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# Selected zoonotic pathogens and diseases identified at the AHL, 2000 - 2004

*Beverly McEwen, Durda Slavic, Davor Ojkic, Susy Carman, Peter Lusis*

Many new, emerging, and re-emerging diseases of people are caused by pathogens originating from animals, or are shared between people and animals. The AHL plays an important role in public health by identifying zoonotic pathogens: in 2004, AHL staff identified ***Eastern equine encephalitis virus*** **(EEEV), methicillin-resistant *Staphylococcus aureus* (MRSA), and *West Nile virus* infection in horses.** Targeted surveillance for MRSA in horses resulted in the increase of isolates. Other selected zoonotic pathogens isolated and/or identified at the AHL are given in Tables 1 and 2. These are numerator data reliant upon submission biases to the diagnostic laboratory and cannot be regarded as population prevalence estimates.

*Salmonella sp., Streptococcus sp.* infections, and *Cryptosporidium sp.* are the most frequently identified zoonotic pathogens at the AHL since 1999. Occupational exposure to pigs and horses is a risk factor for *S. suis* and *S. zooepidemicus* infections. *West Nile virus* continues to be identified by PCR in non-domestic species and occasionally in horses by IgM ELISA. The dramatic increase in *Leptospira spp.* seropositive dogs is partially due to increased submissions; canine Leptospirosis will be updated in the June 2005 AHL Newsletter. As part of our commitment to animal health surveillance, the AHL validated or developed new tests in 2004, such as Giardia ELISA, EEEV PCR. *AHL*

Table 1. Selected zoonotic pathogens isolated and/or identified at the AHL, 2000-2004

| Agent | Bovine | Swine | Equine | Ovine | Caprine | Chicken | Turkey | Canine | Feline | Other | **2004** | 2003 | 2002 | 2001 | 2000 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Bordetella bronchiseptica* | | 39 | 2 | | 1 | | 1 | 8 | 8 | 19 | **78** | 84 | | 84 | 77 |
| *Campylobacter coli/ jejuni/ fetus subsp. fetus* | | | | 2 | | | | 22 | 1 | 1 | **26** | 31 | 33 | 36 | 14 |
| *Chlamydophila psittaci* | | | | 3 | 2 | | | | | 1 | **6** | 19 | 10 | 12 | 21 |
| *Coxiella burnetii* (Q fever) | 1 | | | 1 | 2 | | | | | | **4** | 6 | 6 | 7 | 4 |
| *Cryptosporidium sp.* | 55 | 3 | | | 2 | | | | | 1 | **61** | 129 | 199 | 160 | 120 |
| *Eastern equine encephalitis virus* | | | 2 | | | | | | | | **2** | 10 | 0 | 0 | 0 |
| *Francisella tularensis* | | | | | | | | | | 1 | **1** | 1 | 1 | 3 | 6 |
| *Giardia sp.* | 4 | | | | | | | 9 | | | **13** | 10 | 4 | 19 | 26 |
| *Listeria monocytogenes* | 19 | 1 | 2 | 5 | 2 | | | | | 5 | **34** | 27 | 37 | 44 | 34 |
| Methicillin-resistant *Staphylococcus aureus* | | | 131 | | | | | 2 | | | **133** | 73 | 13 | 1 | - |
| *Mycobacterium bovis* | | | | | | | | | | | **0** | 0 | 1 | 0 | 0 |
| Rabies | | | | | | | | | | | **0** | 0 | 0 | 5 | 1 |
| *Salmonella sp.* | 97 | 234 | 126 | 1 | 7 | 51 | 47 | 5 | | 72 | **640** | 824 | 716 | 565 | 754 |
| *Streptococcus suis* | 5 | 435 | | 4 | 2 | | | | | 1 | **447** | 392 | 477 | 380 | 560 |
| *Streptococcus equisimilis* | 1 | 111 | 25 | | | | 1 | | | 2 | **140** | 111 | 144 | 126 | 154 |
| *Streptococcus zooepidemicus* | 5 | 13 | 162 | 3 | | | | 1 | 1 | 1 | **186** | 211 | 222 | 175 | 162 |
| *Toxoplasma sp.* | | | | 6 | 1 | | | | 2 | 1 | **10** | 5 | 12 | 12 | 8 |
| *West Nile virus* | | | 10 | | | | | | | 148 | **158** | 173 | 91 | | |
| *Yersinia enterocolitica* | 1 | 4 | | | | | | | | 3 | **8** | 8 | 5 | 8 | 2 |

