University of Guelph LABORATORY SERVICES AHL Newsletter
ANIMAL HEALTH LABORATORY GUELPH KEMPTVILLE RIDGETOWN

## Volume 3, Number 1

## In this issue:



## What's happening at the AHL?

- The AHL **client service questionnaire** was sent to a cross section of our clientele in late Nov/98 - 23 of 38 questionnaires were completed and returned. Our overall satisfaction rating was "very good". We strive for continuous quality improvement, and will work on turnaround times (some tests inherently take considerable time, e.g. virus isolation) and improve our billing procedures.
- **Strategic Plan -** Input has been received from a wide variety of stakeholders this winter on the draft of the 5-year strategic plan for the AHL, including private practitioners and commodity groups. The plan is being revised and finalized by the Steering Committee of Dr. Leslie Woodcock of OMAF, Dr. Pat Shewen of the OVC, and Dr. Grant Maxie of the AHL, with facilitation from Dr. Brent Matthew, and is to take effect on May 1, 1999.
- The revised **AHL Fee Schedule** - to take effect May 1, 1999 - will be mailed in early April.
- Parasitology update - **Dr. Andrew Peregrine**, veterinary parasitologist in OVC Pathobiology, continues to provide professional diagnostic support to the AHL parasitology lab. He is available for consultations on Neospora and on canine heartworm, but not for general enquiries.
- Please note that **the submitter is responsible for charges on an AHL case.** Rapid and accurate billing demands that we do not change the "bill to" information after entry into VADDS.
- Please note that, effective immediately, **the AHL Purolator 'incoming collect' number has been changed to 0966901.** Thank you for making this change.
- Our **out-of-hours telephone number** at AHL-Guelph is (519) 824-4120, **ext 4552**(not 4501 or 4502 as used previously). Out- of-hours staff are also available at cell phone **# (519) 823-6146.**
- Effective with the February statements, we will be **charging interest on overdue accounts** - 1.5%/month, 18% per annum.
- Feedback for the AHL? Please feel free to call, fax, or E-mail us at any of our labs.

## Feedback for the AHL?

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# SWINE

## K88 coliform enteritis in swine

***Drs. Gaylan Josephson, Nonie Smart, Beverly McEwen, Tony van Dreumel, Jiggs Gough, Murray Hazlett***

Enteric infections in swine with K88-positive *E. coli* have increased significantly since the fall of 1996 (Figure 1). In 1998, there were 232 cases submitted to the AHL from 196 herds, compared to 47 cases from 46 herds in 1996. The slight decrease in the frequency of K88-positive coliform cases in December has occurred each December since 1995. The median age of infected pigs in 1998 was 21 days (range: a few days to 12 weeks), exactly twice the median age of infected pigs in 1995. **K88-positive coliform enteritis occurs most frequently in the post-weaning pig after segregated early weaning (SEW) has occurred at 14-19 days.** Losses may occur anytime, but are often reported when SEW diets are changed from Phase I to II or Phase II to III.

Histories indicate that pigs are often found dead without premonitory signs of diarrhea, or if diarrhea is present, it is unresponsive to treatment with common antibiotics. Necropsy lesions include: dehydration, venous infarction of the gastric fundus, and fluid-filled small intestines.

Fibrinous or necrotizing colitis, which grossly resembles swine dysentery, is present in many pigs. Large numbers of bacilli attached to the small intestinal brush border and fibrinocellular exudate laden with bacilli and adherent to colonic erosions are often present histologically.

Resistance of K88-positive *E. coli* isolates to individual antimicrobials is listed in Table 1. Of 198 K88-positive *E. coli* isolates, 197 were resistant to at least one antibiotic, however, most were resistant to multiple antimicrobials (Figure 2). Seventy isolates were multiresistant to the combination of clindamycin, tetracycline, spectinomycin, and sulfisoxazole. The same combination, plus resistance to either ampicillin or trimethoprim-sulfa, was present in an additional 20 isolates. There were 4 isolates resistant to all of the antimicrobials tested.

