



AHL Newsletter

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Best practices in submitting specimens to the Animal Health Laboratory

Jim Fairles, Linda McCaig

In order to obtain the best quality results from specimens received, it is important that care is taken in preparation of specimens for shipment. This requires adequate sample handling and packaging as it is processed and transported to the laboratory. We receive specimens in many different ways including commercial courier and client drop off. **It is always a concern when samples are leaking on arrival.** Veterinary diagnostic specimens are covered under the Transportation of Dangerous Goods regulations as *UN3373, BIOLOGICAL SUBSTANCE, CATEGORY B*. **This requires basic standards of shipping and it is up to you, the shipper, to make sure that you comply with these standards.** These principles apply whether you are dropping specimens off yourself, handing the specimen to a same day courier who will submit directly to the lab or packaging and shipping by overnight courier. If a specimen leaks during shipment, the courier may refuse to pick up further packages. Listed below are some pointers for shipment taken from our AHL Packaging and Shipping Guidelines:

<http://www.guelphlabservices.com/files/AHL/AHL%20Packaging%20and%20Shipping%20Info/AHLPackagingandShippingInformation%20October%202010.pdf>

- Submit tissues in separate, properly labeled, sealed Whirl-Pak bags.
- For large tissue specimens, we recommend triple-bagging.
- Collect fluid and feces in screw-capped plastic bottles and place in Whirl-Pak bags
- Submit blood and serum in vials that have screw cap lids or tape pop-off lids and place in leakproof bags
- Place history sheet in a **separate plastic bag or the pocket of a biohazard bag** with the specimens inside.
- Jars of formalin for histology are provided by AHL. (supplied with absorbent material and Whirl-Pak bag).
- Sterile vials for mastitis herd testing are available to purchase from Vet Purchasing. Item 1721510.
- Use mailing containers that meet standards for Transportation of Dangerous Goods, Type 1B packaging:
 - ✦ watertight inner packaging surrounded by absorbent material (e.g., screw cap vial and paper towel).
 - ✦ watertight secondary inner packaging (e.g., Whirl-Pak bag).
 - ✦ sturdy outer (corrugated cardboard) packaging.
- Frozen specimens: Place between ice packs – gel type – NO ICE CUBES, wrap in 5-6 layers of newspaper, and place inside insulated container.

If shipping larger specimens that potentially may leak, rather than cardboard outer packaging please use a plastic container of suitable size. (AHL will return the plastic or insulated container to you)

Attention to detail when shipping specimens to prevent leakage will aid in accuracy of diagnostic testing and prevent cross contamination, provide for safety with all of those involved in handling the specimen, as well as maintain the ability to continue to ship specimens to the laboratory with minimal preparation. AHL

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Selected zoonotic pathogens and diseases identified at the AHL, 2010

Beverly McEwen, Durda Slavic, Davor Ojkic, Josepha DeLay, Hugh Cai, Margaret Stalker, Murray Hazlett, Marina Brash

Many new, emerging, and re-emerging diseases of people are caused by pathogens originating from animals, or are shared between people and animals. The AHL plays an important role in public health by identifying zoonotic pathogens (Tables 1 and 2). These are numerator data reliant upon submission biases to the diagnostic laboratory, and cannot be regarded as population prevalence estimates. Monitoring programs are not included.

The zoonotic pathogens most frequently identified at the AHL since 1999 are *Leptospira* spp., *Salmonella* sp., *Streptococcus* sp., and *Cryptosporidium* sp. Occupational exposure to pigs and horses is a risk factor for *S. suis* and *S.*

zooepidemicus infections.

The dramatic increase in cases of *Coxiella burnetii* and *Chlamydophila* sp. is due to the increased surveillance for these pathogens in the small ruminant abortion project. *Chlamydophila* sp. was also identified in a parrot. Veterinarians must report suspected cases of *Chlamydophila psittaci* to the Ontario Ministry of Health and Long-Term Care.

