UNIVERSITY of GUELPH LABORATORY SERVICES AHL Newsletter
ANIMAL HEALTH LABORATORY GUELPH KEMPTVILLE RIDGETOWN

Volume 4 Issue 1

In this issue:

What's happening at the AHL?

- We have purchased a Bayer **Advia 120 automated hematology analyzer**. This state-of-the-art instrument will replace our Technicon H1, and will provide faster turnaround on testing and greater reliability of results. We are currently establishing reference ranges for the Advia. We're very pleased to have been able to make this capital re-investment in the AHL.

- **Y2K** was uneventful at the AHL and generally throughout the Laboratory Services Division and the whole of the University of Guelph. All of our computer programs had been upgraded, and we saw no deleterious effects on service.

- The AHL has a **new website URL - http://ahl.uoguelph.ca** - for easier bookmarking (note that the www. prefix is not needed). We have added the current Fee Schedule, and will keep the site continuously updated. We would be happy to have your comments on the website.

- Congratulations to **Dr. Marie Archambault**, AHL veterinary bacteriologist, who successfully defended her Ph.D. thesis on "Investigation of the hemoglobin-iron acquisition system at the cell surface of *Actinobacillus pleuropneumoniae*" in Dec, 1999, at the University of Montreal.

- Dr. Doug Key participated in the "Provincial, Territorial, and Local **Pandemic Influenza** Contingency Planning Meeting", Jan 27/28.

- The AHL has been asked to assist Health Canada's zoonotic disease laboratory with collection of serum samples from domestic poultry flocks that are reared outdoors near large bodies of water, as part of **West Nile-like virus** surveillance.

- We isolated an **H3N2 swine influenza virus** from lung tissues from two pigs in January, and identified it as the A/Swine/Colorado/77 H3N2 subtype by hemagglutination inhibition testing. The virus was isolated in eggs, but not in primary pig kidney, MDCK or MARC cell lines. The submitted tissues came from a nursery and a finisher pig from two separate barns within the same production loop. Respiratory signs were evident in several other barns within this loop, but not in any of the farrowing units.

A progress report on the Animal Health Laboratory

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Q: How did the AHL originate?

A: The Animal Health Laboratory (AHL) came into being on April 1, 1997, as a major unit of the

Laboratory Services Division, University of Guelph (UofG) as a consequence of the Enhanced Partnership between the UofG and the Ontario Ministry of Agriculture (OMAF). The AHL is the successor to the Veterinary Laboratory Services Branch of OMAF in combination with the diagnostic labs of the Ontario Veterinary College. As part of the contract with OMAF, the AHL provides disease surveillance information for the province.

Q: What is the track record of the AHL to date?

A: We have fulfilled our mission of providing diagnostic, consultative, and surveillance services and supporting teaching and research through, for example, successfully integrating the diagnostic labs of the VLSB and the OVC, implementing a new computer system (VADDS), implementing and updating annually a fee schedule with individual test pricing, producing a quarterly newsletter, and continuously upgrading and reinvesting in technology (e.g. addition of PCR tests, new serology tests, new equipment).

Q: How has the AHL contributed to animal disease surveillance in Ontario?

A: The AHL supports provincial, national, and international trade by providing data to OMAF and CFIA. As a member of the Ontario Animal Health Surveillance Network (OAHSN, website = www.gov.on.ca/OMAF/english/infores/oahsn), the AHL is a key provider of reliable diagnostic information about the status of existing and emerging diseases in Ontario. The AHL processes about 40,000 case submissions per year, and performs about 700,000 tests on these accessions. Our food animal accessions originate from about 5150 cattle herds, 1290 swine herds, >1000 poultry flocks, and 450 small ruminant herds/flocks.

Q: What new and emerging diseases have the AHL detected?

A: Recent examples include: **in 1997**, identified multiply resistant *Salmonella typhimurium* DT104 in Ontario livestock and poultry, identified circovirus in Ontario swine (implicated as the cause of post-weaning multisystemic wasting syndrome), and Eastern equine encephalomyelitis; **in 1998**, confirmation of a cluster of cases of clostridial myositis in cattle, identification of chlamydiosis in birds in large retail aviaries, identification of the highly pathogenic J strain of myeloid leukosis in broiler breeders, identified an increase in K88 positive *E. coli* in swine; **in 1999**, caprine adenovirus, hepatic lipidosis in Galloway cattle, tuberculosis in elk, bovine botulism outbreak, infectious bronchitis outbreak, increasing trend of *Salmonella typhimurium* DT104 in cattle and swine, H3N2 swine influenza, increase in EVR stillbirths.

Q: How does the AHL support public health?

A: We detect and report important zoonotic pathogens, e.g. multiply antimicrobial resistant *Salmonella typhimurium* DT104 and other agents through routine diagnostic submissions and within our current antimicrobial resistance project. Selected human enteropathogens are reported to Health Canada through the Health Infostructure Support Program. We work closely with other agencies in detecting emerging diseases, e.g. West Nile-like virus.

Q: What challenges lie ahead?

A: As does any organization, the AHL must respond effectively to client needs and provide the best possible value with limited resources. We must continually review and control costs and develop alternative sources of funding. We must maintain current essential testing services while dealing with

emerging diseases and reinvesting in new technology. We welcome your suggestions on improving our services.

