Animal Health Centre NEWSLETTER

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Ministry of Agriculture and Food

Diagnostic Diary Vol. 9, No. 3. *The Animal Health Centre of British Columbia: a diagnostic laboratory accredited by the American Association of Veterinary Laboratory Diagnosticians.* **December, 1999**

CONTENTS

Editor:

J.W. Coates

Contributors to this edition of Diagnostic Diary are:

R. Lewis, S. Raverty, J. Coates, D. McIntosh

From The Chief Veterinarian

R.J. Lewis

Many readers are aware of the controversies regarding salmon farming in British Columbia. The industry has tremendous potential to become a major contributor to our agricultural economy but critics have identified environmental issues and disease concerns as reasons for prohibiting such expansion. The government has reviewed the recommendations of the Salmon Aquaculture Review (SAR) and allowed for limited development of salmon aquaculture over the next two years while putting steps in place to address potential disease and environmental issues. The Animal Health Centre has developed a number of new sophisticated procedures for the identification of a wide variety of disease - causing agents in fish. Similarly, our fish health veterinarian in Courtenay is working with industry, industry veterinarians, and the Ministry of Fisheries to develop an active disease surveillance program. The objective is to provide a monitoring system that will provide full confidence to the public that a viable aquaculture industry can develop without endangering wild fish stocks.

It has been often said that change is the only constant and recent restructuring events in the agriculture division of the Ministry of Agriculture attest to that statement. The former Animal Industry Branch has been divided to allow for the formation of a new Animal Health Branch. The Animal Health Branch combines the Animal Health Centre and the Health Management and Regulatory section. The formation of the new Animal Health Branch ensures a strong profile for animal health activities and demonstrates the strong commitment by the Ministry to the essential services we provide to our clients.

On behalf of the Animal Health Branch, we wish all our clients and associates Best Wishes for a very Merry Christmas. May you and your families enjoy the best of health and prosperity for 2000!

RJL

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

Equine serum tests for selenium and Vitamin E offered at the AHC:

Please keep in mind that our toxicology section tests equine blood sera (along with other species) for the trace element selenium, and for vitamin E (serum vitamin A l is also available). Cost per test is 2.00 for selenium, and, if requested, 5.00 for vitamins A/E. There is a 15.00 accession fee, plus GST, added to this cost. The accession fee is a one-time charge, and does not affect multiple submissions from the same owner.

Short cuts from the PM Room

J.Coates

Actinobaccillus suis was cultured from multiple tissues of a 39 kg feeder pig, where there had been several acute losses. On necropsy, most significant lesions noted were in the trachea and lung. The trachea was filled with pink froth extending into the bronchi. Lung tissue was edematous, failed to collapse upon opening the chest, and was diffusely congested to hemorrhagic on cut section. In some cases of *A suis* pneumonia, severe pleuritis may also be seen. Microscopy revealed a severe, suppurative, interstitial pneumonia, with many dense colonies of small bacilli noted within the alveolar septal capillaries.

A. suis is an established pathogen in swine that may cause septicemia, pneumonia, *n*ephritis, or arthritis (Gyles & Thoen: *Pathogenesis of Bacterial Infections in Animals,* 2nd ed, 1993). This small, gram-negative coccobacillus may produce post mortem lesions in swine resembling those of *Streptococcus suis, Hemophilus parasuis*, or *Actinobacillus pleuropneumoniae*, which are differentials to consider when making a tentative diagnosis. In swine, *A. suis* infection may also be confused clinically in some instances with acute erysipelas. *A. suis* can infect pigs of all ages, but infection is most common in young animals. Infection occurs via the aerosol route or by close contact. In conventional swine, *A. suis* (an opportunistic pathogen) is a commensal organism in the tonsils and upper respiratory tract. High-health status herds are more likely to experience problems with this organism.

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A few **wild raccoons** from the Vancouver and Sunshine Coast areas have been submitted to the AHC recently for necropsy, following observation of illness or unusual clinical signs. One of these animals, which was showing signs of disorientation prior to death, had a nonsuppurative encephalitis microscopically, from which **canine distemper virus** was isolated. Another raccoon died after a brief episode of diarrhea; microscopically, lesions within the intestinal tract were consistent with **parvovirus** infection; raccoon parvovirus was identified by electron microscopy.

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Tissues from a laboratory **nude (athymic) mouse** was submitted for necropsy at the AHC, following ongoing losses in a mouse colony. The most significant lesion was in the liver, where there was multifocally extensive necrosis, with formation of numerous large syncytial cells. The liver changes were characteristic of **coronaviral Mouse Hepatitis Virus.** The animal also had an encephalitis; less severe tissue inflammatory changes were noted in a few other organs.

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Acute cardiomyopathy was diagnosed in **juvenile mink** from a mink ranch that was experiencing increased losses. More than 125 animals from a group of 3500 juveniles died over a month. Hearts from affected mink revealed random, multifocal areas of acute myocardial necrosis, and associated local hemmorhage. The underlying mechanism of the heart changes suggested a primary vascular lesion, or microangiopathy.