Prior to 2008, the numbers of isolates were tabulated, however, due to the increasing number of tests for selected pathogens, the number of cases will now be documented. For data prior to 2008, please refer to previous editions of the AHL newsletter. AHL

Table 1. Cases with selected zoonotic pathogens isolated and/or identified at the AHL, 2008-2010

Agent	Cattle	Swine	Horses	Sheep	Goats	Chicken	Turkey	Dogs	Cats	Other	2010	2009	2008
<i>Blastomyces dermatitidis</i>								5			5	10	Not done
<i>Bordetella bronchiseptica</i>		38	5					5	1	5	54	60	52
<i>Campylobacter coli/jejuni/fetus subsp. fetus</i>				16	1	1		2		4	24	14	14
<i>Chlamydophila</i> sp.				33	24					1	58	29	10
<i>Clostridium difficile</i>	3	10	17							1	31	24	25
<i>Coxiella burnetii</i> (Q fever)	3			71	33					8	115	9	15
<i>Cryptosporidium</i> sp.	136	1	1	1	11					7	157	128	144
<i>Eastern equine encephalitis virus*</i>		12									12	11	12
<i>Giardia</i> sp.	16		1	1	2			39			60	55	56
<i>Listeria monocytogenes</i>	3			10	6						19	18	14
Methicillin-resistant <i>Staphylococcus aureus</i> (MRSA)			72							2	74	36	51
<i>Mycobacterium tuberculosis</i>											0	0	1
Rabies										3	3	8	4
<i>Salmonella</i> sp.	38	60	12	4	1	92	15	12		27	256	281	322
<i>Streptobacillus moniliformis</i> (rat bite fever)											0	0	1
<i>Streptococcus suis</i>	1	106			1					2	110	120	158
<i>Streptococcus equisimilis</i>		17	23					4	1	2	48	43	76
<i>Streptococcus zooepidemicus</i>	4		142	1				3	1	1	152	117	108
<i>Toxoplasma</i> sp.				15	4			1	1	1	22	19	8
West Nile virus										7	7	6	70
<i>Yersinia enterocolitica</i>		2									2	0	6

Table 2. *Leptospira* spp. seropositive cases identified at AHL, 2010, microscopic agglutination test (MAT)

<i>Leptospira</i> spp. serovar*	Cattle	Swine	Horses	Dogs	Other & not specified
<i>L. autumnalis</i>	4	17	17	22	0
<i>L. bratislava</i>	9	26	22	13	1
<i>L. canicola</i>	5	4	3	9	0
<i>L. grippityphosa</i>	7	9	7	45	1
<i>L. hardjo</i>	6	0	0	0	0
<i>L. icterohaemorrhagiae</i>	19	16	13	15	1
<i>L. pomona</i>	0	22	21	40	2

* Some cases are from other provinces

AHL Lab Reports

RUMINANTS

An outbreak of verotoxigenic/enterohemorrhagic (VTEC/EHEC) *E. coli* in calves

Maria Spinato, Durda Slavic, Reny Lothrop

A group of 70 female neonatal calves assembled from multiple sources developed voluminous mucoid diarrhea mixed with flecks of blood. Husbandry on this farm was excellent, and dehydrated animals received appropriate fluid and electrolyte therapy. Five calves with severe dysentery died, despite aggressive fluid and antimicrobial therapy. Although the calves continued to eat, fecal consistency remained soft, and several of the animals eventually became emaciated, and had to be euthanized.

A 12-day-old calf was euthanized and submitted to the AHL necropsy laboratory for post mortem examination. The animal was in thin body condition, markedly dehydrated, and had extensive perineal fecal soiling. Internally, fat stores were depleted. Small intestines were pale and contained fluid to creamy ingesta. Cecum and spiral colon were distended by gas, and contained scant granular tan flecks and a thin film of creamy fluid. Rectum contained fluid feces. Mesenteric lymph nodes were slightly enlarged and pale.