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CATTLE

Placental hemorrhage and stillbirths in dairy calves

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Over one year, 13 calves (approximately 50% of those born to first-calf Holstein heifers bred artificially (AI) to the same bull) were stillborn at term, or died very shortly after birth. Calves that appeared normal at birth and calves born to older cows bred to the same bull survived with no apparent ill effects.

Four stillborn calves and placentas were submitted to the AHL-Kemptville. Postmortem findings included severe hemorrhage of cotyledons, unclotted blood in thoracic cavities, and severe congestion and perivascular hemorrhages in most tissues. Blood smears from one stillborn calf showed adequate numbers of platelets, and bone marrow smears were very cellular with many megakaryocytes and active myeloid and erythroid series. Bacteriology, virology, serology and other testing was negative.

A full blood coagulation factor screen, including prothrombin time, partial thromboplastin time, fibrinogen, and Factors VII, VIII, IX, X and XI, was done on 7 related cows, 8 heifers and 5 'normal' calves from the affected herd. The profile of the cows and heifers was normal, but **in the calves, vitamin K dependent proteins (Factors VII, IX and X) were 25-30% below normal levels.**

Stillbirths stopped after cows and heifers were given intramuscular injections of 1.5 mg vitamin K/kg 1-2 weeks before calving. One stillbirth with typical lesions occurred from a heifer treated only two days before calving.

Hemorrhage resulting from vitamin K deficiency is common in human infants, and many physicians therefore routinely administer vitamin K to infants (1). Vitamin K deficiency in cattle has not been reported unless associated with hepatopathy, toxic (T2 mycotoxin) or other extrinsic factors such as antibiotic therapy, which were not identified in this herd. Spontaneous fatal hemorrhage in perinatal calves is very rare, because healthy calves, unlike lambs and piglets, develop adult levels of vitamin K dependent clotting factors within 24 hours of birth (1). **Because vitamin K dependent factors were reduced in surviving calves from this herd, a toxic factor was suspected but not identified.**

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Typing of bovine viral diarrhea virus isolates, 1998 and 1999

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Over the past 2 years, the AHL virology section has recovered 365 bovine viral diarrhea virus (BVDV) isolates from various bovine specimens submitted for virus isolation. Of these 365 isolates, 74 (20.4%) were cytopathic, recovered from animals with mucosal disease. The remaining 289 (79.6%) isolates were noncytopathic viruses, recovered from animals persistently infected with BVDV and those with various acute disease processes. This segregation of biotypes is similar to our findings in 1994 and 1995, at the end of the outbreak of severe acute BVD in Ontario (1).

Over these same past 2 years, we have provided genotyping for all BVDV isolates. The results of this genotyping for individual years are summarized in Table 1. Overall, **194 (53.2%) isolates were determined to be BVDV type 1, and 169 (46.3%) were BVDV type 2**. There were 2 (0.5%) isolates in 1998 that appeared to contain a mixture of BVDV types 1 and 2. These proportions of genotypes are similar to results reported by others (2).

Table 1. Genotyping of AHL isolates of BVDV, 1998 and 1999

BVD virus type	1998	1999	Total 98/99
BVD type 1			
Cytopathic	26(13.1%)*	23(13.9%)	49(13.4%)
Noncytopathic	85(42.7%)	60(36.1%)	145(39.7%)
BVD type 2			
Cytopathic	15(7.5%)	10(6%)	25(6.9%)
Noncytopathic	71(35.7%)	73(44%)	144(39.5%)
BVD type 1-2 mixture	2(1%)	0	2(0.5%)
Total for each year	199(100%)	166(100%)	365(100%)

***** proportion of the total for each year in brackets

The majority of these isolates were typed using a panel of 4 monoclonal antibodies. However, 27.1% of all BVDV isolates, including 17.2% of all BVDV type 1 viruses and 38.8% of all BVD type 2 viruses, could not be typed using these monoclonal antibodies. These BVDV isolates were subsequently typed using PCR technology (3).

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POULTRY

Ontario broiler chicken disease summary for 1999

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Yearly summaries of specific disease entities are produced from the AHL database of pathology diagnoses made at the Guelph, Kemptville and Ridgetown AHL labs. The following are some highlights of the more frequently diagnosed conditions or diseases of broilers.

Bacterial infections were the most common causes of mortality, and occurred throughout the growing period. During the first 2 weeks, **bacterial omphalitis** and **yolk-sacculitis** are frequent diagnoses. *Escherichia coli* are the bacteria most frequently isolated, with occasional isolates of *Salmonella, Pseudomonas* or *Klebsiella*. Other conditions occurring during the first 2 weeks include **starve-outs, dehydration** and **mycotic pneumonia.** Mycotic pneumonia, which is usually due to *Aspergillus* sp., is an indication of hatching or brooding facility contamination by fungi and fungal spores.

