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Animal Health Centre

Ministry of Agriculture, Food and Fisheries

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From the Director

Avian influenza, was initially diagnosed by the Animal Health Centre in mid- February (subsequently confirmed as H7N3 highly pathogenic AI) with the last positive case identification in mid May. Repopulation of the Fraser Valley poultry industry began in early July. Ultimately, over 17 million birds were destroyed, most through normal processing channels, and total economic losses are estimated at $380 million. This outbreak and eradication exercise emphasizes the importance of animals to our economy but, of course, pure economics fail to adequately address how difficult this has been on producers, processors, and others directly and indirectly involved with the poultry industry.

Following the eradication of AI, all levels of government and the industry have been actively focusing on how we can better prepare for the next disease incursion whether it should occur in poultry or another livestock industry. The culmination of these examinations was a major AI Forum in Abbotsford in late October sponsored by the Canadian Food Inspection Agency; the BC Ministry of Agriculture, Food and Fisheries; and the poultry industry. A series of action items resulted from that meeting and much work remains to be done. Many positive steps were identified and will be taken to improve the critical infrastructure necessary to respond to a foreign animal disease incursion.

The diagnosis of bovine spongiform encephalopathy (BSE or mad cow disease) in Canada in May 2003 has caused extreme financial difficulties for the entire cattle industry due to the US border closure to our live cattle export market. To ensure that Canada returns to its rightful place as a well-recognized source of healthy animals and animal products, we must demonstrate to the global community that if BSE is present in this country it is at a very low level (<1 animal per million). Active surveillance must be done across the country to provide this level of confidence to our trading partners. The Animal Health Centre has dedicated an area of the laboratory to begin BSE testing for BC animals. Renovations have been completed, a contract technician has been hired and sent for training at the National Centre for Foreign Animal Disease (NCFAD) Laboratory in Winnipeg. Our bio-containment has been approved by the CFIA and we are ready to begin BSE testing using the rapid Western Blot test. Testing within the province will speed up results reporting to BC submitters. Suspect positive samples will be forwarded to the NCFAD for confirmatory testing. As our contribution to the national testing and surveillance program, British Columbia must analyze approximately 1900 samples from targeted high risk animals in 2005.

On behalf of the entire staff of the Animal Health Branch, we wish you all the best for a happy, productive, and satisfying New Year in 2005.

TOLL FREE NUMBER AND WEB SITE:

Please note that the Animal Health Centre has a toll free number: 1-800-661-9903. Keep this in mind, if calling long distance. This Newsletter, and other information from the AHC, may also be found on the Internet at our web site : http://www.agf.gov.bc.ca./croplive/anhlth/ahc

Increased AHC laboratory isolations of *Salmonella dublin*:

S Byrne PhD,
Head, Bacteriology

Salmonella dublin is a bovine adapted serotype which causes serious invasive disease and has human disease potential. Herd symptoms include death in calves and young animals and abortion in adult cattle. Carrier animals may harbor *S. dublin* in the lymph nodes and internal organs and will periodically shed the bacteria in feces and milk. Neonatal calves may shed *S. dublin* without any evidence of disease.

Human infections may lead to septicemia, osteomyelitis, meningitis and death, and are usually associated with the consumption of raw milk. *S. dublin* is more likely to cause invasive disease in humans - approximately 70% of human infections result in invasive disease compared to approximately 6% for human infections with *S typhimurium* (Vugia et al. 2004).

Since August of this year, we have isolated *S dublin* from five bovids (table 1) as compared to only 4 isolates between the years 2001-2003. Four of the recent isolates were recovered from animals in the Fraser Valley and one from Vancouver Island (case C). *S. dublin* was isolated in large numbers from extra-intestinal tissues in four of the five cases illustrating the invasive nature of this serotype.

All isolates were sent to the British Columbia Centre for Disease Control for pulsed field gel electrophoresis analysis (PFGE) to determine similarity. The strains from cases B, C and D had identical PFGE patterns, whereas the strains from cases A and E were significantly different, each having a unique pattern. The PFGE pattern from cases B, C and D was also identical to that of other *S. dublin* isolates recovered in previous years, suggesting that this organism has been maintained in carrier animals.

Because of the potential for severe human disease and significant economic losses, it is important to identify and treat animals carrying *S. dublin* early in the course of the disease. Detection in carrier animals may require submission of multiple specimens due to the intermittent nature of shedding. In order to reduce transmission to humans, raw milk from these cows should not be consumed and contact by children or immuno-compromised individuals should be curtailed.

