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LABORATORY SERVICES

Animal Health Laboratory

AHL Newsletter

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Lab profile - AHL Toxicology

Nick Schrier, Brent Hoff

- Full service, proficiency-tested, accredited lab (AAVLD, ISO 9001-2008, ISO 17025)
- In-house expertise and experience
  - Brent Hoff, DVM, DVSc, DipTox, Veterinary Toxicologist
  - Nick Schrier, MSc, Analytical Chemist
  - 4 full-time experienced technical staff
- Pesticide analysis, including insecticides, herbicides, rodenticides and avicides in suspect material, GI contents, tissue.
- Environmental pollutants and industrial chemicals, ethylene glycol, heavy metals, chlorinated hydrocarbons.
- Mycotoxin instrumental analysis, aflatoxins (B1, B2, G1, G2), zearalenone, and ochratoxin A by immunoaffinity column cleanup/HPLC, trichothecenes (DON, DAS, T-2, etc.) by GC/MS in a variety of feeds and feed ingredients.
- Mycotoxin ELISA - DON, zearalenone, ochratoxin, aflatoxins, fumonisins.
- Feed additives - monensin, narasin, salinomycin, lasalocid, in feed and GI contents.
- Heavy metals analysis - extensive elemental panels for feed, plant and animal tissue, blood, environmental samples.
- Nutritional elemental analysis, elemental panels for feed, blood, serum, tissue.
- The toxicology section has a wide range of high tech analytical instrumentation - GC/MS, LC/MS, ICP/MS - available to investigate any suspect food or companion animal toxicoses and mineral deficiencies.

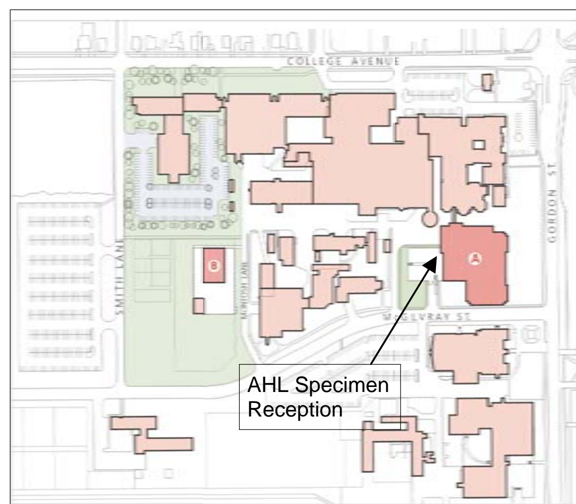
For full list of tests, please see our AHL Fee Schedule, or contact Dr. Hoff bhoff@uoguelph.ca or Nick Schrier nschrier@uoguelph.ca *AHL*

(Continued on page 28 - Photo of the Toxicology team)

We're moving!!

- The Animal Health Laboratory and the Department of Pathobiology will be moving into our new building **during the month of October, 2010.**
- We have possession of the building as of Sept 27, and some equipment and furnishings will already be in place.
- Throughout October, we will be moving labs and offices sequentially, attempting to cause as little disruption to client service as possible.
- We plan to keep all labs functional throughout the move, but there may be unplanned delays.
- If you have any rush submissions during the period of the move, feel free to call us and discuss options.

We look forward to serving you from our new facility!



How are we doing? Quality audits

Nadine Ryan

Evaluation is an integral part of our quality management system. We evaluate ourselves continuously through our internal audit program, inter-laboratory sample comparison and proficiency testing programs. As well, we are evaluated by professionals via external assessments of the AHL - see bullets below. We successfully passed all inspections. **The AHL quality system and testing meet international standards and we welcome external audits to help us improve.**