Vitamin E levels measured in 3 livers from these animals were marginal, at 8.8 to 11.8 ppm; normal or adequate levels are reported at 25-500 ppm (Puls, *Vitamin Levels in Animal Health*, 1994). Liver selenium levels were adequate. Many affected animals showing early clinical signs were spared by injecting them intramuscularly with a vitamin E- selenium preparation.

The owner suspected rancid fish feed, which comprised up to 25% of the ration, as the cause of the dietary vitamin E deficiency. Increasing ranciditiy of the fish meal was thought to be oxidizing and destroying the vitamin E supplement premixed into the ration. Losses occurred during the warmer months of July and August. In addition to injecting ill mink with vitamin E and selenium, vitamin E levels in the diet were markedly increased. Rancid feed was replaced from the diet as quickly as possible. Losses continued for about month, but slowly tapered off as dietary vitamin E levels were increased.

Usually, vitamin E deficiency in mink is described as causing a syndrome of "yellow fat disease", or so-called steatitis (inflammation of fatty tissue). This condition was not observed in these juvenile mink. No skeletal muscle lesions were detected; mature mink were not affected.

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So-called **colitis X syndrome, or colonic dysbacteriosis**, was diagnosed in a young standardbred horse. The animal had been recently castrated, and had suffered post-surgical partial intestinal evisceration and trauma following removal of a retained testicle. It was then transported to a veterinary hospital for extensive medical therapy; then transported again, 3 weeks later, to a racing stable where it was reintroduced immediately to training.

The horse developed acute diarrhea shortly after arrival at the track, and soon succumbed despite treatment. Necropsy revealed severe necrotizing and suppurative inflammation of the cecum and ventral colon.

Bacterial cultures of the colon and associated lymph nodes yielded primarily E. coli; cultures were *Salmonella sp.* negative. Chemotherapeutic agents given orally by the veterinarian following onset of diarrhea may have influenced culture efforts, including culture of *C. perfringens*. Nevertheless, toxin from ***C. difficile*** was readily detected in loops of watery cecal/colic samples via an ELISA test.

* * * * *

Adenoviral inclusion body hepatitis has been recently diagnosed in flocks of young broilers, averaging 2 – 3 weeks of age, where there were complaints of increased mortality. Microscopically, fixed liver tissue submitted for examination revealed diffuse inflammation and widespread hepatic necrosis, with disruption of normal liver

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architecture. Large eosinophilic intranuclear inclusion bodies were readily seen in liver cells undergoing degeneration.

Adenoviral inclusion body hepatitis in broilers may occur as a result of immunosuppression following earlier exposure of the chicks to Infectious Bursal Disease (IBD) virus. Recently however, it appears that there are strains of adenovirus that are acting as primary pathogens, without the contributing influence of IBD immunosuppression.

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Ionophore toxicity was diagnosed in a large group of 23 week-old turkeys; 1600 died in only a few days. Clinically, the birds were unable to stand, and collapsed on the barn floor. Necropsy examination was unremarkable, other than variable edema and congestion noted in some adductor muscles. Microscopic examination revealed acute, patchy, degeneration and necrosis of the adductor muscle of the hind limbs, accompanied with interstitial edema and mild, acute, inflammation. No significant heart lesions were detected in the birds examined.

Qualitative analysis of **crop contents** from these birds revealed the presence of the ionophores monensin and naracin. Naracin is toxic to turkeys at any level, and should not be in turkey feed at any time. Quantitative anlaysis of feed samples also indicated the presence of monensin (.45 mg/kgm) in trace amounts. These birds had inadvertently been fed a ration contaminated with feed intended for use in broiler chickens, which are much less sensitive to the potentially toxic effects of ionophores. In chickens, these substances are used primarily as coccidiostats. Turkeys over 10 weeks of age should not be fed ionophores.

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Septicemic **fowl cholera**, caused by the bacterial organism ***Pasteurella multocida***, was diagnosed in a large group of 19 week-old turkeys in which there had been acute, ongoing losses, with high morbidity and mortality. *P. multocida* was cultured from multiple organs, including enlarged spleens.

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Erysipelothrix rhusiopathiae was cultured from the livers and enlarged spleens of ducks aged 31 wks suffering from acute deaths. Microscopic examination of livers also indicated the presence of chronic-active hepatitis in some of the birds, in conjunction with hepatic and splenic amyloidosis. The acute nature of the mortality in this flock suggests that the culture of *Erysipelothrix* was significant in these birds.

Hepatic amyloidosis is an interesting phenomenon in commercially raised ducks; most authors attribute the condition to ongoing, excessive antigenic stimulation of the liver to a variety of microbial organism such as *E. coli* within the birds' environment, which, by nature of the birds, may often become very moist or sloppy, and heavily contaminated with bacteria. The presence of hepatic amyloidosis, coupled with chronic active hepatitis, may well have predisposed these birds to bacterial septicemia caused by *Erysipelothrix*.

