UNIVERSITY of GUELPH LABORATORY SERVICES
AHL Newsletter
ANIMAL HEALTH LABORATORY GUELPH KEMPTVILLE RIDGETOWN

Volume 4, Issue 2

In this issue:

What's happening at the AHL?

Retirements:

- **Ev Grift**, supervisor of the AHL clinical pathology section, retired effective May 31, after 27 years of service in the OVC and 3 yrs with the AHL.
- **Dennis Thivierge**, technician in the AHL-Kemptville lab, retires May 31, after 26 yrs of service

in VLSB and 3 yrs with the AHL.
- **Vivian Martineau**, customer service representative in the AHL-Kemptville lab, retires June 30, after 25 yrs of service in VLSB and 3 yrs with the AHL.

Dr. Peter Lusis will transfer to the AHL-Guelph lab, effective June 1, as a veterinary pathologist, mammalian/avian. Dr. Lusis has 29 yrs combined service with VSB/VLSB/AHL in Kemptville.

Dr. Josepha Delay joins the AHL as a veterinary pathologist, mammalian, on June 1. Dr. Delay practiced in New Brunswick for 7 years after graduation from OVC in 1988, and earned her DVSc from OVC in 1999 in anatomic pathology.

Inserted in this newsletter is a color poster on *"Packaging and shipping lab submissions*". Please feel free to post in your shipping area. The handout is also available on our website at **http://ahl.uoguelph.ca**

The AHL successfully passed the recent audit of the American Association of Veterinary Laboratory Diagnosticians (AAVLD) and has been **fully accredited** as a full-service lab for all species, for another 5 years.

Updates to the May 1, 2000 **Fee Schedule**:

1. **Acute/convalescent paired serology** charges apply only to HI and VN tests conducted on the pair at the same time. ELISA and AGID tests are done on single samples, and each test is charged separately.

2. We no longer offer **flow cytometry** on a routine basis. Please consult the Clinical Immunology section for quotes on research samples.

Change in format of LSD client statements

In response to requests from our clients, Lab Services will be changing the format of our monthly client statements to resemble a "credit card" style statement. **Effective for the month ending**

May 31, 2000, customer statements will be issued in a "balance forward" format, which gives the total of all the outstanding transactions from prior months. All transactions that have been posted in the current month will be shown on the statement, i.e. all invoices, payments, credit notes, debit notes, etc.

Possible delays in US referral testing

We received notification from the courier customs broker at the US border that, effective immediately, new processes are in place. We anticipate that most samples won't be held up for long. However, in some cases, the researcher testing our samples must supply more information. *If we experience problems we will notify the clinics involved.* We apologize for any inconvenience to our clients.

Centralization of the Animal Health Laboratory

***Grant Maxie** DVM PhD Diplomate ACVP, Animal Health Laboratory-Guelph*

In response to strategic planning consultations with stakeholders over the past 18 months, and in order to

better serve you, our client, the Animal Health Laboratory (AHL) made the business decision to centralize testing activities at the AHL lab in Guelph as of June 1, 2000. We believe that this increase in the breadth and depth of services for testing, consultation, and surveillance will meet the needs of our chief clients and stakeholders, namely veterinarians and livestock and poultry producers. The budget of the AHL is not being cut, but some positions are being re-allocated to Guelph from the AHL labs at Ridgetown and Kemptville.

Services will be expanded at AHL-Guelph through increased technical/clerical staffing in specimen reception, anatomic pathology, histotechnology, immunology, bacteriology, and clinical pathology. The results will include:

- improved turnaround times on critical analyses, e.g. clinical pathology and bacteriology,
- extended hours of service,
- increased consistency of testing, and
- enhanced test development.

New professional positions in Guelph include

- a *mammalian/ avian pathologist*,
- a *microbiologist/ immunologist*, and
- a *swine surveillance specialist/pathologist* (currently at Exeter).

