

Mr. George Howlett retired, effective Nov. 26, 1999, after 30 years of service to OMAF and the

University. George worked for 19 years in the VSB/VLSB toxicology section, and for the last 11 years was the fieldperson in the Ontario Hatchery and Supply Flock Policy (OHSFP) program. We wish George well in his retirement. The new OHSFP fieldperson is Ms. Lynn Henry, who has worked in the specimen room/avian necropsy areas.

The renovations of our **molecular biology** facilities are now complete, and we are expanding our spectrum of PCR tests.

Christmas hours at the AHL: We are closed Mon, Dec 27, Tues Dec 28, Thurs Dec 30, Fri Dec 31. We will have a skeleton staff on Weds Dec 29 0830-1630; full staffing on Mon Jan 3. AHL -Guelph is available for emergency necropsies on all holidays but Christmas Day.

The third case of the mid-Atlantic strain of **raccoon rabies** was identified in September by CFIA, near Oxford Station, Ont. For current information on the point infection control program, see the Ontario Ministry of Natural Resources website at <u>www.mnr.gov.on.ca/mnr/rabies</u>

The AHL underwent an **accreditation site visit** in Oct by a team from the American Association of Veterinary Laboratory Diagnosticians (AAVLD), an event which occurs every 5 years. The VLSB/AHL has been successfully accredited by the AAVLD as a full-service diagnostic lab since 1994.

The AHL Toxicology section has implemented a new test for **cholinesterase activity**, used as a measure of exposure to cholinesterase inhibitors (organophosphates, carbamates).

The AHL Immunology section successfully passed the proficiency testing by USDA NVSL for **Johne's disease** IDEXX ELISA.

CATTLE

Mycoplasma bovis - passive hemagglutination testing

Tony van Dreumel, DVM MSc Diplomate ACVP, AHL-Guelph; Lois Parker, BSc, AHL- Guelph.

Mycoplasma bovis may cause mastitis in dairy herds, arthritis and pneumonia in both dairy and beef calves and decubital abscesses in dairy calves(1). **AHL-Guelph provides both culture of** *M. bovis* **and a test for antibodies in serum and joint fluid [passive hemagglutination test (PHA)].** The PHA test is used primarily to look for seroconversion in acute and convalescent serum samples in animals with pneumonia and arthritis. Fluid from arthritic joints may be submitted for both culture and the PHA test. Antibodies in the joint fluid may prevent the growth of the organism, but a positive titer would suggest that *M. bovis* was the infecting organism.

Fresh or frozen milk samples should be submitted from suspect mastitis cases. Swabs from decubital abscesses may be submitted in regular transport medium. The mycoplasma laboratory occasionally receives requests for serology on cows with reproductive problems - *M. bovis* is rarely associated with natural infections of the reproductive tract. *Ureaplasma diversum* is more commonly associated with such infections, resulting in granular vulvitis, infertility and abortions (2). The laboratory provides culture and identification of *Ureaplasma* sp., but no serological test.

References.

1. Kinde H, et al. *Mycoplasma bovis* associated with decubital abscesses in Holstein calves. J Vet Diagn Invest 1993; 5: 194-197.

2. Ruhnke HL. Mycoplasmas associated with bovine genital tract infections. In: "Mycoplasmosis in

Animals. Laboratory Diagnosis" ed. Whitford HW, Rosenbusch RF, Lauerman LH. pp56-62. Iowa State University Press, Ames, Iowa, First Edition, 1994.

Hepatic lipodystrophy in two Galloway calves

Murray Hazlett, DVM DVSc Diplomate ACVP, AHL-Guelph; Joel Rumney, BSc DVM, North Simcoe Veterinary Services, Midland, ON.

A 5-week-old Galloway calf was examined because of weakness, ataxia and lethargy. The animal was found to have a large firm liver and profound icterus. A second herdmate of similar age and signs was also examined. The owner reported having lost occasional calves over the past several years with a similar clinical picture. Tissues submitted for histopathology from both calves revealed extensive dissecting fibrosis with some cholangiolar hyperplasia and isolation of hepatocytes into islands of 1 to 40 cells. **Most hepatocytes contained a large lipid vacuole occupying 50-90% of the cytoplasm**. Liver copper and molybdenum levels where within normal limits.

The history and lesions seen in these two calves are similar to those reported in hepatic lipodystrophy of Galloway calves (1). In this report, calves born alive usually died between 2 and 4 months of age. Initially, affected calves were normal, but became lethargic and stuporous, and sucking attempts became weak. The small numbers of calves involved did not allow the investigators to determine if the disease was genetic; they could not rule out a storage disease.

This syndrome has been recognized in pedigree Galloway calves since 1965, however it has not, to our knowledge, been identified in North America.

Reference

1. Macleod NSM, Allison CJ. Hepatic lipodystrophy of pedigree Galloway calves. Vet Rec 1999; 144:143-145.

Congenital brain edema in two Hereford calves

Murray Hazlett, DVM DVSc Diplomate ACVP, AHL-Guelph; *Robert Tremblay*, DVM DVSc Diplomate ACVIM, Boehringer Ingelheim, Guelph, ON; *Alexander de Lahunta* DVM PhD, Cornell University, Ithaca, NY, USA; *Harold Kloeze*, DVM DVSc, Twin County Veterinary Services, Owen Sound, ON.

A producer purchased 24 Hereford cows; two of their calves had whole body tremors at birth. These tremors gradually diminished, but the calves performed poorly and either died or were euthanized before they were 6 weeks old. Two calves were submitted for post-mortem examination. One had a mesenteric torsion as cause of death, the second was euthanized. Both had mild asymmetry of the cerebellar folia.

Microscopic examination at all levels of the brain revealed generalized spongiosis of grey and white matter which was related to extensive astrocytic swelling in the grey matter. In the white matter, similar astrocytic swelling was present, along with significant hypomyelination. Virus isolation and immunoperoxidase testing for BVD virus were negative.

Numerous neurologic conditions are described in Hereford calves. These conditions can occur in grade cattle as well as purebred, particularly if there is inbreeding within the herd. The syndromes of hereditary neurologic disease described in Hereford cattle include: hereditary neuraxial edema (Cordy, 1969), inherited congenital myoclonus of polled Hereford calves (Blood and Gay, 1971), and shaker calf syndrome (Rousseaux, et al., 1985). In 1974, Jolly described congenital brain edema of Hereford calves (1) as an autosomal recessive disorder - lesions described in this paper best fit the clinical signs and

lesions seen in animals from this farm.