Table 2. *Leptospira spp*. seropositive samples (≥1:320) identified at AHL, 2000 – 2004, microscopic agglutination test (MAT)

| *Leptospira spp*. serovar | Bovine | Swine | Equine | Canine | Feline | **2004** | 2003 | 2002 | 2001 | 2000 |
|---|---|---|---|---|---|---|---|---|---|---|
| *L. autumnalis* | | | 1 | 225 | | **226** | 122 | 46 | 32 | 50 |
| *L. bratislava* | | 116 | 46 | 166 | 1 | **329** | 151 | 139 | 120 | 88 |
| *L .canicola* | | | | 56 | | **56** | 17 | 4 | 6 | |
| *L. grippotyphosa* | | 58 | 16 | 122 | | **196** | 76 | 29 | 44 | 32 |
| *L. hardjo* | 57 | | | 3 | | **60** | 34 | 30 | 21 | 7 |
| *L. icterohaemorrhagiae* | 51 | | 1 | 59 | | **111** | 122 | 163 | 289 | 140 |
| *L. pomona* | 18 | 12 | 20 | 78 | | **128** | 122 | 105 | 110 | 112 |
| **Total** | **126** | **186** | **84** | **709** | **1** | **1106** | **644** | **516** | **622** | **429** |

# POULTRY

## Summary of AHL pathology diagnoses for Ontario poultry, 2003-2004

*Emily Martin, Brian Binnington, Sameh Youssef, Jan Shapiro, Davor Ojkic, Bev McEwen*

This summary of the pathology diagnoses made from December 2003 to December 2004 is drawn from the yearly summaries produced from the AHL database of diagnoses made at the Guelph and Kemptville laboratories.

### Broiler chickens

**Bursal lymphoid depletion** continues to be a frequent finding in birds ranging in age from 10 d to 7 wk, with the majority of birds being 3-4 wk of age. Bursal lymphoid depletion is a non-specific finding that can have multiple causes including infection with infectious bursal disease virus (IBDV) and severe septicemia. Bursal lymphoid depletion was frequently diagnosed with concurrent infections including bacterial septicemia (primarily *E. coli*) and coccidiosis, however, the most common concurrent disease in 2004 was **inclusion body hepatitis** (IBH) associated with a fowl adenovirus (FAdV). IBH can occur alone, but primary immunosuppressive events (e.g. IBDV infection, *Chicken anemia virus* (CAV)) may enhance the ability of adenoviruses to produce IBH. Outbreaks of IBH have occurred yearly in Ontario, and in the past 2 years the AHL has also received submissions from outbreaks in Manitoba, Alberta, and British Columbia. The main strains found in Canada include FAdV-8a strains TR-59 and T-8, FAdV-11 strain 380, and FAdV-7 strain x11a.

In Nov. and Dec. 2004, a cluster of **infectious laryngotracheitis** (ILT) cases occurred on 5 broiler farms and 1 small layer operation in southwestern Ontario. Slight changes in disease trends included an increase in coccidial enteritis, whereas necrotic enteritis cases decreased. Other diseases diagnosed consistently were *E. coli* septicemia, yolk sacculitis, arthritis, rickets, spiking mortality, and cellulitis.