Mass mortality of racer (*Coluber constrictor*) and garter snakes (*Thamnophis sirtalis*) at White Lake, British Columbia:

— *Stephen A Raverty (AHC); Walt Klenne (BC Ministry of Forestry), and John Richardson (Department of Forest Sciences, UBC).*

During an ongoing, three year investigation into the ecology of tiger salamander (*Ambylostoma tigrinum*) populations at White Lake situated in the southern interior of British Columbia, five to ten adult, moribund racers (*Coluber constrictor*) and commmon garter snakes (*Thamnophis sirtalis*) were observed during daily site visits between July 20 and August 6, 1999. The snakes were found either partially submerged within shallow water or lying along a gently sloping sandy or densely vegetated embankment, within five meters from shore. Both live and dead snakes were recovered. Live snakes were readily approached, non-responsive and lethargic, in contrast to previous interactions experienced with racers and common garter snakes. On physical manipulation, some snakes exuded a clear mucous discharge from the oral or nasal cavities (nares).

Aquatic insects such as water boatmen (*Family Corixidae*) remained abundant on these sites, as did shorebirds such as Killdeer (*Charadrius vociferus*). Wilson's phalaropes (*Phalaropus tricolor*) were commonly observed foraging within shallow water along the periphery of the lake, with no untoward effects observed.

White Lake is a shallow, semi-permanent lake that periodically dries to a salt basin in late summer (a *playa* lake). However, with the abundant precipitation last winter and a cooler spring this year, the lake water remained unusually high throughout the summer months. Because none of the examined snakes had gastric or intestinal ingesta, the phenomenon of elevated water levels - coupled with reduced ambient temperatures and the possibility of a concurrent lack of prey for the snakes at the time of emergence may have contributed significantly to the observed morbidity.

On gross and microscopic examination of the submitted samples, the most consistent lesion was kidney urate deposition, which in most animals was severe and would have contributed significantly to impaired renal function. Based on extrapolation from captive snakes and other reptiles, prime differentials in this case would be dehydration, kidney toxicity, heavy metals (lead), vitamin A deficiency, neoplasia and glomerular disease.

Water samples from 3 sites around the lake were submitted to Environment Canada Laboratories, Pacific Environmental Science Center. Analysis disclosed a number of aberrations. Conductivity, a measure of the sodium and chloride content, was between 22,900 and 30,000 *u*S/cm (normal values range up to 100*u*S/cm); alkalinity was increased, with pH at 9.52-9.56 (normal reference values between 6.5-9.0). With such profound changes in water chemistry, submergence within or consumption of this water may have resulted in severe dehydration, with subsequent urate deposition in kidney tissue.

Although aerobic culture and electron microscopy of submitted snakes proved unremarkable, histopathology of selected snakes disclosed variable numbers of gastrointestinal cestodes, respiratory and intestinal nematodes (worms), and in one animal, gastric cryptosporidiosis (*Cryptosporidium serpentes*). As this last condition (cryptosporidiosis) may pose a significant health threat to snakes in captive situations, additional field surveys were conducted September 13 and 15 to assess the possible prevalence of this condition in the population. However, no snakes were detected within the area. *C. serpentes* is not considered a human health threat, and the risk to local livestock is remote. Due to the importance of snakes within ecosystems, additional field investigations are scheduled for next year to assess the health status of this population.

Listeriosis in farmed chinchilla in British Columbia:

— S. Raverty (AHC) and D. McIntosh (AHC)

Fur bearing animal submissions to the Animal Health Centre have increased incrementally from 4 accessions in 1994 to 7 cases in 1995, 12 in 1997, 15 in 1998 and 14 submissions as of November, 1999. Farmed chinchilla comprise 5 to 25% of the annual submissions, with degenerative and infectious diseases accounting for the majority of the diagnoses.

In June/99, 3 pelted, reproductively quiescent, female chinchillas were presented with a history of a recent change in feed, followed by a liquid, fetid, diarrhea, and increased mortality in the colony. All 3 animals were moderately fleshed . The most salient lesions were diffusely dilated small and large intestines, which contained variably amounts of liquid, green-grey, fluid ingesta. Histopathology of multiple segments of bowel revealed small clusters of neutrophils scattered within the lamina propria and submucosa, with occasional dilation of lymphatic vessels (lymphangiectasia). Special stains disclosed variable numbers of gram negative rods (bacilli) overlying the apical aspect of the mucosa.

Based on heavy yields of *Escherichia coli* on bacterial culture, a possible **dysbiosis** was presumptively diagnosed. Clinically affected animals in the colony were treated empirically with enrofloxacin. On treatment, mortalities initially subsided. However, within 2 weeks of cessation of medication there was recrudescence of clinical signs, and 4 additional chinchillas were presented for necropsy on July 12, 1999.

Gross examination of the second group disclosed focally disseminated necrosis of multiple viscera. Throughout the liver, spleen, mesenteric lymph nodes, and along the serosal surface of the small intestine, there were white, round, punctate foci. Microscopic evaluation demonstrated necrotizing microabscessation, pyogranulomas, and occasional fibrogranulomas in representative tissues.