To facilitate the improved services offered from the Guelph AHL facility, **the AHL-Ridgetown lab will close May 31, 2000**. Beginning June 1, clients of the AHL-Ridgetown facility should follow these procedures:

- All samples formerly submitted to Ridgetown will now be submitted directly to Guelph. AHL-Guelph will continue to accept 'incoming collect' samples sent via Purolator. Please use the University of Guelph account # 0966901.
- Given the possible challenges in shipping intact large animal carcasses to Guelph, the AHL is conducting necropsy update sessions for veterinarians on request.
- For further information on sample submission, please contact AHL-Guelph sample reception at 519-824-4120, ext 4518 or 6071 (Ms. Linda McCaig, supervisor).

As part of the centralization, **the AHL-Kemptville location will be reduced in size effective May 31, 2000.** The Kemptville lab will continue to accept animals for gross post-mortem - *please call ahead* (613-258-8320). **Dr. Jan Shapiro**, who is bilingual, will provide necropsy services, coordination of results on these PM cases, and lab outreach/disease surveillance for veterinarians in eastern Ontario. She will be assisted by **Mr. Alan Darch** as prosector/client service representative.

- Clients who already ship specimens to AHL by courier should now ship directly to Guelph. AHL-Guelph will continue to accept 'incoming collect' samples sent via Purolator. Please use the University of Guelph account # 0966901.
- For those who drop off specimens at the AHL-Kemptville lab and require further information on sample submission, please contact AHL-Kemptville at 613-258-8320, or AHL-Guelph sample reception at 519-824-4120, ext 4518 or 6071 (Ms. Linda McCaig, supervisor).

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CATTLE

Silage-associated abortion

Spoiled silage associated with beef cattle abortions

Peter Lusis *DVM MSc, Animal Health Laboratory-Kemptville;* ***James Ferrier*** *DVM, Perth, Ontario;* ***Peter Warder*** *DVM, Coldwater, Ontario.*

Over 3 weeks, the first 4 cows to calve in a ~30 cow, fully vaccinated, natural-bred, beef herd aborted near term or delivered stillborn calves. The first two fetuses were not examined, but the next two were submitted to the AHL-Kemptville laboratory for necropsy.

One fetus was autolysed with no gross lesions evident, but on histopathology, there was extensive hepatic necrosis, with many rod-shaped bacterial colonies in lesions and blood vessels in various tissues. *Listeria monocytogenes* bacteria were isolated from the lung and stomach contents. The other fetus had no visible gross lesions, but on histopathology, there was diffuse bronchopneumonia with mild focal hepatic fibrosis. *Bacillus licheniformis* bacteria were isolated from lung and stomach contents.

Serology from affected dams and the bull for *Neospora*, *Leptospira*, IBR and BVD was negative. The cows were in excellent body condition and did not appear to be ill at the time of abortions but retained their placentas. A layer of moldy, spoiled silage had been fed, presumably for some time, before the abortions. After the spoiled silage was removed from the ration, all cows delivered healthy calves.

Listeria monocytogenes **and** ***Bacillus licheniformis*** **abortions in cattle have both been associated with the feeding of spoiled silage.** Listeriosis is also a significant human health hazard, especially to pregnant or immunosuppressed people.

References

1. Atkinson E. Isolation of *Bacillus licheniformis* from silage associated with abortion in beef suckler herds. J Appl Bacteriol 1985; 59: 6.

2. Kirkbride CA. Laboratory Diagnosis of Livestock Abortion. 3rd Edition. Iowa State University Press. 1990: 33.

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POULTRY

Ontario broiler breeder, layer, and turkey disease summary for 1999

Brian Binnington *DVM DipPath Diplomate ACVP, Animal Health Laboratory-Guelph*

Yearly summaries of specific disease entities are produced from the AHL database of pathology

diagnoses made at the Guelph, Kemptville and Ridgetown AHL labs. The following are some highlights of the more frequently diagnosed conditions or diseases of broiler breeders.