Bacterial septicemia occurs throughout the growing period and results in inflammatory lesions in a variety of body tissues including lungs, air sacs, joints and visceral organs. *E. coli* are the bacteria most frequently isolated, wheras other bacteria such as *Salmonella* are only occasionally identified. The antimicrobial susceptibility of these bacteria is very variable, therefore susceptibility testing of each isolate is required - extrapolation of data from previous isolates is not reliable. **Cellulitis** - an inflammatory condition involving subcutaneous tissues usually of the ventral portions of the body - was frequently identified in market-age birds. Cutaneous trauma (scratching) and certain serotypes of *E. coli* are considered to be important factors in these infections. **Arthritis** due to bacterial infections of the leg joints was most frequently seen in the last 3 weeks of the grow-out period. *E. coli* were isolated in 80% and *Staphylococcus aureus* in 20% of the cases. **Viral arthritis due to reovirus** remains a rare cause of arthritis, with only one case identified in US- hatched chicks last year. In some cases of bacterial septicemia, there appears to be a concurrent respiratory viral infection.

During the fall and winter, **infectious bronchitis virus** (IBV) was associated with acute respiratory disease, with or without secondary bacterial septicemia in near market aged birds. Last year, several different farms experienced a sudden rise in mortality at 4 to 5 weeks of age with birds that appeared depressed and dehydrated. These birds had pale, swollen kidneys on postmortem examination and microscopic lesions of severe nephritis from which a coronavirus compatible with IBV was isolated. Specific strains of IBV can cause nephritis, but until this past year this syndrome has rarely been identified in Ontario chickens.

Proventriculitis, which may be due to viral infections, was identified more frequently last year in flocks with a history of poor-doing birds and/or increased mortalities. **Acute death syndrome ('flip-overs')** and **ascites** continue to be frequent causes of mortalities in birds greater than 1 week of age. Enteric diseases such as **coccidiosis**, particularly cecal coccidiosis, and **necrotic enteritis** due to *Clostridium* spp. continue to cause mortality, usually in birds greater than 3 weeks of age. **Inclusion body hepatitis** due to an adenovirus infection caused mortality in birds 2 weeks of age and older. This disease has been a repeating problem on some farms. Evidence of bursal damage suggestive of **infectious bursal disease** viral challenge was seen in some of the birds with viral hepatitis.

Weak legs and locomotion problems continued to be common reasons for postmortem submissions. Conditions such as **tibial dyschondroplasia, valgus/varus deformity, tibial rotation, rickets** and occasional **spondylolisthesis ('kinky back')** reflect complex etiologies, which include genetic factors, nutrition, rapid growth rates and concurrent diseases. **Botulism** was an occasional cause of weakness and mortality in broilers.

Spiking mortality associated with hypoglycemia was diagnosed in some flocks which experienced a sudden increase in mortalities at 1 to 3 weeks of age. Recent work by researchers in Georgia demonstrated that continuous lighting programs, infection with a virus (arena-like virus), and stressors such as fasting for 2-5 hours or chilling could consistently reproduce the syndrome (1). Controlled light/dark programs prevented the experimental production of the syndrome (2).

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Error in feeding: serious effects in a small layer flock

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When a veterinary practice dedicated to small animals or large, food-producing animals is asked for advice on poultry, pigeons or show fowl, practitioners may not know where to turn. You might request help from the Animal Health Laboratory, or the Ontario Association of Poultry Veterinarians [contact: Dr. Rachel Ouckama, (905) 885-6337].

A 25% mortality had occurred in a small layer flock of 100 Rhode Island Red chickens in the previous 6 weeks. The greatest losses were in 5-month-old pullets that were slow coming into production. A similar problem had occurred a year ago, but it had not been investigated. The producer's practitioner conducted a necropsy and noted fibrinous pericarditis and swollen kidneys. Formalinized tissues were sent to the AHL-Ridgetown, and pericarditis, severe tubular necrosis, and urate nephrosis were observed. Additional birds were submitted to the AHL and similar lesions were found - there was a heavy white precipitate over the heart, liver and mesentery; the kidneys were distended with a large amount of a chalky precipitate. The diagnosis of urate nephrosis was confirmed.

The pullets had been purchased as day-old chicks and fed a starter diet. **At an early age, the pullets were switched to the layer diet that the hens received.** A commercial layer diet (recommended for pullets and hens in production) has a calcium level of about 3.5%. A commercial grower diet (recommended for pullets before they come into production) has a calcium level of about 0.9% (1). Excess dietary calcium, in particular if combined with low available dietary phosphorus, fed to growing pullets has caused urolithiasis in experimental trials (2). Excess absorbed calcium is excreted through the kidneys; high levels cause ureteral and renal impaction, resulting in nephrosis (3). Feeding errors occur occasionally in commercial flocks when the wrong feed is given to pullets or hens. **Too much calcium fed to pullets can result in urate nephrosis, and conversely, if a grower diet is given to**

laying hens, severe osteoporosis can occur. A marked difference in susceptibility to urolithiasis caused by high-calcium diets has been described between two strains of leghorn chickens (2). Water deprivation, some strains of infectious bronchitis virus, and nephrotoxic mycotoxins also should be considered as causes of urolithiasis (2).

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Necrotic enteritis in poultry

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Necrotic enteritis is a worldwide poultry disease known to be caused by the bacterium *Clostridium perfringens* type A or less commonly type C. Type A produces alpha toxin, and type C can produce both alpha and beta toxin. These toxins are thought to cause the characteristic mucosal necrosis seen in this disease.