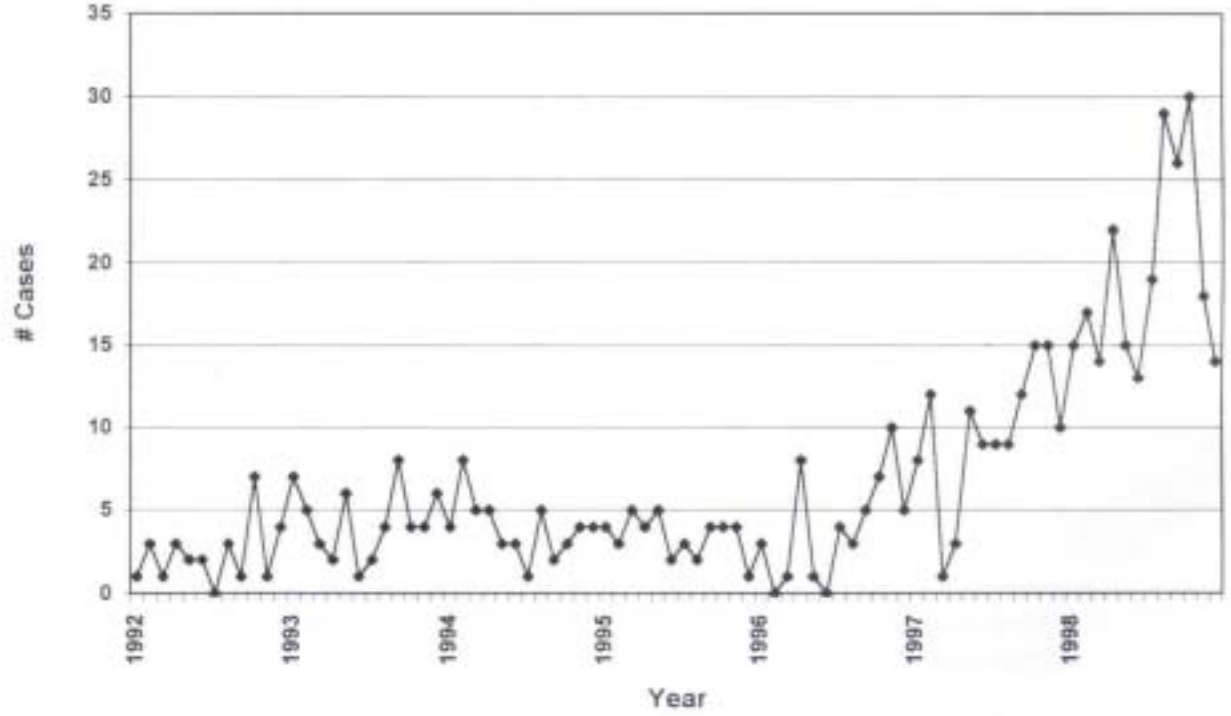


Figure 1. K88-positive *E.coli* cases in swine

Table 1. Resistance of K88-positive *E. coli* isolates to individual antimicrobials.

| Antimicrobial | Proportion resistant |
|---|---|
| **Clindamycin** | **1** |
| **Tetracycline** | **0.94** |
| **Spectinomycin** | **0.86** |
| **Sulfisoxazole** | **0.88** |
| **Ampicillin** | **0.32** |
| **Neomycin** | **0.24** |
| **Trimethoprim-sulfa** | **0.21** |
| **Tobramycin** | **0.1** |
| **Cephalothin** | **0.09** |
| **Gentamicin** | **0.08** |

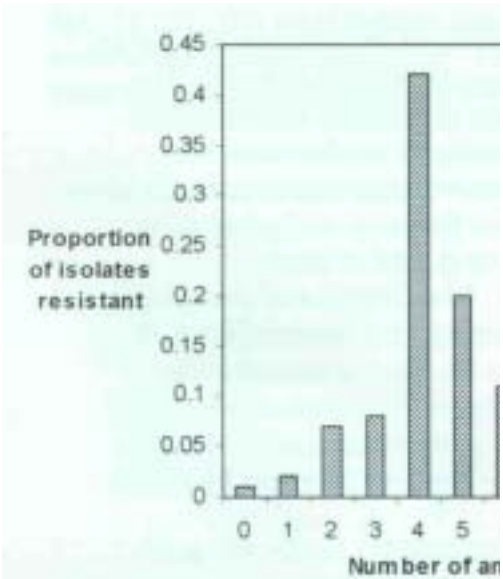


Figure 2. Antimicrobial resistance
*E.coli*

## Viral encephalitis in a swine loop

***Drs. Jeff Caswell, Gaylan Josephson, Susy Carman, Mr. Andrew Moore, Dr. Marg Stalker***

Non-suppurative encephalitis, probably of viral origin, is diagnosed in swine several times each year by pathologists at the Animal Health Laboratory, University of Guelph. Identification of a specific causative agent is often difficult, even from very acute cases.