Broiler breeders

Surveys of mortalities in breeder hens often demonstrated a variety of problems in any one submission of birds. Bacterial infections accounted for the majority of the morbidity and mortalities in these birds. **Septicemia** (60% due to *E. coli*), **arthritis** and **tenosynovitis** (*E. coli* and *Staphylococcus aureus*), **osteomyelitis, cellulitis, pododermatitis, pneumonia** and **peritonitis** were the more common inflammatory conditions seen in these hens. The release of yolks and eggs into the body cavity or ascending infection of the oviduct by *E. coli* bacteria with retention of eggs and yolks resulted in death due to peritonitis of birds in lay. Nutritional and metabolic diseases such as **hypocalcemia/osteomalacia** and **fatty liver/hemorrhage syndrome** caused occasional in-lay deaths. Intestinal infections due to **coccidiosis** and **necrotic enteritis** due to clostridia bacteria were identified with greater frequency in cases of sudden mortality last year. The most frequently diagnosed neoplasms were **myelocytomas** that affected a variety of tissues including liver, spleen, bone, kidney and ovary. This neoplasm is associated with the J subgroup of the **avian leukosis** viruses. The incidence of this tumor appears to decreasing as the J virus eradication programs instituted in primary breeders is decreasing the incidence of this vertically transmitted virus in breeding stocks. However, the J subgroup of viruses can also be transmitted horizontally amongst chicks, therefore, eradication is not likely to occur quickly.

Layer chickens

Nutritional and metabolic problems continued to be causes of morbidity and mortality in laying birds. Calcium depletion resulting in **hypocalcemia/osteomalacia (cage layer fatigue)** and **fatty liver/hemorrhage syndrome** were the most frequently diagnosed conditions in laying birds. Calcium depletion occurred during peak production, however, in several flocks it was identified in young birds recently in production. Errors in replacement pullet management and nutrition were considered to be the initiating problem in these young birds. **Peritonitis** associated with internal release of yolks and eggs or due to an ascending *E. coli* oviduct infection were frequent findings in laying birds. Kidney disease characterized by **urate nephrosis/visceral gout** was not as frequent this past year. Dehydration is the most likely cause of this problem, however, high calcium - low phosphorus diets, and rarely infections by strains of infectious bronchitis virus, have been implicated in the past. Production drops associated with variant strains of **infectious bronchitis virus** were a significant problem in some flocks during the colder months. Cases of **coccidiosis,** with or without **necrotic enteritis** due to clostridia, were diagnosed more frequently. These enteric diseases usually occurred in young in-lay birds housed in battery cages, suggesting inadequate immunity to coccidia in the replacement pullets. **Hepatitis/splenomegaly (hemorrhagic, necrotizing hepatitis)** is a liver disease of uncertain cause that continued to occur in birds which are usually 35 weeks or older. The number of submissions with this form of liver disease has continued to decline over the last few years. Neoplastic diseases associated with **Marek's disease** virus or the **leukosis complex** viruses were identified less frequently than in previous years.

Turkeys

Mortalities in young turkey poults often reflected problems starting in the brooder areas with **starve-outs, litter-picking** and **dehydration** evident on postmortem examination. Bacterial contamination of the young poults during hatching, transport or placement resulted in frequent submissions with **omphalitis** and/or **yolk sacculitis.** *E. coli* and *Salmonella* sp. were the most frequent isolates from these bacterial infections. Variable antimicrobial susceptibility patterns were evident with isolates of multiple drug resistant *E. coli* and *Salmonella* being identified. Contamination of young poults by fungal spores (usually *Aspergillus* sp.) resulted in cases of **mycotic pneumonia** (brooder pneumonia). Bacterial

septicemia was usually due to *E. coli* (most often older than 2 weeks of age) and less frequently by *Salmonella* sp. (usually less than 3 weeks of age). Several flocks experienced increased mortalities, diarrhea, runting and rickets-like bone problems in poults 3-5 weeks of age. Enteritis in these birds was associated with a **corona-like virus,** bacteria (*Salmonella*), and coccidia in some birds. This virus has not shown cross-reaction with known turkey coronaviruses and further characterization of this corona-like virus is continuing. Bacterial respiratory disease was a significant problem in some flocks. ***Bordetella avium*** was isolated from a few cases of conjunctivitis, sinusitis and tracheitis **(turkey coryza).** *Bordetella* may have been involved in other respiratory disease outbreaks, however, it can be difficult to isolate when other bacteria are present in the sampled tissues. Severe respiratory disease in growing meat turkeys, as well as male and female breeders, was associated with **ORT (*Ornithobacterium rhinotracheale*)** bacteria. This organism is apparently increasing in frequency as a significant respiratory pathogen in Ontario turkeys.