Gram stains of intestinal content consistently reveal large numbers of gram-positive bacterial rods, and *Clostridium perfringens* grows readily on blood agar plates within 24 hours of inoculation, making diagnosis relatively easy.

Clostridium perfringens is an environmental organism, but it is also found in intestinal content. It is thought that increased numbers of organisms may result from changes in the nature of the ration that allow for their proliferation. Fish meal, barley, and high levels of wheat are known to exacerbate the disease (1). Damage to the intestinal mucosa is another predisposing factor to disease and can include concurrent coccidia infestation and/or ingestion of high-fiber litter.

The disease is seen most commonly in young broilers (2-5 weeks of age), but it also occurs in older pullets and layers (1). Although clinical signs such as inappetance, depression, diarrhea, reluctance to move, and ruffled appearance may occur, it is more common to see increased sudden mortality. Grossly, the lesions typically involve the small bowel, although cecal and hepatic lesions have been noted on occasion. The mid-small intestine typically contains a pseudo-membranous or diphtheritic membrane, occasionally accompanied by flecks of blood but no outright hemorrhage.

The histologic lesions include focal areas of superficial villus necrosis, fibrin aggregation with necrotic cell debris, heterophil infiltration, and bacterial rods colonizing the denuded surface. Antibiotics such as penicillin or bacitracin are commonly included in the feed or used to successfully treat affected birds.

Although rarely reported in ratites, we had a recent case involving sudden death of a 5-month-old ostrich. The lesions were mainly localized to the paired ceca with lesser involvement of the jejunum. There was necrotizing typhlitis with colonization by clostridial rods; *C. perfringens* type A was isolated

on blood agar and seen on gram-stained tissue sections. All other organ systems were normal. Differential diagnoses in this species included salmonellosis, spirochetosis, and histomoniasis.

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SWINE

Manure gas poisoning of 140 pigs

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One hundred-and-forty of 1200, 50-100 kg pigs were found dead 3 hours after liquid manure was pumped from an underfloor pit in a naturally ventilated barn. The wind was from the west, and all dead pigs were in pens located on the east side of the barn. Fourteen dead pigs were submitted to the AHL-Kemptville for necropsy.

All pigs were in excellent body condition, had extensive unilateral, ventral or dorsal red-blue skin discoloration, and exhibited severe pulmonary congestion and edema. Histopathology revealed severe pulmonary congestion and interlobular and alveolar edema, severe congestion of meningeal and other vessels, and moderate perivascular cerebral edema.

Surviving pigs appeared normal, and no respiratory or other complications were reported within a few weeks of this incident.

Manure gas contains hydrogen sulfide, methane, ammonia, carbon dioxide, and other gases and vapors that are produced by the anaerobic decomposition of feces and urine. They can reach levels of 1000 ppm or more when released from liquid manure during agitation. Exposure to these gases (especially hydrogen sulfide) can cause peracute death in humans, swine and cattle. In swine, hydrogen sulfide causes severe distress, eye irritation and drooling at 250 ppm, and cyanosis, convulsions and death at 1000 ppm.

Manure pits should never be flushed or agitated in closed spaces. Even in winter, fans should run at full capacity with doors and windows open, preferably with a brisk wind blowing. If at all possible, all animals should be removed from the building.

At concentrations higher than 200 ppm, hydrogen sulfide may paralyze the olfactory apparatus and become undetectable by smell - it is very important not to attempt to 'rescue' affected pigs because exposure to manure gas can cause peracute death in humans.

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Colibacillosis in pigs in 1999

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Colibacillosis is a major cause of illness and death in neonatal, young and post-weaning piglets. The disease is usually caused by enterotoxigenic strains of *Escherichia coli* (ETEC), although enteropathogenic *E. co*li (EPEC) strains may also occasionally cause the disease. Diarrhea is typically fluid and profuse, and frequently results in severe dehydration and circulatory shock.

ETEC produce **fimbrial adhesins** named K88 (F4), K99 (F5), 987P (F6), F41 and F18 that mediate the adherence of the bacterium to the mucosal surface. **Enterotoxins** produced by the ETEC strains pathogenic to pigs include heat-labile enterotoxin (LT), and/or heat-stable enterotoxins STa or STb. Some strains, although less common, produce a **Shiga-like toxin** (SLT-IIe) and may cause edema disease in addition to diarrhea. Also uncommon are strains that produce no toxins, but efface the microvilli of the epithelial cells to which they attach; such strains contain *eae* genes and are known as EPEC.

ETEC-expressing K88 or F18 account for essentially all post-weaning colibacillosis in pigs. **K88-positive ETEC is believed to be responsible for the majority of neonatal colibacillosis as well** (1-3). Because many strains of *E. coli* isolated from animals are nonpathogenic, it is important to identify the virulence factors produced by ETEC or EPEC strains to establish the etiology of diarrhea. Detection of the genes that encode those virulence factors will soon be available at the AHL in collaboration with the Ontario Veterinary College.