A sucrose fecal wet mount identified 2+ *Cryptosporidium sp.*, and real-time RT-PCR and immunohistochemical staining for BVDV were negative. Bacterial culture was negative for *Salmonella sp.* Routine screening for viral pathogens was not performed due to the protracted period of clinical illness. Histologic examination of intestinal sections was hampered by autolysis and sloughing of villus epithelium. However, intact enterocytes located at the villus-crypt junction of the jejunum and ileum were heavily colonized by several layers of bacterial coccobacilli. Similarly aggregated bacteria were closely attached to the brush border surface of enterocytes lining glands in the spiral colon. Cryptosporidial organisms were also present within the microvillus border of jejunal enterocytes. Mild accompanying inflammation was characterized by rare crypt abscesses, and a few neutrophils infiltrating the lamina propria. Congested colonic sections contained occasional thrombosed mucosal capillaries.

Based on the histologic feature of “attaching” bacteria found in large intestine in this calf, further molecular typing of *E. coli* isolates was requested. PCR detection of *eaeA* (intimin), *hlyA* (enterohemolysin), and *stx1* (Shiga toxin 1) genes confirmed that *E. coli* isolates were consistent with a verotoxigenic/enterohemorrhagic (VTEC/EHEC) strain. In addition, it was also confirmed by PCR that these isolates belong to O111 serotype. *E. coli* O111 together with *E. coli*

O157 are well known *E. coli* serotypes associated with the severe disease in humans such as hemorrhagic colitis (HC) and hemolytic uremia syndrome (HUS). Since cattle lack the vascular receptor for Shiga toxin(s), they do not develop HC or HUS. However, there is some evidence that VTEC/EHEC isolates are capable of causing severe enteritis in calves 1-5 weeks of age, related to direct epithelial injury. As a result, it was concluded that the VTEC/EHEC O111 isolate likely contributed to the clinical disease in this calf, together with *Cryptosporidium sp.* infection.

Different types of *E. coli* can cause a range of intestinal and systemic disease syndromes in calves. Recent advances in molecular tools facilitate the differentiation between normal intestinal flora and pathogenic strains. **Genotyping of *E. coli* isolates should be considered in cases of non-responsive diarrhea or dysentery in calves 1-5 weeks of age, in order to identify VTEC/EHEC and other pathogenic strains.** During this particular outbreak of calf enteritis, one of the barn staff developed transient diarrhea, nausea and abdominal discomfort. As veterinarians and participants in the “One Health” strategic vision, it behooves us all to be aware of the public health significance of calf enteric diseases, such as cryptosporidiosis and VTEC/EHEC.

AHL

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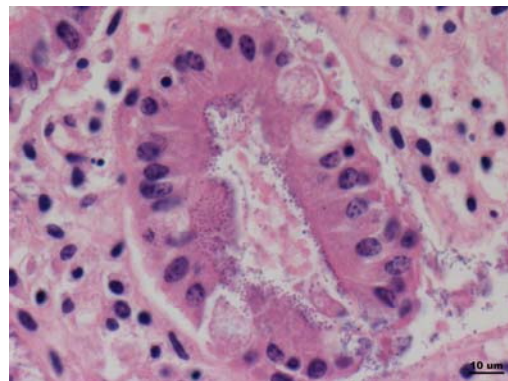


Figure 1. *E. coli* “attached” to the brush border of intestinal enterocytes (courtesy of M. Hazlett).