Table 1

Case	Date	Host	Organ	Bacterial Load
A	2004/08/23	2 month calf	Small intestine, liver, lung, umbilicus	3+
B	2004/09/17	Holstein fetus	Lung and Stomach	4+
C	2004/10/4	Holstein fetus	Small intestine, lung, placenta	4+
D	2004/10/21	5 yr Holstein	Spleen, lung, liver, small intestine	4+
E	2004/10/25	7 day Holstein	Small intestine	Few

Reference:

Vugia DJ, Samuel M, Farley MM, Marcus R, Shiferaw B, Shallow S, Smith K, Angulo FJ; Emerging Infections Program FoodNet Working Group. Invasive Salmonella infections in the United States, FoodNet, 1996-1999: incidence, serotype distribution, and outcome. *Clin Infect Dis. 2004 Apr 15;38 Suppl 3:S149-56.*

An outbreak of severe respiratory disease and mastitis in a dairy herd:

D McIntosh

A large dairy herd experienced an acute outbreak of severe bovine respiratory disease (BRD) in adult cows that resulted in several deaths. The cows died from acute bronchopneumonia as a result of a combined infection with Bovine Respiratory Syncytial Virus (BRSV), *Mannheimia hemolytica*, and *Mycoplasma bovis*. Within two weeks following the BRD outbreak, several cows developed severe mycoplasma mastitis and had to be destroyed.

This disease outbreak followed the introduction of a clean-up bull into the herd. Whether the bull was a carrier of the mycoplasma or any of the other infectious agents is undetermined; however, most *Mycoplamsma bovis* infections are introduced into a herd by clinically healthy carrier cattle.

Severe pneumonia can occur especially if other respiratory bacterial or viral pathogens are present as there appears to be a synergy between *M bovis* and these other pathogens, particularly if there is co-infection with Bovine Virus Diarrhea Virus (BVD).

M bovis infections are often difficult to eradicate once introduced into a herd. Infected cattle can shed the mycoplasma from the respiratory tract for several months. Treatment of the respiratory infection is frequently unrewarding and there is no treatment for the mastitis. *Mycoplasma sp* arthritis can occur concurrently or as a sequel to pneumonia or mastitis.

This case emphasizes the need for vigilance when introducing new cattle into a herd.

Review of equine cases submitted to the AHC from Jan 1 to Oct 15, 2004

A Britton

A total of 94 cases were received from owners or equine practitioners over the review period. Ten abortion cases were received including two caused by Equine Herpes Virus, one with non-specific placentitis, one with goiter, and another with placental degeneration and mineralization. The remaining abortion cases had no pathognomonic lesions. Two young foals with septicemia were necropsied, one caused by *Actinbacillus suis* and the other of undetermined origin.

Diagnoses made in horses under five years of age included Wobbler syndrome; chronic fibrosing cardiomyopathy; a miniature donkey with hyperlipemia syndrome; a perforating ileal ulcer; severe right atrial bacterial vegetative endocarditis with chronic fibrosing pneumonia; severe pleuropneumonia and pericarditis with right heart failure and hepatic abscessation; one animal had myocarditis, and another arthritis. There were three diagnosed cases of trauma.

Sixteen gastrointestinal cases were examined on post mortem, including one animal with colitis X; two colitis cases linked to impaction; colitis with gastric rupture; colonic perforation; colonic impaction with perforation; cecal displacement with colonic torsion; three intestinal strangulations due to pedunculated lipomas; two cases of small intestinal volvulus; large colon torsion; one case with perforating ileal ulcer; one with a gastric ulcer; and, finally, one colic of undiagnosed cause.

There were five cases of laminitis with ventral rotation of the coffin bone.

Five cases of myelopathy were diagnosed in horses with neurological signs referable to the spinal cord. One severe case of equine protozoal myelitis was diagnosed, with positive CSF (cerebrospinal fluid) titre for *Sarcocystic neurona*.

There were two cases of bacterial dermatitis, one with cellulitis, and one horse was observed with marked peripheral edema.

Tumors diagnosed included cutaneous lymphoma, sarcoid, and vulvar squamous cell carcinoma. One horse was diagnosed with abdominal adenocarcinoma and another with pituitary adenoma.

