BRITISH COLUMBIA

Animal Health Centre

Ministry of Agriculture, Food and Fisheries

Diagnostic Diary Vol. 14, No. 1. *The Animal Health Centre of British Columbia: a diagnostic laboratory accredited by the American Association of Veterinary Laboratory Diagnosticians.* **January 2004**

CONTENTS

In this issue:

Editor:

J.W. Coates

Mailing Address:

Ministry of Agriculture,
Food and Fisheries
Animal Health Centre
1767 Angus Campbell Road
Abbotsford, BC V3G 2M3

From the Director

R.J. Lewis:

Following the identification of bovine spongiform encephalopathy (BSE or mad cow disease) in Alberta in May, there has been increased interest in the transmissible spongiform encephalopathies (TSE). This may be an opportune time to present a brief overview for you.

An abnormality in a normal protein termed a prion is considered to be the cause of the various TSE. The TSE with which we are concerned are BSE, CWD (chronic wasting disease in deer), and scrapie in sheep. Creutzfeldt-Jakob Disease (CJD) is a spontaneously occurring TSE affecting humans and variant Creutzfeldt-Jakob Disease (vCJD) is the human disease that has been associated with BSE.

Strain typing for the TSE is done by a mouse bioassay. BSE and vCJD have been shown to be the same strain; whereas CWD is different from other TSE. Many differences have been shown between CJD and variant CJD. There are numerous strains of scrapie in sheep (at least two occur in Canada) which are also different from BSE.

Many tests for diagnosing the disease in live animals are currently under development but none of these has yet been validated. To inactivate these abnormal prions requires temperatures greater than 1000^0 C and prions have been shown to survive in ash resulting from 600^0 C incineration. There is evidence to indicate that the higher the dose of prion, the shorter the incubation period. (A very low dose could help to explain the long delay before the first case was recognized in Canada.)

A new European Food Standards Agency will take over classifying countries relating to their risk for BSE and Canada will fall into category 3 (defined as likely to have BSE but not detected or detected at a low level). The USA will probably also be in this category due to trade with Canada. The Canadian Food Inspection Agency (CFIA) is examining options regarding the feed ban and the final choice will be made in consultation with industry, provinces, and trading partners.

The CFIA is also evaluating BSE surveillance options, each of which will require different levels of testing. Any statements that may be made

Continued Page 2

regarding the prevalence of BSE in Canada will be determined by the intensity of the surveillance program. The preferred option at this time is to test all adult cattle over 30 months of age with clinical signs consistent with BSE or other nervous system disease. In addition, BSE testing will be done on animals that are found dead; down and unable to rise; presented for emergency slaughter; or condemned at ante-mortem inspection before slaughter. No final decision has been made on the enhanced surveillance program; we need to know the level of testing that will be required and then can determine the provincial commitment to testing. The CFIA is seeking feedback from the global animal health agency (OIE) regarding acceptable testing levels. One option may be to return to a negative state more rapidly by increasing testing beyond the levels required by the OIE. A decision is expected within the next few months.

Update (Jan 09, 2004): Following the recent identification of BSE in Washington state, the CFIA has announced enhancements to BSE surveillance. The plan is to examine approximately 8,000 animals from the *high risk target group* in 2004 with this number increasing to approximately 30,000 in following years. There may be significant difficulties associated with attempting to procure such samples. Agriculture and Agri-Food Canada is presently evaluating the economic costs of various options regarding the ruminant feed ban. Presently the three options under consideration are a) maintaining the status quo i.e. no ruminant materials destined for ruminant feed b) no bovine specified risk materials in any animal feed and c) the exclusion of all ruminant materials in all animal feed products.

As noted on our letterhead, the Animal Health Centre is accredited as a full service diagnostic laboratory by the American Association of Veterinary Laboratory Diagnosticians (AAVLD). Following many months of intense preparation by all staff members and extensive documentation of our testing procedures, an on site evaluation team from the AAVLD visited the laboratory for three days in early November. The visit was very thorough and the site team also had the opportunity to meet and discuss our services with several veterinarians who frequently use our facilities. We are honoured to be one of only two Canadian accredited laboratories and look forward to the final report from the AAVLD Accreditation Committee; this is expected in mid-February.

On behalf of the entire staff of the Animal Health Branch, allow me to extend our wishes to you for a happy and safe holiday season and our very best wishes for 2004!