Based on necropsy evaluation, prime differentials for these lesions included *Listeria monocytogenes*, *Salmonella spp* and *Yersinia pseudotuberculosis*. Special stains failed to demonstrate any significant pathogens following culture of intestinal contents either on blood agar, or on MacConkey's agar; crystal violet media selective for *Salmonella spp* and *Campylobacter spp* specific media were negative. Cold enrichment culture yielded heavy growth of ***Listeria monocytogeneses*** from the liver, small intestine, and mesenteric lymph nodes. Based upon the pathology, a diagnosis of **disseminated, visceral listeriosis** was made.

L. monocytogenes is a facultatively anaerobic, gram positive bacillus, which is found widespread in the environment (soils, pasture, silage and water), and infects a number of wild and domestic animal species. Human infections are generally acquired via ingestion of contaminated and unpasteurized milk, dairy products, or uncooked vegetables.

In this case, based on the extent of intestinal involvement, an oral route of infection, possibly associated with contaminated feed or bedding, was considered the principle route of exposure. Recommended control measures included prompt removal of suspect bedding and feed, disinfection of enclosures, removal of dying and dead animals, and improved rodent surveillance. The isolates were strongly sensitive to erythromycin, gentomycin and penicillin, moderately sensitive to novobiocin and tetracycline, and marginally sensitive to cloxacillin and enrofloxacin. A **bacterin** was prepared for use in more valued stock, which were inoculated at the tail base; however, for logistical reasons, al animals in the facility could not be vaccinated.

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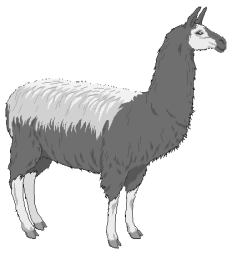
Listeriosis in farmed chinchilla in British Columbia: *(Cont'd)*

Despite reported epizootics and an inherent susceptibility of chinchillas to listeriosis, this is considered the first, culture-confirmed case in B.C. since the AHC has been in service. An epizootic is an outbreak of disease with large numbers of ill animals (high morbidity), which is only occasionally present in an animal community. Following antimicrobial therapy and vaccination, additional sampling has failed to demonstrate any further evidence of this disease at the chinchilla ranch.

Fetal myocardial degeneration and necrosis associated with vitamin E deficiency in an adult Llama (*Lama glama*)

— SA Raverty (AHC) and T Jacobson (Jacobson: Reimer Veterinary Clinic, Kelowna, BC)



Tissues were submitted from an adult, mid to near term, female Llama (*Lama glama*) with a history of acute mortality. Post mortem examination disclosed severe degenerative changes within the myocardium; throughout the ventricles, there were multifocally extensive regions of pallor which on sectioning were contracted, firm, white, and occasionally gritty. As this was the second adult llama to succumb with similar gross findings, representative tissues were forwarded to the AHC for evaluation.

The most salient microscopic lesions were localized within the heart. Immediately below the endocardium and randomly throughout the ventricular myocardium, there were multifocally extensive regions of myocardial degeneration and necrosis. Marked plexiform accumulations of dense, mature fibrous connective tissue were also noted, which peripherally entrapped degenerate and viable myocardial fibers that were multifocally punctuated by dense, mineral deposits. The lung exhibited variably extensive alveolar flooding with hypocellular proteinaceous fluid and within the liver there was marked, diffuse, centrilobular to midzonal sinusoidal congestion, with attendant compression of hepatic cords.

Based on the initial microscopic findings, a diagnosis was made of myocardial degeneration and necrosis possibly associated with **vitamin E/selenium deficiency**. Additional blood samples were obtained from a representative cohort of the flock. Follow-up blood submissions from 5 animals disclosed moderate to severe deficiency in serum vitamin E levels while selenium values were essentially normal. Vitamin E levels were measured respectively at <10, 110, 100, 40, and 70 mcg/dl (normal reference range: 150-600 mcg/dl).

Vitamin E interacts with selenium as a major antioxidant and free radical scavenger in the body. In adult llamas, deficiency is typically associated with myocardial degeneration, necrosis and fibrosis, with occasional ischemic necrosis of the extremities. In this case, feed analysis of the summer and fall forage disclosed a severe reduction in vitamin E levels.

It is important to realize that treatment of clinically affected animals will not resolve the pre-existing myocardial lesions. Nevertheless, treatment may prevent further worsening of the condition. Blood sampling (for serum analysis) to assess specific markers of myocardial degeneration may be considered. In larger herds, periodic serum samples taken from animals of different age groups is recommended, to ensure optimal levels of vitamin E in the forage.

Renal *Parvicapsula minibicornis* in wild sockeye salmon (*Oncorhynchus nerka*) from the Harrison and Adam Rivers, British Columbia

*— S Raverty (AHC), D Kieser, J Bagshaw, and S St Hilaire**

** Kieser, Bagshaw, & St Hilaire: Fish Pathology Program, Department of Fisheries and Oceans, Nanaimo, BC*

Sockeye salmon (*Oncorhynchus nerka*) are 1 of 7 wild Pacific salmon species and are considered one of the most valued Pacific northwest, fisheries commodities. This species ranges throughout the north Pacific rim, from the Columbia River, Oregon, along the west coast of British Columbia, to Bristol Bay, Alaska, with abundant stocks reported eastward around the Kamchatka peninsula and northern Okhotsk Sea, Japan.

