UNIVERSITY of GUELPH
LABORATORY SERVICES

Animal Health Laboratory

AHL Newsletter

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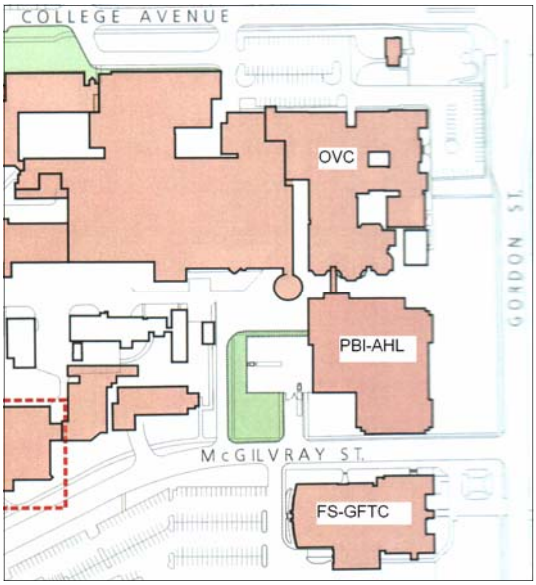
In this issue:

Progress on the new Pathobiology-AHL building

Planning is moving forward for the new Pathobiology-AHL building, to be located facing Gordon Street in Guelph, between the main building of OVC and the Food Science Building (see map).

- Major funding for the building has been provided by the Canadian Food Inspection Agency ($37.3 million) and the Ontario Ministry of Agriculture, Food and Rural Affairs ($25 million).
- Robbie/Young + Wright have been selected as the architects, and building design is expected to be completed over the next year.
- Occupancy is scheduled for fall 2010.

We look forward to improved biosecurity and biocontainment, better specimen flow and workflow for the AHL, and improved teaching and research space for the Department of Pathobiology. The capabilities of the AHL to detect new, emerging, and foreign animal diseases will be expanded by such features as integral enhanced containment level 2 (CL2) laboratory space.
AHL



Update on the new AHL LIMS

Our laboratory information management system (LIMS) - "Sapphire", from LabVantage - was implemented on May 7. We continue to streamline functions and enhance reports. Projects that are now underway include:

- Improving our invoicing process - invoices now follow completed cases on the next business day, (if tests are added to the case after initial testing is complete, then subsequent invoices will be generated rather than having to make a new case) and monthly statements roll up all cases reported and billed during the previous month - electronic faxing/ emailing of invoices is being built, and should be ready by September.
- Refining the formatting of reports, including indicating the report status as 'results pending', 'interim', or 'final'– adding tests to final reports creates more than 1 'final', so these will be dated.
- We are now able to expand services to include on-line submission as well as Web access to results - please let us know if you are interested. New printed submission forms are on their way this fall as well.
- Our new LIMS will feed into a data warehouse, which will enhance the surveillance capabilities of OMAFRA.

To sign up for access to the client-only section of the AHL website and Web access to your results, please contact:

Dr. Jim Fairles, AHL Client Services Veterinarian (519) 824-4120, ext. 54611 jfairles@lsd.uoguelph.ca

Or: **Ms. Josie Dobing**, Client Services (519) 824-4120, ext. 56071 jdobing@lsd.uoguelph.ca

Client feedback on these recent changes and suggestions for future improvements are most welcome! Thank for you for patience during this major changeover. *AHL*

AHL Newsletter
September 2007 - Volume 11, Number 3

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Editorial Assistant: **Ms. Helen Oliver**

The *AHL Newsletter* is published quarterly (March, June, September, December) by the Animal Health Laboratory, Laboratory Services Division, University of Guelph.

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

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Our continued thanks to all of the non-author AHL clerical, technical, and professional staff who contribute to the generation of results reported in the AHL Newsletter.

