Laboratory Services Division
Animal Health Laboratory
University of Guelph
Changing Lives Improving Life

AHL Newsletter

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In this issue:

Dr. Susy Carman retires

Dr. Susy Carman, AHL mammalian virologist, is retiring effective December 31, 2012. Susy joined the Veterinary Laboratory Services Branch of OMAFRA in 1985. She continued in this position with the transfer in 1997 to the Animal Health Laboratory at the University of Guelph. Susy has been a tireless worker in her field, and in collaboration with VLSB/AHL technical staff over the course of 27 years, greatly expanded our offerings of mammalian virology tests. Her work included the development of many molecular biology tests in support of the animal industries of Ontario. She recently supervised the certification of 7 of our virology tests to the ISO/IEC 17025 standard, audited by the Standards Council of Canada.

We will miss Susy's wealth of expertise, and wish her all the best in her retirement. *AHL*

Season's Greetings from the staff of the Animal Health Laboratory

2012/13 Holiday laboratory hours

Date	Hours	Notes
Fri Dec 21	Guelph/Kemptville normal hours	Guelph/Kemptville - full staff
Sat Dec 22	Guelph - 9 - 5, Kemptville - closed	Usual Saturday services
Sun Dec 23	Guelph - 9 - 5, Kemptville - closed	Usual Sunday services
Mon Dec 24	Guelph - 9 - 5, Kemptville - 8:15 - 4:30	Open with limited staffing and testing
Tues Dec 25	**CLOSED – Christmas Day**	
Wed Dec 26	Guelph - 9 - 5, Kemptville closed	Usual statutory holiday services
Thurs Dec 27	Guelph - 9 - 5, Kemptville - 8:15 - 4:30	Open with limited staffing and testing
Fri Dec 28	Guelph - 9 - 5, Kemptville - 8:15 - 4:30	Open with limited staffing and testing
Sat Dec 29	Guelph - 9 - 5, Kemptville - closed	Usual Saturday services
Sun Dec 30	Guelph - 9 - 5, Kemptville - closed	Usual Sunday services
Mon Dec 31	Guelph - 9 - 5, Kemptville - 8:15 - 4:30	Open with limited staffing and testing
Tues Jan 1	Guelph - 9 - 5, Kemptville - closed	Usual statutory holiday services
Wed Jan 2	Guelph/Kemptville normal hours	Guelph/Kemptville - full staff

Guelph and Kemptville drop box and/or refrigerator are available 365/24/7 for specimen drop off.

Usual Saturday services include: specimen receiving, emergency mammalian necropsies, full bacteriology setup and reporting, as well as clinical pathology testing.

Statutory holiday services and usual Sunday services include: specimen receiving, emergency mammalian necropsies, and basic bacteriology setup.

Please call the laboratory for "**limited testing**" details.

Arcanobacterium pyogenes has been renamed *Trueperella pyogenes*

Durda Slavic

Arcanobacterium pyogenes formerly known as ***Corynebacterium pyogenes*** and ***Actinomyces pyogenes*** underwent recent taxonomic evaluation resulting in its name change (again). Based on the phylogenetic and chemotaxonomic differences within the members of the *Arcanobacterium* genus, this genus was further divided into two new genera, namely *Arcanobacterium* and *Trueperella*. *Arcanobacterium haemolyticum* is the type species of *Arcanobacterium* genus, whereas *Trueperella* (*Arcanobacterium*) *pyogenes* is the type species of the *Trueperella* genus. This taxonomic change will be reflected in AHL reports starting in January 2013. *AHL*

AHL offers a TaqMan real-time PCR for the detection of *Geomyces destructans*, the pathogen of bat white nose syndrome

Hugh Cai, Hamid Haghighi, Pat Bell-Rogers

After providing a SYBR-green based real-time PCR for the detection of *Geomyces destructans* for the past 6 months, the AHL will offer an additional TaqMan probe based real-time PCR for the same purpose starting November 1, 2012. The SYBR-green PCR was developed for field sample testing for over a year by Dr. Dorothee Bienzle's group at the Department of Pathobiology, University of Guelph, and was then transferred to the AHL in March 2012.