Continued Page 4

Injured horses included five carpal fractures, four sesamoid fractures, one fracture of the third metacarpal; one fracture of the third metatarsal; one patellar fracture; one tibial fracture; one skull fracture; one scapular fracture; and one fracture of a lumbar vertebra.

Other cases included a superficial flexor tendon rupture, a suspensory desmitis, a horse with navicular bursitis, and one animal with radial nerve paralysis.

Eleven cases of liver disease were observed; two of these exhibited bridging fibrosis, one animal had hepatitis and splenitis, and another demonstrated hepatic lipidosis.

Diseases of the respiratory tract included severe, unexplained pulmonary hemorrhage in one horse; probable fatal exercise-induced pulmonary hemorrhage in a racehorse; pneumonia in another mature animal; and bronchiolitis with myocardial degeneration in another. There were two cases of laryngitis and *Strep equi zooepidemicus* was isolated from one of these.

Diagnosed kidney lesions included chronic pyelonephritis with ipsilateral ureteral obstruction observed in an older mare. Brain mineralization was observed in a horse with a history of colic and 'falling down'. Sudden death in one horse went unexplained, and another had hoof wall separation linked to selenosis (chronic selenium poisoning). There were also two cases of arthritis.

Due to the closure of the Abbotsford transfer station in September, increased numbers of horses were submitted to the Animal Health Centre for necropsy and disposal. These are included in this above report.

Suspected Vitamin D toxicosis in mink:

J Coates

More than a hundred juvenile mink gradually went completely off their feed over a period of approximately ten days. Several animals became lethargic and died, despite the owner replacing the feed source completely with a poultry-based ration. A week prior to their loss of appetite and becoming ill, the owner had placed the mink on a ration comprised completely of fish offal.

Post mortem findings were inconclusive, although the specimens examined had empty stomachs and extensive melena (dark stools stained with altered blood), together with dilated gall bladders. Multi-organ changes or lesions observed on microscopy were consistent with vitamin D poisoning or toxicosis. The most significant change on microscopy was widespread metastatic mineralization of lung, heart, and kidney, with subsequent loss of functional tissue.

Fish offal that had been fed to the mink included liver tissue. Fish liver is known to store large quantities of the fat-soluble vitamins A, D, and E. As in this case, the toxic threshold of vitamin D may be exceeded if fish livers are fed in quantity for even a short period of time. Interestingly, in a much earlier case involving pigs that had been fed fish offal containing livers, vitamin A toxicity had been observed by this writer [(1)].

In this case, fresh-frozen fish offal was fed over a fairly short time period. The likelihood of significant loss of vitamin D activity from oxidation during storage was remote.

To avoid exposure to potentially toxic levels of fat-soluble vitamins found in fish offal, various techniques have been suggested (1). These include separation and removal of the fat soluble layer that contains potentially toxic levels of vitamins A and D in liquefied fish silage prior to feeding; alternatively, the silage may be treated by heating, fermentation, and oxidation. Separation and removal of the livers from fish offal at time of harvest and prior to feeding is another effective approach, although this method is labor intensive.

This case of suspected vitamin D toxicity in mink is an example of animal toxicity resulting from exposure to abnormally high levels of a compound (vitamin D) that is an essential nutrient when ingested in adequate amounts.

1. Coates JW, Holbek NE, Beames RM, Puls RW, O'Brien P. Gastric ulceration and suspected vitamin A toxicosis in grower pigs fed fish silage. *Can Vet Jour 1998; 39: 167-1*

Hemorrhagic Pneumonia (*Pseudomonas aeruginosa)* in Juvenile Mink:

J Coates

Four juvenile mink were submitted for post mortem, following ongoing ranch losses. The most significant and consistent finding was severe hemorrhagic pneumonia. Lung lobes were often semi-firm or consolidated. The heart sac and chest cavity frequently contained an excess of serous to bloody (sanguinous) fluid.

The bacterial organism *Pseudomonas aeruginosa* was cultured from lung tissue in heavy growth. Microscopically, lung tissue was flooded with inflammatory cells (neutrophils), proteinaceous fluid, fibrin, and red blood cells. Tissues from the animals were negative for Aleutian Disease (AD). *Pseudomonas aeruginosa* is a well-recognized cause of hemorrhagic pneumonia in mink.

Severe Vitamin A Deficiency in Chickens:

J Coates

A single chicken form the Peace River District was submitted for necropsy by a veterinarian, following ongoing losses in a small outdoor poultry flock of 20 week-old layers. The clinical history indicated that the birds were being fed a barley ration, with little or no vitamin or protein supplement.