❖❖❖❖❖❖❖❖❖❖

Computer System Update:

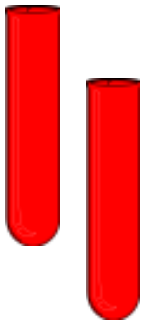
R. Lewis

Our new computer system now allows us to send reports to you via electronic mail. If you would like your laboratory reports to be mailed to you electronically, please inform our front office staff and provide them with the e-mail address to which you would like your reports sent. We hope this will allow us to provide reports to you sooner and provide greater convenience to you for record storage.

❖❖❖❖❖❖❖❖❖❖

Adequate blood needed for mineral and vitamin analysis requests:

R Davis



Please send two tubes of blood for trace mineral analysis. Quite frequently, a full mineral package is requested on a specimen, but only a single tube of blood (or serum) is submitted. We have always tried to provide as many tests as possible from the small specimen, but we need most of the serum for selenium testing (e.g. 2.5 mL). Also with two tubes of blood this will allow us to do further testing, if requested, at a later date and will allow for repeat testing, if any result is questionable.

If we don't have enough sample, we will try to contact the submitter for instructions as to which tests are most important. We will attempt to assay selenium on as little as 1 mL, but our method has not been validated on smaller quantities. This could be a problem when low values for selenium are expected, such as in horses.

"Royal blue top" trace mineral tubes are the preferred collection tube because the special stopper formulation has low levels of trace elements. "Red top" tubes are also frequently used and are probably suitable; however, there may be some leaching of trace elements into the serum. "Clot activator" and "serum separator" (SST) should be avoided.

Serum for vitamin testing should be separated as soon as possible after collection and frozen if not shipped immediately. Please do not let samples sit on "SST".

❖❖❖❖❖❖❖❖❖❖

Equine Roundup

A Britton

From January to August 2003, 63 equine cases were completed at the AHC.

There were 12 abortions/perinatal deaths including seven of unknown cause (idiopathic), one with placentitis, one of bacterial origin, one with liver disease (hepatopathy) and one with an extremely large, disfiguring naso-sinal cyst.

Six cases of gastrointestinal tract disease were comprised of three cases of colitis, one gastric rupture, one gastrosplenic entrapment with intestinal rupture, and one post-surgical colonic torsion. Two cases of bacterial pneumonia and two cases of diaphragmatic hernia were observed in mature horses.

Nine foals were necropsied; six were septicemic or had bacterial pneumonia; one had undiagnosed blindness; one had nutritional myopathy, and one had a cardiac anomaly with ruptured bladder.

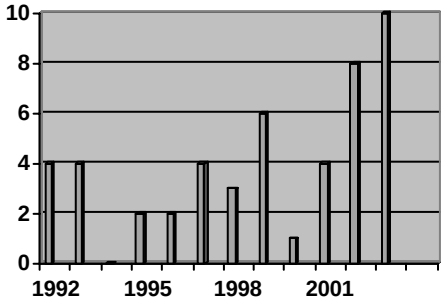
Four central nervous system (CNS) cases yielded varied diagnoses: one wobbler syndrome, one equine degenerative myeloencephalomalacia, one Dandy-Walker syndrome, and one undiagnosed CNS case. Musculo-skeletal lesions ranged from two foundered horses, two racehorses with humeral fractures, a geriatric horse with osteoarthritis in the hip, and a 19 year-old mare with lumbar spondylosis.

Skin cases included equine sarcoid, equine canker, non-specific dermatitis, and normal skin. One case of serous atrophy and suspected starvation was seen. Other cases diagnosed were a horse with thymoma, one with multicentric lymphosarcoma, and another with hemoperitoneum and a ruptured spleen. A horse with severe kidney disease (nephropathy) was also diagnosed.

Continued Page 4

Liver disease diagnosed in horses at the AHC:

From January to August 2003, the AHC diagnosed liver disease in 10 mature horses or donkeys. This reflected an increase in the frequency of equine liver disease compared to other years. A review of AHC records from 1992 to 2002 revealed a total of 45 cases of liver disease diagnosed in horses or donkeys, with an apparent increase in cases in 2002 and 2003.



The historic diagnoses of liver disease were categorized as follows:

Diagnosis	No. of Cases 1992 to 2002/ **2003**	% of Cases 1992 to 2002/**2003**
Fatty Liver	5/**0**	11/**0**
Hepatopathy	15/**7**	34/**70**
Hepatitis	25/**3**	55/**30**

In 2003, 7/10 cases were diagnosed as hepatopathy (liver disease) and 3/10 were diagnosed as hepatitis (inflammation of the liver). Of the seven hepatopathy cases, three originated in the southern Okanagan and four were from the lower mainland/Fraser Valley.