### Broiler breeder chickens

**Bacterial septicemia** was the primary diagnosis for broiler breeders from 3 d to 48 wk of age. *E. coli* was the primary isolate, followed by *Staphylococcus aureus*. The next most common diagnosis was **arthritis/tenosynovitis**, with *E. coli* and *S. aureus* isolated individually or in combination. There were 5 cases of *Fowlpox virus* infection (cutaneous and diphtheritic forms) in unvaccinated flocks. Some of these cases were suspected to be associated with movement of people between related farms. A number of other diseases were consistently but less frequently diagnosed in broiler breeders, including cellulitis, pododermatitis, peritonitis, hepatitis, and urate nephrosis. Diseases that have decreased over the last few years include neoplasms (leukosis).

### Layer chickens

Nutritional disease, primarily **osteomalacia** (cage layer fatigue), continues to be the most common diagnosis in layers at the AHL. Osteomalacia develops after prolonged calcium depletion leading to softened bones, bone fractures, and tendon ruptures. Conditions consistently diagnosed include peritonitis (yolk, bacteria), septicemia (*E. coli* and *S. aureus*, alone or in combination), necrotic enteritis and coccidiosis. We made occasional diagnoses of Marek's disease and peripheral neuropathy of white leghorns. Peripheral neuropathy can cause lameness and increased mortality with inflammatory lesions in peripheral nerves but not in the brain or spinal cord. Diagnoses of hepatitis/splenomegaly decreased in 2004. Avian strains of hepatitis E virus may play a role in this condition, however, the role of other factors is also under investigation. Other disease diagnoses that have decreased over the last few years include neoplasms (leukosis, other), infectious bronchitis (IB), and fatty liver hemorrhage syndrome.

### Turkeys

**Bacterial septicemia** continues to be the primary diagnoses for turkeys ranging in age from 2 d to 18 wk of age. *E. coli* is the primary isolate; other organisms include *S. aureus* and *Pseudomonas aeruginosa*. Other conditions consistently diagnosed include enteritis (nonspecific, parasitic), hepatitis, long-bone deformities, round heart, and myopathy. Decreases were observed in cases of enteritis (necrotic, coccidia), aspergillosis, candidiasis, yolk sacculitis, starve-outs, arthritis and rickets. There was one case of histomoniasis in 2-mo-old birds, one case of botulism in 16-wk-old males, and 2 cases of erysipelas in 14-wk-old males. Disease diagnoses that have decreased and remained low over the past several years include *Bordetella avium* and *Ornithobacterium rhinotracheale*. *AHL*

# SWINE

## *Porcine circovirus* type 2-associated conditions in pigs

*Tony van Dreumel, Gaylan Josephson, Peter Lusis*

In the past few months, we have seen several cases of severe *Porcine circovirus* type 2 (PCV2)-induced **enterocolitis** in pigs. Affected pigs have watery diarrhea and loss of condition, both of which are poorly responsive to treatment. Gross lesions resemble those seen with *Lawsonia intracellularis* enteropathy and consist of thickening of the jejunum, ileum and colon. On histopathology, there are large numbers of lymphocytes in the villus mucosa and prominent lympho/plasma/histiocytic infiltrates in the lamina propria and submucosa. Lymphocytolysis and lymphoid depletion of the submucosal follicles, and grape-like intracytoplasmic inclusions in affected cells and follicles may also be observed. Immunohistochemical staining (IHC) shows circovirus antigen in proprial macrophages and the myenteric plexus (Fig. 1).

PCV-2 should be considered in the differential diagnosis of the porcine respiratory disease complex and of enterocolitis in grower and finisher pigs