During the most recent spawning run of the Harrison and Adam rivers, British Columbia, large numbers of dead, adult salmon were observed during late August, through September and October. Most losses were incurred prior to spawning. In a collaborative effort with the Fish Pathology Program, Department of Fisheries and Oceans (DFO), Nanaimo, BC, an investigation into the cause of death of these animals was initiated.

Five adult salmon were presented September 29, 1999 in good body condition and aside from multiple superficial cutaneous abrasions and lacerations attributed to physical excoriation associated with migration, and small numbers of adult nematode parasites (*Anisakis spp*) within the coelomic cavity of 4 fish, there were no other overt internal or external lesions. Aerobic bacterial and viral culture of multiple tissues disclosed no significant pathogens. Trace mineral analysis of liver and skeletal muscle proved within normal, in-house limits, for adult Pacific salmon.

Histopathology of all six salmon revealed consistent lesions in the kidney. Throughout the posterior kidney, the capillaries of virtually all the renal glomeruli were massively distended and occluded by dense accumulations of immature protozoal parasites. Small numbers of similar developmental stages were observed within the gills of selected fish, and an early developmental stage of the parasite was noted in the lateral ventricle of the brain in a single fish.

To assess the development of this parasite and possible associated pathology, follow-up collection and submission of 5 additional fish was conducted on Oct 27, 1999. Each of the submitted fish exhibited more profound changes within the kidney. In addition to developmental stages within the renal glomerular tufts, large numbers of mature, parasitic spores were noted within the lumen of numerous tubules. In more severely affected fish, there was diffuse thickening of the glomerular basement membrane, likely a sequela to persistent antigenic stimulation associated with the parasites and immune complex deposition; in addition, there were scattered tubules with focal to segmental coagulative necrosis of renal tubular epithelia with occasional proteinaceous, tubular casts.

The parasite was speciated by scientists at the Pacific Biological Station, DFO as *Parvicapsula minibicornis*, a myxosporean pathogen previously observed in sockeye salmon from Horsefly River in 1978 and described in spawning wild, sockeye salmon stocks from Weaver creek in November, 1996. The life cycle of this parasite has not yet been determined. However, based on extrapolation from other related, myxosporean species, it is likely complex, requiring an intermediate tubificid worm stage.

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Renal *Parvicapsula minibicornis* in wild sockeye salmon (*Oncorhynchus nerka*) from the Harrison and Adam Rivers, British Columbia *(Cont'd)*

While there has been considerable mention of possible recruitment of this parasite from production stocks off the BC coast in the lay media, there is no evidence of transmission between farmed Atlantic and Sockeye salmon. Based on review of case material submitted to our respective diagnostic services and discussions with aquaculture veterinarians, this parasite has not been previously detected in farmed Atlantic salmon along the British Columbia coast and the source of infection remains unknown.

Efforts to further characterize the life cycle, distribution and transmission of this organism are ongoing at the DFO and more results are to follow.

Suspect adverse vaccine reactions in farmed Atlantic salmon:

— S Raverty (AHC), J Thornton (Bayotek/ Microtek), and J Brocklebank (Brocklebank Mobile Services)

Historically, adverse vaccine reactions in farmed fish stocks have been associated with a chronic, fibrosing and melanizing peritonitis, which can contribute to downgrading and potential financial loss at the time of harvest and processing. However, over the course of the last year, the AHC has been involved with 4 investigations of peracute to acute mortalities associated with intraperitoneal vaccination of commercially available vaccine preparations.

From a clinical perspective, fresh water or recently introduced salt water smolts are seined into a holding facility, then transferred to an anesthetic bath and vaccinated intraperitoneally. Signs of adverse reactions are usually evident either within 30-60 minutes, or 1-2 days post vaccination. Characteristically, a large proportion of injected fish exhibit blackened, discolored skin, are unresponsive to external stimuli, rise to the surface of the water and exhibit sudden, incremental increases in mortality.

Gross examination of fish often reveals nonspecific findings of visceral congestion, hyperemia with occasional petechiae and ecchymoses, and the presence of free vaccine dispersed throughout the peritoneal cavity.

Vaccine samples in all 4 episodes were cultured both aerobically and anaerobically. All but 1 case failed to yield any significant pathogens. In one submission, the vaccine bag had been opened, and *Pseudomonas spp* contamination of the contents had likely occurred after injection. Vaccination was discontinued in 1 of 4 submitted cases, as soon as mortalities were noted.

Lipopolysaccharides (LPS) are incorporated into vaccine preparation to evoke a protective immune response. Paradoxically, while fish appear to be innately resistant to LPS, excess levels may be deleterious. To exclude the possibility of excess LPS in vaccine preparations, samples have been submitted to the Department of Veterinary Pathology, Ontario Veterinary College, for determination of endotoxin titers. Assays for endotoxin or lipopolysaccharide have been conducted in all 4 cases, with elevated levels from 1.25 to 2.50 ug/ml detected in only one instance. This value represents only a single-fold increase in dilution; however, this test result is not considered statistically significant, due to lack of sensitivity within the assay.