Liver disease is a frequently reported problem in the horse. The liver performs a large number of metabolic functions and has a large regenerative capacity. Hepatic dysfunction does not become clinically apparent until approximately 60 to 80 % of liver capacity is lost. Affected horses will most commonly present with weight loss, icterus (jaundice) and behavioural changes. Icterus is more commonly associated with acute hepatic failure and may be absent in cases of chronic liver disease prior to exhaustion of hepatic reserve capacity. Horses with liver disease may also develop gastric impaction and rupture, photosensitization, laryngeal paralysis and bleeding disorders.

Bacterial hepatitis in the mature horse usually occurs secondary to ascending infection via the biliary tree. Although primary ascending infection can occur, it is believed that predisposing causes which lead to bile stasis, such as choleliths (bile stones), ileus or enteritis (inflammation of the intestine), are most common. In the acute phase, the horse will develop cholangitis (inflammation of the bile ducts) or cholangiohepatitis (inflammation of the bile ducts and the liver tissue immediately surrounding the bile ducts). If the infection resolves, the disease will progress no further; however, if the infection is not brought under control, the inflammation can progress to chronic liver disease characterized by self-perpetuating inflammation and fibrosis.

Hepatotoxicity in the horse in British Columbia is most commonly attributed to a handful of toxins. Alsike clover is a poorly documented toxin in the horse. Exposure to the plant appears to be associated with hepatotoxicity. It is unclear whether the plant itself is toxic or whether an exogenous toxin is responsible for the liver damage. Alsike clover is cultivated in some areas of northern BC and Alberta and most cases described in the literature have

Continued Page 5

been associated with feeding of alsike clover hay. The liver lesions typically surround bile ducts and are characterized by marked fibrosis and bile duct hyperplasia with progressive loss of liver cells. Unlike cholangiohepatitis, there is minimal inflammation.

Tansy Ragwort is another hepatotoxin reported to affect horses. The plant is unpalatable and as such is unlikely to cause acute equine hepatotoxicity; however, the toxin is cumulative and toxicity can occur due to long term low level exposure. The toxic principle is a pyrrolizidine alkaloid in the plant which damages liver cells, preventing cell division, and causes a characteristic appearance microscopically. The changes in the liver are similar to Alsike Clover poisoning in character and distribution within the liver. Tansy Ragwort toxicity will also exhibit giant cells due to interference with cell division by the toxin.

Aflatoxin is another possible hepatotoxin, although horses are not considered a highly susceptible species. The toxin is produced by the fungus *Aspergillus flavus* and found in spoiled grains. Depending on the amounts consumed, the disease can present either as acute or chronic disease. Acute disease is uncommon in horses and distinct microscopically from the other hepatotoxins. Chronic disease is difficult to distinguish from pyrrolizidine alkaloids. Like pyrrolizidine alkaloid, aflatoxin interferes with cell division and leads to the presence of giant cells.

❖❖❖❖❖❖❖❖❖❖

Horse triplets

A Britton

i. A 10 month-old horse was diagnosed with severe verminous typhlocolitis (inflammation of the cecum and colon caused by worms). The entire mucosal surface of the cecum and large colon was swollen and ulcerated. Thousands of small red worms typical of *Strongylus spp* were present in the lumen. Microscopically, there were massive numbers of strongyle larvae in the affected mucosa and submucosa. Deworming programs should target strongyle larvae and adults. Drug rotation should be pursued to minimize the potential for development of parasite resistance. Deworming medications should always be coupled with good manure management to reduce further pasture contamination from nematode (worm)fr eggs.

ii. A two month-old foal was diagnosed with acute bronchopneumonia and sepsis. *Streptococcus equi subs. zooepidemicus* was isolated from the lung, brain and spleen. This bacterium is a common opportunistic pathogen of horses and may cause pneumonia and sepsis in foals. The organism usually requires a predisposing factor such as viral infection or stress to cause disease.

iii. A 15 year-old horse presented with a history of neurological signs. On gross necropsy, a firm mass was observed within the skull that involved the left cribriform plate of the ethmoid and basisphenoid bones, the optic chiasm, left optic canal and optic nerve, left nasal passage, left frontal sinus, and the left olfactory lobe of the brain. Microscopically, the naso-sinal tumour was compatible with anaplastic carcinoma. The tumor aggressively infiltrated local structures and readily spread through bone.