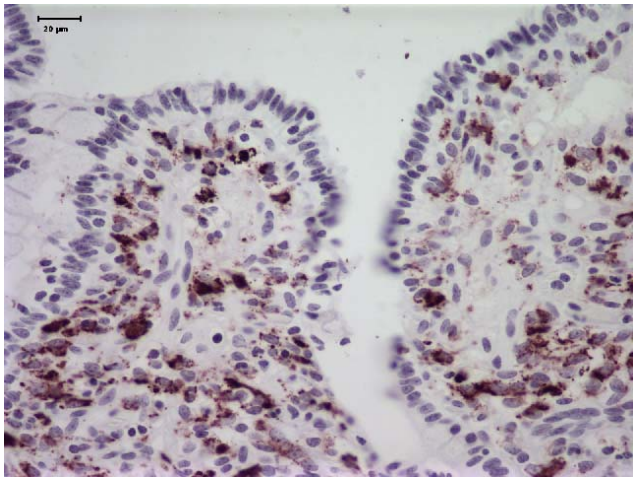
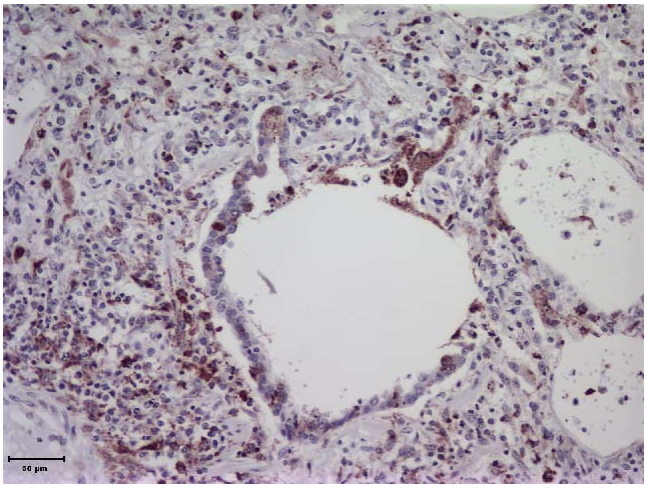


Figure 1. Intense staining of macrophages in the villus lamina propria of small intestine. IHC stain for PCV-2 antigen.

In many cases, the initial presenting sign is a chronic, deep, rasping cough that appears 2-3 weeks into the grower phase. Closer examination of late nursery pigs may identify the presence of a few poor-doing pigs, as well as some puffing animals. Death losses have ranged as high as 15% of animals placed. In some cases, surviving pigs continue to gain weight as expected, being marketed at normal times. However, in most cases, many of the affected pigs that survive do poorly and, if they actually do reach market weight, do so at a much later date than their cohorts. Post mortem findings in affected pigs also include various degrees of **bronchointerstitial pneumonia**, with lungs failing to collapse on opening the thorax. Microscopic findings include marked monocytic interstitial reaction in some lobules. Mild peribronchial and perivascular lympho/histiocytic cuffing is also found. Mild necrotizing bronchiolitis, characteristic of swine influenza virus infection, is occasionally observed. In some submissions, laboratory tests have failed to identify the presence of other pulmonary pathogens such as *Streptococcus suis*, *Mycoplasma hyopneumoniae*, swine influenza virus, and PRRSV. PCV2 has been identified by PCR and subsequent restriction fragment length polymorphism from affected lungs. In addition, IHC staining has identified the presence of large amounts of PCV2 antigen associated with the lung lesions (Fig. 2).

Figure 2. Pig lung showing PCV-2 antigen in bronchiolar epithelial cells and alveolar macrophages.



Affected animals may have other lesions associated with PCV type 2 infection, including lymphadenopathy, hepatitis, and interstitial nephritis.

PCV-2 should be considered in the differential diagnosis of the porcine respiratory disease complex and of enterocolitis in grower and finisher pigs. *AHL*

# PRRSV outbreak in southwestern Ontario

*Susy Carman, Beverly McEwen, Gaylan Josephson, Jim Fairles*

During the fall of 2004, many veterinarians in southwestern Ontario recognized an increase in the number of swine herds that "broke" with PRRSV infection. In some swine herds, the clinical disease was mild with seroconversion as the only indicator of infection, whereas in others the clinical disease was severe with abortion storms in sows. Figure 1 shows the total number of cases submitted to the AHL for PRRSV diagnosis using PCR and the total number of these cases found to be positive, using 3-month intervals from January 2001 to December 2004. These data do not include monitoring cases, where semen was the sample submitted.