Reference

1. Jolly RD. Congenital brain oedema of Hereford calves. J Pathol 1974; 114:199-204.

Neosporosis

Peter Lusis, DVM MSc, AHL- Kemptville; **Andrew Peregrine**, BVMS PhD DVM, Ontario Veterinary College, Guelph, ON.

What is neosporosis?

This is a disease caused by *Neospora caninum*, a protozoan that closely resembles *Toxoplasma* and *Sarcocystis* spp. Sporadic infections have occurred in several species (including dogs, sheep, goats), but *Neospora* infection is most important as a cause of abortion in cattle.

How is it spread?

More than 95% of spread is by vertical transmission. Calves born to infected cows are often infected prenatally and remain infected for life, passing the infection on to many of their offspring. The dog is a definitive host, but there is no direct evidence from the field that they spread the infection. They may excrete oocysts for several days after ingesting infected tissue. Cats do not transmit *Neospora*, but wild canids, rodents, etc. have not been definitely ruled out.

How is it diagnosed?

Serologic tests are available, and seropositivity identifies infected animals, but the clinical significance of infection is yet to be determined. Many seropositive cows do not abort, however the incidence of *Neospora* abortion is generally significantly higher in seropositive animals. The presence of *Neospora* organisms in association with typical histologic lesions in fetal brain, liver, skeletal muscle, and heart is strongly supportive of *Neospora* as the cause of abortion.

Should seropositive cows be culled?

Seropositive cows remain infected for life, infect many of their offspring, and may be culled a few months earlier than seronegative cows for non-abortion related reasons, but culling and replacement costs may exceed benefits in many cases. Seropositive heifer calves should not be used for replacements if possible. However, some workers have suggested that seronegative animals may be more susceptible to horizontal transmission of the parasite. The culling decision thus remains controversial.

What can be done to control neosporosis?

Restrict access of dogs and other animals to aborted fetuses, placentas, and cattle feed and water. Serotest replacement heifer calves and prepurchase cows.

Is Neospora a public health risk?

There is serological evidence of exposure of humans to *N. caninum*, but the significance of human exposure to, and possible infection with, *N. caninum* is unknown.

References

1. Dubey JP. Recent advances in *Neospora* and neosporosis. Vet Parasitol 1999; 84: 349-367.

POULTRY

Botulism in poultry

Shelley Newman, DVM DVSc Diplomate ACVP, AHL-Guelph.

In the past six months, we have had reason to suspect botulism as a causative agent of neurologic signs in broiler flocks in Ontario with increased frequency. Its occurrence in confined broiler flocks has been previously documented, although originally it was seen most commonly in free- ranging and wild birds. The disease is the result of ingestion of preformed *Clostridium botulinum* exotoxin, and in poultry this is most commonly type C toxin. The toxin is produced under anaerobic conditions and at 10 - 47 degrees C. With high doses of toxin, the onset of clinical signs is rapid (hours) but can require several days with lesser doses.

When it occurs in confined flocks the question is always: "what is the source of the toxin?"

Type C organisms readily grow in the gastrointestinal tract of chickens but also in wild birds. As a result, the organism is commonly found in the environment around poultry farms. The main factor enhancing spread of the organism is the resistance of the spores to environmental insult and thus their continued persistence. Botulism is thought to occur typically as a result of ingestion of preformed toxin. Its source can be in carcasses, food or water contamination, maggots, insect larvae (such as beetles) and other invertebrates. Additionally, there is a form known as toxico-infection that results from production of the toxin within the chicken's intestinal tract. Toxin production occurs in association with other unknown factors to produce clinical disease.

How does one confirm the diagnosis?

Affected chickens typically show signs of progressive paralysis, with involvement of limbs, neck, and eyelids being most common. Morbidity and mortality may vary depending on the amount of toxin ingested. Unlike other disease conditions, there is an absence of gross and microscopic lesions and this condition often becomes a diagnosis by exclusion. Occasionally, there may be signs that birds have cannibalized penmates, and certainly dead birds represent a valid source for growth of the organism and production of the preformed toxin.

The standard means of confirming the diagnosis is by mouse bioassay for toxin, preferably in serum or less suitably in crop or gastrointestinal contents from moribund live birds. Mice typically succumb to the effects of the toxin within 48 hours of inoculation. The test is run here in the Bacteriology Unit at the Animal Health Laboratory for a fee of \$100. The turnaround time varies from 2 - 4 days depending on the absence or presence of clinical signs in the inoculated mice.

What are the management strategies?

1. Minimize contact with possible infected sources, i.e. remove dead chickens from the house promptly and maintain hygiene of the water and food sources.

- 2. Use of sodium selenite and vitamins A, D3 and E.
- 3. Antibiotics: bacitracin, streptomycin, chlortetracycline or penicillin.
- 4. Antitoxin difficult to source.
- 5. Adequate disinfection of affected premises.
- 6. Feed lower energy diets.
- 7. Adequate fly control.
- 8. Use of a vaccination protocol too expensive in commercial poultry.

Chicken anemia virus serology

Doug Key, DVM MSc DVSc, AHL-Guelph.

We are currently evaluating the IDEXX Laboratories chicken anemia virus serological ELISA that has been licensed by the USDA. Seroconversion with this test is detected at between 10 and 14 days post-

infection. The results will be accompanied by a statement outlining the method for interpretation of the results, which are reported as **sample to negative (S/N) ratios.** It is important to understand that the method of interpretation is inverse to how we normally think, as **samples with an S/N** <u>value of less</u> <u>than 0.6 are positive</u>. Samples with S/N values of <u>equal to or greater than 0.6 are negative</u>.

We will be reporting the individual values for each sample along with a "positive" or "negative" beside each sample. In an experimental trial, birds vaccinated at 10 weeks of age showed:

- <u>negative</u> S/N values of 0.8 to 1.2 initially,
- positive S/N values of 0.0 to 0.3 at 6 weeks post-vaccination, and
- positive S/N values of 0.0 to 0.1 at 8 weeks post-vaccination.