Carcinomas of the naso-sinal passages in the horse may present as squamous cell carcinoma, adenocarcinoma, or anaplastic undifferentiated carcinoma. Prognosis is poor in most cases due to locally invasive tumour behaviour.

❖❖❖❖❖❖❖❖❖❖

Histomoniasis (Blackhead) in pheasants:

J Coates

Histomoniasis (blackhead), a serious parasitic disease caused by the parasite *Histomonas meleagridis*, was diagnosed in a single six month-old peacock. The flock consisted of ten birds and two others had died previously. According to the owner, the birds became listless, lost weight, and finally died. There was severe thickening of the cecal mucosa and muscular wall, with the presence of cheesy cores within the cecal lumen. Liver tissue showed mild, random, pale foci. Microscopically, severe inflammation and ulceration of the cecal mucosa and muscular layers, with numerous protozoal histomonads were seen within the tissue – lesions that are diagnostic of avian histomoniasis. The parasite was also found within random areas of severe liver inflammation.

The owner was advised immediately of the post mortem results. Since healthy chickens often carry infected cecal worms (*Heterakis gallinarum*), the practice of ranging chickens with turkeys (or with pheasants, peacocks, or other galliformes) should be avoided. Grouse and quail may also bring the infection to turkey yards. Chickens may occasionally suffer a milder form of the disease. The protozoan is spread through soil, with eggs of the cecal worm (*Heterakis gallinarum*) and three different species of earthworms acting as hosts that can transport the parasite over distances. Since *H. gallinarum* eggs can survive in soil for many months or even years, turkeys should not be placed on ground contaminated by chickens. Direct transmission via exposure to contaminated fecal matter may also occur. Rotation of outdoor ranges for turkeys and other susceptible species (pheasants, peacocks, for example), will help to reduce the density of soil contamination with infected feces.

Effective treatment of this condition in sick birds has been severely compromised following the withdrawal of one of the most effective medications from the market. However, some compounds such as nitarsone are still available for prevention and control.

❖❖❖❖❖❖❖❖❖❖

Canine Cryptococcosis – *C. neoformans var gatti:*

A. Britton

A mature male dog with a history of chronic rhinitis refractory to treatment and acute onset of CNS signs characterized by seizures was received for necropsy. The dog was suffering from chronic fungal rhinitis which had disseminated to other organs, including the lungs, but had most significantly infiltrated the brain. The mycotic agent was identified as *Cryptococcus neoformans var. gattii.*

Cryptococcus neoformans var. gattii is a common cause of crytococcosis in humans and animals, primarily in tropical and subtropical climates. The organism is reported to have an affinity for Eucalyptus trees. In the late 1990's, *C. neoformans var. gattii* was identified in several terrestrial and marine animals in the vicinity of southeastern Vancouver Island. The organism occurred in the bark of Eucalyptus trees on the eastern coast of Vancouver Island and a new endemic area for the organism was identified.

Cryptococcus neoformans var. gattii is found in the soil and in tree bark . It is transmitted by aerosolization of infected soil, feces and decaying bark. Direct transmission from infected individuals is considered less likely as the organism is most virulent in its acapsular form. Acapsular forms are predominantly found in free-living organisms in soil and bark. Once the organism gains entrance via aerosolization in an individual, it forms a

Continued Page 7

thick capsule which protects it from the host's immune response. This capsule also makes the organism less likely to infect a new host. However, it should be noted that this organism is a pathogen for both animals and man. While individuals who are immuno-compromised are most at risk for development of disease, it is prudent for all individuals to minimize the risks from exposure to affected soils or decaying bark.

At this time it is unclear where the dog contracted the infection. He had been to Vancouver Island on a number of occasions and may have contracted the infection there. Studies are presently being conducted to determine the source of the infection.

❖❖❖❖❖❖❖❖❖❖

Cryptococcus neoformans var gattii in a mature llama:

*J Coates, T Maarhuis**

Cryptococcal meningoencephalitis (inflammation of the brain) was diagnosed in a mature llama from the eastern Fraser Valley. Over several days, the animal gradually developed signs referable to the central nervous system including inappetence, lethargy, blindness, staggering, 'goose-stepping', and finally recumbency. The animal was euthanized in a terminal state. Post mortem revealed nothing specific, although lungs were diffusely congested and slightly meaty on cut section. Microscopically, numerous organisms consistent with *Cryptococcus* sp were identified in lung tissue and brain, associated with interstitial pneumonia and encephalitis, respectively. The organism was grown on Sabouraud's agar, and finally identified as *Cryptoccus neoformans var gattii* via serological testing. Additional PCR analysis of the agent for molecular typing is to follow.