Our case submission information reflects the PRRSV outbreak recognized by swine practitioners in the fall of 2004, with **an increase in the total number of cases submitted for testing by PCR, an increase in the total number positive for PRRSV, and an increase in the percent PRRSV-positive cases to 55%.** The graph also shows an increase in percent PRRSV-positive cases beginning in the fall of 2003, with 50% positive for PRRSV. This increase was originally interpreted as the result of increased testing of serum by PCR for surveillance programs, but may or may not have been the start of the outbreak.

Gene sequence analysis of the ORF5 envelope gene for 42 PRRSV strains identified from 2003 to 2004 shows the outbreak not to be from a single source (except for one integrated loop), with viruses distributed over 8 phylogenetic branches, including 2 vaccine-like groups. *AHL*

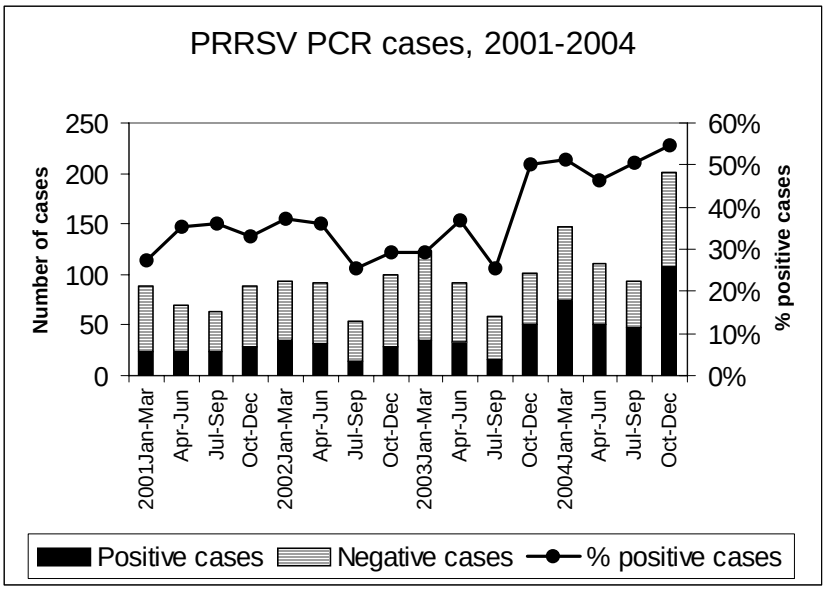



Figure 1. PRRSV PCR submissions tested by the AHL, 2001-2004, with % positive cases.

# AHL adds indirect IgM immunofluorescence (IFA) test for PRRSV

*Susy Carman*

The AHL now offers indirect immunofluorescence (IFA) testing for the evaluation of **both IgM and IgG antibody for the North American strains of PRRSV** in swine sera. Using the IFA test, IgM antibody can be detected in pigs as early as 5 days post infection (dpi). IgM antibody peaks at 14-21 dpi, and rapidly declines to undetectable levels by 35-42 dpi. IgG antibody can be detected in the IFA at 7-11 dpi, compared to 9-11 dpi for the ELISA. Although IFA IgG antibody arrives earlier, it persists for a relatively shorter time (4-5 mo) compared to IgG demonstrated by ELISA (4 to >10 mo).

**The IDEXX ELISA is the best herd-based test to use for serological monitoring of swine herds**. **The IFA tests for IgM and IgG are best used to evaluate IDEXX PRRSV singleton positive reactors in PRRSV-negative swine herds.**

The IFA tests are performed in 8-chamber cell culture slides at a single 1:20 serum dilution, using both a virus-infected chamber and an uninfected negative control chamber for each serum. The result is reported as positive or negative. These tests will be run on **Wednesdays and Fridays**, on the days following routine PRRSV IDEXX ELISA testing, for a fee of **$9 per test**. *AHL*

# HORSES

## Possible alsike clover toxicosis in a group of horses

*Murray Hazlett, Robert Wright, Graham Dingwell*

Out of a group of 9 mature Quarter Horses, two 2-yr-olds were ill and over the period of a weekend died. One horse was found down in its stall, stood when examined, but was ataxic. It was eating 12 h later, but was found dead the next day. The second horse was off feed for a few days, icteric, and was bilirubinuric and hypoglycemic before death. Both originated from western Canada, approximately 1 yr previously. Horses were housed indoors in a renovated bank barn set up into large pens containing 2-3 animals each, and were turned out into large paddocks as weather permitted.