- April 2009. On behalf of the American Association of Veterinary Laboratory Diagnosticians (AAVLD), 4 experienced veterinary laboratory assessors audited all sections of AHL over 2 days - the **AAVLD standard**, "*Requirements for an accredited veterinary medical diagnostic laboratory*", is the World Organization for Animal Health (OIE) version of the ISO/IEC 17025 standard. The AAVLD Accreditation Committee granted the AHL full accreditation for all species.
- September 2009. AHL Molecular Biology and Toxicology sections were assessed by Standards Council of Canada (SCC) to the **ISO/IEC 17025 standard** "*General requirements for the competence of testing and calibration laboratories*" for 7 tests listed on the University of Guelph, Laboratory Services scope of accreditation. All audit findings were resolved within 45 days.
- January 2010. BSI, our ISO 9001 registrar, assessed AHL Guelph and Kemptville to the **ISO 9001:2008 quality standard** "*Quality management systems – requirements*". The AHL met all requirements and their assessors' comments on AHL laboratory sections: "Overall process is well managed".
- April 2010. The Canadian Food Inspection Agency (**CFIA**) Retrovirology Centre of Expertise inspected the equine infectious anemia (EIA) test. The CFIA auditor examined 2 years of EIA records and observed the performance of the test. AHL successfully maintained the CFIA accreditation of the EIA test. The auditor's comment: "Good application of ISO/IEC 17025 guidelines."
- July, 2010. The Section Chief Veterinarian, Department of Animal and Plant Inspection & Quarantine, Hangzhou Entry-Exit Inspection & Quarantine Bureau of The People's Republic of **China**, inspected AHL swine influenza testing prior to China accepting AHL test results to support shipment of pigs to China.
- The AHL participates in inter-laboratory sample comparison and proficiency sample programs. In the 2009/2010 fiscal year, the AHL participated in 50 biological proficiency panels with a success rate of 100%.

These internal and external evaluations give us confidence in our laboratory testing and identify areas that need improvement. We welcome your own evaluation through our annual client survey and daily communication. *AHL*

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Editor: **Grant Maxie,** DVM, PhD, Diplomate ACVP
Editorial Assistant: **Ms. Helen Oliver**

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Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends. *All material is copyright 2010. Ideas and opinions expressed herein do not necessarily reflect the opinions of the University or the editor.*
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Mailing address & contact information:
(**please return all undeliverable Canadian addresses to**:)
Animal Health Laboratory
Laboratory Services Division, University of Guelph
Box 3612, Guelph, Ontario, Canada N1H 6R8
Phone: (519) 824-4120 ext. 54538; fax: (519) 821-8072
Email: holiver@uoguelph.ca

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Contributors to this issue:
From Laboratory Services / Animal Health Laboratory:
Pam Brown, BSc
Hugh Cai, DVM, MSc, DVSc
Susy Carman, DVM, PhD
Patsy Graham, DipEnvEng
Murray Hazlett, DVM, DVSc, Diplomate ACVP
Brent Hoff, DVM, DVSc, DipTox
Helen Kocmarek, BSc Hons, MLT
Andrew Moore, MSc
Kristiina Ruotsalo, DVM, DVSc, Diplomate ACVP
Nadine Ryan, BSc
Nick Schrier, MSc
Maria Spinato, DVM, DVSc, MBA, Diplomate ACVP

Other contributors:
Michael Taylor DVM, OVCTH; **Fernanda Castillo-Alcala**, DVM, DVSc, **Jeff Caswell**, DVM, DVSc, PhD, **Dale Smith** DVM DVSc, Pathobiology; **Ken Bateman**, DVM, MSc, Population Medicine, OVC
Erin Wilson, DVM, Lindsay, ON

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.