Lancet fluke, *Dicrocoelium dendriticum*, (continued from page 24)

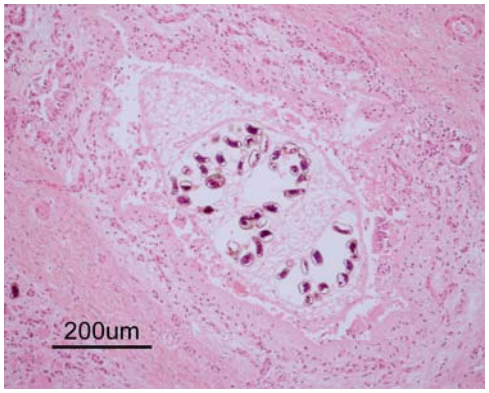

Figure 1: Cross-section of the lancet fluke, *Dicrocoelium dendriticum* within the lumen of a large bile duct, surrounded by a zone of fibrosis. Note the numerous, brown, thick-shelled eggs.

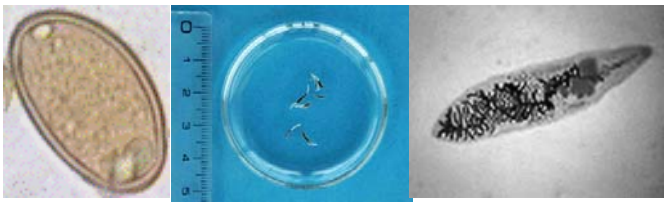

Figure 2: *Dicrocoelium dendriticum* egg (40 x 25 µm) and mature flukes (6-10 x 1.5-2.5 mm).

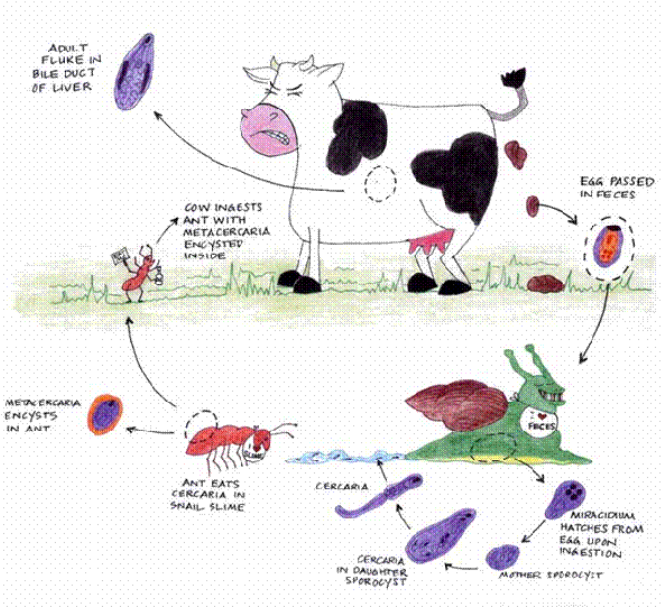

Figure 3: Life cycle of *Dicrocoelium dendriticum* (Image used with the permission of Meghan Petrucci and Thomas P. Buckelew, Ph.D. Professor, Department of Biological and Environmental Sciences, California University of Pennsylvania, California, Pa 15419 http://workforce.cup.edu/Buckelew/)

Accurate and complete submissions data are important contributions to surveillance

Bruce McNab, Jim Fairles, Beverly McEwen

Aggregate animal health surveillance data are increasingly important to trade and the prioritization of research and programs. They also help veterinarians place their client data into context. In fact, surveillance was the theme of the Canadian Association of Veterinary Epidemiology and Preventive Medicine meetings recently held in Edmonton.

Diagnostic laboratory data are an important source of surveillance information. The Animal Health Laboratory's new Laboratory Information Management System (LIMS) and more extensive syndromic coding of submissions will significantly enhance the surveillance system's ability to identify changes or trends in aggregate data at the farm, practice and provincial levels.