Rickets-like bone disease (field rickets) that was associated with enteric diseases was identified occasionally in young birds. **Spontaneous turkey cardiomyopathy (round heart disease)** was a sporadic cause of death in young poults.

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Ornithobacterium rhinotracheale isolates from turkeys, 1998 and 1999

Marie Archambault *DMV MSc PhD,* ***Shelley Newman*** *DVM DVSc Diplomate ACVP,* ***Beverly McEwen*** *DVM MSc PhD Diplomate ACVP,* ***Brian Binnington*** *DVM DipPath Diplomate ACVP, Animal Health Laboratory-Guelph.*

Over the past two years, 56 isolates of *Ornithobacterium rhinotracheale* (ORT) were recovered from 19 poultry cases at the AHL bacteriology laboratory. In Ontario, ORT infections were usually characterized by respiratory signs (sneezing, coughing) and an increased mortality rate in turkey flocks. The AHL had no ORT isolations from Ontario chickens during this period. ORT is a recently discovered gram-negative bacterium. It was previously described as a *Pasteurella*-like organism, *Kingella* sp., Taxon 28 and is now classified in the rRNA superfamily V (1).

The first known isolation of ORT was made in Germany in 1981 from turkeys with respiratory tract infection (1) but the organism was not officially described until 1994 (2). Twelve distinct serotypes have been identified; of which serotype A is the most prevalent (3-4). ORT is now of worldwide distribution in commercial poultry and it is also found in wild birds. The infection can be transmitted horizontally by aerosol, as well as vertically through eggs. There is no known public health significance.

Airsacculitis and pneumonia are the most common features of infection with ORT. Therefore, lungs and air sacs are the best tissues from which to isolate the organism. Laboratory isolation is done on blood agar and identification is performed using a set of biochemical tests (3-4).

At the AHL, antimicrobial susceptibility testing was performed upon request on 15 ORT isolates using the disk diffusion method. Interpretation of the *in vitro* susceptibility findings should be done with caution because standards have not been established for this organism. *Pasteurellaceae* NCCLS guidelines have to be used. **Of these isolates, 9 (60%) have been found susceptible *in vitro* to ampicillin, 12 (86%) to erythromycin, and 10 (67%) to tetracycline; 13 (87%) isolates were resistant to trimethoprim-sulfamethoxazole and 10 (67%) to cephalothin. Variable susceptibilities**

were observed with gentamicin and penicillin (Table 1).

Therapeutic treatment of ORT infection can be difficult because acquired resistance against the regular antibiotics is very common (4). In suspected cases of ORT, identification of bacterial isolates and antimicrobial susceptibility testing is

recommended. Treatment of ORT is then based on the recommendations of a practicing veterinarian following assessment of the farm situation. Antibiotics have been utilized as part of an ORT treatment program either singly or in a combination of oxytetracyclines in the water, chlortetracycline in the feed, or spectinomycin, ceftiofur, and penicillin by injection (3). **ORT infection in a turkey flock was recently reported in Ontario** and this outbreak responded well to treatment with neomycin-tetracycline in the water and oxytetracycline in the feed followed by penicillin G potassium in the drinking water (5). Vaccination with autogenous bacterins has been successful in reducing clinical signs, but success depends on the adjuvant used (4). Currently, there is no commercially available vaccine (3).

Table 1. Antimicrobial susceptibility testing of ORT isolates during 1998 and 1999.

Organism	*Antimicrobial agents and numbers of susceptible, intermediate and resistant isolates*																				
	AMP			*CEP*			*ERY**			*GEN*			*PEN*			*TET*			*TMS*		
ORT	*S*	*I*	*R*	*S*	*I*	*R*	*S*	*I*	*R*	*S*	*I*	*R*	*S*	*I*	*R*	*S*	*I*	*R*	*S*	*I*	*R*
	9	*3*	*3*	*2*	*3*	*10*	*12*	*1*	*1*	*5*	*6*	*4*	*6*	*4*	*5*	*10*	*3*	*2*	*1*	*1*	*13*

S = Susceptible, I = Intermediate, R = Resistant

*Fourteen ORT isolates were tested for erythromycin

AMP, Ampicillin; CEP, Cephalothin; ERY, Erythromycin; GEN, Gentamicin; PEN, Penicillin; TET, Tetracycline; TMS, Trimethoprim- sulfamethoxazole.