In September, 2012, a TaqMan probe based real-time PCR was published (Muller et al., 2012), and was reported to be specific, agreeing with histologic diagnoses for all 91 previously analyzed bat skin samples with no false positives from a panel of 54 closely related fungal isolates. We compared the TaqMan PCR to our current SYBR-green PCR by testing a panel of field samples. Both methods had similar performance, with the TaqMan PCR being slightly more sensitive.

The AHL is now ready to offer the TaqMan probe based real-time PCR for the detection of *G. destructans* with the SYBR-green real-time PCR as a backup assay. Please submit swab or tissue samples to the Molecular Biology Lab using test code "gdsPCR". *AHL*

Reference

Muller LK, et al. Bat white-nose syndrome: A real-time TaqMan polymerase chain reaction test targeting the intergenic spacer region of *Geomyces destructans*. Mycologia. 2012 Sep 6. [Epub ahead of print]

AHL Newsletter
December, 2012 - Volume 16, Number 4

Editor: **Grant Maxie,** DVM, PhD, Diplomate ACVP
Editorial Assistants: **Helen Oliver, April Nejedly**

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.

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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Conspicuous by its absence - the importance of brain removal in field necropsies

Josepha DeLay, Maria Spinato, Ken Bateman, Jeff Rau

Gross and histologic examination of brain is very important in necropsy cases with a history of neurologic signs, in cases of unexpected death, or in situations where gross lesions identified in other organs are not sufficiently severe to have resulted in debility or death. Because of the inherent difficulty of brain removal, the procedure is often avoided or overlooked, and important diagnoses may be missed. Similar issues arise if only 1-2 sections from brain are submitted, rather than the entire brain – an example is the difficulty in diagnosing listeriosis, which targets brainstem, if samples only from cerebral cortex are submitted for histopathology.

Practitioners have 2 options for brain submission to the diagnostic lab:

1. Submit the intact head to the diagnostic lab. Brain removal and appropriate sampling will be performed at the lab. For head removal, disarticulate the atlanto-occipital joint beginning at the ventral aspect of joint. The joint is located at the level of the base of the ear and just caudal to the ramus of the mandible. Flexion and extension of the joint may aid in its location. Cut through skin and soft tissue ventral and lateral to the joint with a large knife, sever the brainstem-spinal cord junction at the joint, and disarticulate by applying leverage between the articular surfaces of occipital condyles and the first cervical vertebra (C1).

The head may be delivered directly to the lab or shipped by usual courier methods. To prevent fluid leakage during transport, please ensure that the head is wrapped in 2 separate, individually sealed plastic bags and placed in a sufficiently heavy box or sealed plastic container. Several cold packs should be placed around the head to slow autolysis.

2. Remove the brain at the time of necropsy. This technique uses an axe to open the skull and requires that the carcass is placed on a firm but shock-absorbing surface, such as dirt / pasture or straw pack (NOT concrete). BE SAFE – ensure that other people and animals are well away from the area, and the axe operator should be wearing appropriate footwear (steel-toed boots) and protective eyewear. Keep the head attached to the body for brain removal. Using the landmarks outlined (Figure 1) and an axe, remove the skull cap by directing the axe cut from the poll toward the front of the skull, just dorsal to the eye. In most cases, this will require several axe strikes. An alternative to consider is the use of a battery-powered reciprocating saw ("Sawzall") with a 9 or 12 inch blade (depending on the age of the animal) with 5 or 6 teeth per inch. The dorsal aspect of cerebral hemispheres and cerebellum may be severed in the process, however sufficient brain should remain intact for sampling (Figure 2). With the skull cap removed, scoop the brain out of the skull, cutting cranial nerves at the ventral aspect the brain. Remember to include cerebellum and brainstem, which will be caudal to the exposed cerebral cortex and may still be obscured by a section of occipital bone.

Make a mid-sagittal cut in the brain once it is removed from the skull. Place one half in a large Whirl-Pak bag and chill. Make several transverse, partial-thickness incisions in the remaining half, and place in formalin – the incisions will allow better fixation of a large brain. Submit both fresh chilled and formalin-fixed brain to the diagnostic lab.