It is recognized that laboratory submissions are not representative of the population. However, they do provide an important perspective of situations in which veterinarians are sufficiently concerned to submit samples. The **accurate completion of ALL information, on each and every laboratory submission, is an important contribution** submitters can make to their profession and clients, in support of industry and the public. This includes ALL submission form information concerning: clinic, farm premises, animal and sample identification; numbers of herd size, at risk, sick, dead; date and duration of problem; breed, age, sex, body systems affected, reason for testing, syndromic check-offs, etc. We request that you designate one or a few key people in your clinic (as works best in your clinic) to be responsible for ensuring that all AHL submission forms are filled in accurately and completely.

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We recognize that for logistical reasons, clinic-personnel actually completing submission-forms may not always be as familiar with the farm and case information as would otherwise be ideal. Also we recognize that the same premises may sometimes be recorded over time, as John Doe, or J. Doe, or Doe Farms, etc. Unfortunately, this can lead to erroneous counts of aggregate data in computer systems.

Starting this fall, new AHL submission forms will be distributed to take full advantage of the new AHL LIMS data system. Those forms will include a space to record a unique farm premises ID number. **We ask that you fill in a consistent unique premises ID number or code in the premises ID box on all submissions over time, from each unique farm premises**. If appropriate, your clinic may choose to use its own unique farm premises ID number system. When combined with your clinic ID in LIMS, this will produce a unique premises ID, even if other clinics happen to use the same premises number for one of their clients in another part of the province.

In most cases, it may be appropriate to consistently use the 10-digit premises phone number (land line) in the premises ID box in the AHL submission forms. This has the advantage of being a unique premises ID system that already exits, for which people know their number. The purpose of this is NOT to enable the AHL or OMAFRA to phone farms directly, as we will always work through the submitting veterinarian if additional information is needed. However, for the purposes of accurately aggregating data over time, it will allow us and you (in most, but not all instances), to consistently group data from the same premises. This consistent unique premises ID number (of your own creation or 10-digit phone number) will also allow the AHL to provide you with aggregate data about your clients and help you with easier electronic search capabilities. *AHL*

AHL Lab Reports

RUMINANTS

Hepatic infection with the lancet fluke, *Dicrocoelium dendriticum*, in an Ontario sheep flock

Margaret J. Stalker, Gary Halbert, Paula Menzies, Tony van Dreumel, Ian K. Barker, Andrew S. Peregrine

Tissues were submitted to the AHL from a 4-year-old Rideau-cross ewe from a flock of 25 pastured in Dufferin County, Ontario. The ewe had experienced 4 d of inappetance and depression leading to recumbency prior to death. On post-mortem examination, the ewe was noted to be mildly icteric with a nodular-appearing liver, dark congested kidneys, and melenic feces in the colon. **The tentative clinical diagnosis was copper toxicosis**, although a thorough review of management procedures and pasture failed to implicate a potential source of excess copper. Histopathology of the liver revealed chronic cholangiohepatitis characterized by periportal fibrosis and bile duct proliferation, with cross-sections of trematodes and numerous trematode eggs within the lumens of large bile ducts (Figure 1, p. 22). The eggs were ovoid, approximately 45 µm in length, with thick, brown operculate shells, morphologically consistent with *Dicrocoelium dendriticum*, the so-called lancet fluke (Figure 2). In addition, there was concurrent acute hepatopathy, with hepatocyte single cell necrosis, intracanalicular bile plugs, and acute nephropathy with hemoglobinuria, typical of acute copper toxicosis. ICP analysis of frozen liver revealed elevated hepatic copper concentrations (360 ug/g; reference interval 25-100 ug/g). In this case, copper accumulation was presumed to be secondary, associated with reduced normal biliary copper excretion in this chronically scarred liver, rather than excessive dietary copper intake. Dissection of frozen liver tissue available from this ewe revealed numerous small (<1cm long) transparent white flukes within the lumens of bile ducts. Examination of liver available from a second sheep from this flock that had died earlier in the year from pregnancy toxaemia revealed the presence of similar parasites in bile ducts.