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Suspect adverse vaccine reactions in farmed Atlantic salmon: *(Cont'd)*

A prospective investigation comparing 2 identical vaccines from different manufacturers was undertaken. In one challenge, vaccination evoked an anticipated lymphohistiocytic reaction within the peritoneal cavity, whereas, the intraperitoneal vaccination of the suspect vaccine resulted in a severe fibrinosuppurative peritonitis with scattered, degenerate, exfoliated mesothelial cells. A difference in the adjuvant incorporated into the latter vaccine likely account for this process.

The lack of a clinically defined acute to peracute, adverse vaccine reaction has significantly impeded diagnostic evaluation of suspect cases. Clinical suspicion should be heightened whenever there is mass mortality associated with vaccination. Thorough evaluation of underlying diseases to exclude any other processes should be pursued. In one of the above cases, there was a fulminating septicemia associated with furunculosis. In an additional submission severe internal parasitism was observed, which likely contributed directly or indirectly to the observed fish losses.

At present, the diagnosis of an adverse vaccine reaction is contingent not only on historical data, but also on exclusion of any additional, underlying factors. The possibility of sub-optimal water quality at the time of anesthesia, status of smoltification, fish strain, and physical trauma incurred at the time of handling the stock, cannot be discounted. As well, physical changes associated with emulsion preparation, partitioning of the vaccine components and other factors, warrant evaluation.

Suspect cases should be brought to the attention of field veterinarians or diagnostic laboratories, and appropriate clinical work-ups initiated.

Tall fescue and endophyte toxicosis: a hazard to grazing cattle:

Tall fescue toxicity linked to the fungal alkakloid **ergovaline** has recently occurred in cattle in B.C., notably within the Peace River District. The following extracts are from an article on the subject, which we offer to our readers.

"…**Endophyte** is a fungus that lives inside the grass plant. The relationship between grass and endophyte is symbiotic, that is, they both benefit. Although endophyte does not harm grass, it produces toxins that are harmful to livestock. Since it does not affect the appearance of the grass plant, its presence can be detected only by laboratory analysis.

Some grass varieties grown for turf seed have high levels of endophyte. The reason is that infected plants can have increased growth, increased drought tolerance, and resistance to certain insects - qualities for which plant breeders select.

"…Endophyte is transmitted only by seed, and its entire life cycle takes place inside plant tissues. A plant does not become infected from its neighbours. Therefore, a stand of a non-infected variety (of tall fescue) will remain non-infected. If it is over-seeded with an infected variety, only the new plants will be infected. A stand of an infected variety cannot be cured with an application of fungicide.

Avoiding endophyte toxins: "…Fields planted with certified seed of zero- or low-endophyte forage varieties of tall fescue or ryegrass should be safe to graze or cut for hay. To keep them endophyte-free, livestock producers should not over-seed with infected turf varieties.

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Tall fescue and endophyte toxicosis: a hazard to grazing cattle: *(Cont'd)*

Ergovaline, the toxin produced by the tall fescue endophyte, is concentrated in the reproductive parts of the grass plant, especially the seedheads (and seed screenings). Toxicity is not reduced by pelleting the screenings. Therefore, feeding seed screenings from unknown grass seed fields is risky.

"…Ergovaline, however, is still present after several years of storage. Storing infected grass seed straw or screenings doesn't make them safer to feed. Also, making silage does not reduce toxicity, although ammoniating straw does…"

Testing: Oregon State University can test straw, seed screenings, or pasture grass for ergovaline… It is important to take a representative sample… To sample a pasture, take grass plants from at least 20 locations… Seal samples in a polyethylene

Freezer bag and label them (include your address)…Send them, along with your payment, to the address below:

Veterinary Diagnostic Laboratory
PO Box 429
Corvallis OR 97339 USA.

The lab phone number for endophyte questions is (541) 737-6541."

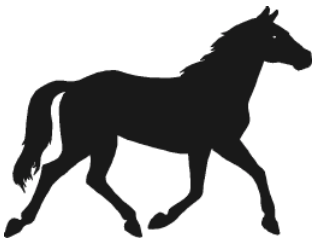
Summary: " In summary, livestock producers in the Peace should avoid feeding seed screenings and forage aftermath from turf-type tall fescue fields. All livestock producers in the province growing or thinking of growing tall fescue for forage should plant only a zero- or low-endophyte forage-type variety. Currently, such varieties being sold in B.C. include Atlas, ASU Triumph, Barcel, Courtenay, Dovey, Festorina, Fuego, Maximize, Montebello, Seine, and Stef. Finally, dairy producers at the coast have been growing and feeding forage-type tall fescues for over ten years with no livestock problems."