If rabies, BSE, CWD, or scrapie is suspected, you must contact your local CFIA office and submit samples according to their instructions. If these diseases are included in the differential diagnosis but are not strongly suspected, consult your local CFIA District Veterinarian regarding your options before submitting the brain or head to the AHL for diagnosis. *AHL*

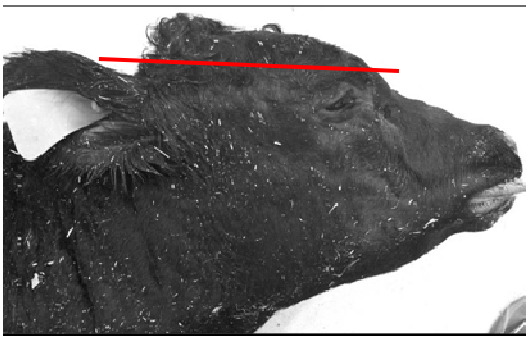

Figure 1. Location of cut to remove skull cap.

For more detail on field necropsies, please refer to our LabNote: http://guelphlabservices.com/files/LabNote02FieldNecropsyJuly2012.pdf

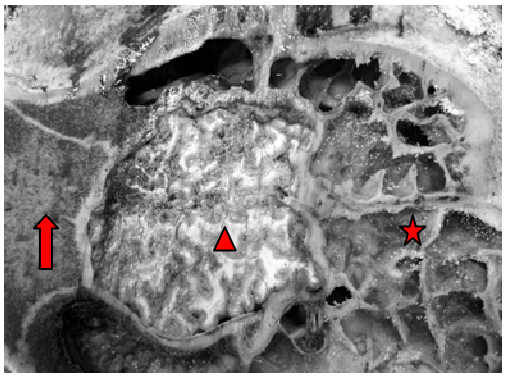

Figure 2. Dorsal aspect of skull following removal of skull cap. Cerebral cortex is visible (arrowhead); cerebellum and brainstem are ventral to an intact segment of occipital bone (arrow). Frontal sinus and ethmoid turbinates are visible to the right of (rostral to) cerebral hemispheres (star).

AHL Lab Reports

RUMINANTS

Nitrate poisoning in cattle

Brent Hoff, Nick Schrier, Margaret Stalker, Maria Spinato, Ken Metzger

A herd of 30 beef cattle was removed from summer pasture and housed in a yard overnight. The following morning, 4 cows were found dead, 1 animal was recumbent, and 3 others had clinical signs of ataxia, weakness, tremors and blindness. Nitrate toxicity was suspected by the referring veterinarian upon noting the chocolate-brown discoloration of blood samples. One of the dead cows was submitted to the AHL for post-mortem examination. Additional fresh and fixed tissues from a second dead cow, feed, and water were also submitted. Other than a slight dark discoloration of heart blood, muscle and mammary gland, no significant gross lesions were identified at necropsy. Elevated concentrations of nitrate were found in heart blood (351 ug/g) and ocular fluid (225 ug/g), consistent with a diagnosis of nitrate toxicosis. **The cattle had been fed *Sorghum halepense*, commonly called Johnsongrass, a plant that accumulates nitrates when stressed by drought or early frost.**

Nitrate poisoning in cattle may be caused by consumption of excessive amounts of nitrate/nitrite in various pasture forages and weeds, hay, and silage, as well as contaminated drinking water, lubricating oil, or agricultural fertilizers. Cattle are especially susceptible to nitrate poisoning because the ruminal flora reduce nitrate to nitrite, then to ammonia which is used for microbial growth. Nitrite is 10 times more toxic than nitrate, causing oxidation of ferrous iron in hemoglobin to the ferric state, forming methemoglobin, which cannot bind oxygen. As the percentage of methemoglobin in the blood rises, the blood turns chocolate brown, and tissues begin to enter an oxygen starved state. Clinical signs include weakness, staggering gait, collapse, dyspnea, tachycardia and cyanosis. Abortions due to nitrate toxicosis are also reported. Plasma is the preferred ante-mortem sample for diagnosis, because some plasma-protein bound nitrite could be lost in clotted blood. Suitable post-mortem samples include ocular fluid, fetal thoracic fluid or abomasal content, and maternal uterine fluid.