The primary hosts of the lancet fluke are sheep, cattle, deer and rabbits, in which they inhabit the bile ducts and gallbladder, usually causing few overt lesions. The life cycle is intriguing, and differs from other flukes in that there are 2 obligate intermediate hosts, both widely distributed inhabitants of dry land, rather than aquatic environments (Figure 3). Fluke eggs are passed in the primary hosts' feces and hatch when ingested by the first intermediate host, various species of land snail. The miracidia released in the snail's gut develop into sporocysts, then cercariae, and are extruded from the respiratory system of the snail in small slime balls, which are then ingested by brown ants of the genus *Formica*. Metacercariae develop primarily within the ant's hemocoel, although a few reach the subesophageal ganglion, where they trigger a specific behavioural change signalled by cool temperatures. The infected ant, which would normally seek the warmth of its nest in the evening as air temperatures drop, instead is compelled to climb up and clamp its mandibles on the tips of vegetation, increasing the likelihood of ingestion by grazing herbivores. In the primary host, metacercariae hatch in the small intestine, and young flukes migrate up the main bile duct to inhabit smaller ducts in the liver. The prepatent period is 10-12 wk, and the hermaphroditic flukes are long-lived, surviving in the final host for several years. Eggs can also survive for months on dry pasture, and this, coupled with the wide distribution of the terrestrial intermediate hosts, makes control difficult once the parasite becomes established in an area.

In our experience, this is the first published report of hepatic infection with the lancet fluke in Ontario sheep; the actual prevalence and distribution of infection across the province is unknown. A retrospective search of the Ontario Veterinary College pathology case files from 1964-1980 and the AHL pathology database from 1988-2007 revealed only 3 previous diagnoses of hepatic *Dicrocoelium* infection, all in the last 5 y; the first in a pair of Saanen does from the Newmarket area in 2002; the second, a 5-year-old ewe from the Schomberg area in 2003; and most recently, slaughter plant condemnation of affected livers from 2 Holstein cattle pastured in Middlesex County in 2006. The source of the apparent recent appearance of this parasite in Ontario pastures is unknown. White-tailed deer are also known to be primary hosts, and may serve as a potential source of spread of the parasite in the province, while the secondary hosts are ubiquitous in southern Ontario. The clinical significance of infection is usually minimal, however, heavy infestations may cause clinical hepatic disease in small ruminants as evidenced in this case report. In cattle, the chief concern is the potential for liver condemnations at slaughter.

Control measures include removing animals from pastures with high levels of contamination, as control of intermediate hosts is impractical. Anthelmintic therapy with albendazole (Valbazen) or fenbendazole (Panacur, Safeguard), at elevated dose rates, has been reported to be effective in reducing parasite loads. *AHL* *(continued on page 22)*

AVIAN SPECIES

Summary of AHL pathology diagnoses for Ontario poultry, 2006

Emily Martin, Marina Brash, Brian Binnington, Katie Welch, Jan Shapiro, Beverly McEwen

The following is a summary of the pathology diagnoses made during 2006, drawn from the yearly summaries produced from the AHL database of pathology diagnoses made at the AHL-Guelph and AHL-Kemptville laboratories.

Broiler chickens

The top 2 diagnoses in 2006 were **inclusion body hepatitis** (IBH, caused by *Fowl adenovirus*) and **bursal lymphoid depletion**. Bursal lymphoid depletion is a non-specific finding that can have multiple causes including primary infection with viral or bacterial agents or multiple concurrent infections. Both IBH and bursal lymphoid depletion started to increase at the lab around 2002 and 2003 with a higher increase in bursal lymphoid depletion noted at that time. Since then, the diagnosis of IBH has increased and remained steady whereas bursal lymphoid depletion peaked and has declined slightly. In the fall of 2006, an IBH project was initiated and at this time all 50 samples have been collected and further virology testing and data analysis is ongoing.

The next most common broiler diagnoses included *Escherichia coli (E. coli)* septicemia and yolk sacculitis. Other ongoing diagnoses included coccidial and necrotic enteritis, rickets, ascites, spiking mortality syndrome, and cellulitis.

In 2006, 1 case with Infectious laryngotracheitis virus (ILTV) and 1 case with *Avian paramyxovirus 1* (Newcastle disease virus) were identified, and in both cases the virus was found to be indistinguishable from a vaccine virus.