PCR and IHC tests currently available at the AHL

PCR (polymerase chain reaction)

16S rRNA gene sequencing for bacterial ID
Actinobacillus pleuropneumoniae - PCR
Aleutian disease virus - PCR
Avian adenovirus - PCR
Avian bornavirus - RT-PCR
Bluetongue virus - multiplex real-time RT-PCR
Brachyspira hyodysenteriae - PCR
Brachyspira hyodysenteriae and *Brachyspira pilosicoli* - PCR profile
Brachyspira pilosicoli - PCR
Brachyspira spp - PCR
Canine distemper virus - real-time RT-PCR
Chlamydophila (*Chlamydia*) spp - PCR
Chlamydophila abortus - real-time PCR
Chlamydophila psittaci - real-time PCR
Coxiella burnetii - real-time PCR
E. coli, VTEC (verotoxigenic) typing, O157/O111 - PCR
E. coli, VTEC PCR
Equid herpesvirus 1 - real-time PCR
Equine encephalitis virus (EEEV/WEEV) - RT-PCR
Infectious bursal disease virus - RT-PCR
Infectious bronchitis virus - RT-PCR
Infectious laryngotracheitis virus (ILTV, *Gallid herpesvirus 1*) - PCR
Influenza A virus, avian, H5, food animal - real-time RT-PCR
Influenza A virus, avian, H7, food animal - real-time RT-PCR
Influenza A virus, avian, matrix - real-time RT-PCR
Influenza A virus, canine - real-time RT-PCR
Influenza A virus, equine - real-time RT-PCR
Influenza A virus, swine - real-time RT-PCR
Influenza A virus, swine - RT-PCR, gel, virus typing
Mycobacterium paratuberculosis - PCR
Mycobacterium spp.16S - PCR
Mycoplasma bovis - real-time PCR
Mycoplasma haemocanis/haematoparvum - PCR
Mycoplasma haemofelis and *M. haemominutum* - PCR
Mycoplasma hyopneumoniae - PCR
Mycoplasma iowae RAPD typing
Mycoplasma iowae - real-time PCR
Newcastle disease virus - RT-PCR
PRRSV, 609 bp - RT-PCR/RFLP
PRRSV, 716 bp - RT-PCR/RFLP
PRRSV, 933 bp - RT-PCR/RFLP
PRRSV, Next Generation NorthAm/Eur - rt-RT-PCR (Tetracore)
PRRSV, ORF5 - gene seqencing, RT-PCR
Salmonella Enteritidis - PCR
Salmonella spp- PCR
Salmonella Typhimurium DT104 - PCR
Scrapie resistance PrP genotyping, codons 136, 154, 171 - real-time PCR
Streptococcus equi, companion/other - SeM gene PCR
Tritrichomonas foetus - PCR
West Nile virus - real-time RT-PCR

Immunohistochemistry - infectious agents

Bovine coronavirus (CCV)
Bovine herpesvirus 1 (BoHV-1, IBRV)
Bovine respiratory syncytial virus (BRSV)
Bovine viral diarrhea virus (BVDV)
Canine coronavirus (CCV)
Canine distemper virus (CDV)
Canine parvovirus 2 (CPV-2)
Chlamydophila spp
Equid herpesvirus 1 (EHV-1)
Feline coronavirus (FCoV)
Feline panleukopenia virus (Feline parvovirus)
Influenza A virus
Leptospira spp
Mycoplasma bovis
Neospora caninum
Porcine circovirus 2 (PCV-2)
Porcine reproductive and respiratory virus (PRRSV)
Transmissible gastroenteritis virus (TGEV)
Toxoplasma gondii
West Nile virus (monoclonal / polyclonal)

Immunohistochemistry - cell markers

Actin, muscle
Actin, smooth muscle
Calcitonin
CD18 (canine)
CD 18 (feline)
CD3
CD31
CD79a
CD117 (c-kit)
Chromogranin
Cytokeratins: pan CK (AE1/AE3), CK7, CK20, HMW CK
Desmin
FVIII-related antigen (von Willebrand factor)
Glial fibrillary acidic protein (GFAP)
Hepatocyte marker (HepPar 1)
Ki-67
Lambda light chains
Melan A
Major histocompatibility complex II (MHC II)
Neuron-specific enolase (NSE)
S100
Synaptophysin
Thyroglobulin
Thyroid transcription factor (TTF)
Tryptase
Uroplakin
Vimentin

AHL Lab Reports

RUMINANTS

Mycoplasma bovis in respiratory disease of feedlot cattle

Jeff Caswell, Ken Bateman, Hugh Cai, Fernanda Castillo-Alcala

(Abstract reprinted with permission from Vet Clin Food Anim 2010;26:365–379.)