Editor's note: These comments, written by agriculturalist Don Bates on fungal toxicity linked to tall fescue, were taken, with grateful appreciation, from **The Forager***, newsletter of the B.C. Forage Council; Sept. 1999 issue, Volume 8, No. 4.*

Bovine Spongiform Encephalopathy Survey:

Over the last decade, considerable interest has emerged regarding transmissible spongiform encephalopathies and in particular, bovine spongiform encephalopathy (BSE). As part of an ongoing, cross-Canada national survey for BSE, staff of the AHC routinely sample and microscopically review all brain sections from cattle over 2 years of age for any histopathological evidence of this disease. To expand the number of samples reviewed by AHC staff, we request the assistance of practitioners who conduct field post mortems on adult cattle. Whenever possible, your collection and submission of brain samples (particularly the **medulla and brain stem**) for later BSE evaluation would be appreciated.

Equine Erosive dermatitis revisited:



" Our June 1999 *CEPTOR* reported cases of erosive stomatitis in a group of Ontario horses. Further investigation has linked the condition to bedding. The wood shavings were made from maruba, a tree in the *Simaroubaceae* family. Exposure to maruba resulted in hemoconcentration, hyper-bilirubinemia, and a rise in hepatic enzymes. Exposed horses developed erosive stomatitis and died as a result of contact with this material.

The *Simaroubaceae* family is largely tropical and semitropical trees. Maruba (*Simarouba amara*) is a tropical hardwood tree from South America. One plant from that family…. *Ailanthus altissima*, is grown as an ornamental tree in warmer parts of Canada. It apparently causes dermatitis in humans and is suspected of causing gastro-enteritis.

To avoid similar problems, horse owners should know the source of all bedding materials and ask about the tree (wood) species included in the shavings before using the product."

— From ***CEPTOR***, ***Animal Health News***, newsletter of the Ontario Ministry of Agriculture, Food, and Rural Affairs; October /99: vol 7, no.3.

Botulism toxin and its discovery:

"Irrelevant Historical Note: The toxic basis for botulism was identified in 1895 by Emille Van Ermengem, a professor at the University of Ghent, at a funeral music festival in the town of Ellezelles. Musicians became ill following a meal of tainted ham. This brings to two the number of reasons people might wish to avoid such festivals."

Ed.'s note: *this brief, enlightening, and morbidly amusing comment was included as an addendum to an Abstract presented by Dr. Donal O'Toole of Wyoming State University, at the 1999 Western Conference of Veterinary Diagnostic Pathologists, at Saskatoon. The Abstract and case discussion focused on an outbreak of botulism in 14 horses. Botulinum toxin type A was detected in alfalfa hay, via the mouse bioassay test.*

Equine Notes from the Western Conference of Veterinary Diagnostic Pathologists:

Editor's note: this past September, the Western Conference was held at Saskatoon. The topic of discussion was "pathology of equines". The moderator was Dr. Beth Valentine, of Oregon State University. A special session was also held at the end of the conference on equine eye disease, under the tutelage of Dr. Cullen, of the WCVM. Below are condensed versions of abstracts from 3 of the 46 different case abstracts presented at this conference.

1. Equine polysaccharide storage myopathy (*case 39*):

Ed.'s note: Case discussed was a 2yr Quarter horse with colic that arrived recumbent in the trailer, and exhibited "neurologic signs" (stumbling, falling) prior to admission for abdominal surgery… the horse eventually died from a difficult recovery in which cervical vertebrae were fractured.

On necropsy, microscopic sections of semimembranosus muscle demonstrated mild fiber size variation with fiber hypertrophy more obvious than fiber atrophy…Numerous fibers contained one or more sarcolemmal rounded vacuoles. A moderate number of fibers contained small multiple "lakes" of material… that was PAS positive and amylase resistant (complex polysaccharide)… scattered interstitial macrophages containing PAS positive amylase resistant complex polysaccharide were also noted.

"…. The semimembranosus muscle findings are characteristic of equine polysaccharide storage myopathy (EPSSM) in an early stage (consistent with the age of the horse), and the presence of interstitial macrophages is indicative of prior segmental necrosis of fibers containing complex polysaccharide. This underlying myopathy is thought to have been the cause for increased muscle enzyme levels at admission. Other speculations on this case are that neuromuscular weakness and pain may have contributed to the physical findings."

— from the Abstract presented by moderator B. Valentine, of Oregon State University.

2. Equine sarcoids (*case 34*):

" Equine sarcoids are unique, locally aggressive fibroblastic skin tumors in horses different from papilloma, fibroma, and fibrosarcoma. They are not only the most common skin tumors (about 70%), but also the most frequently observed tumor (about 40%) of horses, donkeys and mules. Therefore equine sarcoids are considered as semi-malignant tumors. The etiology of equine sarcoids is not yet fully understood. Skin injuries seem to be an important cofactor in the development of this tumor… it was possible to show that equine sarcoids contain DNA sequences of bovine papilloma viruses (BPV)… Studies using PCR and direct sequencing confirmed the view that BPV-1 and BPV-2 play an important role in the development of equine sarcoids…

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"…The studies confirm the opinion that equine sarcoids are the result of a nonproductive infection of an alien, non-permissive host with BPV. They explain also the high recurrence rate after incomplete surgical removal. PCR is shown to be an ideal tool to affirm the histopathological diagnosis of "sarcoid" especially in early stages of sarcoid development or severely ulcerated and inflamed equine skin lesions."