Gross post mortem findings indicated emaciation and wasting of breast muscle, renal (kidney) gout with accumulations of urate crystals and an empty gastrointestinal tract. Prominent small white nodules were observed along the mucosal membrane of the esophagus. The bursa of the bird was slightly enlarged and had a central caseous (cheesy) core.

Microscopic examination indicated severe squamous metaplasia in several tissues: the glandular tissue of the esophagus, ductal transitional epithelium within the kidney, and pseudo-stratified columnar epithelium within the bursa. Squamous metaplasia refers to the transition of normal glandular tissue to an abnormal form. This pathologic process is virtually diagnostic for hypovitaminosis A induced by a primary nutritional deficiency or, in some instances, secondary to ingestion of toxic compounds.

The level of vitamin A in the liver tissue was so low as to be not measurable. The renal gout, emaciation, and loss of appetite observed in this single bird were all secondary to severe nutritional deficiency, particularly hypovitaminosis A. Bone strength was adequate on gross post mortem examination, and growth plates appeared normal.

Barley is not an adequate sole constituent for any animal's diet, and this is especially so in young, growing and metabolically active birds or other animals. The veterinarian was advised of the laboratory findings, and the owner was advised to feed the birds a nutritionally balanced ration. Meanwhile, the birds were given a comprehensive vitamin and electrolyte preparation in their drinking water for about a week, pending arrival of the new ration.

Guidelines for the Submission of Specimens for Bacteriology:

In the Bacteriology laboratory we isolate and identify a wide variety of bacterial and fungal pathogens. Many of these organisms are fragile and will die off quickly if not handled appropriately, while others require special conditions for growth. Cultured bacterial pathogens are susceptible to overgrowth by contaminants. To ensure diagnostic relevance, specimens submitted for bacteriology require careful collection and handling, along with excellent communication with laboratory staff. We have listed the following suggestions to assure the diagnostic value of samples:

i. General guidelines for all specimens:

Submit specimens with a brief history and a tentative diagnosis. Indicate if you wish culture and sensitivities, or culture alone. If you suspect a specific pathogen please state this clearly with your submission. Many organisms require special media and atmospheric conditions or prolonged incubation and may not be recovered under the conditions used for routine culture. Others will only be detected using PCR. If you suspect an organism which may cause human disease, clearly state this, so that people handling your specimens will take appropriate protective measures. If this is a follow-up case, please mention pathogens previously isolated.

Obtain specimens from animals before antibiotic treatment, since bacteriology on specimens from treated animals is often useless and may be misleading.

Take specimens from living or recently dead animals. After death, bacteria from the gastrointestinal tract rapidly invade and contaminate tissues.

Protect specimens from contamination and use aseptic technique for collection of specimens. It is best to take other tissue specimens before opening the gastrointestinal tract. Keep each tissue in a separate, labeled container.

Submit sufficient amounts of specimen, but not too much. Blocks of tissue should be approximately 4 cm^3, while several milliliters of pus, exudate, or feces are sufficient.

Avoid submitting specimens on dry swabs. Many bacteria die quickly due to desiccation, and anaerobic bacteria will not withstand exposure to oxygen. If you must use swabs for collecting specimens, use a swab transport system with no antibiotics (viral transport media is not acceptable). To maintain viability of bacteria and prevent contaminant overgrowth, keep the swab/transport medium cold, but not frozen.

Place <u>each</u> specimen in a sterile leak-proof screw cap jar. Ensure the container is clearly labeled with the name of the owner, the tissue enclosed, the date of collection, and any other identifiers required. 'Whirlpak' bags, plastic gloves or sleeves, are not appropriate for specimen submission.

Do not submit syringes with the needle attached as this is a danger to yourself, the people opening the packages, and other laboratory personnel . For fluids collected by syringe, transfer the fluid to a red topped serum tube. Do not put specimens to be cultured into tubes containing EDTA (purple top) because this chemical is toxic to some organisms.

Transport specimens to the laboratory immediately in an insulated container. Use sufficient ice packs to keep the specimens at 4°C for 48 hours or until the sample reaches the lab. Freezing is not recommended for bacteriology specimens.

ii. Submission of milk samples for bovine mastitis:

Collect samples as soon as possible after mastitis is noticed and prior to administration of antibiotics. Wash dirt off and dry with clean cloth. Wipe the teats and sphincters with 70% ethyl alcohol to allow for drying. The first squirt of milk from each teat should be discarded. For a composite sample a small amount of milk from each quarter should be collected into a labelled sterile snap cap or screw cap tube. Whirlpak bags may leak in transport. Transport to the laboratory on ice as quickly as possible.