Layer chickens

Osteomalacia (cage layer fatigue), a nutritional disease, was the most common diagnosis followed by **visceral urate deposition and urate nephrosis** (dehydration). Necrotic enteritis (*Clostridium perfringens*) and coccidiosis continued to be identified. Hepatitis/splenomegaly was diagnosed occasionally. Encephalomyelitis compatible with avian encephalomyelitis (AE) was diagnosed in a group of 2-week-old leghorn pullets that had low antibody titres to *Avian encephalomyelitis virus* (AEV), even though the breeder flock was vaccinated. There was 1 other suspect case of AE in 11-day-old birds, also with a history of breeder flock vaccination. One case of ILT was identified in leghorn pullets and was found to be vaccine-related. Very few tumors were diagnosed in 2006.

Broiler breeders

Arthritis/tenosynovitis remained the most common diagnosis, with a variety of agents involved (*Staphylococcus aureus*, *Pseudomonas aeruginosa*, *Enterococcus sp.*). Several cases of bacterial osteomyelitis were diagnosed and a *Streptococcus sp.* was isolated on bacterial culture. Half of the bacterial osteomyelitis cases involved the vertebral column. In the US, *Enterococcus cecorum* has recently been isolated from cases of vertebral osteomyelitis. Other ongoing diagnoses included peritonitis (yolk, bacterial), pneumonia (*E. coli*) and septicemia (*Pasteurella sp.*, *E. coli*). Very few tumors were identified in 2006.

Turkeys

***E. coli* septicemia** and **yolk sacculitis** due to *E. coli*, *Staphylococcus aureus*, *Staphylococcus intermedius*, *Pseudomonas aeruginosa* and *Klebsiella sp.* were the most common turkey diagnoses in 2006. Coccidial enteritis, bacterial pneumonia (*E. coli*, *Staphylococcus sp.*), and round heart (spontaneous turkey cardiomyopathy) were also common diagnoses. Several cases of histomoniasis (blackhead, due to *Histomonas meleagridis*) were identified in birds on range and 4 cases of erysipelas due to *Erysipelothrix rhusiopathiae* were also identified. Mycotic pneumonia continued to be identified, and 1 case of mycotic meningoencephalitis was found in a group of 2-week-old meat turkeys. Ongoing diagnoses included necrotizing enteritis, arthritis, and rickets. Disease diagnoses that have remained low over the last several years included *Bordetella avium* and *Ornithobacterium rhinotracheale* infections.

Pigeons

Late in 2006, a number of farmed pigeons were identified with ***Pigeon paramyxovirus 1*** (PPMV-1, pigeon Newcastle disease). This pigeon version of paramyxovirus is not a reportable disease, however, poultry producers should be aware that this organism exists in Ontario and should take appropriate biosecurity measures to avoid contact with both commercial and wild pigeons. *AHL*

SWINE

PRRSV infection is ongoing in Ontario swine herds in 2007

Susy Carman, Beverly McEwen, Jim Fairles

For Jan-Dec 2006, 33% (619/1842) of all swine cases tested were identified as PRRSV-PCR positive (Fig. 1), which is less than the 45% (682/1536) that tested positive from Jan-Dec 2005. Although the proportion of PRRSV-PCR positive cases has declined since the outbreak began in the fall of 2004, the total number of PRRSV-PCR positive cases increased from 285/553 (52%) cases in 2004, to 619/1842 (33%) cases in 2005 and 682/1536 (45%) cases in 2006. **For Jan-Jun 2007 the proportion of positive cases has remain at about 31%** (294/940).