Positive S/N values of 0.3 are reported to be comparable to virus neutralization (VN) titers of 1:64 to 1:256, and positive S/N values of less than 0.1 are equivalent to VN titers of 1:1024 to1:2048.

This new test **will be run on a weekly to biweekly basis depending on demand, and the fee will be \$2.25 per sample.** If you have any questions regarding this test, please contact Dr. Doug Key at 519-824-4120, ext. 4524.

An outbreak of fowl cholera in pheasants

Jiggs Gough, DVM Dip Path, AHL-Ridgetown.

In October 1999, 30 5-month-old ringneck pheasants in a pen of 1000 died overnight. The rest of the birds in the pen and the 1000 in the adjoining pen looked healthy. Two pheasants were submitted to the Animal Health Laboratory, Ridgetown for necropsy. The birds were in good body condition. There were subcutaneous hemorrhages over the head, thorax and thighs. The livers were firm and friable with focal hemorrhages in the liver of one bird, and white pinpoint foci in the liver of the other. Spleens were enlarged and lungs were congested. There were hemorrhages in the mucosa of proventriculi. Intestinal contents were bloody, with occasional focal transmural hemorrhage. Microscopically there was severe acute coagulation necrosis in the spleens with large numbers of bacteria present. Focal hemorrhage and single cell necrosis were severe in the livers. Hemorrhage and inflammation in the mucosa of the proventriculi were marked. There were large numbers of a mixture of inflammatory cells in the air sacs.

Large numbers of *Pasteurella multocida* were cultured from lung, spleen and liver; the bacterium was sensitive to many antimicrobials - the *P. multocida* is being serotyped. Birds in the affected pen were treated with penicillin in the water for 6 days; losses stopped after 8 days. After 11 days, 2 more pheasants died and the remaining birds in this pen were treated with tetracycline in their water. **Fifty pheasants and 3 of the 25 wild turkeys that were in this pen died during the outbreak.**

Fowl cholera (FC) (avian pasteurellosis, hemorrhagic septicemia) is a contagious disease affecting domesticated and wild birds that is more prevalent in the late summer, fall and winter (1). The virulence of *P. multocida* is complex and variable depending on the strain and host. The bacteria may enter through the pharynx, upper air passages, conjunctiva or cutaneous wounds (1). The disease has been reported in a wide range of avian hosts, but turkeys are most susceptible and losses may be very high. Losses in chickens are usually in laying flocks, as chickens less than 16 weeks of age are generally quite resistant. Domestic geese and ducks are also highly susceptible. Epornitics in wild waterfowl with very heavy losses have been reported, and in some areas FC is enzootic in waterfowl (1). Chronically infected birds are considered a major source of infection. An acute outbreak in wild pheasants was reported in 1964 (1).

Reference

1) Rimler RB, Glisson JR. Fowl cholera. In: Calnek BW ed. Diseases of Poultry, 10th ed. Ames, Iowa: Iowa State Univ Pr 1997: 143-159.

SWINE

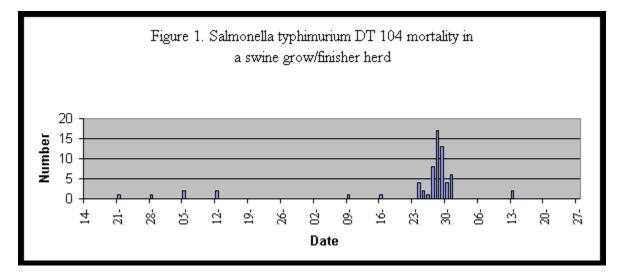
A *Salmonella typhimurium* DT 104 outbreak in a swine finishing operation

Gaylan Josephson, DVM Dip Path, AHL-Exeter; Paul Morris, DVM, Wellesley, ON; Marie Archambault, DVM MSc, AHL-Guelph.

This case involves a group of 1200 finisher pigs in a naturally ventilated barn. This operation is part of a multi-site, segregated early weaning (SEW) unit.On August 19, 1999, the herd was given feed medicated with chlortetracycline because of a marked increase in coughing. An 80 kg pig died 5

days later, and necropsy revealed fibrinous pneumonia. Losses continued and, within 4 days, a total of 15 animals had died. Watery diarrhea was noted, with yellow pools of liquid feces apparent within the pens. Necropsy of several dead pigs revealed splenomegaly, watery intestinal contents, enlarged ileocecal lymph nodes and pulmonary congestion. Tissue samples were submitted to the AHL-Guelph for testing.

Salmonellosis was suspected and the herd was given a water medication consisting of Neomix and Onycin. Individual animals were treated with Trivetrin. Within 2 days of starting antimicrobial treatment, the pigs showed considerable improvement, although a few pigs were still clinically ill. Death losses stopped within 4 days, and there was no recurrence of the problem before the animals were shipped to market. A total of 65 pigs from this group of 1200 died during the 105 day grow/finisher phase, with 55 animals dying during this single outbreak. (See Figure 1)



Salmonella typhimurium phage type 104 was cultured from lung, liver, spleen and feces of dead pigs, and from rectal swabs of scouring animals. The isolate was sensitive to neomycin, gentamicin and trimethoprim-sulfa, but was resistant to ampicillin, tetracycline, sulfisoxazole and spectinomycin.

H3N2 influenza A virus in pigs

Susy Carman¹, Gaylan Josephson¹, Carol Stansfield², John Weber², Rob Bildfell¹, Tony van Dreumel¹

¹ AHL-Guelph; ² Health Canada, Laboratory Center for Disease Control, Winnipeg, MB.

Influenza A viruses have a segmented gene with eight distinct strands of RNA. Exchange of genomic segments can occur during simultaneous infection of a host with different influenza viruses, resulting in the generation of "reassortant" viruses. Pigs have a weak species-specific barrier against infection by human and avian influenza A viruses, such that interspecies transmission and genetic reassortment between classical swine, human and avian viruses occurs, favoring transmission and replication in swine. As a result, swine can be considered reservoirs for human influenza A virus strains.