References

1. Hinz KH, Blome C, Ryll M. Acute exudative pneumonia and airsacculitis associated with *Ornithobacterium rhinotracheale* in turkeys. Vet Rec 1994; 135: 233-234.

2. Vandamme P, Segers P, Vancanneyt M, et al. *Ornithobacterium rhinotracheale* gen. Nov., sp. nov., isolated from the avian respiratory tract. Int J Syst Bacteriol 1994; 44: 24-37.

3. Chin, RP, Droual R. *Ornithobacterium rhinotracheale* infection. In: Calnek BW ed. Diseases of Poultry, 10th ed. Ames, Iowa: Iowa State Univ Press, 1997: 1012-1015.

4. Van Empel PCM, Hafez HM. *Ornithobacterium rhinotracheale*: a review. Avian Pathology 1999; 28: 217-227.

5. Abdul-Aziz TA, Weber L. *Ornithobacterium rhinotracheale* infection in a turkey flock in Ontario. Can Vet J 1999; 40: 349-350.

We thank the bacteriology staff for their technical assistance.

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SWINE

PRRS outbreak in a partially vaccinated herd

Cathy Templeton *DVM,* ***Janet Alsop*** *DVM, Listowel, Ontario;* ***Gaylan Josephson*** *DVM DipPath,* ***Gary Thomson*** *DVM MSc,* ***Marie Archambault*** *DMV MSc PhD, Animal Health Laboratory-Guelph*

Between April 12 and 19, 2000, 40 sows aborted in a 1600 sow, farrow-to-early-wean herd. Since that time, a further 60 sows have aborted. Most of the abortions have been late term, i.e. 7 - 14 days before due date, although there were some abortions in sows due in 30 days. Sows were febrile and off feed when they aborted.

There were a few mummified fetuses or stillbirths in the litters >109 days of gestation, but the piglets appeared to be weak and did not suckle well. There was no evidence of "thumping" pigs. Initially, most of the late term aborting and farrowing sows had milk. In the last 3 weeks, approximately half of the litters born at term have starved out and die. The other 50% of litters appear normal.

The sow herd was diagnosed as being "unstable" for PRRSV in December 1999, based on routine monitoring of sera using the IDEXX antibody ELISA. Prior to that time, gilts had been vaccinated against PRRSV twice prior to entry and boostered on entry, but the sow herd had not been routinely vaccinated. On February 4, all sows bred between Dec 15, 1999 and Feb 4, 2000 were vaccinated against PRRSV. Since that time, sows to be retained and re-bred have been vaccinated against PRRSV at weaning. They have been routinely vaccinated for leptospirosis/parvovirus/erysipelas at weaning. All gilts are vaccinated twice, 3 weeks apart for lepto/parvo/erysipelas after arrival at the farm. There had been no history of PRRS in this herd since it was established in 1994-95.

Diagnostic testing has ruled out mycotoxins in the feed, and other major causes of abortion, such as leptospirosis and parvovirus infection. Significant histological lesions were not observed in tissues from 17 fetuses from 4 aborted litters nor were pathogenic bacteria isolated from these fetuses. Aborted sows have shown dramatic increases in PRRSV serum antibody levels, with the average mean of 9 acute samples being 0.772, and that of the corresponding convalescent samples being 2.208. PCR has identified the presence of field strain-like PRRSV in fetal tissues and sera.

Losses in the herd are continuing. The rate of abortions has slowed slightly, but farrowing room losses continue. Conception rates, as diagnosed by ultrasound, are only 50% of previous levels.

To date, piglets in the off-site nurseries have not shown clinical signs of the respiratory form of PRRS, nor have there been signs in animals in the gilt barn, which is connected to the sow barn by a passageway. **Farm biosecurity is considered to be good, and the source of infection is not known**. It is probable that reproductive-associated losses related to PRRSV may last as long as 16 weeks.