Mycoplasma bovis has emerged as an important cause of respiratory disease and lameness in beef cattle. Pulmonary infection is uncommon when calves arrive in feedlots, but is widespread after several weeks. Clinical features do not differentiate *M. bovis* from other causes of bacterial pneumonia, although chronic respiratory disease, recurrent treatment failure, and lameness caused by arthritis or tenosynovitis are suggestive.

Mycoplasma bovis causes chronic caseonecrotic bronchopneumonia, characterized by cranioventral pulmonary consolidation with multiple raised white friable foci of caseous necrosis. Although *M. bovis* probably causes other forms of bronchopneumonia, its importance in such situations has been difficult to assess. It can act as a primary pathogen, yet many cases are co-infected with other bacteria or viruses, and evidence suggests that *M. bovis* colonizes and perpetuates lung lesions that were initiated by other bacteria, such as *Mannheimia haemolytica*. BVDV appears to predispose to bacterial pneumonia generally rather than to *M. bovis* pneumonia in particular.

Simply identifying *M. bovis* in the lung of a calf with pneumonia does not necessarily indicate that *M. bovis* was the cause, and the diagnosis is based on gross, histologic, and immunohistochemical findings. *Mycoplasma bovis* elicits a robust humoral immune response, but the resulting antibodies are not protective because of the variable surface proteins, and vaccines have not yet been shown to prevent disease. *Mycoplasma bovis* infections are responsible for a high proportion of the chronic disease occurring in feedlots, and the welfare of such animals is an important aspect of feedlot health management.

Copper toxicosis in goat kids due to ingestion of copper sulfate

Maria Spinato, Erin Wilson, Brent Hoff

Several 2- to 4-month-old kids on a large dairy goat operation were found dead without prior signs of illness. The referring veterinarian performed field necropsies on three dead kids, and noted that the carcasses were markedly icteric. Livers were subjectively enlarged and green-bronze. Fresh and formalin-fixed tissue samples were collected and submitted to the AHL in Guelph.

No pathogenic bacteria were isolated. Histologic examination of liver revealed dissociation of hepatic cords, widespread single cell necrosis, and regional acute periacinar necrosis. Cholestasis was a prominent feature, with extensive plugging of bile canaliculi in affected zones. A diagnosis of acute hepatic necrosis with marked cholestasis was made, most compatible with exposure to a hepatotoxic substance. The most commonly implicated toxins in small ruminants in Ontario include copper, and phytotoxins such as blue-green algae and certain *Brassica* spp. plants.

Upon further inquiry, the producer reported administering copper sulfate in the drinking water of the young kids for topical therapy of a severe outbreak of contagious ecthyma (orf). **Samples of formalin-fixed liver are suitable for mineral analysis**, and the remaining pieces of liver were forwarded to our toxicology laboratory - the copper level was 3,800 ug/g (ppm) dry weight. Normal reference intervals for copper level in caprine liver are estimated to be 75-450 ug/g (ppm) dry weight; values greater than 1,000 ug/g (ppm) would be indicative of potential toxicity.

Goats are more resistant to copper toxicity than sheep, and are considered similar to cattle in their ability to tolerate and store comparatively higher levels of ingested copper. The most common sources of excess copper are inappropriate trace mineral formulations (high copper and/or low molybdenum), feeding of poultry litter, or ingestion of copper sulfate, which is widely used in agricultural operations as an algicide, copper supplement, and footbath treatment for foot rot. Historically, copper sulfate has also been used as an anthelmintic in small ruminants, until its role in inducing copper toxicity in sheep was realized.