Various strains of both H1N1 and H3N2 influenza A viruses, with genome segments contributed from swine, human and avian influenza viruses, have co-circulated in European pigs since the mid 1970's. In contrast, in North America serological surveillance of swine in the USA during 1976 to 1977 demonstrated a high prevalence of antibody to the classical H1N1 strain of swine influenza, but only a low prevalence of antibody to H3N2 strains. H3N2 viruses were believed to be uncommon in North American swine and to cause only mild disease. As well, the majority of isolates recovered from swine with clinical disease were H1N1 strains. In 1988, an H3N2 influenza virus was found in swine in Quebec with severe clinical disease; this H3N2 strain was similar to a human strain that had circulated in 1975.

In 1995, we tested sera, collected from 1989 to 1992 from 107 Ontario pigs representing 12 different herds with a clinical history of pneumonia, coughing or abortions, using hemagglutination-inhibition (HI) assays to determine the prevalence of antibody to selected influenza A virus strains. We found 53% of these sera had antibody to classical H1N1 A/Swine/Wisconsin/49/76. Approximately 17% had antibody to human H3N2 A/Beijing/353/89. This virus was circulating in humans in Ontario at the time the swine sera were collected. About 4% of these Ontario swine sera had antibody to human H1N1 A/Taiwan/1/86. None had antibody to the H3N2 Quebec strain.

Despite the ongoing presence of antibody to H3N2 viruses in Ontario swine, only classical H1N1 isolates had been recovered from Ontario swine prior to 1997. In January 1997, the AHL isolated an H3N2 virus from a one-week-old piglet with pneumonia. On histologic examination of a section from the consolidated cranioventral area of the lung, there was a neutrophilic reaction in the lumens of bronchioles. Bronchioles were often denuded or lined by cuboidal to flattened epithelial cells. Alveoli were filled by protein-rich fluid and neutrophils with fewer macrophages and rare multinucleated giant cells. No significant pathogenic bacteria were demonstrated in the lung. Using formalin-fixed paraffinembedded lung tissue, avidin-biotin immunoperoxidase (IP) tests (Prairie Diagnostic Services, Saskatoon) were positive for both porcine reproductive and respiratory syndrome virus and influenza A virus, with extensive alveolar and bronchiolar staining for influenza A virus antigens. Similar IP tests for circovirus were negative.

A cytopathic hemagglutinating agent was recovered from the lung in primary pig kidney cell cultures after 3 blind passages. Infected cell cultures were positive for influenza A virus using monoclonal antibodies in fluorescent antibody assays. However, the virus could not be typed using HI assays, or reference viruses and antisera for classical H1N1 swine influenza virus or for H3N2 Quebec influenza virus. Hence virus- infected cell culture supernatant was forwarded to Health Canada for typing. There the virus was typed, using reference human H3N2 viruses and antisera in HI tests, to be similar to human H3N2 A/Wuhan/359/95 and H3N2 A/Nanchang/933/95 strains. The Wuhan H3N2 human virus

strain was the most common strain recovered from humans in Ontario during the 1996 to 1997 influenza season. This new swine H3N2 isolate has been designated A/Swine/Ontario/00130/97.

We continue to recover H1N1 isolates from Ontario swine. Although current serological submissions reveal evidence of H3N2 (Quebec strain-like) in Ontario swine, we have not recovered any other H3N2 virus strains since 1997. However, H3N2 influenza A virus strains have been recovered from swine from North Carolina, Iowa, Minnesota and Texas in 1998 (1,2). These viruses have been associated with severe respiratory disease in affected swine herds. As with European H3N2 strains, they have been shown to be reassortant viruses (2), incorporating genes from classical swine H1N1 influenza virus and a recent human H3N2 influenza virus (North Carolina) or the genes of classical swine, human-like and avian influenza A viruses (Iowa, Minnesota, Texas).

Since these new H3N2 viruses vary antigenically from each other, and there is no cross-reaction with the more homogeneous classical H1N1 isolates in HI tests, a separate HI serology test should be performed to look for specific antibody to each of these influenza A isolates.

Hence the AHL now offers three HI tests for antibody to influenza A viruses in swine:

1. H1N1 serology test using the A/swine/Ontario/81 strain as antigen for classical H1N1 swine influenza A virus

2. H3N2 serology test using the A/swine/Colorado/77 strain as antigen for the Quebec strain of swine influenza A.

3. H3N2 serology test using the A/swine/Texas/98 strain as antigen for strains from the midwestern USA. This new test is more difficult to perform and hence will cost \$4/animal, rather than \$3/animal as for other influenza A serology tests.

We are continuing work on the development of an HI test to identify antibodies for the A/Ontario/00130/07 and North Carolina influenza A virus strains.

References

1. Janke BH, Yoon K-J, Halbur PG, et al. Isolation of a new subtype (H3N2) of swine influenza virus from pigs with respiratory disease in the North Central US. Proc IX International Symposium, World Assoc Vet Lab Diagnosticians 1999:51.

2. Zhou NN, Senne DA, Landgraf JS, et al. Genetic reassortment of avian, swine and human influenza A viruses in American pigs. J Virology 1999; 73: 8851-8856.

HORSES

News Note from ProMed:

West Nile virus, an exotic mosquito-borne virus, has claimed at least 6 human lives in New York City, plus an elderly Toronto man who visited Queens, and caused the death of at least 2 horses and euthanasia of about 10 more horses on Long Island, NY. The virus, which cycles between infected birds and mosquitoes, is believed to have entered the US in infected birds. The West Nile arbovirus is closely related to St. Louis encephalitis virus. Signs of the viral infection in horses include lethargy, weakness in the hind quarters, stumbling and loss of coordination, lack of awareness, head tilt and twitch, convulsions, circling, hyperexcitability, paralysis, and coma. *MGM*

Lawsonia intracellularis infection in a group of foals

Murray Hazlett, DVM DVSc Diplomate ACVP, AHL-Guelph; *Marie Archambault*, DVM MSc, AHL-Guelph; *John Prescott*, VetMB PhD, Ontario Veterinary College; *Brent Hoff*, DVM DVSc, AHL-

Guelph.

Four of eight weanling foals developed anorexia, lethargy, and hypoproteinemia over a 3-week period. One 7-month-old foal died and was submitted for necropsy at the AHL in Guelph. The foal was underweight, but had fat stores and no dependent edema. **The foal had severe proliferation of the mucosa of the distal 90% of the small intestine**, with some hemorrhage and local peritonitis of the associated serosa. Lesions seen were similar to those in pigs with proliferative enteritis caused by *Lawsonia intracellularis*. Modified acid-fast smears of mucosa revealed large numbers of acid-fast *Campylobacter*-like organisms within cells, typical in staining and morphological appearance to *Lawsonia intracellularis*. Silver-stained histopathology sections showed the bacteria in enterocyte cytoplasm, usually oriented at the luminal margin of the cell.