Please direct any questions about sample collection/transport or special testing to the bacteriology laboratory (604)556-3125.

i. Sandra Etheridge, Histology section:

Sandra Etheridge was recently appointed as senior technologist in the AHC Histology Section. Sandra received her Bachelor of Science Degree from UBC in 1991. She later worked at the S.P.C.A in Vancouver for a short period before entering B.C.I.T., graduating as a certified Medical Laboratory Technologist in 1994. Ms Etheridge then worked briefly with the Red Cross Society, and also at the R.C.M.P. Forensic Lab. in Vancouver. Other work experience included a brief stint as medical technologist at Richmond Hospital and full-time employment at Vancouver General Hospital. Sandra gained extensive work experience at several hospital histology laboratories prior to her appointment to the Animal Health Centre.

Sandra and her husband find that they have little free time, with their energies divided between the family business in Chilliwack (*Steelhead Electric*) and her new role as AHC senior histology technologist. Both love their home in the Fraser Valley and the close proximity of their families.

Sandra finds her responsibilities at the AHC interesting not only because of her duties in the Histology section, but also because of her ongoing interest with animals.

Sandra's resourcefulness, energy, and leadership has contributed greatly to the Histology Section and to re-establishing immunochemistry (IHC) at the AHC. We sincerely welcome Ms Etheridge to the Animal Health Centre.

ii. Dr Gary Marty, Fish Pathologist

Dr. Gary Marty was recently appointed Fish Pathologist with the Animal Health Branch. Dr. Marty has an extensive background in fish diseases, and for the last several years was a research pathologist at the University of California at Davis.

Gary's professional qualifications include a Bachelor's and Master's degree in Fisheries Biology, the Doctor of Veterinary Medicine degree, and a PhD in Comparative Pathology. Dr. Marty is also certified as a veterinary pathologist by the American Collegeof Veterinary Pathologists. Gary has extensive background teaching veterinary students and a large number of scientific publications to his credit, including significant involvement with the Exxon Valdez oil spill in Alaska.

Dr. Marty will be working closely with Dr. Joanne Constantine, our Fish Health Veterinarian in Courtenay, providing diagnostic support to the Ministry's Fish Health Auditing and Surveillance program as well as to practicing fish veterinarians on diagnostic challenges.

Gary is very interested in outdoor activities including cycling, hiking, and kayaking. We anticipate that he will have at least some opportunity to participate in these activities!

Please join the staff of the Animal Health Branch in welcoming Dr Marty to the Abbotsford Agriculture Centre and the Ministry.

Mr Chastek, horsetail, and fiddleheads:

Chastek's paralysis was named after the Minnesota fox farmer that first noticed the condition clinically, in his foxes. Various fish such as carp contain thiaminase activity. Thiamin deficiency may occur in mink and other fur-bearing animals when large amounts of raw fish are used as feed.*

Horsetail (*E arvense*) is a common thiaminase-containing weed in most areas of the U.S. and Canada. Cases of horse poisonings in North America have been documented. Hay containing 20% or more of horsetail may produce symptoms of thiamine deficiency in horses in 2-5 weeks.*

Bracken fern is a potential human health hazard… "The carcinogenic activity of bracken is highest at the crosier stage. The young fiddleheads (crosiers) are consumed as a delicacy in Japan, China and Korea. It has been suggested that the high incidence of stomach cancer in Japan could be partially due to consumption of bracken. In Japan, bracken is processed by boiling in alkaline solution or pickled in salt…… while processing treatments markedly reduced the carcinogenicity, the processed bracken was still carcinogenic." *

* PR Cheeke: *Natural Toxicants in Feeds, Forages, and Poisonous Plants*, 2nd ed.

The examination of dirt:

"Dirt is evidence of the imperfections of life, a constant reminder of change and decay. It is the dark side of all human activities – human, because it is only in our judgements that things are dirty; there is no such material as absolute dirt."

— Terence McLaughlin, English writer and scientist, from *Dirt: A Social History as Seen Through the Uses and Abuses of Dirt,* 1971. *(*from*The Quotable Scientist* by LA Horvitz, McGraw-Hill; published 2000).