In this goat herd, copper sulfate had been safely used for several years in the drinking water to hasten the healing of vesicles and scabs caused by Orf virus, and toxicity had not been reported previously. This year, the contagious ecthyma outbreak was particularly severe, and factors such as overcrowding, increased dosing, and excessive drinking during the recent heat wave are believed to have contributed to ingestion of toxic levels of copper. Approximately 2% of this year's kid crop died of acute copper toxicosis prior to removal of copper sulfate from the drinking water, and 1-2 kids per week are reported to be dying of complications related to chronic toxicosis. AHL

SWINE

New swine Rotavirus group A/C real-time RT-PCR and swine Rotavirus group B RT-PCR tests available at the AHL

Susy Carman

Rotavirus group A accounts for 50% of rotavirus diarrhea in pigs. The remaining rotavirus infections are a result of *Rotavirus group B* and *Rotavirus group C* infections. These viruses cannot be cultivated in cell culture, such that no diagnostic immunological reagents are available to demonstrate these enteric viruses. PCR offers an important improvement in the ability to diagnose rotavirus infections in swine. The AHL now offers PCR tests for Rotavirus group A, B, and C. The protocols for these tests and *Rotavirus group B* and *C* positive control gut homogenates were a gift from the University of Minnesota. These tests were developed and initially validated in their laboratories.

1. ***Rotavirus group A/C* real time RT-PCR.** This new multiplex real-time RT-PCR detects and differentiates both Rotavirus group A and Rotavirus group C using 3 real-time probes. The test is performed using frozen feces, intestinal contents, or jejunum. Swabs are not acceptable. The cost is $40 per test.

2. ***Rotavirus group B* RT-PCR**. This gel based RT-PCR detects only Rotavirus group B. The test is performed using frozen feces, intestinal contents, or jejunum. Swabs are not acceptable. The cost is $29 per test.

For more information on the use of these PCR tests, please contact Dr. Susy Carman at 519-824-4120 ext 54551 or at scarman@uoguelph.ca *AHL*

Porcine rotavirus – a pathologist's perspective

Murray Hazlett

A histologic diagnosis of porcine rotavirus is often frustrating for practitioners and pathologists alike! Submission of a good, non-autolyzed specimen tends too often to end up with a diagnosis of "Atrophic enteritis, likely viral (TGE or rotavirus)". Although we do have an excellent immunohistochemistry (IHC) test for TGE, we do not have an IHC test for rotavirus in pigs. Our commonly used rotavirus latex agglutination test (RLA) will detect only group A rotavirus, which accounts for only about 50% of rotavirus infections in swine.

Careful histologic examination of freshly fixed small intestine may occasionally reveal typical syncytial cells within the epithelium (villus epithelial cells stuck together) (Figure 1), which, when seen, are associated with rotaviral infections. These are evident only occasionally, and then only if the samples are from very early in the disease. When these are present, the pathologist will be almost certain they are dealing with rotavirus, even with a negative RLA. Because of these problems, **we are delighted to have new tests that will detect group A, B and C rotavirus.**

As a reminder of the importance of careful sample selection, **it is important to select animals for testing in the early stages of infection, within the first 24 hrs of the onset of diarrhea,** as infected cells are rapidly lysed and lost, and therefore unavailable to support a diagnosis. In addition, compare the 2 photos shown, one being freshly fixed tissue (Figure 1) taken immediately after death, and the other being autolyzed intestine (Figure 2) taken a few hours after death, and you can see why it is important to fix intestine as soon as possible after death to increase chances of a definitive diagnosis. We would have no chance of seeing syncytial cells in the second specimen. *AHL*

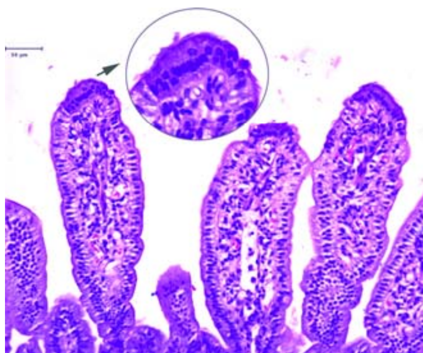

Fig 1. Syncytial cells (darker, small clumps of cells) at villus tips of small intestine. Inset is close-up of same.