Over the past 16 months, we have examined multiple pigs from a large swine loop that continues to experience an ongoing neurological problem in post-weaning pigs. This operation consists of 7-8000 sows in several breeding herds which supply piglets to multiple, off-site, segregated early weaning nurseries at 16-18 days of age. The neurological syndrome has not been identified in all of the nurseries, nor in progeny from all of the sow herds. To achieve rapid filling of the nurseries, co-mingling has been practiced in several of the barns. However, losses have occurred in both single-source and co-mingled nurseries. In affected nurseries, the maximum prevalence was 40 cases per 1000 pigs at risk. Piglets most commonly developed neurologic disease at 10 to 14 days after entry, although pigs up to 65 days of age have been affected.

Clinical signs were described as being similar to those seen with meningitis caused by *Streptococcus suis*, and were characterized by depression, anorexia, ataxia, recumbency, convulsions and death. Vomiting was not a feature of the disease. Barn managers were able to differentiate, at a very early stage, pigs that were affected with this syndrome from those with streptococcal meningitis.

Gross lesions were not noted in the brain, but lymphocytic encephalomyelitis was a consistent histologic lesion. The inflammatory changes were most severe in the brainstem and spinal cord, but also affected the cerebellum and cerebrum. These lesions were consistent with viral encephalomyelitis due to hemagglutinating encephalomyelitis virus (HEV) or less likely, porcine enterovirus (Teschen-Talfan disease).

Pseudorabies was considered unlikely based on the sporadic nature of the disease in the herd. A neurovirulent form of the PRRS virus has recently been described (1), but serologic and fluorescent antibody tests for PRRS on several cases in this herd were negative.

Numerous attempts to isolate bacterial and viral pathogens from brain, spinal cord, and lung were unsuccessful. The inability to isolate the virus may be caused by the presence of virus-neutralizing antibody in affected pigs. These antibodies would not neutralize virus present within neurons, and hence not arrest the development of disease, yet antibodies could neutralize extracellular virus and prevent the infection of cells in tissue culture. Indeed, sera of many pigs with encephalitis contained antibody to HEV. Serum titers to HEV were variable in both affected and unaffected pigs, ranging from negative to 1:8192. Interpretation of serological titers for single sera is difficult because the virus is endemic in most herds. As well, affected piglets may have already developed high levels of antibody by the time illness is noted, making evaluation of titers from paired sera also potentially unrewarding (2).

Examination of thin sections of brain from an acutely affected animal using transmission electron microscopy revealed viral particles in the cytosol and in membrane-bound cytoplasmic vesicles. The spherical particles were single or in groups of up to seven, averaged 106 nm diameter, and had an electron-dense 69 nm central core separated from the outer membrane by an electron-lucent layer. **These viral particles were of comparable morphology to previous descriptions of HEV in tissues (3)**. Serologic investigation of the herd is ongoing to further define the diagnosis of HEV and to assist in control of the disease in this loop.

Infection with HEV is widespread in swine herds, with most piglets receiving colostral antibodies to

HEV. Subsequent infection with the virus in the presence of these maternal antibodies does not result in clinical disease (2). Clinical disease may occur if piglets do not receive adequate colostral protection. Disease severity may also be related to the virulence of the specific strain of virus (2).Previous reports of HEV indicate neurological disease and/or a vomiting and wasting syndrome, usually in piglets less than three weeks of age. However, HEV has been reported in pigs up to 50 days of age (2). Several factors may explain the occurrence in older pigs in this herd. First, these are herds of relatively high health status that purchase gilts from external sources. There may be sub-populations of gilts that have not previously been infected with the virus, and colostrum from these naive dams would not contain protective levels of antibody to HEV. Offspring from these gilts would be susceptible to disease after entry to a contaminated nursery. Second, neonates which receive a low but protective level of maternal antibody may become susceptible as the antibody titer declines. Finally, because all piglets leave the breeding facility at an early age, there may not be adequate circulation of virus in the breeding herd to ensure a continuing high level of antibody in colostrum from older sows. **Control of the disease in this herd will focus on identifying groups of gilts and/or sows with low antibody titers, and implementing management changes to ensure that these animals are exposed to the virus prior to farrowing, to ensure that all dams pass protective colostral immunity to their offspring.**