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HORSES

Equine abortion update

Beverly McEwen *DVM MSc PhD Diplomate ACVP,* ***Tony van Dreumel*** *DVM MSc Diplomate ACVP, Animal Health Laboratory-Guelph;* ***Bob Wright*** *BSc (Agr) DVM, Veterinary Science, OMAF, Fergus, Ontario.*

During the last year, there was a 37% increase in equine abortions submitted to AHL pathology compared to 98/99. The 89 cases were submitted from 85 owners. Fourteen breeds were represented, with Standardbred (41.9%) and Thoroughbred (30.2%) the most frequently submitted.

Infectious abortion collectively comprised 37% of the total cases, followed by non-infectious causes

of abortion, primarily umbilical torsion (13.6%).Since 1999, there have been 7 submissions of edematous placenta, which was accompanied in 3 cases by an aborted or stillborn foal. **Placental edema has been associated with fescue toxicity which is a form of ergot alkaloid toxicity.** Ergot alkaloids may be produced by certain fungal endophytes which infect a variety of forage grasses and grain. These alkaloids exert toxic effects primarily on the reproductive tract of mares and have been associated with prolonged gestation and thickened edematous placentas. Foals may be small, weak or stillborn (1). **Submission of foals, entire placenta, and providing placental weight are important for diagnosis.** Further investigations of this problem in Ontario are ongoing.

Reference

1. Bacon, CW. Fungal endophytes, other fungi and their metabolites as extrinsic factors of grass quality. In Fahey G (ed.) Forage, Quality, Evaluation and Utilization. American Society of Agronomy, Inc., Madison WI 1994: 336.

Table 1. Pathology diagnoses of equine abortion/placental lesions, 95/96 to 99/00.

	95/96	96/97	97/98	98/99	99/00
# Cases submitted	**81**	**69**	**46**	**64**	**89**
Pathology diagnoses (N, %)					
EVR	**8 (9.9%)**	**0**	**1 (2.2%)**	**9 (14.1%)**	**8 (8.9%)**
Non-viral infectious abortion combined	**15 (18.5%)**	**13 (18.8%)**	**7 (15.2%)**	**17 (26.6%)**	**25 (28.1%)**
Streptococcus zooepidemicus	**7**	**3**	**2**	**1**	**5**
Staphylococcus aureus	**0**	**0**	**1**	**0**	**3**
Streptococcus equisimilis	**0**	**0**	**1**	**2**	**0**
Ehrlichia risticii	**1**	**1**	**0**	**0**	**0**
Leptospira spp.	**0**	**0**	**0**	**3**	**0**
Miscellaneous bacteria/fungi	**1**	**7**	**2**	**2**	**8**
Mycotic	**0**	**0**	**1**	**1**	**0**
Infectious, etiology unknown	**6**	**2**	**0**	**0**	**2**
Placentitis	**0**	**0**	**0**	**8**	**7**
Umbilical torsion	**3 (3.7%)**	**14 (20.3%)**	**10 (21.7%)**	**8 (12.5%)**	**12 (13.5%)**
Placental edema* (presumptive alkaloid-associated)	0	0	**1 (2%)**	**4 (6.3%)**	**3 (3.4%)**
Placental insufficiency/ hemorrhage	**0**	**0**	**1**	**1**	**0**

Placental mineralization	**0**	**0**	**1**	**1**	**0**
Placental adenomatous/cystic hyperplasia	**0**	**0**	**1**	**0**	**1**
Dystocia/stillbirth	**6**	**1**	**0**	**2**	**5**
Congenital anomalies	**5**	**1**	**0**	**0**	**0**
Idiopathic	**44 (54.3%)**	**40 (58.0%)**	**28 (60.9%)**	**24 (37.5%)**	**38 (42.7%)**

* 3 cases submitted with stillborn or aborted foals; other cases placenta submitted with

history of weak (2), premature (1) and normal (1) foals.