SWINE

PRRSV PCR-positive testing report for 2001 to 2010

Susy Carman, Beverly McEwen, Jim Fairles

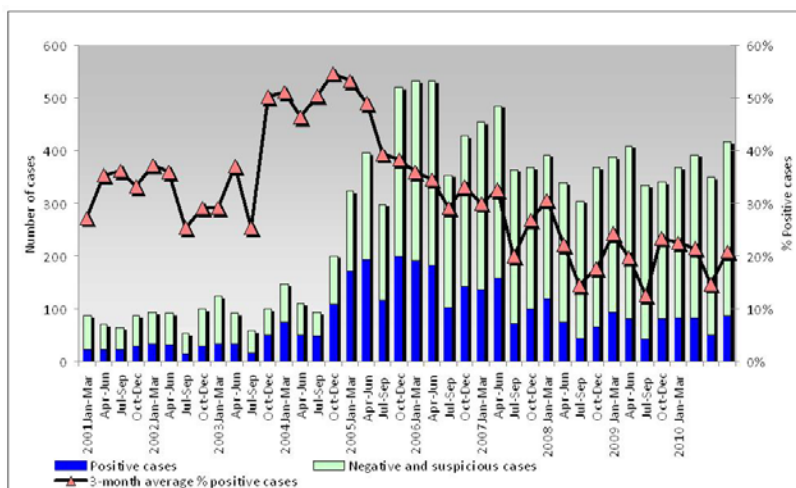
Since the height of the PRRSV outbreak in Ontario swine in 2004, the “annual average” of PRRSV PCR-positive swine cases presented to the AHL has gradually declined from 51% in 2004, to 20% in 2010 (Figure below). Now that the epidemic has declined, there are apparent seasonal rises in winter and falls in the summer. For the last quarter of 2010, there is a slight increase in the proportion of PRRSV PCR-positive cases from 15% in the summer to 21% in the last quarter. These data do not include semen monitoring cases. However it was not possible to exclude serum or blood swab monitoring cases, such that some testing represents monitoring of swine herds with ongoing infections, rather than new outbreaks.

Gel-based ORF5 RFLP typing for PRRSV strains, as determined using Wesley’s RFLP typing criteria, includes only 1 MluI, 8 HincII, and 3 SacII restriction enzyme patterns. However in the last few years there has been significant genetic drift in the ORF5 gene sequence, with many strains now being reported to be “undetermined” at this restriction HincII site, when gel-based typing is used. Although RFLP type names are a convenient way to track viruses within the same herd, full ORF 5 sequence homology provides a better comparison.

For comparison of PRRSV strains, we strongly recommend that you request “PRRSV ORF5 gene sequencing”, which compares the % sequence homology over the entire ORF 5 gene sequence, rather than gel-based ORF5 RFLP typing, which compares

only 6 nucleotides at each restriction site. A comparison of the % homology over the entire 603 base ORF5 genome is the best comparison. Each comparison is rooted to the current sequence.

When PRRSV ORF5 sequencing is requested, the AHL also provides the Minnesota based “sequence predicted RFLP typing classification”. Based on the original Wesley system, this classification extends the RFLP typing classification to include 4 MluI patterns, 58 HincII patterns, and 8 SacII restriction enzyme patterns. More patterns are being identified every month. These can only be determined following sequencing, and using the sequence to predict the RFLP type. Viruses of different RFLP type can have almost identical sequences. Conversely, viruses with the same RFLP type can have very different sequence homology. AHL



AVIAN/FUR/EXOTIC SPECIES

First report of *Enterococcus cecorum* infection in meat turkeys in Ontario

Marina Brash, Mike Joyce, Durda Slavic

Fourteen day-old commercial turkeys were submitted for necropsy because approximately 1% of the flock was exhibiting signs of lameness. At necropsy, birds were noted to be dehydrated and were in poor body condition with litter in their gizzards. There was femoral head necrosis and fibrinous airsacculitis. Swabs of the femoral head lesions were submitted to the AHL for culture. Moderate numbers of *Enterococcus cecorum* and low numbers of *Escherichia coli* were recovered from one of the swabs.

This is the first isolation of *Enterococcus cecorum* from femoral head necrosis lesions in meat turkeys in Ontario. In turkeys, *E. cecorum* was previously isolated only from septicemia cases in 2 commercial flocks in Pennsylvania, but with no bone or joint lesions identified and no signs of lameness.

While *E. cecorum* infection is well recognized in broiler and broiler breeder flocks in Ontario since its first description in 2008, it remains to be seen what impact this bacterium will have on turkey flocks in Ontario. AHL

Psittacine beak and feather disease: a review *Hein Snyman, Marina Brash*

Psittacine beak and feather disease (PBFD), caused by the circovirus *Beak and feather disease virus* (BFDV), although uncommon in North America, is extremely common in southern Africa, Australasia and Europe and poses a risk every time a new bird is purchased or imported. The disease affects over 40 psittacine species, especially old world parrot species (African and Australasian parrots), including African grey parrots (*Psittacus erithacus*), ringneck parakeets (*Psittacula spp.*), cockatoos (*Cacatuidae*), African lovebirds (*Agapornis spp.*), Eclectus parrots (*Eclectus roratus*), as well as lorries and lorikeets (*Lorinae*).