Submissions made to the AHL are usually related directly to piglet diarrhea, and ETEC organisms are frequently isolated. In calendar year 1999, ETEC organisms were isolated from 290 submissions, with 226 isolates in pure culture and 64 found in combination(s) (Tables 1 and 2). It is desirable to characterize the *E. coli* isolate obtained from animals to provide evidence that the isolate is virulent. Characteristics that suggest virulence include serogroup, fimbriae, and exotoxins. While complete serotyping includes determination of the O (somatic), K (capsular), H (flagellar) and F (fimbrial) antigens, diagnostic laboratory serotyping is often reduced to one or two classes of antigens and to a limited spectrum of antisera. This should be adequate, since in a given region, pathogenic serotypes maintain their antigenic characteristics.

The serogrouping utilized by the AHL identifies the O (pools 1N and 2N) and F (K88 and K99) antigen groups. ETEC isolates are categorized into a specific serotype pool (1N or 2N), with each pool containing serotypes of *E. coli* known to cause diarrhea in certain age groups. **Pool 1N** contains the serotypes O8:K"S16", O8:K25, O9:K28, O9:K30, O9:K35, O9:K103, O9:K"79-416", O20:K101, O64:K"V142", and O8:K+. These serogroups are most commonly associated with diarrhea in neonatal

piglets and calves. **Pool 2N** serogroup contains those strains associated with diarrhea in older and post-weaning pigs, and with edema disease. This 2N pool contains the serotypes O138:K81, O139:K82, O141:K85ab, O141:K85ac, O45ac:K"E65", O157:K"V17", O115:K"V165", O8:K"X105", O?:K48 and O149:K91. The antisera against **K88 (F4)** and **K99 (F5)** are used by the AHL to detect strains of ETEC organisms that produce the fimbrial adhesins most commonly associated with diarrhea.

Table 1. Number of isolates of ETEC in 1999 at AHL laboratories.

Serotype	No. of isolates*
Pool 1N	58
Pool 2N	99
K88	194
K99	4

* In some submissions, more than 1 serotype was identified.

Table 2. Ages of submitted pigs with ETEC.

Serotype	No. of isolates	Average age**	Age range
Pool 1N	39	9 d	1-14 d
Pool 2N	48	5.7 wk	1d - 16 wk
K88	139	3.9 wk	2d - 16 wk
Pool 1N, pool 2N	6	7 d	1 - 14 d
K88, pool 1N	10	8 d	1 - 24 d
K88, pool 2N	44	4.6 wk	1 - 11 wk
K99, pool 1N	3	5.3 d	na
K88, K99, pool 2N	1	4 wk	na

** when included in history. na = data not available

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Sudden death of two mature sows

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Two mature gestating sows (parities 4-5) died unexpectedly in mid-September in a 1500 sow farrow-to-wean operation. The herd had been established for several years, and the most recent disease problem was an outbreak of TGE in 1996. Annual animal inventory turnover was approximately 30-35%, with sows often retained longer, because of the genetic potential of their offspring. Incoming gilts are vaccinated against PRRSV during their quarantine/acclimatization period, but sow vaccination is not practiced. PRRS has not been identified as a problem in this herd. Sows are routinely vaccinated pre-farrowing with an *Escherichia coli*, rotavirus and *Clostridium perfringens* type C bacterin/vaccine/ toxoid.

Autolysis was quite marked in both sows, with decomposition of tissues noted as being "quite advanced considering the length of time elapsed since death" by the veterinarian who performed an on-farm examination. One liver was bronze, and emphysematous while the other one was congested and necrotic. A section of skeletal muscle showed marked interstitial edema and congestion that extended into the adjacent adipose tissue. Large numbers of clostridia-like rods were evident in the affected areas. A liver swab was submitted for culture and fluorescent antibody staining for clostridia. *Clostridium perfringens* was isolated from the liver. Fluorescent antibody staining of direct smears identified *Clostridium novyi* and *chauvoei*, but a stronger reaction was observed with *C. novyi*. Based on previous clinical experience, the practitioner instituted a two-week regime of in-feed antibiotics. There were no further losses.

Clostridium novyi is an anaerobic, large, gram-positive, spore-forming rod bacterium. To types A to C, which differ biochemically, epidemiologically, and pathogenically, some would add *Clostridium haemolyticum* as Type D. Type A is common in soil. Types A and B occur in normal intestine and liver of herbivores. All enter their hosts by ingestion or wound infection. The organism produces highly potent exotoxins. Type A is implicated in gas gangrene of humans and wound infections in animals (myonecrosis). Type B causes infectious necrotic hepatitis ('black disease') of sheep and cattle, rarely horses and swine. Type C is the reported cause of osteomyelitis of water buffalo in Southeast Asia. Type D causes bacillary hemoglobinuria ('red-water') of cattle and sheep.

Clostridial toxemia as a cause of sudden death in sows is infrequently reported in the literature (1-4). An obvious feature of pigs that had died from this disease was the bronze color of the liver and the presence of large numbers of small gas bubbles in the substance of the organ when cut (3). The type involved in swine infections was identified as type B and/or A (3). Isolation and typing of the organism are difficult since it has the most fastidious growth requirements of the clostridia commonly encountered in swine (3). Studies have reported using zinc bacitracin to reduce mortality, and disposal of carcasses by incineration or deep burial may reduce the contamination of the environment by spores (4).