These data do not include semen monitoring cases. However, it was not possible to exclude serum monitoring cases, such that increases in testing and PRRSV-positive cases may represent enhanced monitoring of swine herds with ongoing infections, rather than new outbreaks. *AHL*

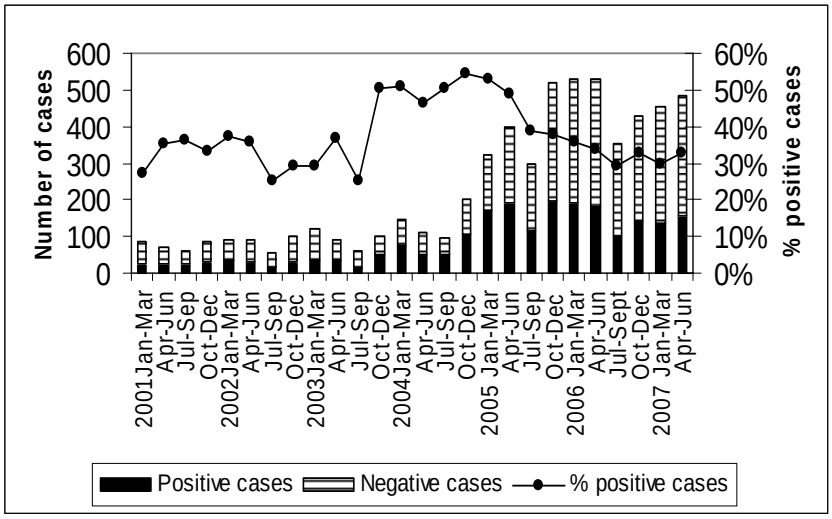

Figure 1. PRRSV PCR submissions tested by the AHL April 1, 2001 to June 30, 2007, with semen monitoring removed.

HORSES

Rabies in a donkey *Josepha DeLay*

A 2-year-old castrated male donkey was euthanized and submitted for necropsy following a 3-day history of self-mutilation, progressive ataxia, muscle fasciculations, inability to urinate, and eventual collapse. No significant gross lesions were evident at necropsy, other than areas of exudative dermatitis corresponding to the sites of self-trauma. Rabies was the primary clinical differential diagnosis. Rabies antigen was identified in brain sections from this donkey by immunofluorescence testing performed by CFIA. Histologically, severe nonsuppurative inflammation was present multifocally at all levels of brain except cerebral cortex, and in spinal cord.

This case highlights the importance of including rabies among the differential diagnoses in neurologic cases involving horses and other species in Ontario. Complacency is inevitable because rabies is diagnosed infrequently in horses; however, the public health implications of rabies and other zoonotic pathogens producing neurologic disease make it imperative to consider these agents. *West Nile virus* and *Eastern equine encephalitis virus* were additional zoonotic pathogens considered in this case, as well as equine protozoal myelitis, cauda equine syndrome, bacterial encephalomyelitis, and trauma.

Rabies has been diagnosed in 7 additional animals submitted to the AHL for necropsy since 1997, including 4 cattle (1 calf, 3 mature cows), 1 mature horse, 1 sheep, and 1 mature unvaccinated dog. Clinical signs of rabies can vary widely in all species and can easily mimic other more common neurologic diseases, and exclusion of rabies on the basis of clinical signs alone can be dangerous. In horses, rabies can mimic colic or lameness referable to a single limb; more common clinical signs include ataxia, paresis, lethargy, and behavioral changes including depression and, less commonly, aggression. Self-mutilation, as seen in case, is sometimes evident in cases of equine rabies.

(continued on page 27)

Rabies in a donkey - continued from page 26

The importance of adequate personal protection and safe tissue handling cannot be overemphasized in equine neurologic cases. At the AHL, necropsies of potentially infectious neurologic cases are conducted in an area of the post-mortem room with little human traffic. Pathologists and technicians wear full-coverage coveralls and HEPA-filter hoods with face shields, to prevent exposure to aerosolized virus and splashes, and Kevlar gloves. Power saws are not used, to prevent aerosolization of virus. Rabies and other zoonotic viruses are tested for and excluded prior to proceeding with additional tests, in order to avoid unnecessary and potentially dangerous exposure of personnel to infectious tissues. We do proceed with histologic examination of tissues, as formalin will neutralize *Rabies virus* and other zoonotic pathogens, rendering formalin-fixed tissues safe for processing. *AHL*

Four interesting equine cases from eastern Ontario *Jan Shapiro, Brian Binnington*

We are reporting 4 interesting equine necropsy cases which were submitted to the Animal Health Laboratory in Kemptville in the spring of 2007.