Infection of young birds occurs through ingestion, inhalation or via the cloaca. The incubation period can be as short as 21 da. The bursa of Fabricius is infected first, resulting in lymphoid necrosis and severe immunosuppression. The virus spreads to the liver, thymus and bone marrow and a second round of virus replication occurs. BFDV is epitheliotropic and spreads to the skin, feathers and beak resulting in the typical clinical signs. In an infected bird, the virus is shed in massive quantities from all epithelial surfaces, so contaminated feces/feather dust/dander result in horizontal spread between birds as well as from the hen to her chicks during crop feeding. Vertical transmission is suspected.

Clinical disease is seen in peracute, acute and chronic forms. **Peracute disease** is observed in young chicks from the first few weeks to the first few months of age, and occurs especially in African grey parrots, ringneck parakeets, and cockatoos. Often there will be no premonitory signs, and chicks will simply be found dead in the nest. Clinical signs may include lethargy, inappetence and rapid weight loss, vomiting, regurgitation, diarrhea. Death results from secondary septicemia, pneumonia and enteritis. **Acute disease** usually affects fledglings after the first moult, when deformed and broken feathers as well as feather shaft hemorrhages become evident. Birds develop secondary infections due to immunosuppression and die. The acute disease occurs differently in African grey parrots and they will be extremely weak, with yellow urates, and are panleukopenic and anemic - all of these birds die within 1 wk of presentation.

Chronic disease is the more classical form, with progressive feather loss, dystrophy and feather discoloration (Fig 1). Loss of powder down is another early sign of infection. As well, beak necrosis and deformation occur, and cracks and fractures form within the beak. Secondary infections ensue and most birds will die within 6-12 mo of presentation. Often these birds have a normal white cell count. **Chronically infected birds shed large amounts of virus and pose a significant risk to other pet birds as well as exotic zoo collections.** Treatment consists of supportive care and treatment of secondary bacterial and fungal infections.

On necropsy, most lesions are related to secondary infections. The histological evaluation of tissues, especially the bursa of Fabricius and diseased feather follicles, is extremely important. **The characteristic lesions include severe necrosis of bursal lymphocytes and typical intracy-**

toplasmic circoviral inclusions (Figs 2, 3). Similar inclusions are often observed within diseased feather follicles.

No commercial vaccine is available, so prevention remains the key for the control of this disease. All new birds should be screened for BFDV and other viruses prior to or within a week of purchase, preferably through PCR testing of EDTA blood and cloacal and choanal swabs. All birds that feather pluck or birds with abnormal feathers should have a feather biopsy taken and submitted for histological evaluation. The diagnosis of PBFD is based on a positive PCR test and compatible clinical signs. Birds that have no clinical signs but are PCR-positive are considered infected and should be isolated and PCR retested in 2-3 months. If this subsequent PCR test is negative, the bird is considered to have been infected with BFDV, experienced a transient viremia and mounted an effective immune response. Birds that still test positive are chronically infected and are chronic carriers. Rarely, chronically infected birds can test PCR-negative if no virus is actively circulating in the blood, and feather biopsies of affected follicles may be helpful.

The virus is extremely hardy and may persist in the environment for up to 6 mo. Adequate biosecurity includes the quarantine of all new introductions for 4 to 6 wk, and the use of new or cleaned and disinfected cages when transporting birds. The quarantine site should have dedicated utensils, equipment and personnel. *AHL*

Figure 1. African Grey parrot with chronic PBFD. Progressive feather loss around ears and under wings. Feathers are dystrophic and discolored. The beak is shiny because of loss of the powder down. Photo courtesy of Dr. D Elliott, Bird and Exotic Animal Hospital, South Africa

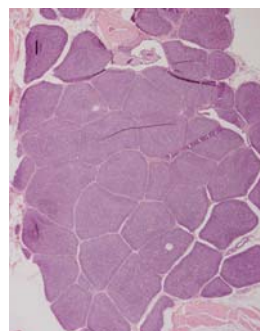
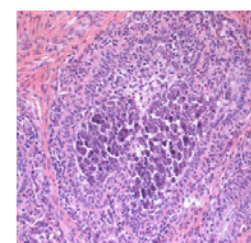


Figure 2. Histology of normal follicles in bursa of Fabricius.