Although great care must be taken in trying to interpret the bacteriological findings, since *C. novyi* is an expected postmortem invader, **the identification of clostridia, along with the history of sudden death, rapid postmortem decomposition of internal organs, and the presence of gas bubbles in the liver are suggestive of death due to clostridial infection.**

We thank the bacteriology staff for their technical assistance.

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HORSES

Outbreak of an atypical *Streptococcus equi* infection on a horse breeding farm

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An upper respiratory disease outbreak started on a Southwestern Ontario horse breeding farm in early December and has been ongoing since then. The farm is a boarding/breeding/foaling operation with roughly 60 horses. Initially, a group of yearlings arrived from a training facility for lay-up with two individuals affected. These yearlings were dull, intermittently pyrexic and had small draining submandibular abscesses. These horses were isolated. One to two weeks following the arrival of the affected individuals, several others in the yearling lay-up group developed pyrexia and/or abscessation.

Typically, affected animals are mildly to severely pyrexic and dull with normal lung sounds but increased upper tracheal sounds and sensitivity to palpation of the larynx/throatlatch area. Coughing has not been seen consistently and has varied in severity. Fevers have been as high as 41^{o}C (105.5^{o}F) and abscesses have ranged from 10 - 20 cm diameter with some extending up to the ear. Many affected horses were pyrexic and dull for variable periods but recovered without abscessation. Affected individuals were isolated and fevers were treated with label doses of flunixin or phenylbutazone. Severely affected horses received penicillin G. **As the outbreak has progressed, all age groups have been affected including weanlings, yearlings and older healthy resident individuals.** Morbidity is estimated at 50% with 0% mortality. Yearlings have been the most affected group.

Samples for culture were collected and submitted to the AHL-Guelph. The isolates were first identified as *Streptococcus dysgalactiae* subspecies *equisimilis*. Upon further testing using API 20 STREP (BioMerieux) and the sugar ribose, some strains identified previously as *S. equisimilis* were confirmed to be atypical *S. equi*. Their identity as *S. equi* was also confirmed by immunoblot demonstration of the characteristic *S. equi* M protein by Dr. Timoney, University of Kentucky. We therefore conclude that some horses were infected with both *S. equi* and *S. equisimilis*.

The laboratory differentiation of these two bacteria is traditionally based on the fermentation of lactose, sorbitol, and trehalose. Lancefield group C streptococci which fail to ferment any of these sugars are identified as *S. equi* and those able to ferment trehalose are identified as *S. equisimilis*. The isolates recovered from the abscess fermented the sugar trehalose but only on initial subculture. Although uncommon, atypical isolates of *S. equi* able to ferment trehalose and/or lactose have been reported but these atypical strains are unable to ferment ribose (1). As a result of these atypical biochemical results, ribose fermentation will be added to the standard three sugar fermentation tests done at the AHL. This

will prevent the misidentification of an atypical *S. equi* as *S. equisimilis.* **On the basis of the culture results and clinical signs, a final etiologic diagnosis of atypical *S. equi*-induced abscesses was made.** Further study of these unusual atypical *S. equi* continues in the laboratory of Dr. Timoney.

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COMPANION ANIMALS

Bilateral nephrolithiasis with renal failure in a dog

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An 8-year-old spayed female, spaniel/beagle-cross dog was euthanized after 8 days of anorexia and deteriorating condition. Abdominal palpation was painful, but vomiting or other signs were not observed, and she was passing small quantities of urine. During this period, blood urea increased from 32 to 46 mmol/L (ref = 3-11) and creatinine increased from 219 to 617 µmol/L (ref = 63-124).

On gross postmortem examination, she was in very fat body condition. **Each kidney contained a ~6 X 4 cm, smooth, hard, white chalky struvite (magnesium ammonium phosphate hexahydrate) calculus with less than 10% of the renal cortex remaining.** No visible calculi were present in the urinary bladder. Mineralization, ulcers or other uremia-related lesions were not evident in the mouth, stomach or other tissues.

On histopathology, renal cortical lesions varied from severe cortical interstitial fibrosis with prominent lymphocytic infiltrates to subtotal cortical atrophy with no glomeruli and only a few dilated tubules containing proteinaceous casts remaining. Occasional microliths were present in the mucosa of the urinary bladder. No mineralization, ulceration or other lesions commonly associated with uremia were present in kidneys or other tissues.

Nephrolithiasis accounts for less than 3% of urinary calculi in dogs and cats (1). Struvite calculi commonly follow urinary tract infections. Surgical and/or 'dissolution' techniques have been used for treatment of nephrolithiasis (2), but it is unlikely that this dog could have survived, because of extensive destruction of renal tissue.

This case is unusual because of the absence of typical uremic signs and lesions in spite of very extensive loss of renal tissue, and the absence of cystic calculi, which usually accompany nephrolithiasis.