Lawsonia intracellularis has been described in a number of cases as a cause of proliferative enteropathy in foals since 1997 at the AHL. Most cases are in single animals - in this case, a group of foals was likely involved. The vector for this organism in horses is unknown.

We recommended that other foals in this group be treated with oral erythromycin and rifampin, at doses usually used for the treatment of *Rhodococcus equi*.

COMPANION ANIMALS

Sudden death following administration of oral milbemycin/lufenuron to a microfilaremic dog

Margaret Stalker, DVM PhD Diplomate- ACVP, AHL-Guelph; Andrew Peregrine, BVMS PhD DVM, Ontario Veterinary College.

A 4.5-year-old spayed female Basset hound experienced sudden onset of respiratory distress, panting, cyanosis, mucoid stools, and hemoptysis within one hour of administration of oral lufenuron (10mg/kg)/milbemycin oxime (0.5 mg/kg). The dog did not respond to supportive therapy (oxygen, IV epinephrine, dexamethasone), progressed into a shock-like state, and died within one hour of onset of clinical signs. The dog had been presented to a veterinarian approximately 10 hours prior, for routine physical exam and vaccinations - physical findings had been unremarkable. The dog had never traveled outside Ontario, had not been given any form of heartworm preventive medication in previous years, and was not tested for heartworm infection prior to administration of the medication this year.

Significant findings on post-mortem included marked pulmonary congestion, edema and hemorrhage. Eight adult heartworms (*Dirofilaria immitis*) were present within the lumen of the right ventricle of the heart. Histologically, there was marked congestion of alveolar capillaries, with occasional fibrin thrombi, and extensive alveolar edema with focal intra-alveolar hemorrhage. Numerous microfilariae were present within capillary lumens, and occasionally free within alveoli.

Mild to moderate transient reactions (pallor, labored respiration, coughing, lethargy) have been reported in microfilaremic dogs administered milbemycin; these are thought to be related to the microfilaricidal effects of milbemycin at the preventive dosage. Fatalities have not been reported either in toxicology studies, or in the published veterinary literature, to our knowledge. Whereas milbemycin is routinely used as a microfilaricide, and serious adverse reactions are apparently rare, **this case underlines the importance of heartworm testing prior to prophylaxis with milbemycin**.

Ethylene glycol (antifreeze) poisoning of a cat

Jiggs Gough, DVM Dip Path, AHL-Ridgetown.

In September, 1999, a male, one-year-old domestic shorthair cat was submitted to the Animal Health Laboratory, Ridgetown for necropsy. The cat had been taken to the veterinarian 5 days earlier with vomiting and depression. The owner was very concerned because several cats in the neighborhood had died recently. The biochemical profile revealed azotemia as well as an electrolyte imbalance. Urine production was very low. Despite treatment, the cat's condition deteriorated and he was euthanized.

At necropsy there was marked subcutaneous, pulmonary and hepatic edema. Abdominal, pleural and pericardial fluid were approximately 200, 100 and 20 mL respectively. Microscopically there was widespread nephrosis. A moderate number of tubules contained grey to light-yellow crystals arranged in sheaves and rosettes that were birefringent in polarized light. The diagnosis was ethylene glycol (antifreeze) toxicosis.

All animals are susceptible to ethylene glycol (EG) toxicity, but it occurs most commonly in dogs and cats. Most intoxications are associated with engine antifreeze because it is widely available, has a sweet taste, and the lethal dose is small (1). Another factor is the lack of public awareness that results in improper storage and disposal. Poisoning by EG commonly occurs in the fall when antifreeze is changed or replenished.

The minimum lethal dose of EG for a cat is 1.4 mL/kg body weight and 4.4 mL/kg body weight for dog (1). It is rapidly absorbed from the gastrointestinal tract, and in dogs peak blood concentration occurs within 3 hours of ingestion. Fifty per cent is excreted unchanged in the urine. However, a series of oxidation reactions results in toxic metabolites that cause acidosis and nephrosis. Calcium oxalate crystals form in renal tubules. They are birefringent in polarized light and large numbers of crystals are virtually pathognomonic of ethylene glycol poisoning (2).

Clinical signs of EG poisoning are dose- and time-dependent. The first signs resemble those caused by ethanol intoxication: vomiting, depression, ataxia, polydipsia and polyuria. Signs of oliguric acute renal failure and acidosis (lethargy, anorexia, dehydration, vomiting, seizures) usually develop in 12-14 hours in cats and 36-72 hours in dogs (1). Prognosis varies inversely with the time elapsed between ingestion and initiation of treatment. Treatment is aimed at decreasing absorption of ingested EG, increasing excretion and preventing metabolism of EG, and correcting the ensuing metabolic acidosis.

References

1. Aiello S E. ed. The Merck Veterinary Manual. 8th ed Philadelphia: National Publishing. 1998: 2035-2038.

2. Maxie MG. The urinary system. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals 4th ed. vol 2 San Diego, California: Academic Press 1993: 491-492.

Preliminary results from the Canadian Veterinary Urolith Centre

Doreen Houston¹, Jim Patterson¹, Andrew Moore², Sandy Smith², Michael Favrin², Michael Villagonzalo², Brent Hoff³

¹ Veterinary Medical Diets, Guelph, ON; ² Canadian Veterinary Urolith Center, Lab Services Division, University of Guelph; ³ AHL-Guelph On February 1, 1998, Veterinary Medical Diets (VMD) partnered with the Laboratory Services Division to establish the Canadian Veterinary Urolith Centre (CVUC), located at 95 Stone Road West in Guelph. The CVUC is capable of analyzing urinary crystals and calculi from dogs and cats with a variety of microscopic techniques including:

- Polarized light microscopy
- Fourier transform infrared spectroscopy
- Scanning electron microscopy
- X-ray microanalysis
- X-ray diffraction (available to assist with problematic cases)

Within the first 16 months, the CVUC received and analyzed over 5000 submissions from all parts of Canada. Twenty-four percent of stones submitted came from Western Canada (British Columbia, Alberta, Manitoba); 48% from Ontario; 17% from Quebec; 11% from Eastern Canada (Nova Scotia, New Brunswick, Prince Edward Island and Newfoundland).