Figure 3. Histology of typical PBFD circoviral inclusions in a depleted bursal follicle.



HORSES

Ontario Racing Commission Death Registry: 2003-2010 necropsy summaries *Josepha DeLay*

The Ontario Racing Commission Death Registry continues to provide excellent data regarding the causes of morbidity and mortality in racehorses in this province. Necropsy requests by the ORC have become more selective in the past 3 years, with a shift in emphasis to non-fracture cases and with increased complexity (Table 1). Breed distribution of submissions in 2010 was Standardbred 22 (69%), Thoroughbred 8 (25%), Quarter Horse 2 (6%). *AHL*

Table 1. Necropsy diagnoses of ORC Death Registry submissions by body system, 2003-2010.

Diagnosis by body system:	2003	2004	2005	2006	2007	2008	2009	2010
Fracture / limbs	53 (42%)	69 (49%)	48 (44%)	42 (39%)	54 (44%)	16 (31%)	4 (9%)	9 (28%)
Fracture / other	10	4	7	13	10	5	0	3
Non-fracture musculoskeletal	8	6	6	8	6	5	2	3
Gastrointestinal	15	19	17	16	18	5	4	7
Respiratory	21	17	9	11	16	9	21	6
Cardiovascular	5	6	5	5	2	4	6	2
CNS	6	11	7	4	1	1	2	0
Integumentary	0	0	1	2	2	1	1	0
Renal	0	2	0	0	2	0	1	0
Hematopoietic	2	1	1	0	0	0	0	0
Whole body conditions	1	7	5	2	9	0	4	0
Cause of death undetermined	4 (3.2%)	0 (0%)	4 (3.6%)	4 (3.7%)	2 (1.6%)	5 (9.8%)	0 (0%)	2 (6%)
Total	125	142	110	107	122	51	45	32

Equine abortion due to infection with *Candida parapsilosis*

Janet Shapiro, Durda Slavic, Beverly McEwen, Brian Binnington

A 26 cm long, 670 gram, 4-month gestation Warmblood equine fetus and placenta were submitted from a mare that had aborted outside in Ontario in December. The mare showed no prodromal or postdromal signs. This was the only mare in the group of 2 that had aborted. There were no specific gross lesions of the fetus or placenta. Histopathologic findings consisted of mild subacute bronchointerstitial pneumonia and multifocal placentitis, and there were large numbers of intralesional organisms morphologically consistent with yeast in lung and placenta. Yeast were also present in moderate numbers in the small intestinal lumen. **Moderate to large numbers of yeast identified as *Candida parapsilosis* were isolated from stomach content, lung and placenta.** *Candida parapsilosis* has been implicated in sporadic natural cases of equine endometritis.

Mycotic abortion of horses is an infrequent and sporadic event, having been diagnosed at the AHL in 0-2.1% of equine abortions in any year since 1993. From Jan 1, 2001 and Dec 31, 2010, 8 of 693 (1.2%) equine fetuses and/or their complete placenta, submitted to the AHL for necropsy, were diagnosed with mycotic infections. There was 1 case each of *Mucor* spp, *Candida* spp, *Aspergillus fumigatus*, and *Candida parapsilosis*, 1 case each of unidentified yeast and an unidentified fungal infection, and 2 cases in

which mycotic agents were seen histologically in placenta and/or fetal tissues in inflammatory lesions, but were not isolated. This is similar to retrospective reports from other laboratories (Michigan State Animal Health Diagnostic Laboratory, 1985-1996- mycotic agents implicated in 1.2 % of cases (*Mucor* spp, *Aspergillus* spp), Pathology Section of the Animal Health Trust, Newmarket, UK-1988-1997- mycotic agents implicated in 1.4% of cases (*Aspergillus* spp, *Absidia* spp, mixed bacterial, and mycotic), and Livestock Disease Diagnostic Center, University of Kentucky, 1986-1991- mycotic agents implicated in 1.7% of cases (*Aspergillus* spp, mucoraceous fungi, *Histoplasma capsulatum*, *Candida* spp).