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Trichobezoars in two Pomeranian puppies

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A 7-week-old female Pomeranian puppy had vomited several times the day before she was presented to the veterinarian in a seizure. Hypoglycemia was marked. She responded briefly to intravenous glucose but soon relapsed to a non-responsive state. That evening she vomited several times and died overnight. The next day, the only other pup in the litter was presented with a similar history of vomiting, weakness and crying. Despite oral treatment for hypoglycemia and ascarids, the pup died later in the day.

Necropsy findings were similar in both pups. In the first pup, the distal esophagus was markedly dilated and completely blocked by an impacted mass of hair 6 cm by 2 cm. The stomach and intestine contained only a small amount of fluid. In the second pup, the distal esophagus was dilated and the mucosa was dark. A large, firm mass of impacted hair filled the stomach and extended 5 cm into the duodenum. The mass filled the lumen so tightly that when an attempt was made to extract it through the incision in the stomach an intussusception of the duodenum occurred.

The bitch was probably the source of the hair. Her abdomen had been clipped before she whelped but the owner reported that she had really "blown her coat" (undergone an effluvium) after the pups were born. Why the pups would ingest the hair is not understood. This is a very interesting case and both authors would be pleased to hear from anyone with a similar experience.

Trichobezoars, or hairballs, are concretions of hair formed in the digestive tract. Although they are not uncommon in ruminants, rabbits and laboratory rats, references to them in dogs are rare. Signs of trichobezoars are nonspecific and may mimic those of other gastrointestinal conditions. **The "Rapunzel syndrome" in humans refers to a rare form of gastric trichobezoar with a tail that can extend to or beyond the ileocecal valve.** In children, they are associated with pica and psychiatric disorders; only 12 such cases had been reported in the literature. The mass may be removed by gastrotomy and multiple enterotomies or via laparoscopy (1). Fresh pineapple juice, which contains the enzyme bromelain, capable of digesting hair, has been successful in treating hairballs in rabbits (2). Hairballs have been controlled in laboratory rabbits by increasing the roughage in the diet and administering a commercial remedy for hairballs in cats on a regular basis.

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SMALL RUMINANTS

Viral diarrhea in young goats

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Diarrhea in kid goats may be due to both nutritional and infectious causes (1). Nutritional causes of diarrhea include overfeeding of milk, use of poor quality milk replacers, switching to cow milk replacers, and sudden changes in feeding routine. Goat kids may be predisposed to infectious diarrhea by overcrowding and poor sanitation, with increased incidence during extreme weather conditions. A number of pathogens can induce diarrhea in kid goats. The most common pathogens in goats less than 3 weeks of age include cryptosporidia, enteropathogenic *Escherichia coli*, *Salmonella*, rotavirus (group A and non-group A), and coronavirus. Kids older than 3 weeks of age may also suffer from coccidiosis, enterotoxemia, and yersiniosis.

The AHL virology section identified caprine adenovirus in a 2-to-3 week-old kid that was submitted for necropsy. This milking goat herd had a problem with diarrhea in 2-week-old kids. Of 30 kids in the affected age group, 20 were sick and 4 had died. The submitted goat kid was dehydrated and had no fat stores. Distal small intestine and large intestine were dilated with watery green fluid. Microscopically, there was severe acute cryptal necrosis in most areas. Both intracytoplasmic and intranuclear structures suspicious for inclusion bodies were seen. Caprine adenovirus was isolated. Occasional *Cryptosporidia* spp. were seen on fecal examination.

Adenoviruses have been reported as a cause of enteritis in kids from a dairy herd in California (2), in which 50 kids died with diarrhea and had epithelial necrosis of the small intestine and colon. This goat adenovirus was determined to be a new serotype designated goat adenovirus type 2. The adenovirus recovered from the Ontario goat has been forwarded to the same laboratory (National Animal Diseases Center, USDA, Ames, Iowa) for typing.

As well, **the AHL has identified group A rotavirus as the cause of diarrhea in kid goats in Ontario.** Goats aged 4 days to 5 weeks have been affected. These goats scour, and may have distended abdomens and abdominal pain. Histological evaluation of the small intestine shows mild to severe diffuse and patchy villus atrophy and fusion. Several other agents were also identified in these neonates, including cryptosporidia, *Eimeria* spp, and *E. coli*. Lactogenic immunity is important for protection from disease due to rotavirus, and reduces viral replication. Kids with lactogenic immunity may be protected from diarrhea even though rotavirus can be found in their feces.

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Iodism and goiter in a herd of goats

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In 1999, a producer encountered several does and kids with enlarged thyroid glands. The producer had fed the does a dairy ration from a local feed mill from February until December 1999, when the does were dried off. All goats had free-choice loose blue salt, along with a mineral block available at all times.

In January 2000, several weak kids were born. One doe had three kids, born dead. Most of the does and many of the kids had enlarged thyroid glands. Thyroxine (TT4) levels on two of the does were 102 and 204 nmol/L (ref. <70 nmol/L). Iodine levels in two milk samples were 2.02 and 2.46 ppm (ref. 0.07-0.40 ppm). A milk sample taken a year earlier, before the clinical problem began and held frozen, contained <0.10 ppm iodine.

Dietary goiter in goats is usually caused by iodine deficiency or by the ingestion of goitrogens. In addition, elemental cobalt deficiency (and thus vitamin B_{12} deficiency) has produced elevated thyroxine levels accompanied by marked hypertrophy of the thyroid gland.