While many plants contain some nitrate, excessive amounts are more likely to occur under conditions of excessive fertilization and/or stress during the growing season. **The drought conditions experienced in Southern Ontario this past summer may have stressed susceptible plant species, resulting in excessive accumulation of nitrates.** Feed analysis for nitrate and cyanide levels is advisable prior to feeding known nitrate-accumulating forages, particularly sorghums, to ruminants. *AHL*

The small ruminant abortion project wraps up

Murray Hazlett, Rebeccah McDowall, Josepha DeLay, Margaret Stalker, Beverly McEwen, Tony van Dreumel, Maria Spinato, Brian Binnington, Durda Slavic, Susy Carman, Hugh Cai

The project is completed and final results have been prepared for publication. We would like to thank all of the veterinarians and producers who helped by submitting material, as well as all of the lab staff who worked on the results. In total, we examined 163 sheep and 96 goat abortion submissions. The final diagnosis was determined by the pathologist after reviewing all the lesions and testing on a particular case (Table 1).

Interestingly, **both *Coxiella burnetii* and *Chlamydophila abortus* were identified in many cases by rtPCR testing, but were not always felt to be significant**. *C. burnetii* was detected in 69% of sheep abortions and 75% of goat abortions, but when found, was felt to be significant when found in 6.7% (sheep) and 15% (goat) submissions. For *C. abortus* 26% of sheep and 59% of goat submissions were positive when tested. Pathologists felt a positive result was significant in only 38% (sheep) and 63%(goat) of submissions. *Toxoplasma gondii* was also commonly identified with other pathogens.

C. burnetii positive samples are being analyzed in another ASHI project carried out by the Agriculture and Food Lab, our sister lab, to check for possible differences among the *C. burnetii*. This may shed some light on why *C. burnetii* seems to sometimes be a bystander, and sometimes be a primary pathogen. *AHL*

Table 1. Agents identified in sheep and goat abortion cases.

Final diagnosis	Sheep (cases, %) n=163	Goats (cases, %) n=96
Toxoplasma gondii	31 (19%)	16 (17%)
Coxiella burnetii	12 (7.4%)	15 (16%)
Chlamydophila abortus	19 (12%)	36 (37%)
Campylobacter spp	21(13%)	0
Salmonella spp	3 (1.8%)	0
Listeria monocytogenes	1 (0.6%)	1 (1%)
Other bacteria	6 (3.6%)	4 (4%)
BVD virus	1 (0.6%)	0

SWINE

New real-time PCR test for *Porcine parvovirus* available at the AHL

Susy Carman, Li Ge

The AHL now offers a real-time PCR for *Porcine parvovirus*. This PCR was initially developed at Iowa State University. We have validated this test for use at the AHL using 20 *Porcine parvovirus* cell culture isolates from aborted fetuses presented to the AHL from 1988 to 2011.

This real-time PCR will replace the indirect fluorescent antibody test. For more information, please contact Dr. Susy Carman 519-824-4120, ext. 54551 or Dr. Davor Ojkic, ext 54524.

scarman@uoguelph.ca dojkic@uoguelph.ca *AHL*

- Send lung from aborted fetuses.
- Fetuses with crown-rump length less than 16 cm are preferred.
- Please ship fresh or frozen samples on ice packs.
- Testing is performed weekly.
- Test code **ppvrt.**
- **The fee is $31.00 per test**.

OASV Fall Conference 2012

The annual fall conference of the Ontario Association of Swine Veterinarians (OASV) was held in Guelph on October 12 and 13. Approximately 40 veterinarians and students attended a half-day session at the Animal Health Laboratory that included an update on swine disease diagnoses in Ontario in 2011 and 2012; a review of PCR testing and comparison with results obtained by other test methods; and histologic techniques and the impact of tissue sampling on histopathology results.

A pathology demonstration lab and discussion focussed on practical tips for field necropsies, with emphasis on sampling and testing for swine respiratory and gastrointestinal disease. AHL presenters and contributors included Drs. Grant Maxie, Jim Fairles, Hugh Cai, Murray Hazlett, Josepha DeLay, and Beverly McEwen, and Josie Given. *AHL*

Swine necropsy lab presenters Dr. Terri O'Sullivan, OASV vice president; Dr. Josepha DeLay, AHL; Dr. Murray Hazlett, AHL.

Involvement of the AHL in swine surveillance and disease investigation and eradication *Jim Fairles*

The AHL is pleased to be involved with a wide variety of projects and programs aimed at the early identification, reduction and eradication of swine diseases. Listed below are some of these ongoing endeavours.