To date, 70% of the submissions have been canine and 30% feline. In both dogs and cats, struvite submissions outnumbered oxalate submissions. In dogs, 50% of the submissions were struvite, whereas 38% were oxalate. In cats, 55% of submissions were struvite and 36% oxalate.

Canine data: Females outnumbered males approximately 9:1 in struvite stone submissions. Males outnumbered females approximately 2:1 in calcium oxalate stone submissions. Composition of urinary calculi by canine breed and sex from the top six breed submissions is summarized in Table 1. Although mixed breed dogs accounted for the majority (19.6%) of canine submissions, there was over-representation of cases from 5 breeds of small purebred dogs (Miniature Schnauzer, Bichon Frise, Shih Tzu, Lhasa Apso, and Yorkshire terrier). Additionally, note the strong tendency for males in these breeds to form oxalate stones and the females, struvite stones. Dalmatian dogs were at greatest risk for development of urate stones with the male Dalmatian at highest risk

Feline data: The majority of stones and urethral plugs came from domestic short hair and domestic long hair cats (88.5%) followed by Himalayan (5.5%), Persian (3%), and Siamese (3%) cats. Males outnumbered females 1.6:1 in struvite (includes both urethral plugs and stones) and 1.8:1 in calcium oxalate submissions.

For more information about stone analysis conducted through the Canadian Veterinary Urolith Centre (CVUC) call Veterinary Medical Diets toll-free at 1-800-567-8900.

Table 1. Urolith composition by breed and sex for the 6 most commonly affected canine breeds.

UROLITH COMPOSITION

SEX ^a	# CALCULI		I	Calcium oxalate			Struvite		Urates	0	Other	
%	#		%		#		%	#		%		
chnauzer		Μ	213	184	86.4	15	7.0	7	3.3	7	3.3	
		F	318	147	46.2	160	50.3	4	1.3	7	2.2	
•		Μ	119	108	90.8	4	3.4	1	0.8	6	5	
	%	% #	SEX ^a # CALCUL % # chnauzer M F	SEX ^a # CALCULI % # % chnauzer M 213 F 318	SEX ^a # CALCULI Calciu % # % chnauzer M 213 184 F 318 147	SEX ^a # CALCULI Calcium oxala % # % # hnauzer M 213 184 86.4 F 318 147 46.2	% # % # % # % # % M 213 184 86.4 15 F 318 147 46.2 160	SEX ^a # CALCULI Calcium oxalate Struv % # % # % hnauzer M 213 184 86.4 15 7.0 F 318 147 46.2 160 50.3	SEX ^a # CALCULI Calcium oxalate Struvite % # % # % # hnauzer M 213 184 86.4 15 7.0 7 F 318 147 46.2 160 50.3 4	SEX ^a # CALCULI Calcium oxalate Struvite Urates % # % # % # hnauzer M 213 184 86.4 15 7.0 7 3.3 F 318 147 46.2 160 50.3 4 1.3	SEX ^a # CALCULI Calcium oxalate Struvite Urates O % # % # % # % % # % # % # % % # % # % # % % # % # % # % % # % # % # % % # 184 86.4 15 7.0 7 3.3 7 F 318 147 46.2 160 50.3 4 1.3 7	

	F	275	44	16.0	224	81.5	0	0	7	2.5
Shih Tzu	Μ	112	67	59.8	25	22.3	10	8.9	10	8.9
	F	275	41	14.9	210	76.4	2	0.7	22	8.0
Lhasa Apso	Μ	102	88	86.3	8	7.8	0	0	6	5.9
	F	110	35	31.8	66	60.0	1	0.9	8	7.3
Yorkshire terrier	Μ	73	60	82.2	6	8.2	5	6.8	2	2.7
	F	37	13	35.1	22	59.5	0	0	2	5.4
Dalmatian	Μ	155	1	0.6	4	2.6	147	94.8	3	1.9
	F	15	0	0	4	26.7	10	66.7	1	6.7
Totals		1804	788		748		187		81	

^{**a**} M = male, F = female

Streptococcal toxic shock in a cat

Peter Lusis, DVM MSc, AHL-Kemptville.

A 1-year-old female cat died 5 days after she was spayed and declawed, one day after she had been discharged from the clinic. The owner reported that she had appeared a bit 'groggy' when discharged.

Necropsy revealed acute peritonitis, with ~10 mL of thick red exudate in the peritoneal cavity and ~6 mL of serosanguineous fluid in the thoracic cavity. Surgical sites and sutures appeared normal, with no evidence of cellulitis, suppuration or other complications. Histopathology revealed severe multifocal peritonitis and pleuritis, acute thymic and splenic necrosis, and septicemia, with many coccoid bacteria in blood vessels. Heavy pure cultures of beta-hemolytic group G streptococci (*Streptococcus canis*) were cultured from thoracic and abdominal fluids and spleen.

S. canis is commonly isolated from the throat, tonsils, skin and lymph node abscesses of cats, and rarely causes septicemia in kittens, but *S. canis* septicemia has apparently not been reported in adult cats. **This unusual case of streptococcal septicemia in an adult cat resembles the S. canis toxic shock syndrome increasingly recognized in dogs** (1,2), although necrotizing fasciitis was not a feature of the current case.

References

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

2. Miller CW, Prescott JF, Mathews KA, et al: Streptococcal toxic shock syndrome in dogs. J Am Vet Med Assoc 1996; 209:1421-1426.

SMALL RUMINANTS

Salmonella typhimurium DT 104 in a group of feedlot sheep

Jiggs Gough, DVM Dip Path, AHL-Ridgetown; Beverly McEwen, DVM PhD Diplomate ACVP, AHL-Guelph.