Iodine poisoning does not occur under natural conditions, as the toxic dose of iodine is very high. **In this case, it is likely that a source of excess iodine was ingested, as the thyroxine levels were elevated and the iodine levels in milk samples were markedly elevated.** A source of the excess iodine has not been identified.

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Outbreak of *Streptococcus zooepidemicus* on a goat farm

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At the beginning of January, a group of fresh does was experiencing acute mastitis, this problem has been ongoing since then. Clinical signs of abnormal milk, swollen glands and discomfort were similar in all. **One hundred and twenty-five animals were at risk and 17 experienced clinical mastitis.** A number of does examined had moderate abrasions to the teat end.

Five milk samples were collected for culture, and the isolates were identified as *Streptococcus equi* subspecies *zooepidemicus*. The isolates were confirmed as group C streptococci by the latex agglutination test (BBL). Three milk samples contained pure cultures of *S. zooepidemicus.* One contained *S. zooepidemicus* and *Staphylococcus aureus*, and the fifth one showed no bacterial growth. Susceptibility testing revealed *S. zooepidemicus* susceptible to penicillin, novobiocin/penicillin, erythromycin, ampicillin, cephalothin, and oxacillin; but resistant to tetracycline. The *S. aureus* isolate was susceptible to novobiocin/penicillin, cephalothin, and oxacillin, but resistant to ampicillin, erythromycin, pirlimycin, penicillin and tetracycline.

Affected individuals were isolated and treated with CEFA-LAK (cephalosporin). As the outbreak has progressed, more cases have occurred and morbidity is estimated at 12% with 0% mortality. Further investigation concluded that this problem is likely related to the liners of the milkers. The owner has

been advised to replace these, but also to have the mechanical operation of the milking system, vacuum pressure, pulsation rate, etc. examined.

***Streptococcus zooepidemicus*, an opportunistic pathogen, has been known to cause severe mastitis that can go on to affect a large part of the herd** (1). This organism is also a threat to human health; immunocompromised patients are more at risk. Human cases of septicemia, meningitis and glomerulonephritis have been reported (2,3). Unpasteurized milk should not be consumed, as zoonotic organisms may be present.

We thank the bacteriology staff of the AHL for their technical assistance.

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CERVIDS

Malignant catarrhal fever in Sika deer (*Cervus nippon*)

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Over the course of three weeks, six Sika deer from a small private collection of 19 deer died following a brief clinical course of depression and bloody diarrhea. Some affected animals exhibited convulsions shortly before death. Post- mortem findings included hemorrhagic intestinal and ruminal lymph nodes, and hemorrhages within the spiral colon and cecum. Differential diagnoses included bacterial septicemias, e.g. *Salmonella* spp., *Yersinia pseudotuberculosis*; viral diseases, e.g. malignant catarrhal fever (MCF), adenovirus; and bluetongue, epizootic hemorrhagic disease, both exotic to Ontario].

No significant bacterial pathogens were cultured from various organs. However, histologic examination of tissues revealed **severe hemorrhagic colitis, with marked vasculitis** characterized by endothelial cell degeneration, fibrinoid necrosis and perivascular infiltration by mixed inflammatory cells. Based on clinical signs and lesions, a tentative diagnosis of MCF was made.

MCF is an important viral disease of many wild and captive/farmed deer species, as well as bison and domestic cattle; Sika deer are highly susceptible, as are red deer, Père David's deer, rusa, Varasingha deer, chital, white-tailed deer, mule deer, roe deer, reindeer and moose. Fallow deer appear to be resistant to the disease.

Typical history includes contact with sheep, which serve as well-adapted asymptomatic reservoirs for the sheep-associated MCF virus, ovine herpesvirus-2 (OHV-2). Deer are also susceptible to alcelaphine

herpesvirus-1 (AHV-1), shed by African wildebeest, a potential problem in zoological collections. In this case, OHV-2 was identified; the source was likely sheep that shared a common fenceline with the deer.

Clinical signs in deer include conjunctivitis, nasal discharge, ulceration of the muzzle and mouth, depression and bloody diarrhea (1). The disease is invariably fatal. Diagnosis is made based on typical histologic lesions. **While the virus cannot be isolated, definitive diagnosis can be made by PCR on frozen postmortem tissues, preferably lymph nodes and spleen, or chilled EDTA blood from clinical cases** (sent to NVSL, Ames, Iowa).

No effective treatment is available for affected deer. Mortality is 100%; morbidity also can approach 100%. The disease is not considered to be contagious amongst deer. Prevention involves avoiding any contact between susceptible species and reservoir hosts. Farm workers who handle deer should avoid any contact with sheep.

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Animal Health Laboratory Accreditations:
American Association of Veterinary Laboratory Diagnosticians (AAVLD) (lab system)
Thyroid Registry of the Orthopedic Foundation for Animals Inc. (OFA) (thyroid function)
Canadian Food Inspection Agency (CFIA) (EIA)
Canadian Association of Environmental Analytical Laboratories (CAEAL) (metals)
ISO 9002 registered (toxicology)

Comments? Suggestions?

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