— From the Abstract presented by Jens Teifke, Washington State U.

3. Monensin toxicosis in a miniature horse (*case 1*):

"…This horse came from a group of 17 animals, four of which showed clinical signs of lethargy, anorexia, recumbency and convulsions. Three of the 4 animals died. Vaccination history included current inoculations for eastern and western equine encephalitis, tetanus, and equine influenza… Nutritional regimen included dry hay and pelleted feed.

Gross pathological findings included abundant straw-colored fluid in the pericardial and pleural cavity, and diffuse, bilateral pulmonary congestion and edema. Microscopic examination of cardiac muscle revealed a severe, extensive, necrotizing and histiocytic myocarditis. Based on the histopathology, feed was analyzed for monensin residues…identified 90 ppm monensin, a level that is roughly equal to 41 grams/ton of feed. The recommended level of monensin in feed for cattle is 25-30 grams/ton.

Horses are extremely sensitive to the cardiotoxic effects of monensin. The LD50 for the horse is 2-3 mg/kg with a single dose toxicity of 1-2 mg/kg. An ionophore, monensin is an active compound produced by ***Streptomyces cinnamonensis***. It is used both as a growth promotant in ruminants and a coccidiostat in poultry. Toxic doses interfere with intercellular potassium and calcium transport and lead to mitochondrial swelling and failure."

— From the Abstract presented by N.W. Dyer, North Dakota State U. Diagnostic Laboratory.

Humans and the domination of nature:

"…Our ability to humanize the world has caught up with our historical, human-centred sense of dominance, and it is the extent of our contemporary ability to dominate nature that is behind today's extreme ecological movements.

"…We need a new debate about our role in nature, one that steers us away from both the extreme fervour of tree slashers and the excesses of technological progress and bulldozing development. Eco-vandalism is as abhorrent as any violent anti-social act, yet we need to understand the fundamental alienation from which these militant acts develop.

Human progress through science, technology and development can express the finest attributes of human ambition, but we also need to balance the benefits of these advances with the losses they induce by preventing us from connecting with nature.

We have an unfortunate ability to react to individual problems rather than acting from a fundamental ethic. Cloned trees, a pesticide spray, or the loss of salmon stimulate interest groups to action, but as a society we have not come to grips with our relationship to nature that underlies and unites these issues. Until we engage in this discussion, we can expect that polarized response to environmental issues will continue to occupy the public agenda."

— From the essay *Humans struggle with their polluting place in nature*, in the Nov 12/99 *Vancouver Sun***,** by Mark Winston, professor of biological sciences at Simon Fraser University.

The importance of dung beetles:

"…One of the causes of fly pestilences that in warmer months used to plague parts of Australia, after the introduction of cattle in 1788, was the absence of native species of dung beetle, able and willing to dispose of the daily tonnages of cow manure. Since most of the native Australian species are accustomed to dealing solely with the feces of marsupials, conditions in that continent became increasingly unpleasant. It was asserted that in some parts the pats "almost merged into one continuous habitat." It was only after the 1970s, when an extensive Commonwealth Scientific and Industrial Research Organization program was started in which some twenty selected species of African dung beetles were introduced, that Australians managed to ameliorate the problem. In some places, the population of dung flies… were reduced by… 90 per cent, a partially successful biocontrol. Unfortunately, for these insects, and indirectly for ourselves, cattle vermifuges… which finish up in the feces, may wipe out the dung beetles along with the parasites."

— From the book *Merde: Excursions in Scientific, Cultural, and Socio-Historical Coprology*, by Ralph A Lewin. Random House publishers, New York, 1999.

A Poet's truths:

"…Wee, sleekit, cow'rin, tim'rous beastie,
Oh, what a panic's in thy breastie!
Thou need na start awa sae hasty,
Wi' bickering brattle!
I wad be laith to rin an' chase thee
Wi' murd'ring pattle!

I'm truly sorry man's dominion
Has broken nature's social union,
An' justifies that ill opinion,
Which makes thee startle
At me, thy poor, earth-born companion,
An' fellow-mortal!

"…Thy wee bit housie, too, in ruin!
Its silly wa's the win's are strewin'!
An' naething, now, to big a new ane,
O' foggage green!
An' bleak December's winds ensuin',
Baith snell and keen!…"

"…But, Mousie, thou art no thy lane,
In proving foresight may be vain:
The best laid schemes o' mice and men
Gang oft a-gley,
An' lea'e us not but grief an' pain,
For promis'd joy.

Still thou art blest compar'd wi' me!
The present only toucheth thee:
But och! I backward cast my e'e
On prospects drear!
An' forward, tho' I canna see,
I guess an' fear!"

— *excerpts from the poem* To A Mouse (on turning up her nest with the plough, November, 1785) . *Robert Burns*.