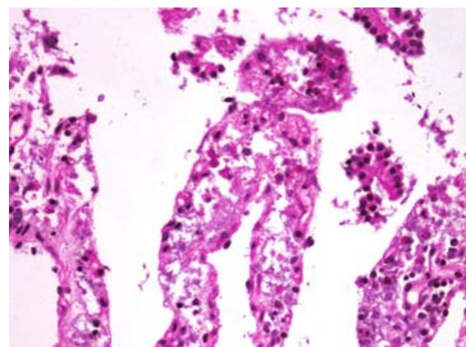

Fig 2. Small intestinal villi with sloughing of enterocytes due to autolysis.

AVIAN/FUR/EXOTIC SPECIES

Potassium chloride as a euthanasia agent in psittacine birds when histopathologic assessment of tissues is desired

Michael Taylor, Dale Smith

Barbiturates and intravenous T-61 are two commonly used agents for the euthanasia of birds but both frequently cause extensive tissue damage, especially to the lungs, interfering with the identification and interpretation of histopathologic changes. A group of researchers from the Avian and Exotic Service, Veterinary Teaching Hospital and the Department of Pathobiology of the Ontario Veterinary College, University of Guelph, including Dr. Raj Raghav, Dr. Michael Taylor, Mr. Mark Guincho and Dr. Dale Smith, evaluated the clinical and histopathological aspects of the use of potassium chloride (KCl) as a euthanasia agent. The use of KCl for euthanasia is acceptable to the American Veterinary Medical Association and the Canadian Council on Animal Care as long as the animal is under general anesthesia at the time of injection.

Their results showed that psittacine birds could be euthanized by intravenous injection of KCl at 3 mEq/kg bodyweight. All birds had been placed under at least a moderate plane of isoflurane anesthesia prior to KCl administration; however some muscular activity was still seen. Tissues including heart and lung showed no artifacts attributable to the euthanasia agent (Figure 1).

In conclusion, when birds are euthanized with the intention of submitting tissues for histopathologic evaluation, **this team of researchers recommends a moderate plane of anesthesia followed by intravenous injection of KCl at 3 mEq/kg bodyweight to improve the diagnostic value of histopathology.**

The full length manuscript entitled "Potassium chloride as a euthanasia agent in psittacine birds: Clinical aspects and consequences for histopathologic assessment" with the details of their findings has been accepted for publication in the Canadian Veterinary Journal. *AHL*