Case 1: A 1-year-old Miniature horse from a herd of 30 died after 4 d of profuse watery diarrhea. No other horses were affected. The horse was in fair body condition with no depot fat stores. The contents of the small intestine, cecum and colon were liquid green-brown. The wall of the distal half of the small intestine was thickened with a prominent ridged appearance of the mucosal and serosal surfaces. Histologically, the jejunal and ileal crypts were elongated and Warthin-Starry silver staining demonstrated a few to numerous argyrophilic comma-shaped bacteria in the cytoplasm of epithelial cells. The gross and microscopic lesions were compatible with **proliferative enteropathy due to *Lawsonia intracellularis*.** Clinical disease is most frequent in 4- to 6-month-old weanlings. The infection route is fecal-oral and it is likely that foals become infected from herdmates shedding the organisms. Various stressors are believed to increase susceptibility to infection. Proliferative enteropathy is sporadically diagnosed in multiple breeds of horses in Ontario.

Case 2: A 3-year old Paint gelding had an acute history of severe diarrhea, edema of the legs and ventral body, and respiratory signs. Weight loss had been reported for 2 mo preceding the other clinical signs. Serology for equine infectious anemia was negative. The horse was in poor body condition, and clear yellow edema fluid was present in the subcutaneous tissues along the ventral portions of the body. The cecum and colon contained liquid brown contents and the mucosa was grey to black with thickened irregular ridges. Mucosal scrapings and wet mount smears demonstrated small-sized larval and adult nematodes. On microscopic examination, there was chronic typhlitis and colitis with localized ulcerations, edema and fibrosis in the mucosa and submucosa. Sections of nematode larvae were present in the lamina propria and in submucosal granulomas. **The typhlocolitis was compatible with larval cyanthostomiasis** caused by the synchronous emergence of hypobiotic L4 larvae in large numbers in late winter and early spring.

Case 3: An 8-month-old zebra had a 10-d history of partial anorexia, depression, gingivitis and severe anemia. At necropsy, the zebra was emaciated, and there was acute moderately severe multifocal erosive stomatitis, gingivitis, gastroenteritis and dermatitis. The liver was mottled and petechiated, and there were scattered white parenchymal foci. Histologically there was acute severe multifocal necrotizing hepatitis, splenitis, dermatitis, gastroenteritis and pneumonia, associated with acute vasculitis and vascular thrombosis. Many cells had intranuclear eosinophilic viral inclusion bodies. **The fluorescent antibody test of lung for *Equid herpesvirus 1* (EHV-1) was positive, and EHV-1 was isolated from pooled tissues**. Systemic herpesviral infection is observed in neonatal foals, and pulmonary endotheliotropic systemic EHV-1 infection has been reported in zebras. However, the lesions in this zebra were not restricted to the lungs, and were even more widespread than is usually seen in foals. Involvement of the skin was particularly dramatic, and is uncommon in reports of horse cases.

Case 4: A 1-day-old Hanoverian colt had a history of not having stood since birth, being weak in the hindquarters, and having large patches of skin sloughing from all pressure points. There was dramatic excessive looseness and tenting of the skin of the head, neck, sternum and lower limbs. There was excessive extensibility and flexibility of multiple joints, including the carpi, fetlocks, pastern, hips and atlanto-occipital joint. Cartilage in the pinnae of both ears was excessively flexible. There were multiple skin tears and large defects over the carpi, the hocks and the withers, with marked separation of the subcutis from deeper soft tissues for several cm around these defects. The soft tissues showed ecchymotic hemorrhage and mild edema. Multiple linear mucosal defects (tears) were in the oral cavity. The hypermotile joints had stretched joint capsule and a moderate excess of clear synovial fluid. Routine histopathology of skin showed loosely packed and disorganized dermal collagen, and in some sections, horizontal splitting through the deep dermis and acute hemorrhage. **The gross and histopathology findings were consistent with a dermal collagen defect disease**. This case has some features in common with a heterogeneous group of connective tissue disorders, Ehlers-Danlos syndrome, which has been reported in humans and other species. Some of the clinical features and age of onset were different than what is reported for hereditary equine regional dermal asthenia. There have been no other cases reported in other foals from this mare or other offspring from this stallion. *AHL*