Ionophore toxicosis in a group of 21 horses and donkeys

Brent Hoff *DVM DVSc DipTox,* ***Tony van Dreumel*** *DVM MSc Diplomate ACVP,* ***Cam Lyttle****, Animal Health Laboratory-Guelph;* ***John Pierce*** *DVM, Earlton, Ontario.*

A northern Ontario farm-based tourist operator found several of his donkeys and horses lethargic and depressed after they were fed a high phosphorus mineral mix. Four of 21 horses, donkeys and mules died within a few days. The first donkey that died was necropsied by the referring veterinarian. Macroscopic lesions consisted of marked pulmonary edema, early hepatic necrosis and myocardial degeneration. There was marked hyaline degeneration and fragmentation of myocardial fibers without evidence of dystrophic mineralization. There was passive congestion of the lung and liver compatible with left and right heart failure. Ionophore toxicity or vitamin E/selenium deficiency was suggested based on the histologic lesions.

A sample of the mineral mix submitted to the AHL toxicology section was negative for arsenic, copper and lead. The tissue selenium levels were within AHL adequate levels. A sample of the mineral mix contained 2900 µg/g of monensin (Rumensin). Horses and donkeys are highly susceptible to ionophore toxicosis. The LD_{50} for horses (most susceptible) is 1-2 mg/kg.

The mineral mix was a cattle mix and monensin was not listed as one of the ingredients. Signs of general cardiovascular collapse, weakness, decreased exercise tolerance; cyanosis, recumbency and death may be seen in susceptible animals. Death may occur during an acute toxic episode or weeks later.

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COMPANION ANIMALS

Streptococcus canis necrotizing fasciitis in a dog

Marg Stalker *DVM PhD Diplomate ACVP,* ***Marie Archambault,*** *DVM PhD, Animal Health Laboratory-Guelph;* ***John Prescott*** *VetMB PhD, Department of Pathobiology, Ontario Veterinary College, Guelph.*

A five-year-old neutered male German Shepherd dog was examined for apparent back pain, tentatively diagnosed as a strain injury. The dog was re-examined 11 days later with weakness, ataxia, pain over the dorsal thorax, and pyrexia; a CBC showed marked neutrophilia. The dog was prescribed enrofloxacin

and ketoprofen. Following a brief improvement in clinical signs, the dog's condition rapidly deteriorated, and five days later he was examined and found to be lethargic, depressed, in right lateral recumbency, unable to rise. An area of swelling in the left axilla was detected. The dog went into cardiopulmonary arrest and died during examination.

On post-mortem examination, there was an extensive area of subcutaneous hemorrhage in the left axilla, with exudation of fibrin and necrotic debris. The lesion extended through subcutaneous tissue into fascial planes, involving the musculature of the dorsal and lateral thorax and left shoulder. **Histology confirmed a locally extensive cellulitis, myositis with necrosis, and numerous coccoid bacteria. Pure cultures of *Streptococcus canis* were isolated from the subcutaneous tissues and filtering organs.** There was also histologic evidence of septicemia, with pulmonary thrombosis, and infarction. A diagnosis of necrotizing fasciitis with streptococcal toxic shock was made.

Streptococcus canis is part of the resident flora of skin and mucosa in dogs and cats, and may cause various opportunistic infections (1). Streptococcal toxic shock syndrome and necrotizing fasciitis caused by *S. canis* have been reported in dogs since the mid-1990's (2,3). In dogs with necrotizing fasciitis, the site of infection is often the leg, neck or ventral thorax, associated with minor dog bites, skin infections or trauma, although a predisposing injury may not be present in many cases (1). **Typical clinical signs include intense pain, localized heat and swelling**, sometimes progressing to necrosis and sloughing of overlying skin. Dogs with necrotizing fasciitis often develop severe systemic disease, with hypotension and septicemia.

Isolates of *S. canis* are usually susceptible to ampicillin, cephalothin, clindamycin, or amoxicillin/clavulanic acid. In this case, as in other reported cases, the dog was treated with a non-steroidal anti-inflammatory drug in combination with enrofloxacin prior to exacerbation of clinical signs (2). It appears that enrofloxacin, with or without anti-inflammatory drugs, may somehow potentiate invasive streptococcal infections, even though some isolates may be susceptible *in vitro* to enrofloxacin. The mechanism of this change, and study of virulence attributes of *S. canis* isolates continues to be the topic of active research in the laboratory of Dr. Prescott.