- OMAFRA animal health surveillance – a product of testing on our laboratory case intake.
- Animal Health Strategic Investment (AHSI) investigations – possible via enhanced OMAFRA support.
- Ontario Swine Health Advisory Board (OSHAB) – PRRSV sequencing database including the Canadian Swine Health Board development of a non-reportable disease surveillance and outbreak detection system using PRRSV as a model.
- Ontario Swine Health Advisory Board – PRRSV Area and Regional Control and Elimination (ARC&E) projects.
- Canadian Swine Health Board - Periweaning failure-to-thrive syndrome (PFTS) in pigs in Ontario.
- Canadian Swine Health Board – Canadian Swine Health Information Network (CSHIN) participation, a national practitioner-based swine disease surveillance and information-sharing network.

HORSES

Top 10 neurologic diseases in Ontario horses identified at the AHL from 2008 to October 2012

Beverly McEwen, Davor Ojkic, Susy Carman, Josepha DeLay, Murray Hazlett, Margaret Stalker, Durda Slavic

Cases from horses with a pathology diagnosis of neurologic disease (n=86) and those submitted for testing to identifyspecific neurologic pathogens (n=563) were reviewed. The specific neurologic pathogens were: *Eastern equine encephalitis virus* (EEEV), *West Nile virus* (WNV), *Rabies virus, Equid herpesvirus 1* (EHV-1) (neuropathic/ non-neuropathic), and *Sarcocystis neurona*. This group of cases comprises 2-3% of all equine submissions to the AHL, with peak submissions occurring in August and September each year. One or more of the above pathogens were identified and/or diagnoses were made in 262 of the 649 submissions (42%). The top 10 pathogens identified and diagnoses from 2008- October 2012 are listed in Table 1. These data represent the frequency of identification of these diseases at the AHL and cannot be considered to be representative of their prevalence in the general population due to submission biases to the laboratory.

Equine protozoal myelitis (EPM, *S. neurona*) was consistently the most common neurologic condition identified whereas the annual frequency of identified viral infections varied considerably. Both the neuropathic and non-neuropathic strains of **EHV-1** were associated with encephalomyelitis.

Idiopathic encephalomyelitis was often described by pathologists as 'mild' and was often considered of dubious significance in the death of the horse. Horses with idiopathic encephalomyelitis were generally older than those having other diseases, with an average age of 11.3 yr (median 10 yr), compared to 6.2 yr (median 5 years) for the viral encephalitides, 8.8 yr (median 7.2 yr) for bacterial, 8.6 yr (median 7 yr) for equine protozoal myelitis cases, and 6.7 yr (median 4 yr) for horses with degenerative myelopathy. The reasons for this are speculative, but may indicate a previous mild or subclinical neurological infection. Also, the horse's age is provided in only a subset of all submissions, which may skew the average values. *AHL*

Table 1. The top 10 pathogens identified and diagnoses in Ontario cases of equine neurological disease submitted to the AHL from 2008 to October 2012.

Diagnosis/pathogen identified	2008	2009	2010	2011	2012 to Oct
Sarcocystis neurona (EPM)	35%	21%	27%	30%	18%
West Nile virus	1%	1%	0%	9%	9%
Idiopathic encephalomyelitis	1%	7%	6%	6%	6%
Degenerative myelopathy	12%	17%	10%	4%	3%
Bacterial infection	4%	1%	2%	4%	3%
Eastern equine encephalitis	4%	3%	3%	4%	0%
Trauma	3%	4%	2%	1%	1%
Hepatic encephalopathy	1%	2%	0%	0%	1%
EHV-1 neuropathic	0%	1%	0%	1%	1%
EHV-1 non-neuropathic	1%	1%	0%	2%	0%

Reminder - EIAV electronic reporting enhancements *Jim Fairles*

Clients continue to receive the standard AHL report indicating that the EIAV ELISA has been completed, but the report now says "**Results to follow and available on-line**". The AHL offers 5 ways to obtain EIA results:

1. **Email** (please indicate on the form and include email address if different than the email address we have on file).
2. **On-line access** to results (view on-line, and print - convenient - no need to call the lab).
   To activate on-line access, please contact us at ahlinfo@uoguelph.ca Telephone: 519-824-4120 ext. 54530.
2. **Fax** (please indicate on form and include fax number).
3. **Courier** (please indicate on the form, additional charge of $5.25/package) (for original).
4. **Canada Post mail** – standard (for original).