Necropsies revealed fluid in the thoracic and peritoneal cavities. Horse 2 had a large hematoma on its liver. Samples were submitted to the AHL for histopathology.

Microscopically, livers of both horses had hyperplasia of bile ducts with accompanying fibrosis and some inflammation (Fig 1). Many bile ducts were distended with bile. Horse 1 had areas of neutrophilic inflammation, and evidence of terminal bacteremia. Horse 2 had large areas of necrosis in the liver, with some neutrophilic inflammation associated with these. Casts, likely hemoglobin, were present in renal tubules of horse 2. Based on histopathology, differential diagnoses included alsike clover, mycotoxins, or other environmental toxicoses.

A subsequent examination of the hay that the horses were being fed revealed the presence of large amounts of alsike clover (15 to 20% of dry matter).

In a review on this disease, Dr Nick Nation concluded that the cause was never fully proven, and given the amount of alsike clover fed to livestock in North America, it is surprising that more cases of this disease have not been identified. It may be, as Dr Nation speculates, that a mycotoxin is involved, or a breakdown product formed with improper curing. A mycotoxin produced by saprophytic or endophytic fungi seems most likely. Last spring and early summer were cooler and wetter than normal, and all of the clover species were more abundant in pastures and hay fields. Slaframine poisoning (slobbers), associated with the fungus commonly called Black Patch, was reported (5 reports) in Ontario in greater numbers than previous years. The cooler wetter weather commonly leads to increased fungal growth and higher than normal mycotoxin concentrations. **We would be interested in hearing of potentially similar problems equine practitioners have encountered.**

Pasture mixes in Ontario often contain alsike clover seed, where it may be as much as 40% of the seed mix. The clover is not native to North America - it was originally imported from Sweden, but is now found throughout Canada and the USA. It grows well on cooler wet clays and is commonly found in northern and eastern Ontario, but was commercially grown in the southwestern area of Ontario where these horses were located. Alsike clover is easily differentiated from red clover by its 1 cm non-apical pink flower, and leaves that are solid green (no white "v" on them; Fig 2). The leaves and flowers are produced along the length of the vertical stem. Red clover also grows vertically but has hairs along the stem and terminates in larger 2-3 cm magenta flowers. There is a distinct white "v" on the leaves of red clover. White clover has a horizontally growing stem along which leaves and flowers are found. In hay, alsike is easily differentiated from red clover by the lack of hairs and a finer stem and position of the flowers. In hay, only the leaves and flowers of white clover are found. *AHL*

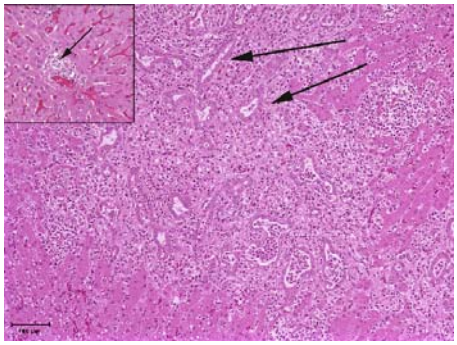


Figure 1. Liver from horse 2 showing affected triad region with proliferating bile ducts (2 large arrows). Top left insert shows normal liver and bile duct (arrow).