Clinical differentiation of viral encephalomyelitis and bacterial meningitis may be difficult. The histological findings are usually characteristic: neutrophil-rich meningitis is often seen with bacterial meningitis, whereas multifocal infiltration of lymphocytes into the brain and spinal cord parenchyma is more typical of viral encephalomyelitis. Identification of the specific etiologic agent causing viral encephalitis is often difficult. Serology has been a recommended method, and has the added benefit of providing information useful in the control of the disease. Other diagnostic techniques may also be useful.

1. Rossow KD, Shivers JL, Yeske PE, et al. Porcine reproductive and respiratory syndrome virus infection in neonatal pigs characterized by marked neurovirulence. Proc Ann Mtg AAVLD 1998: 55. 2. Pensaert MB and Andries K. Hemagglutinating encephalomyelitis virus. In: Leman AD et al. (eds.), Diseases of Swine, 7th
ed., 1992: 268-273. 3. Meyvisch C, Hoorens J. An electron microscopic study of experimentally-induced HEV encephalitis. Vet Pathol 1978; 15: 102- 113.

## *Actinobacillus suis* in swine

**Drs. Gaylan Josephson, Nonie Smart, Beverly McEwen, Jiggs Gough**

*Actinobacillus suis (A. suis)*, a normal inhabitant of the porcine nasal tract, has been identified as a cause of septicemia and sudden death in suckling and recently weaned piglets and in grower/finisher pigs. Less commonly, it has been associated with outbreaks of arthritis, abortion, neurological signs, and skin conditions resembling erysipelas.

From January 1, 1991, to December 30, 1998, there were 359 isolates of *A. suis* from specimens submitted to the Veterinary Laboratory Services Branch/Animal Health Laboratory. Isolations were more frequent during the winter (Jan - Mar) months (Figure 1).

In a review of 81 cases from 1997 and 1998, *A. suis* was isolated from pigs of all age groups: 25 (30.4%) of the affected pigs were under 3 weeks of age, 18 (22.5%) were nursery pigs, and 25 (30.4%) were in the grow/finisher area. The ages of pigs from the 17 other submissions were not given. Pathology diagnoses included: - pneumonia - 28 cases - septicemia - 26 cases - arthritis - 7 cases - meningitis - 3 cases (including 1 bred gilt) - endocarditis - 1 case The organism was also cultured from vaginal swabs that were submitted for an infertility analysis. *Actinobacillus suis* has been identified as a

normal inhabitant of the vagina of sows (1).

Histories in suckling pigs typically mentioned sudden deaths in 2 or more pigs from the same litter. Petechial hemorrhages were often noted throughout the lungs, with larger diffuse areas of hemorrhage and/or edema also noted - *A. suis* organisms were isolated in pure cultures from lungs, and occasionally from brain, spleen and kidney in 19/25 cases. Histological examination of tissues confirmed the presence of a septicemia in these cases. In several submissions with a history of swollen legs and/or joints, *A. suis* was recovered from the fibrinopurulent joint fluid and from swollen, hemorrhagic lymph nodes.

In suckling pigs with a diagnosis of septicemia/pneumonia (19 of the 25 submissions of piglets in this age group), the organism was usually recovered in pure culture, but was occasionally isolated in mixed cultures with *Bordetella bronchiseptica, Streptococcus suis* or *Pasteurella multocida.*