AVIAN/FUR/EXOTIC SPECIES

Reovirus as an etiologic component of current leg problems in Ontario broilers

Emily Martin, Marina Brash, Davor Ojkic

In the last few months, the AHL has had multiple submissions of lame broilers with non-suppurative tenosynovitis composed predominantly of lymphocytes, plasma cells and lymphoid nodules (Figures 1 and 2). Lameness with bacterial etiology usually show suppurative tenosynovitis on histology. When closed hock joints are submitted for PCR testing they are positive for reovirus. On further histology, multiple causes of lameness are often identified, including rickets, tibial dyschondroplasia (TD), and bacteria. There are also mononuclear cell aggregates in the epicardium and/or myocardium of the heart. In cases with bacterial involvement, the synoviae have a mixed cell population including lymphocytes, plasma cells and heterophils (Figure 3). Therefore, the presence of reovirus is considered to be only one of multiple possible etiologies of the lameness.

Clinically, the signs of lameness range from slight difficulty walking to splay leg or leg deformity (Figure 4). By 14 days or older, an affected flock becomes suddenly lame, often with only 1 floor or 1 barn affected. Earlier infections may be missed due to variable clinical signs and difficulty differentiating the causes of lameness on post mortem. The earlier the lameness is noted, the more birds will eventually become affected. Birds can have histologic lesions in the tendons, and reovirus may be identified, but clinical signs may or may not be evident. Flocks that are clinically affected can have losses of up to 10% due to culling. Flocks with inapparent infections can be 300-400 grams behind at processing (poor feed conversion) and have high reovirus titers from blood taken at processing. A similar problem has been identified in the USA.

To further investigate this reovirus, a hock fluid sample was sent to a US lab for further characterization. The sample is positive for reovirus by RT-PCR using the reovirus S1 primer set. The amino acid sequence of the reovirus sigma C is 99% similar to recent field isolates in the southeastern US associated with viral arthritis/tenosynovitis and <50% to reovirus vaccine strains S1133, 1733 and 2408. Further samples have been sent for characterization.

Avian reoviruses (ARV) consist of double stranded non-enveloped RNA. They are ubiquitous in the environment and >80% are considered non-pathogenic, however, some cause a variety of diseases including viral arthritis/tenosynovitis (VA, +/- tendon rupture), growth retardation, respiratory disease, enteric disease, immunosuppression and malabsorption syndrome. **Reoviruses that target joints primarily cause inflammation and rupture of tendons, pericarditis, myocarditis, hydropericardium, uneven growth and increased mortality**. Incubation period and disease production depends on age, immune status, virus pathotype and route of exposure. Reovirus interaction with other infectious agents can impact the nature and severity of reovirus-related disease and vice versa. In meat birds, disease results in increased mortality, VA and poor performance (e.g., poor weight gain, poor feed conversion, uneven growth, downgrading at processing). Breeder flocks with VA experience lameness, increased mortality, decreased egg production, poor hatchability/fertility, and vertical transmission to chicks. Transmission can also be horizontal by the fecal-oral route. Resistance to infection increases with age. Reoviruses survive well in the environment, so methods of control attempt to decrease the viral load (e.g., increased down time, total clean out, etc.). Vaccination with live and killed vaccines in breeders pre-lay prevents egg drop in the breeders and provides MAb to the progeny, but vaccines only protect against homologous serotypes.

Overall, **it is thought that reovirus is another etiologic component that has contributed to the recent increase in leg problems in Ontario broilers.** However, the epidemiology is complex and many factors likely interact to influence the development of clinical signs and disease. *AHL*

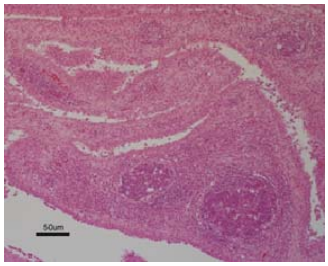

Figure 1. Synoviae showing development of lymphoid nodules in reovirus affected birds. 10X

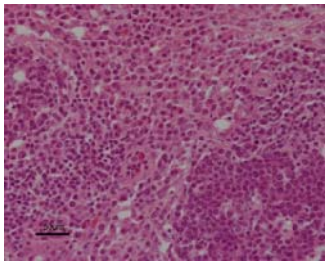

Figure 2. Predominantly lymphocyte and plasma cell populations in the synoviae of birds with reovirus. 40X

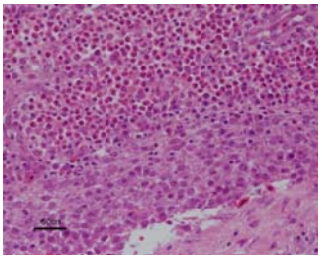

Figure 3. Lymphocyte, plasma cells and heterophils in the synoviae of birds with concurrent reovirus and bacterial infections.