In October 1999, 640 5-month-old lambs were delivered to a farm in southwestern Ontario from western

Canada. Some lambs had scoured during transit. Five days after arrival, morbidity was high; many lambs had fevers of 40-41°C, depression and tenesmus with bloody, fluid feces. Mortality had reached 31 when lambs were submitted to the Animal Health Laboratory, Ridgetown. Findings were similar in the three carcasses. The rumens were full of a dry roughage/grain mixture. The small and large intestines contained turbid yellow to brown fluid. There were focal hemorrhages on the cecal and colonic mucosa. Superficial mucosal necrosis with cryptitis and increased inflammatory cells in the lamina propria and submucosal edema were evident microscopically. Large numbers of *Salmonella* spp. were cultured from the intestine. The organism was resistant to many antimicrobials. It was serotyped by Health Canada, Health of Animals Laboratory, Guelph as *S. typhimurium* antigen 4,5:i:2 phage type 104.

Clinical response to treatment did not correspond to the *in vitro* susceptibility test; although the organism was susceptible to trimethoprim-sulfa, response was poor. One hundred sheep were treated with one injection of florfenicol and clinical improvement was swift, although the organism was resistant on the susceptibility test. About 10 sheep were treated with a second injection. **During the outbreak, 47 lambs died.**

Salmonellosis is not a common disease in sheep, but outbreaks are often severe and may cause heavy losses (1). Predisposing influences are necessary and these are usually provided by circumstances which enforce congregation (1). Holding in a collection yard, transportation over a long distance, deprivation of food and water, and fatigue had stressed these sheep. The owner had been bringing lambs in from western Canada for many years. In 1996, he had severe losses in the first few weeks and *S. typhimurium* DT 104 was isolated. The problem did not occur in 1997 or 1998.

From 1991 to November 3, 1999, *Salmonella* spp. were isolated from sheep 36 times at the Animal Health Laboratory. From 1991-1998, 26 of the 30 isolates were *S. arizonae* and two were *S. typhimurium*. During the first ten months of 1999, *Salmonella* spp have been identified 7 times; *S. arizonae* and *S. typhimurium* have been found once each, and 5 other serotypes have been isolated.

Public and animal health agencies are becoming increasingly concerned about the occurrence of multidrug resistant *S. typhimurium* **DT (definitive type or phage type) 104 that is resistant to at least 5 antimicrobials: ampicillin, chloramphenicol, streptomycin, sulfonamides, and tetracycline (2), also known as R-type ACSSuT (3). This pathogen has been associated with higher hospitalization and mortality rates among people than for other** *Salmonella* **infections (2). In the United Kingdom, surveillance has documented a rapid increase in the prevalence of DT 104 in several species, including cattle, swine, sheep and poultry, during the past decade (3).**

Direct transmission is thought to occur via the fecal-oral route. Indirect transmission can also occur through contaminated feed, water, pasture and wildlife vectors (2). Control measures should include good biosecurity, proper management of waste, animal feed and water, and husbandry practices that prevent the spread of disease from adult animals to young, naive animals (2). Control of rodents has been shown to be very important in the reduction of the organism, although, once introduced, it is difficult to consistently eliminate *Salmonella* spp. from the environment (3). Since the zoonotic potential is great and the organism can cause serious illness, a very important control measure is good hygiene in animal handling that reduces the spread from animal-to-human and human-to-human as well as from animal-to-animal.

References

1. Barker IK, van Dreumel AA, Palmer N. The alimentary system. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animal 4th ed. vol 2. San Diego, California: Academic Press 1993:226.

2. Dargatz DA., et al. "The veterinarian's role in diagnosis, treatment and prevention of multidrug resistant *Salmonella typhimurium* DT 104". USDA-APHIS-VS. 1998.

3. Akkins JE, Hogue AT, et al. Epidemiologic aspects, control, and importance of multiple-drug resistant *Salmonella typhimurium* DT 104 in the United States. J Am Vet Med Assoc 1999; 214: 790-798.

BACTERIOLOGY

Antimicrobial resistance among bacterial isolates from foodproducing animals in Ontario.

Beverly McEwen¹, Nonie Smart¹, Case Poppe², Moira Neale¹, Marie Archambault¹, Alfonso Valdivieso², David Alves³, Bruce McNab³, Abdul Rehmtulla³, Scott McEwen⁴. ¹AHL-Guelph; ²Health of Animals Laboratory, Health Canada, Guelph; ³Ontario Ministry of Agriculture, OMAF; ⁴Department of Population Medicine, Ontario Veterinary College, University of Guelph.

There is increasing concern about the emergence and pattern of antimicrobial resistance among bacteria isolated from foods of animal origin. A collaborative research project involving the Animal Health Laboratory, Health Canada, OMAF, and the Dept. Population Medicine (OVC), to examine patterns of antimicrobial resistance in food-producing animals, will be completed in May 2000.

This study will document patterns of bacterial resistance to antimicrobials commonly used in veterinary and human medicine among approximately 500 isolates from food animal veterinary cases submitted to the Animal Health Laboratory. Similarly, antimicrobial resistance profiles of over 300 isolates obtained from fecal samples of healthy broiler chickens, market hogs, and beef collected at provincially inspected abattoirs from across Ontario will be documented. Antimicrobials will be consistent with those used in the National Antimicrobial Resistance Monitoring System (NARMS) of the United States. Preliminary results were presented in a poster session in Toronto at the 'Agriculture's Role in Managing Antimicrobial Resistance Conference', held October 24-26, 1999.

Determining antimicrobial resistance profiles of bacterial isolates from abattoir and clinical specimens will provide useful baseline information on the frequency and occurrence of antimicrobial resistant bacteria that could potentially reach the consumer. These surveillance data could be used to evaluate the risks associated with the use of antimicrobials in food animals. This research may form the basis of one component of ongoing surveillance of antimicrobial resistance in agriculture.

This project is funded by the Enhanced Food Quality and Safety Research Fund, Ontario Ministry of Agriculture, OMAF.

Bacterial terminology update

Marie Archambault, DVM MSc, AHL-Guelph.

Based on the results of phylogenetic investigations, some bacteria that cause veterinary infections have been reclassified into new genera. These reclassification are summarized in Table 1 below. Please have a look at what is happening in the taxonomy world because you will see these new names on our reports.