For known details on the activities of this organism within the environment, refer to the preceding discussion by Dr. Ann Britton (*Canine crytpococcosis*). This is the first known case of *C. gattii* infection in a domestic animal within the eastern Fraser Valley of British Columbia.

*With thanks to Dr Karen Bartlett of the School of Occupational and Environmental Hygiene, UBC, for the typing of the Cryptococcus sp culture isolated at our laboratory - Ed. * Valley Veterinary Services, Chilliwack.*

❖❖❖❖❖❖❖❖❖❖

IBR abortion in Holstein cattle:

J Coates

A single IBR (infectious bovine rhinotracheitis) abortion was diagnosed in a five-month Holstein fetus from a 90-cow Fraser Valley dairy herd. Microscopically, there were multifocal areas of tissue degeneration (necrosis) within the liver and spleen as well as prominent inflammation of the placenta (placentitis) and lung (pneumonitis). Viral inclusion bodies were not readily detected in the tissues examined microscopically. Viral studies indicated IBR-like cytopathic effect on tissue culture cells. Subsequent fluorescent antibody tests on infected cell cultures were positive for IBR virus. In addition, aborted fetal tissues were positive for IBR via PCR -DNA analysis.

The bovine herpesvirus type-1 (BHV-1 subtypes) or the infectious bovine rhinotracheitis (IBR) virus is an alphaherpesvirus and causes respiratory disease, abortion, conjunctivitis, and other clinical forms of the disease complex (1). Genetic analysis has found at least

Continued Page 8

three distinct subtypes of this virus (OM Radostits, CC Gay, et al: *Veterinary Medicine*, 9th edition; WB Saunders publishers, 2000).

The herd had not been vaccinated for IBR virus. The aborted three year-old cow was in her second pregnancy. In recent discussions later with the veterinary practitioner, no additional abortions have been reported in the herd.

❖❖❖❖❖❖❖❖❖

Mycoplasmal 'pneumoarthritis' in weaned beef calves:

J Coates

In December, a veterinary practitioner submitted fresh specimens from a group of eight month-old weaned, auction-purchased beef crossbred calves that were showing signs of inflammed joints and pneumonia. Treatment with oxytetracycline had been relatively ineffective in controlling the joint problems in the animals.

Laboratory study at the AHC indicated that both lung tissue and submitted joint fluid/clot aspirates were positive for *Mycoplasma bovis* (*M bovis*), via PCR-DNA analysis. Standard bacterial culture of the lung tissue failed to yield any other bacterial pathogens, probably because of heavy antibiotic usage. Viral tissue culture on the very fresh lung and joint specimens was negative. Microscopically, examination of lung tissue revealed a fibrinosuppurative bronchopneumonia with random necrotic foci.

As in most cases of mycoplasma-induced infectious arthritis, response to medication in these calves was unsatisfactory. Radostits at al. indicate that while antibiotics may be effective on culture plates within a laboratory setting (in vitro), they are generally ineffective in clinical field cases (*Veterinary Medicine,* 9th ed., pub. 2000: WB Saunders Co, London & New York). In this case, microscopic examination of lung tissue also showed lesions typical of mycoplasmal pneumonia.

The possible link of M bovis pneumoarthritis with BVD virus is an interesting topic. On the basis of hundreds of submissions, EG Clark of Prairie Diagnostic Services has published information indicating that calves demonstrating ongoing problems of M bovis pneumonia and infectious arthritis may actually be undergoing primary infection to circulating BVD virus.

M bovis has also been linked to bovine conjunctivitis, or inflammation of the eyelids. *M bovis* is a less common cause of contagious mastitis (Radostitis et al). The organism may cause outbreaks which do not respond to therapy and which are difficult to control.

❖❖❖❖❖❖❖❖❖❖

Respiratory disease in nursling piglets:

J Coates

Persistent and debilitating respiratory disease was observed clinically in a large group of nursing pigs, beginning at three weeks of age. Piglets would sniffle, become unthrifty, lose weight, and some would die. Antibiotic therapy was of limited value in controlling the progression of the outbreak. Mortality within the nursery was 7%.