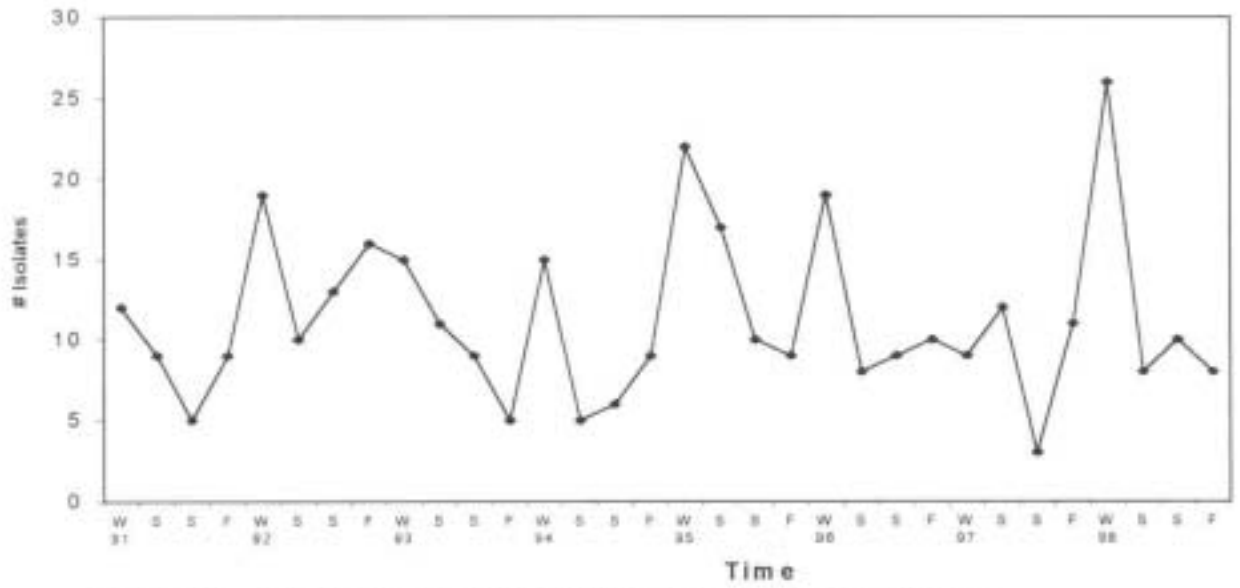


**Figure 1. Seasonal occurrence of *Actinobacillus suis* isolates from swine, 1991-1998**

Sudden unexpected death was the most common history in pigs in the grow/finisher stage. Gross findings commonly included blood-tinged nasal discharge, and areas of hemorrhagic, necrotizing pneumonia, often with extensive fibrinous pleuritis.

In post-weaning or older pigs (n = 43), *A. suis* was isolated in pure culture from 18 (42%). It was isolated in combination with *Streptococcus suis* on 11 occasions (26%), with *Pasteurella multocida* 3 times (7%), and with *Streptococcus suis* and *Pasteurella multocida* 10 times (24%).

***Actinobacillus suis* must be considered as a possible agent in cases of sudden death in pigs of all ages.** In addition, it has been involved in several other syndromes, including arthritis and meningitis.

1. Sanford SE. In: Leman AD, et al, eds. *Diseases of Swine,* 7th ed. 1992: 633-636.

# SMALL ANIMALS

### Lead poisoning in a dog

***Dr. Brent Hoff, Mr. Nick Schrier, AHL Dr. Lisa Carioto, OVC VTH Dr. Melanie Grein, Durham***

An eight-year-old, male, Lhasa Apso dog was presented to the Veterinary Teaching Hospital of the Ontario Veterinary College with a 7-day history of gastrointestinal signs, including anorexia, vomiting, painful cranial abdomen, and diarrhea. The dog also had very mild neurologic signs that consisted of restlessness and frequently getting up and lying down. No blindness or seizures had been noted.

Laboratory analysis revealed a mild, normocytic, normochromic anemia with many nucleated red blood cells and a few red cells with basophilic stippling. Radiography revealed a mass in the stomach. A small piece of metal (3.2 g, 1 cm X 1.5 cm X 0.3 cm, irregular borders, source unknown) was removed from the stomach by endoscopy and identified in the AHL toxicology section as pure lead by elemental composition analysis (X-ray diffraction).

The lead concentration in an EDTA blood sample, taken at the time of endoscopy, was 0.87 mg/L (acceptable level < 0.1 mg/L). In a sample taken a week later, after treatment with calcium EDTA, blood lead had decreased to 0.39 mg/L. By the second week, the dog had improved clinically and went on to make a complete recovery.

Lead poisoning causes many deaths in animals every year in Ontario, and is one of the most common toxicoses encountered in domestic animals, especially in cattle. Over the past five years, we have had 87 requests for lead analysis in dogs and cats, and 18 samples have contained toxic levels of lead. The Animal Health Laboratory offers 24 hr turnaround time on blood lead analysis ($20/sample) on samples as small as 0.2 mL, e.g. birds.