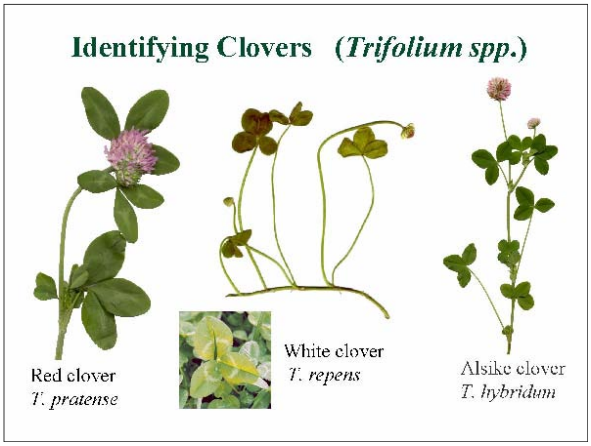



Figure 2. Red, white and alsike clover. From ref 2.

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# COMPANION ANIMALS

## Feline hepatic lipidosis

*Peter Lusis, Brent Hoff*

Two female domestic shorthaired cats aged 3.5 and 10 years, from different premises and with vague clinical histories, were presented for necropsy. Both cats were obese, had severe jaundice and severe hepatic lipidosis. No other significant lesions were evident in the younger cat, but the older cat had advanced glomerulonephritis with glomerulosclerosis.

**Hepatic lipidosis is a relatively common disease of obese, inappetent cats, an is usually fatal if untreated**. It can be primary (idiopathic) or secondary to other diseases such as diabetes or pancreatitis.

In one study, 12 of 15 obese cats developed hepatic lipidosis after fasting for 5-7 weeks, with 30-35% reduction in body weight, reduced serum urea, glucose and albumin. Hyperbilirubinemia and increased hepatic-associated enzyme levels (ALT, AST and ALP) with little or no increase in gamma GT activity are also present, with low PCV, hypokalemia and older age being significantly related to non-survival. In some cases, enzymes may not be significantly elevated. Serum concentrations of β-hydroxybutyrate (BHBA), non-esterified fatty acids (NEFA), and bile acids may be of diagnostic value in suspect cases.

**Because hepatic lipidosis can be confirmed readily using fine-needle aspiration biopsy, anesthesia and surgical biopsy are not required**.

A 50-80% recovery rate can be expected with intensive long term (several weeks) dietary therapy (nasogastric or percutaneous gastric tube feeding). *AHL*

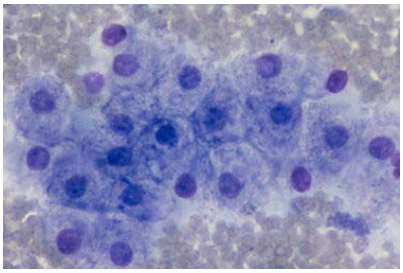

Figure 1. Fine needle aspirate (FNA) of normal liver.

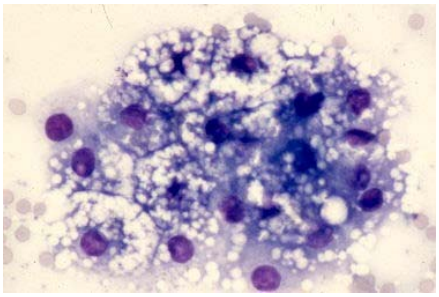

Figure 2. FNA of grade 4/5 fatty liver.

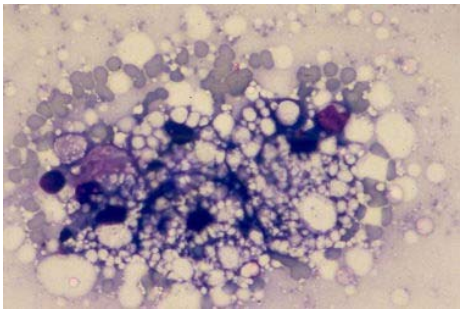

Figure 3. FNA of grade 5/5 fatty liver.

**References**

Biourge VC, et al. Experimental induction of hepatic lipidosis in cats. Am J Vet Res 1994;55:1291-1302.

Center SA, et al. A retrospective study of 77 cats with severe hepatic lipidosis: 1975-1990. J Vet Intern Med 1993;7:349-359.

## The revised AHL submission forms are ready!

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