COMPANION ANIMALS

Screening dogs in Ontario for *Borrelia burgdorferi* and *Ehrlichia canis* should be selective rather than routine

Andrew S. Peregrine, Ian K. Barker, Anthony C.G. Abrams-Ogg, J. Paul Woods

(Originally published in the July 2007 issue of the Canadian Veterinary Journal (volume 48, page 673) - reprinted with permission of the CVMA)

Recently, IDEXX Laboratories and Wyeth Animal Health held workshops across Ontario entitled "Lyme disease for the canine practitioner." At the Kitchener meeting, data were presented from 53,541 dogs screened with the SNAP® 3Dx® Test (IDEXX Laboratories) in 2006. Seventy-five per cent (40,015) were from Ontario, 50 of which were positive for exposure to the Lyme disease agent; the authors concluded that "This study demonstrates that a significant percentage of dogs in Canada have been exposed to *Borrelia burgdorferi*" (1). The take-home message was that Ontario dogs should routinely be screened for *B. burgdorferi* and *Ehrlichia canis*, and that dogs exposed to *B. burgdorferi* should be treated with antibiotics.

In our opinion, the risk of exposure to *B. burgdorferi* in dogs that have not travelled outside Ontario is very low. Adventitious *Ixodes scapularis* (vector ticks dropping off migrating birds) may be encountered uncommonly anywhere in Ontario, but only 10-12% are infected with *B. burgdorferi*. Exposure to *B. burgdorferi* in Ontario is most likely to be associated with travel to locations with established *I. scapularis* populations: Point Pelee, Rondeau, Long Point and Turkey Point (all Lake Erie), Long Point - Prince Edward County, and the Thousand Islands (2). *Ehrlichia canis* infection is an extremely uncommon diagnosis in Ontario dogs that have not travelled, since its vector, *Rhipicephalus sanguineus*, prefers a warmer climate.

The very low risk of infection with *B. burgdorferi* and *E. canis* was reflected in a recent study of sera from dogs with suspicion of tick-borne illness submitted by Ontario and Quebec veterinarians; prevalence of antibody to *B. burgdorferi* and *E. canis* was 2/108 (1.9%) and 1/271 (0.4%), respectively (3). Unfortunately, the travel history was not available for most of the dogs. Over the last 5 yr, there has been no diagnosis of Lyme disease in dogs at the Ontario Veterinary College, and just 1 case of canine ehrlichiosis (travel-associated). This correlates well with the low prevalence of exposure to *B. burgdorferi* (0.12%) and *E. canis* (0.02%) in the Ontario dogs screened with the SNAP 3Dx Test in 2006 (1).

The reported sensitivity and specificity of the SNAP® 3Dx® Test for detection of exposure to *B. burgdorferi* are 94.4% and 99.6%, respectively (4,5), indicating that the test is extremely good. However, if the prevalence of such exposure in Ontario dogs is truly 0.12% (1), **the predictive value of a positive test is 22% (6); that is, more than 75% of positive test results will be false positives**. Thus, unless confirmatory testing is carried out with a different test, unexposed dogs are at risk of being treated unnecessarily with antibiotics. The same concern applies to treatment of dogs with genuine subclinical *B. burgdorferi* infections, because the benefit of treating such dogs is uncertain (7,8).

In summary, **the rationale for routinely screening healthy Ontario dogs for exposure to *B. burgdorferi* and *E. canis* is highly questionable**. Any testing should be based on an evaluation of the clinical syndrome, risk of infection, and benefit of treatment, on a case-by-case basis.

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