## HORSES

### Equine abortions due to leptospirosis in eastern Ontario

***Dr. Jan Shapiro, AHL-Kemptville Dr. John Prescott, OVC Pathobiology Dr. Garth Henry, Russell***

In late November 1998, two Standardbred equine fetuses, aborted in the 3rd trimester of gestation, were submitted to the Animal Health Laboratory in Kemptville for necropsy. The fetuses were from 2 herds on farms located 5 km apart in eastern Ontario. There was no direct contact between horses in the 2 herds, which each consisted of 4-5 Standardbred mares. There was no water source shared between herds. Mares in one herd had daytime access to large fields, but were housed at night; the other herd was always outdoors. Horses in both herds were fed a commercial grain mix and dry hay. Mares were routinely vaccinated prior to breeding, and also were given a series of killed intramuscular equine herpesvirus vaccines during pregnancy. Fetal and placental lesions for both cases were similar. The allantochorion showed severe, acute to subacute necrosis and neutrophilic to mixed inflammatory cell infiltrates in villi and stroma;

vasculitis, vascular thrombi and mineralization were also observed. Fetal lesions consisted of mild acute lymphocytolysis in spleen and lymph nodes, mild lymphocytic and plasmacytic portal infiltrates in liver, mild meningeal or ependymal perivascular infiltrates in brain and, in one fetus, mild acute renal cortical tubular necrosis. Silver stains showed large numbers of tangled spirochetes compatible with leptospires in inflamed placental tissue, including blood vessel walls. Spirochetes were not demonstrated in fetal kidney.

Routine bacteriology, mycology, and virus isolation tests of fetal tissues and placenta were negative. Sera from the 2 aborting mares were tested for leptospiral antibodies using the microagglutination plate

test; they had titers to L. pomona of 1:5120 and 1:2560, to *L. grippotyphosa* of 1:1280 and 1:160, and to *L. bratislava* of 1:640 and 1:5120 respectively. **These titers were interpreted as indicating recent exposure to Leptospira, and supported the diagnosis of leptospiral abortion based on histopathology and silver stain results.** Contact mares on both farms showed no clinical signs of leptospirosis either before or after the abortions. As a precaution however, they were treated prophylactically with oxytetracycline intravenously at a dose of 6 mg/kg SID for 7 days; there have been no subsequent leptospiral abortions to date.

Leptospiral infection as a cause of abortion in horses has received increased attention in the past few years, with reports of series of cases from Kentucky (1), Louisiana (2), and Ontario (3).

There is a high prevalence in Ontario horses of antibodies to serovar bratislava, for which horses are thought to be a maintenance host (4), but abortions are most commonly associated with serovar pomona. The high titers to 3 common serovars make it difficult to determine on the basis of a single test which serovar(s) was responsible for the infections we report here. We suggest that it was pomona, since this showed the highest titers; the other titers may have been paradoxical reactions which occur early in leptospiral infection. Exposure of mares to serovar pomona could be through direct contact with cattle, swine, rodents or wildlife, or exposure to urine from these animals; exposure to grippotyphosa might follow direct or indirect encounter with rodents or other wildlife, including raccoons. Infection of dogs with serovar *grippotyphosa* may be increasing in Ontario, possibly as a result of widespread infection in raccoons (5) . In the herds reported here, there was no known exposure to cattle or swine, but pasturing would have provided opportunities for contact with wildlife urine. The unusually long and mild autumn in eastern Ontario in 1998 may have contributed to increased opportunities for pasture interaction between grazing horses and wildlife.

Diagnosis of this abortion would have been difficult if the placenta had not been submitted, since histological demonstration of leptospires in fetal kidney is inconsistent, and many fetuses have no leptospiral antibodies in body fluids. **This report emphasizes the continuing role of leptospires in animal disease, including abortion in horses, and the likely need for an approved leptospiral vaccine for this species.**

1. Poonacha KB et al. Leptospirosis in equine fetuses, stillborn foals and placentas. Vet Pathol 1993; 30: 362-369 2. Hodgin EC, Miller DA, Lozano F. Leptospira abortion in horses. J Vet Diagn Invest 1989; 1: 283-287 3. Wilkie IW, Prescott JF, Hazlett MJ, Maxie MG, van Dreumel AA. Giant cell hepatitis in four aborted foals: a possible leptospiral infection. Can Vet J 1988; 29: 1003-1004 4. Kitson-Piggot AW, Prescott JF. Leptospirosis in horses in Ontario. Can J Vet Res 1987; 51: 448-451 5. Hrinivich K, Prescott JF. Leptospirosis in 2 unrelated dogs. Can Vet J 1997; 38: 509-510