In general, yeast infections associated with equine reproductive disease (endometritis, embryonic or fetal loss) are opportunistic, requiring predisposing conditions to become established. The origin of these agents is the caudal reproductive tract (vagina, clitoral fossa, contaminated skin of the external genitalia), or fecal contamination associated with pneumovagina or poor perineal conformation. **Antimicrobial administration and progesterone administration are possible predisposing factors, and iatrogenic uterine infections have been suggested.** *AHL*

COMPANION ANIMALS

Gallbladder mucocele in dogs *Margaret Stalker, Beverly McEwen*

Canine gallbladder disease encompasses a spectrum of changes including cholecystitis, cholelithiasis, gallbladder infarction, neoplasia, cystic mucinous hyperplasia, and mucocele. The latter, gallbladder mucocele, was only rarely reported in dogs prior to 2004, and since then has been diagnosed with increasing frequency in the caseload of the Animal Health Laboratory and Department of Pathobiology, OVC. Review of canine surgical biopsy and necropsy diagnoses in 2010 revealed **11 diagnoses of gallbladder mucocele, 6 of which had ruptured causing localized or more extensive bile peritonitis**. All cases were in smaller dog breeds, including 3 Shetland sheepdogs, and single cases in a Pomeranian, Maltese, Border terrier, teacup poodle, Cocker spaniel, Bichon X poodle, Nova Scotia duck tolling retriever, and a small (5 kg) mixed-breed dog.

Gallbladder mucoceles are characterized by intraluminal distension of the gallbladder with green-black, gelatinous, inspissated mucus and bile (Figures 1, 2), which may extend into the biliary tree causing biliary obstruction. A breed predilection for Shetland sheepdogs, Cocker spaniels and Miniature Schnauzers has been reported. **Clinical signs are typically nonspecific, including anorexia, lethargy, vomiting, abdominal pain, diarrhea, and/or jaundice**. Serum biochemistry reveals elevated serum ALT, ALP, AST, GGT and total bilirubin, with leukocytosis, mature neutrophilia and monocytosis on CBC. The ultrasonographic image of the enlarged gallbladder often has a characteristic echogenic, immobile, stellate pattern with a hypoechoic rim, resembling a cross-section of a kiwi-fruit. Histology reveals a gallbladder distended with mucinous secretions, and the lining mucosa is composed of hyperplastic, tall columnar, mucus-secreting epithelial cells.

The etiology of this condition is currently unclear. There appears to be an association with concurrent endocrine disease, as a significant proportion of dogs diagnosed with mucoceles also have hyperadrenocorticism or hypothyroidism, although a causal mechanism is at yet speculative. An association has also been suggested with dyslipidemia, and gallbladder dysmotility. Recent research has uncovered a significant association between this disease and a frameshift insertion mutation in an ABC phospholipid transporter important in canalicular transport and bile formation. This mutation would result in a truncated, dysfunctional transporter and associated alterations in bile composition, specifically reduced transport into bile of protective phosphatidylcholine.

This might increase the likelihood of chronic bile salt-mediated injury to the epithelium lining the gallbladder, resulting in compensatory mucinous hyperplasia. Similar ABC transporter mutations in humans result in severe progressive intrahepatic cholestasis and cholelithiasis, and the dog may offer a spontaneous animal model for translational medicine, with development of medical or dietary treatments beneficial for both species. *AHL*

References

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Figure 1: Surgically excised gallbladder mucocele, courtesy Karla Fernandez.



Figure 2: Bisected gallbladder mucocele, courtesy Andrew Vince.