References

1. DeWinter LM, Low DE, Prescott JF. Virulence of *Streptococcus canis* from canine streptococcal toxic shock syndrome and necrotizing fasciitis. Vet Micro 1999; 70: 95-110.

2. Prescott JF, Miller CW, Mathews KA, Yager JA, DeWinter LM. Update on canine streptococcal toxic shock syndrome and necrotizing fasciitis. Can Vet J 1997; 38: 241-242.

3. Miller CW, Prescott JF, Mathews KA, Betschel SD, Yager JA, Guru V, DeWinter L, Low DE. Streptococcal toxic shock syndrome in dogs. J Am Vet Med Assoc 1996; 209:1421-1426.

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Cyclodiene organochlorine insecticide toxicosis in a dog

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A three-year-old, male soft-coated Wheaten Terrier was hospitalized with drooling, tremors, ataxia, vomiting and seizures. An EDTA blood sample was submitted to the AHL toxicology section for

determination of cholinesterase activity, as some of the clinical signs were compatible with organophosphate (OP) poisoning.

No cholinesterase inhibition was present in the blood sample. A source sample and a sample of stomach content were analyzed using a GC/MS (gas chromatography/mass spectrometry) screen. A compound was identified as endosulfan in both samples. The dog was treated with supportive therapy and diazepam for a possible OP neurotoxicity. The recovery was complete after 24 hours of treatment.

Endosulfan is a cyclodiene organochlorine insecticide (OC; chlorinated hydrocarbon) compound or one of the chlorinated hydrocarbon insecticide class. Others include: aldrin, dieldrin, chlordane, endrin, heptachlor, toxaphene, and lindane. **These compounds are insecticides or acaricides, used for the treatment of the aerial vegetative parts of plants, soil, seeds and animal dips.** The mechanism of neurotoxic action is by competitive inhibition of the binding of GABA at its receptor.

Although not encountered as commonly today as several years ago, occasional exposures of small animals to OC insecticides still occur. In addition to acute toxicosis, residues in the fats of meat and milk are of primary concern in food animals.

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CIPHS UPDATE

Linking animal health and food safety data to human health

Beverly McEwen, Elroy Mann, Aaron Middleton, Frank Pollari, Murray Hazlett, Charlie Fulton, Joseph Odumeru, Mary Halfpenny, Gaston Annamunthodo, Stephen Fraser, Grant Maxie, Patricia Collins

Gaps in the current national surveillance infrastructure between food safety data, animal health data and the occurrence of human health events need to be closed. A collaborative project between the Laboratory for Foodborne Zoonoses, Health Canada the Laboratory Services Division, University of Guelph is amongst the first in North America to integrate information from food, animals and people to improve public health. The AHL and Food Microbiology Laboratory, Laboratory Services Division, University of Guelph were funded by the Health Infostructure Support Program (HISP), Health Canada, to develop a system that will accommodate food safety and animal health data for improved public health surveillance in Canada. The expected completion date of the project is September 2000.

Databases at the Animal Health Laboratory and Food Microbiology Section, Laboratory Services Division, University of Guelph and the Laboratory for Foodborne Zoonoses, Health Canada will be linked. Although these laboratories use distinct computer systems, the data will be integrated using the Laboratory Data Management System developed by the Canadian Integrated Public Health Surveillance System (CIPHS), Health Canada. It provides a way of linking laboratory data with epidemiologic data through local area networks or internet communication.

Emerging food-borne and zoonotic diseases are of increasing concern to health officials. The agricultural, veterinary and medical communities are grappling with issues such as antimicrobial resistance. This prototype pilot moves an integrated, comprehensive surveillance system from vision to reality. It will be possible to determine spatial and temporal relationships between the occurrence of human disease and pathogens in animals or food. Animals may be more effectively used as sentinels of

human disease, and policy makers will have more information to make evidence-based decisions, evaluate control strategies, identify research needs and facilitate planning.

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)

ISO 9002 registered (toxicology)

Comments? Suggestions?

If you have comments or suggestions, or would like to be added to, or removed from, the AHL Newsletter **mailing list**, please send a fax to **Ms. Helen Oliver** at 519-821-8072 or an E-mail to holiver@lsd.uoguelph.ca

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