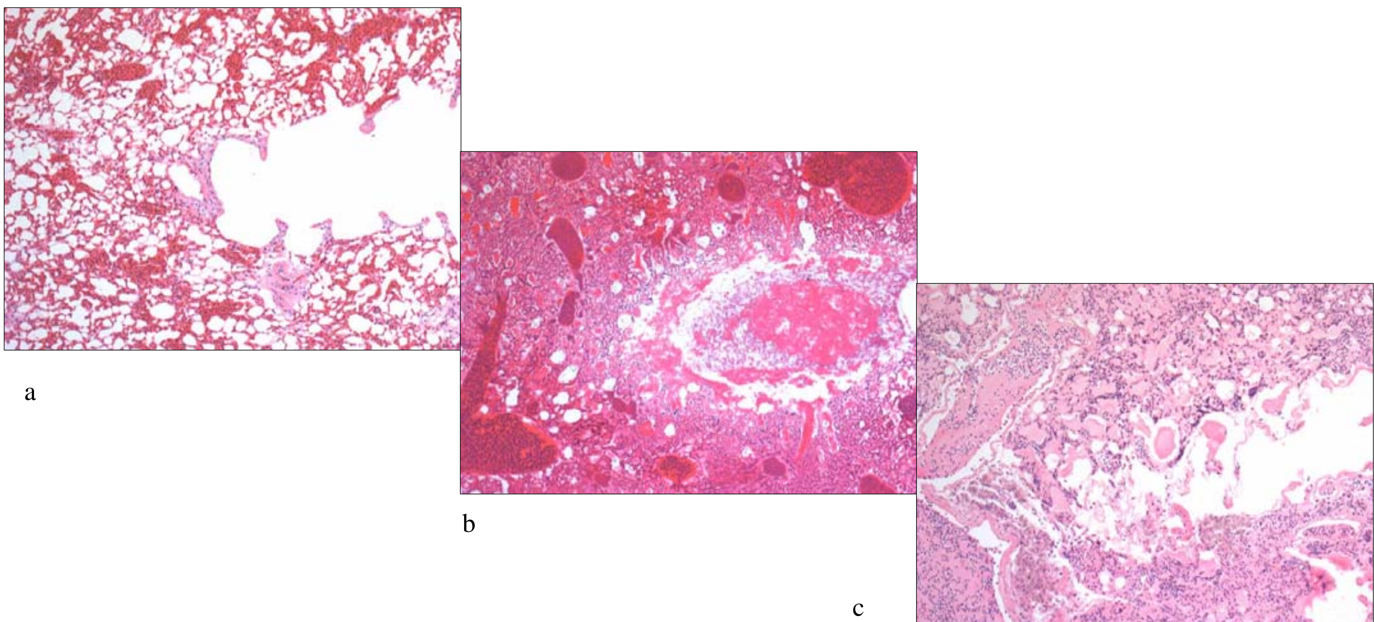

Figure 1. Microscopic images of the lungs of psittacine birds euthanized with 3 different euthanasia agents. Note the excellent preservation and pulmonary anatomic detail seen with (a) potassium chloride (KCl), compared with birds euthanized with (b) pentobarbital sodium, or (c) T-61. The latter 2 agents show lysis of erythrocytes, bare erythrocyte nuclei and nuclear fragments. Eosinophilic granular material and fluid are accumulating in parabronchi, atria and air capillaries.

HORSES

Validation of serum amyloid A (SAA) for evaluation of health and disease in horses

Brent Hoff, Helen Kocmarek, Kristiina Ruotsalo

Serum amyloid A (SAA) is a major equine acute-phase protein that is produced by the liver in response to harmful inflammatory or infectious stimuli. Increased hepatic production occurs within 4 t o 5 hours of a stimulatory event, leading to increased serum levels that are a reflection both of protein production and peripheral catabolism. SAA is postulated to play a role in cholesterol transport, platelet function, neutrophil function and the modulation of inflammatory processes. Increased blood concentrations of SAA are noted in horses suffering from a variety of conditions including viral or bacterial infection, exposure to endo- or exotoxins, inflammatory disease, neoplasia, trauma or surgery.

Baseline SAA values in healthy horses are negligible (often almost 0), but blood concentrations increase rapidly and dramatically (often exceeding 1,000 mg/L) in response to infection and inflammation. Although infectious stimuli generally elicit a greater response than inflammatory stimuli, the increase in blood concentrations is non-specific, and has been documented with a variety of conditions including inflammation involving the airways, gastrointestinal tract, reproductive organs and the musculoskeletal system. Surgical stimuli and parturition have also been shown to result in elevated SAA concentrations. The clearance of SAA from the blood is also rapid, thus the high levels accompanying active inflammatory or infectious conditions will decrease quickly after the eliciting stimulus has subsided, or after successful treatment has been initiated. Relapse or clinical deterioration will result in sustained increases in SAA. Therefore, serial SAA determinations may be used to gauge response to treatment and possibly as a predictor of recovery from illness.