Continued Page 9

Post mortem findings from five affected 28 day-old piglets indicated ongoing pneumonia involving up to 40 % of lung tissue, with minor lung change noted in the least affected animals. One animal also had chronic pleuritis and adhesions to the heart sac. Grossly, lung tissue resembled mycoplasmal pneumonia despite the young age. Microscopically, there was a distinct suppurative rhinitis (inflammation of the nasal turbinate bones and sinuses) as well as a bronchointerstitial type of pneumonia, suggesting viral and bacterial components. Lung tissue was positive for PRRS virus via PCR analysis, and areas of lung injury were also confirmed positive for PRRS virus, via immunohistochemistry. Antibiotic usage likely influenced bacterial culture – no significant organisms were cultured other than *E coli*. Microscopically, no turbinate mucus gland inclusions consistent with Inclusion Body Rhinitis (herpes virus) were detected and electron microscopy of turbinate tissue for porcine herpesvirus was also negative.

PRRS virus continues to threaten nursling piglets when consistent vaccination is not practiced. Antibiotic therapy will be of limited value and only for the control of secondary, opportunistic bacterial agents. Differentials in this case included Inclusion Body Rhinitis (herpes virus), Circovirus 2, *Mycoplasma hyponeumoniae, Bordetella bronchiseptica and Pasteurella multocida,* as well as swine influenza virus. PCR analysis for *M* hyopneumoniae and Circovirus 2 were both negative. PCR analysis and vial egg inoculations were negative for swine influenza. If you are interested in a vaccination control programme for porcine PRRS virus, your veterinary practitioner should be consulted.

❖❖❖❖❖❖❖❖❖❖

Resistance of cattle to scrapie by the oral route:

"Early epidemiological information indicated that bovine spongiform encephalopathy (BSE) originated from scrapie in sheep. The question arose if scrapie in North America would induce a BSE-like disease in cattle. Six years ago, we reported that brain tissue from sheep with scrapie caused a neurologic disease when injected directly into the brains of cattle, but the disease induced was different from BSE as it occurs in the United Kingdom and Europe. Here, we report that cattle fed raw brain or meat and bone meal and tallow prepared from sheep with scrapie remained normal for 8 years after exposure. This work indicates that cattle are highly resistant to North American scrapie by the oral route." Cutlip RC et al. *Can Jour Vet Res* 2001;65:131-132.

❖❖❖❖❖❖❖❖❖❖

Xenotransplantation and the potential risk of xenogenic transmission of porcine viruses:

"The clinical success of allotransplantation and the shortage of donor organs have led to a proposal for the use of animal organs as alternative therapeutic materials for humans. In that regard, swine are preferable to non-human primates as a source of donor organs. A major concern, however, is the potential for transmission of viruses from animals to humans via organ, tissue, or cellular transplantation or via *ex vivo* exposure of humans to porcine biologic materials. Xenotransplantation allows viruses to bypass the normal immunological defense mechanisms of the recipient. Furthermore, the use of

Continued Page 10

immunosuppressive drugs following transplantation may facilitate the xenogenic transmission of zoonotic agents. Of porcine viruses, swine hepatitis E virus does not cause any clinical symptoms in the natural host but is likely a zoonotic agent that can infect humans and cause hepatitis. Porcine circovirus type 1 is prevalent in swine populations with no known association with clinical disease, while circovirus 2 causes post-weaning multi-systemic wasting syndrome. Porcine endogenous retrovirus is integrated into the host chromosomes while porcine cytomegalovirus undergoes latent infection. Two additional porcine herpesviruses have recently been identified in swine and have been named porcine lymphotropic herpesviruses. These herpesviruses can potentially become reactivated in human recipients after xenotransplantation. All in all, there are a number of viruses in swine that are of primary concern to screen and eliminate from xenotransplantation protocols….."

— portion of Abstract with similar title, by Yoo D, and Giulivi A; *Can Jour Vet Res* 2000;64:193-203.

❖❖❖❖❖❖❖❖❖❖

A field trial of *Moraxella bovis* bacterin:

"The primary purpose of these experiments was to evaluate an autogenous *Moraxella bovis* bacterin administered through two separate routes of inoculation. An autogenous vaccine was manufactured by using *M. bovis* recovered from the herd. The bacterin was administered by subcutaneous injection or subconjunctival injection. In each experiment, unvaccinated animals served as controls… There was no statistical difference in development of infectious keratoconjunctivitis between the vaccinated and unvaccinated calves…."