Figure 4. Birds can be down with one leg extending out to the side. These birds can have difficulty getting up and moving.

COMPANION ANIMALS

'What is the most useful test for the diagnosis of hypothyroidism in dogs?'

Kristiina Ruotsalo

This is a common question posed to clinical pathologists. Unfortunately, there is no simple answer, as the test, or test combination, chosen needs to be based upon careful evaluation of the patient's clinical and drug history, clinical signs, and ancillary laboratory data. The diagnostic performance and predictive value of thyroid function tests are greatly enhanced when they are used to confirm the clinician's suspicion of disease, and when other conditions or confounding therapies have been first ruled out or resolved. Considering these factors, common thyroid function tests are briefly reviewed.

Sole determination of **serum total T4** is only diagnostic if the value is within reference intervals or elevated. This is significant as hypothyroidism can be effectively ruled out by a 'normal' result in most cases (keeping in mind that approximately 10% of hypothyroid dogs may have total T4 results within reference intervals due to assay interference by circulating anti-T4 autoantibodies). Be aware of the suppressive effects of nonthyroidal illness, and certain drugs (e.g., sulfa antimicrobials, phenobarbital, glucocorticoids, NSAIDs) on serum total T4 concentrations.

Serum free T4 by equilibrium dialysis or gamma 2-step methods produce equivalent results in dogs and have been shown to be the most accurate single hormone measurement for the diagnosis of hypothyroidism. These tests are more complex to perform and are thus often processed in batches at a slightly greater cost, however in most instances measurement of free T4 will distinguish dogs with low total T4 concentrations attributable to nonthyroidal conditions from those with hypothyroidism. As well, for those dogs with circulating anti-T4 autoantibodies, determination of free T4 by dialysis or gamma 2-step is the only way to accurately assess thyroid status and monitor response to therapy.

The greatest concern regarding the use of **endogenous TSH** on its own for assessment of thyroid function is approximately 15%- 40% of hypothyroid dogs do not have elevated values. However, because increases in TSH in euthyroid dogs are rare, except perhaps during recovery from nonthyroidal illness, an elevated TSH in combination with decreases in total and free T4 increases the diagnostic utility of this test.

T3 is the most potent thyroid hormone at the cellular level, but it is not the predominant circulating thyroid hormone. The sensitivity and accuracy of serum T3 measurement for diagnosis of hypothyroidism is low (except perhaps in sighthounds.)

Thyroglobulin autoantibodies (TgAA) are almost always associated with underlying thyroiditis. However, although positivity for TgAA is highly suggestive of later development of clinical hypothyroidism, a positive result on its own does not necessarily indicate hypothyroidism. Dogs with only positive TgAA and normal total T4, free T4, and TSH values should simply be monitored more frequently for possible progression of disease.

T3 and T4 autoantibodies are also markers of lymphocytic thyroiditis. These autoantibodies are of occasional importance in thyroid testing as they may interfere with thyroid hormone assays, causing variably increased or decreased results, depending upon the method used.

As with all laboratory testing, results must be interpreted in light of clinical signs and other factors which might impact test results. It must also be recognized that discordant results of total T4 or free T4 measurement with TSH and TgAA may reflect intermediate stages of thyroid disease, and that retesting at a later date may be required.
AHL

References:

Ferguson DC. Testing for hypothyroidism in dogs. Vet Clin North Am Small Anim Pract 2007;37:647-669.

Kemppainen RJ, Behrend EN. Diagnosis of canine hypothyroidism. Perspectives from a testing laboratory. Vet Clin North Am Small Anim Pract 2004;31:951–962.

MSU Diagnostic Center for Population & Animal Health website (animalhealth.msu.edu), Frequently asked questions: thyroid function in dogs.