SAA is a valuable adjunct to the other inflammatory markers that are already available in equine medicine. Several studies have indicated that SAA is even more sensitive than the classic inflammatory markers fibrinogen and total leukocyte count; increases in SAA occur much earlier in the course of disease, thus facilitating timely clinical intervention or treatment.

You will notice SAA as additional test offered with the comprehensive equine biochemistry profile. Hopefully this acute phase protein will prove to be a valuable diagnostic tool in your clinical practice. *AHL*

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COMPANION ANIMALS

Isoniazid toxicosis in 2 dogs

Murray Hazlett, Brent Hoff, Nick Schrier, Andrew Moore, Pam Brown, Patsy Graham

Two small breed dogs that were in a fenced yard were found in distress by the owner. One dog was frothing at the mouth, began to seizure, and then died. The second began to show similar signs and died within 1 hr of being admitted to a veterinary clinic. The owner reported seeing the dog eating a hot dog in the back yard. Clinically dog 2 had bradycardia with a pulse of 90 and a capillary refill time of 4 sec. She was ataxic, drooling, with mydriasis and was vomiting bile, with 3 seizure events occurring within 5 min. A tentative diagnosis of organophosphate or carbamate toxicosis was made, however the dog died before treatment was initiated.

Both dogs were submitted for necropsies at the AHL, and were accompanied by a piece of wiener found in the backyard and that had a white granular residue on it.

At necropsy, both dogs were found to be in good condition, well cared for, and had body weights of 6.5 and 6.0 kg. Both had marked acute pulmonary edema and congestion, and stomachs and intestines were empty.

Microscopically, there was acute multifocal hemorrhage in brain, kidney, and thymus. Lack of lesions was felt to be compatible with the clinician's diagnosis of organophosphates or carbamates.

Gas chromatography-mass spectrometry (GC-MS) testing revealed the crystalline material on the hot dog to be isoniazid, also known as isonicotinylhydrazine, a drug used for the treatment of tuberculosis in humans. The drug was not detected in vomitus or tissues from the dogs. Organophosphates and carbamates were not detected.

Although it appears that this is a case of malicious exposure, it is an unusual choice for a toxin due to its limited availability to the general public and because most people are unaware of the sensitivity of dogs to this drug. Although we were unable to detect the drug in the dogs, we suspect this may be due to the prompt elimination of the drug via vomiting, and the relative insensitivity of the test. **The fact that the dog was seen eating the hot dog, and the clinical signs seen in these dogs is supportive of a diagnosis of isoniazid toxicosis.**

In the United States, the National Animal Poison Control Center received 28 calls of isoniazid exposures in dogs and cats between 1987 and 1993. Isoniazid has a low therapeutic margin and produces life-threatening signs in dogs ingesting single 300 mg human tablets. The LD_{50} in dogs is estimated at 50 mg/kg BW, which is probably similar to that for humans. The most consistent clinical signs reported were recurrent clonic-tonic seizures followed by a stuporous state with poor response to stimulus. Ideal treatment combines vitamin B6 (pyridoxine) given as a single IV bolus at an equivalent dose to the amount of ingested isoniazid and anticonvulsants such as 1 mg diazepam/kg BW. This combination acts synergistically to improve GABAergic transmission in the CNS and has proved effective in protecting animals from further convulsions and death, even after several seizure episodes, as often encountered in clinical situations.

On the ASPCA website, isoniazid is listed among the top 10 human medications that poison pets. *AHL*

Reference

Villar D, et al. Treatment of acute isoniazid overdose in dogs. Vet Hum Toxicol. 1995;37:473-477.

From page 21, Lab profile - AHL Toxicology:

AHL Toxicology staff: Back row, left to right: Michael Morrison, Patsy Graham, Nick Schrier, Dr. Brent Hoff. Front row, left to right - Heather Harris, Sue Couling.

AHL Newsletters and LabNotes are available on the Web at - http://ahl.uoguelph.ca