— Davidson HJ and Stokka GL; A filed trial of autogenous *Moraxella bovis* bacterin administered through either subcutaneous or subconjunctival injection on the development of keratoconjunctivitis in a beef herd. *Can Vet Jour* 2003;44:577-580.

❖❖❖❖❖❖❖❖❖❖

Health challenges faced by honeybees:

*P van Westerndorp**

The western honeybee *Apis mellifera*, was introduced to the Americas over 300 years ago. Even though many native insect pollinators existed in North America, virtually all of these were solitary with the exception of bumble bees. As agriculture expanded, so did the dependence on honeybees since the native bees could not meet crop pollination requirements.

Honeybees have assumed a pivotal role in the production of virtually all fruit-bearing crops. While the early settlers could depend on the abundance of native bees, modern agriculture with its mono-cultural practices and use of high-yielding varieties in high-density plantings require huge numbers of pollinating insects during the time of bloom. For example, one hectare of mature high bush blueberry generates some 9 million flowers during its three week blooming season. The value of Canada's annual crop production

Continued Page 11

dependent on honeybee pollination is near $1B. In the United States with its warmer climates, this value has been estimated at over US$14B per year while the value of the US honey crop is only worth several hundred million dollars. For good reason, the honeybee has often been described as the "sparkplug of modern agriculture".

Despite its pivotal role in agriculture, everything is not well with the honeybee. Since the late 1980s, the North American honeybee population has been plagued by the parasitic mite *Varroa destructor*. This ecto-parasitic mite originated from Kalimantan Island (Borneo) where it was a natural parasite of the eastern honeybee *Apis cerana*. In the early 1900s, Varroa was introduced to the Asian mainland and since then, it has spread to all major beekeeping areas of the world. The obligate parasite completes its brood cycle by parasitizing bee brood while adult female mites feed on the hemolymph of adult honeybees. Without intervention, the honeybee colony is doomed.

In the early 1990s, Canadian beekeepers gained access to the pyrethroid, fluvalinate, marketed under the trade name Apistan. This product proved remarkably effective without the dangers of residues or ill effects to the bees. Recently, however, mite resistance has been reported in different parts of Canada, causing beekeepers to resort to less desirable products such as coumaphos. Coumaphos is an organo-phosphate, sold under the trade name CheckMite+ and only temporarily available under PMRA's emergency registration permit. The use of this product typifies the difficulty beekeepers have experienced in controlling Varroa. A great deal of effort is underway in finding alternative control methodologies and products that could replace or at least reduce the application of chemical control products. The development of an integrated pest management (IPM) program may provide the long term protection so badly needed for this economically important insect.

* *Provincial Apiculturalist, British Coloumbia*

❖❖❖❖❖❖❖❖❖❖

Lightning injury in an outdoor swine herd:

"Abstract. Three pigs, weighing 63 kg-70kg each, from a group of 8 pigs in an outdoor pen that was struck by lightning were necropsied. All 3 pigs presented with hind limb paralysis. The only lesions identified were multiple fractures of the last (seventh) lumbar vertebral body and first sacral segment, with dorsal displacement of the sacrum and transaction of the distal spinal cord and spinal nerves. Hemorrhages extended from the fracture sites into muscles immediately surrounding the lumbosacral junction and retroperitoneally into the pelvic cavity…… Because vertebral fractures may be the only lesions and may be grossly subtle in heavily muscled pigs, careful pelvic and vertebral dissection is recommended in cases of suspected lightning strike and electrocution."

— Van ALstine WG, Widmer WR. *Lightning injury in a swine herd.* J Vet Diag Invest 2003; 15: 289-291.

❖❖❖❖❖❖❖❖❖❖

Looking back – piglet castration:

"This is generally understood by every stockraiser, yet there are some points many do not know. For instance, you should use in this operation an antiseptic solution such as Carbolic Acid or Bichloride of Mercury. Wash thoroughly with antiseptic your hands and knife, also the seat of operation and make your incision as low as possible to permit the pus to drain out nicely. If this is not practiced, the pus will become absorbed into the blood, producing blood poison, which may produce death, or at the best will cause the hog to become stunted, whereas, if the operation is performed properly, the hog will thrive, regardless of the shock from the operation. I may add that it is much better to castrate pigs or hogs when their stomach and intestines are empty, and it is always good practice to feed laxative and easily digested foods sparingly after this operation."