Table 1. Bacterial terminology update

New name	Former name	Reference	Infection		
Actinobaculum suis	Eubacterium suis	Int J Syst Bact 47:901, 1997	Urinary tract infection in pigs		
	Actinomyces suis				
Arcanobacterium pyogenes	Actinomyces pyogenes	Int J Syst Bact 47:51, 1997	Suppurative pneumonia, mastitis, endometritis, arthritis, wound infections, and abortion in various species		
	Corynebacterium pyogenes				
Brachyspira hyodysenteriae	Serpulina hyodysenteriae	Int J Syst Bact 48:327, 1998	Enteric infection in pigs (swine dysentery)		
	Treponema hyodysenteriae				
Brachyspira innocens	Serpulina innocens	Int J Syst Bact 48:327, 1998	Enteric infection in pigs		
	Treponema innocens				
Brachyspira pilosicoli	Serpulina pilosicoli	Int J Syst Bact 48:327, 1998	Enteric infection in pigs (colonic spirochetosis)		
Burkholderia mallei	Pseudomonas mallei	Int J Syst Bact 43:398, 1993	Glanders		
			(not reported in Canada)		
Burkholderia pseudomallei	Pseudomonas pseudomallei	Int J Syst Bact 43:398, 1993	Melioidosis or pseudoglanders		
			(not reported in Canada)		
Mannheimia haemolytica	Pasteurella haemolytica	Int J Syst Bact 49:82, 1999	Pneumonia, septicemia, mastitis, mainly in cattle and sheep		
WHO'S NEW AT THE AHL?					

Marie Archambault DMV, MSc AHL Veterinary Bacteriologist

Dr. Marie Archambault graduated with her DMV from the Faculty of veterinary medicine, University of Montreal, St-Hyacinthe, Que, in 1993. Following graduation she continued her studies in veterinary microbiology at the University of Montreal where she was involved in undergraduate teaching as an instructor in veterinary bacteriology, immunology and anatomy. After obtaining her MSc in 1995, Dr Archambault worked as a veterinary bacteriology diagnostician for the Clinical Veterinary Bacteriology Laboratory of the University of Montreal. During this time, she enrolled in a PhD program with the GREMIP (Groupe de Recherche sur les Maladies Infectieuses Porcines). Her focus was investigation of the hemoglobin-iron acquisition system at the cell surface of *Actinobacillus pleuropneumoniae*. Her work led to 3 peer-reviewed scientific publications, a manuscript in preparation, and several professional presentations. Dr. Archambault joined the AHL as our veterinary bacteriologist in August, 1999, and will defend her PhD thesis in December.

Shelley Newman, DVM, DVSc, Diplomate ACVP AHL Veterinary Pathologist, Avian and fur-Bearing Animals

Dr. Shelley Newman is a 1990 DVM graduate of the Ontario Veterinary College, who was in private practice in Ontario for 3 years, and then completed her DVSc at OVC in 1996 on the topic of immune-

mediated glomerular disease in mink, and potentially dogs, as a possible result of vaccination. She then undertook a 2-year appointment at the Virginia-Maryland College of Veterinary Medicine, teaching undergraduate and graduate pathology. During this time, she successfully completed the rigorous examination of the American College of Veterinary Pathologists. Dr. Newman joined the AHL in May, 1999, as veterinary pathologist, avian and fur-bearing animals.

1999 PUBLICATIONS

Dewey CE, **Carman PS, Hazlett M, van Dreumel T, Smart N**. Endemic transmissible gastroenteritis: Difficulty in diagnosis and attempted confirmation using transmission trials. Swine Health and Production, 1999; 7: 73-78.

Duffield T, Peregrine A, **McEwen BJ**, Hietala S. Epidemiology of *Neospora* infection in Ontario Holsteins. Proc Am Assoc Bov Practitioners, Nashville, Tenn. Sept 1999; 238.

Lumsden JH, **Hazlett MJ**. Choosing a laboratory information system: reflections on 'kicking the tires'. Proc 1998 Congress of the International Society for Animal Clinical Biochemistry, Hiroshima, Japan, 1998.

Shapiro JL, Prescott JF, Henry G. Equine abortions in eastern Ontario due to leptospirosis. Can Vet J 1999; 40: 350-351. (A Cross-Canada Disease Report)

Sutton A, Dawson H, Hoff B, Grift E, Shoukri M. Analyte comparisons between 2 clinical chemistry analyzers. Can Vet J 1999; 40: 255-260.

Wilson JB, Pauling GE, **McEwen BJ, Smart N, Carman PS**, Dick CP. A descriptive study of the frequency and characteristics of proliferative enteropathy in swine in Ontario by analyzing routine animal health surveillance data. Can Vet J 1999; 40: 713-717.

1999 PRESENTATIONS-POSTERS

Hazlett MJ, McEwen BJ. Using VADDS for surveillance. VADDS/VetStar annual conference, June 4, 1999, Guelph, Ont.

Hazlett MJ, Halfpenny MT, Few P, Fraser S. Using VADDS at the University of Guelph. VADDS/VetStar annual conference, June 4, 1999, Guelph, Ont.

Mann E, Hockin J, **McEwen** BJ, Odumeru J. Canadian Integrated Public Health Surveillance (CIPHS) System. Presentation at Interagency Food Safety and Nutrition Research Meeting. Oct 4-5, 1999, Winnipeg, Man.

Mann E, **McEwen BJ, Hazlett MJ**, Odumeru J. Linking animal health and food safety data to human health: CIPHS Guelph pilot. Abstract, American Association of Veterinary Medical Colleges (AAVMC) Meeting. July 1999, New Orleans, LA, USA.

McEwen BJ, Smart N, Poppe C, **Neale M, Archambault** M, Valdivieso A, Alves D, McNab B, Rehmtulla A, McEwen S. Antimicrobial resistance among bacterial isolates from food producing animals in Ontario. Poster presentation at "Agriculture's Role in Managing Antimicrobial Resistance Conference", Oct 24-26, 1999, Toronto, Ont.

McEwen BJ, Hazlett MJ. "Surveillance and the Animal Health Laboratory; Tailoring information to capture customers' needs, or What's in the database for me?" Presentation at the Lab Services Division

"Leading Edge" Open House, Oct 21, 1999, Guelph, Ont.

Shapiro JL. "Laboratory submission tips for practitioners". Central Canada Vet Assoc, Fall Conference. Dec 2, 1999, Ottawa, Ont.

Shapiro JL. "Pathology and laboratory testing", in Workshop on Diseases of Meat Rabbits. Dec 8, 1999, Kemptville, Ont.

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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