## Sudden increase in foal stillbirths due to equine herpesvirus

***Drs. Tony van Dreumel, Susy Carman, Jeff Caswell, Marg Stalker, Beverly McEwen***

**Equine herpesvirus (EHV) has been diagnosed as a cause of stillbirth in four Thoroughbred and one Standardbred near-term foals at the AHL-Guelph laboratory during the past two weeks.**

This is a marked increase in EHV-related abortions/stillbirths compared to recent years:

| | ‘92 | ‘93 | ‘94 | ‘95 | ‘96 | ‘97 | ‘98 | ‘99* |
|---|---|---|---|---|---|---|---|---|
| | | | | | | | | |

| n | 5 | 6 | 13 | 5 | 6 | 0 | 2 | 5 |
|---|---|---|---|---|---|---|---|---|

***first 6 weeks of 1999; n = number of stillbirths/abortions**

Gross lesions consisted of congested firm lungs, swollen edematous thymuses, 1-mm grey foci of necrosis in the liver, and prominent splenic lymphoid follicles. All of these lesions were not necessarily present in each foal. Microscopic lesions included necrotizing bronchiolitis, alveolitis, lymphocytolysis in the thymus and splenic follicles, and focal areas of necrosis in the liver and adrenal gland. Typical herpesvirus inclusions were evident in bronchiolar epithelial cells and in hepatocytes. Four foals were tested and found positive for EHV 1/4 (equine abortion virus / equine rhinopneumonitis virus) using the recently introduced direct fluorescent antibody (FA) test. Lung and thymus were the tissues most commonly positive using this test. Virus isolation tests are in progress to determine the EHV type (1 or 4) involved. Most equine herpesvirus abortion cases are due to EHV-1. To avoid abortion storms, all aborted fetuses should be submitted for postmortem examination and the results used to quickly initiate any required management changes. **A diagnosis of EHV infection should alert the farm to the potential for an ensuing EHV epidemic.** Epidemics may follow immediately or be delayed for several months and include both abortion of fetuses and pneumonia in newborn foals. Vaccination, separation of the mares into small groups, isolation of aborting mares with disinfection of the hindquarters to remove virus-infected fetal fluids, prompt removal and incineration of the virus- infected fetus and placenta, restriction of personnel, and disinfection of premises, clothing and handling equipment should be encouraged to decrease horizontal transmission. Since virus-negative aborted fetuses, without diagnostic histological lesions, can occur as a consequence of vasculitis of the maternal uterine endometrium, rather than virus invasion of the fetus, it may be prudent to immediately treat all abortions as due to EHV to reduce the devastating consequences of an EHV abortion storm. **To help avoid EHV abortion, regular vaccination of pregnant mares should be encouraged.** Owners should be made aware that vaccines are not fully effective, especially when mares are stressed. Pregnant mares should be managed to reduce stress, so that recrudescence of latent EHV within the lymphoreticular system is minimized. Mares should be placed in small uncrowded groups as soon as they are checked pregnant. These groups should be maintained without additions or mixing, especially in the latter part of parturition. Mares should be allowed to foal together in the barn in which they are normally housed, rather than being moved through various barns prior to foaling.

1. Bryans JT, Allen GP. Herpesviral diseases of the horse. In: Wittman G, ed. *Herpesvirus Diseases of Cattle, Horses and Pigs.* Kluwer Academic Publishers, Boston. 1989; 176-229.

2. Carrigan M, Cosgrove P, Kirkland P, Sabine M. An outbreak of equid herpesvirus abortion in New South Wales. Equine Vet J 1991; 23: 108-110.

Animal Health Laboratory Accreditations:
American Association of Veterinary Laboratory Diagnosticians (AAVLD) (lab system)
Thyroid Registry of the Orthopedic Foundation for Animals Inc. (OFA) (thyroid function)
Canadian Food Inspection Agency (CFIA) (EIA)
Canadian Association of Environmental Analytical Laboratories (CAEAL) (metals)

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