— Dr Chas J Korinek; *The Veterinarian*, 4th edition, published 1915. With the compliments of the Merchants Bank of Canada, Rural Service Department, Regina, Saskatchewan. Published by the Gerlach-Barklow Co, Joliet, Illinois, USA, and Toronto, Canada. Dr Chas Koronek: graduate of the Ontario Veterinary College of the University of Toronto, Ex-State Veterinarian of Oregon, Ex-President Oregon State University Medical Board, Hon Member Ontario Veterinary Medical Association.

❖❖❖❖❖❖❖❖❖❖

Focus on Staff: Dr Ann Britton

Dr. Ann Britton is a 1981 graduate of the Ontario Veterinary College (OVC), University of Guelph. Upon graduation, Ann practiced in southwestern Ontario in a mixed practice until her return to Guleph in 1982 to be with her husband, Dr Brad Hicks (OVC '82) who was working as a research associate in the OVC pathology department. Dr. Britton completed a Master's degree in the OVC pathology department where she studied the in vitro effects of *Ureaplasma diversum* on bovine embryos. She demonstrated that the organism readily attaches to the bovine zona pellucida and cannot be removed by routine pre-transfer washing. In 1987, after Brad accepted the position of Provincial Fish Health Veterinarian in the BCMAFF, he and Ann moved to BC, settling in Langley. Ann then completed her Ph.D. studies at UBC in reproductive pathology, where she examined the adverse effects of superovulatory regimes on egg and embryo quality.

While conducting her PhD studies at UBC, Ann utilized the AHC's electron microscope in Abbotsford. In addition, she also worked part-time as a diagnostic anatomic pathologist. Later in her Ph.D programme, Dr. Britton accepted a full-time position as diagnostic anatomic pathologist for *The Central Laboratory for Veterinarians* in 1990. In 1993, she resigned to spend more time with her children. In 1999, Dr. Britton returned to work on contract at the Animal Health Centre as a diagnostic pathologist, and accepted a permanent part-time position the following year. Among her many pathological

Continued Page 13

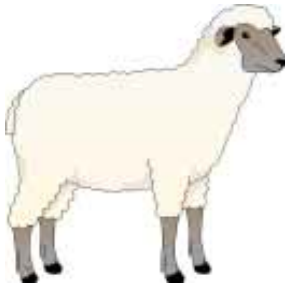
endeavours, Dr Britton retains a specific interest in equine pathology – happily, these have found ample case material for study at the AHC. Readers may benefit from Dr Britton's equine comments on a regular basis in this newsletter, where she writes her own column entitled 'Equine Roundup'.

Aside from her professional interests and duties, Ann is a recreational dressage rider and an avid skier. She enjoys spending time with her family and participating in pony club events and a free style ski club with her children.

With her past experience in large animal practice, her research in bovine reproductive pathology, and previous diagnostic laboratory work, as well as her abiding interest in all things equine - especially of a pathological nature - Ann has many talents and much expertise to offer clients of the AHC. We sincerely welcome Dr Britton to the Animal Health Centre.

❖❖❖❖❖❖❖❖❖❖

Fools rush in where sheep wouldn't:



"Nobody rushes sheep – to perfection or anywhere else. Perhaps if sheep were part of my life, they would impose an order on it and bring out in me the calm patience and good humor so evident in this book. Perhaps you, dear readers who raise sheep or are thinking about it (or raise sheep and *are* thinking about it), can take some pleasure in knowing someone envies you and your enterprise. It's a pleasure to envy you and imagine what sheep raising is like, and if I'm wrong – well, no sheep will ever suffer as a result."

— Garrison Keillor, in the Foreword of *The Sheep Book* by Ron Parker, author; published 1984 by Ballantine Books, New York.

The last word:

" Meliora sequi: This book aspires: we believe that the jack-of-all-trades in our profession, the general surgeon, human or veterinary, will find it useful; so will that drawer of water, the urologist, and that hewer of wood, the orthopedist; so indeed will all workers in experimental biology – all, that is, except those incredible soothsayers of today, the Freudian psychoanalysts. These gentry may not read the stars, but they interpret dreams. True, they do not concentrate upon the entrails of chickens, but they have more than a passing interest in the last inch of the human digestive tube. If you wish to know what to do until the psychiatrist arrives, you won't find it in this book."

— from *Experimental Surgery*, 1964, 5th edition, by J Markowitz, J Archibald, and HG Downie; published by Williams and Wilkins, Baltimore.