Animal Health
PERSPECTIVES

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The changing world of Animal Welfare

By: Greg Douglas, Chief Veterinary Officer, Saskatchewan Ministry of Agriculture

Society wants to be heard on the issue of appropriate care for animals. Increasingly, a well-meaning public is expecting early intervention when animals are in distress. They won't tolerate the notion of an animal being abused or neglected. When the question is asked, people invariably choose the protection of animals.

Saskatchewan residents expect that the livestock industry, government and animal producers support their values. No longer does the public subscribe to the view that animal producers are free to raise their animals in whatever fashion suits them. The public is giving clear direction that they wish to have a role in the oversight of animal care.

How does veterinary medicine rationalize this groundswell of opinion with the fact that the modern world is generations removed from food production? This same public has had no widespread exposure to today's production methods; methods that minimize disease introduction, optimize product quality and provide for comfort as animals grow. Biosecurity in barns, herd health programs and selecting for genetic potential are as foreign to our society as the idea that milk actually comes from the udder of a cow.

How should livestock industry and government proceed?

It's a combination of many strategies. We will need to communicate the reasons for protecting domesticated animals in production systems. Collectively, it will become increasingly important to promote the value of our accessible, affordable, nutritious and safe food supply. There is merit in improving our transparency with current animal management practices. It's an opportunity .to educate the public and illustrate the benefits of science in agriculture.

Animal welfare is a complex issue. Our challenge is to lead progressive, constructive change. Our industry must advance animal husbandry in areas of nutrition. **Too many herds in Saskatchewan suffer through long, cold winters with inadequate feed quality.** In some cases, we must be open to new ways of providing for the animals that we keep. It's imperative that when individuals turn their backs on their animals, our communities respond quickly and appropriately. Collaboratively, we can provide early detection systems that may uncover animals in distress sooner. Together with agencies like the Saskatchewan Society for the Prevention of Cruelty to Animals we can track, monitor, investigate and prosecute cases of animal abuse. We must try to prevent these situations from becoming catastrophic. Communities and residents must be vigilant in reporting suspicious conditions.

We can change animal protection legislation so perpetrators of animal abuse and neglect face tougher penalties. One example might be broadening the definition to include all individuals who inflict harm and cruelty on animals. In cases of serious animal abuse, lifetime bans from keeping animals should be a consideration by our judiciary. Changing legislation, however, has its limitations. **Obviously, our best efforts will be to care for the animals before they are in distress.**

Saskatchewan livestock producers have built a legacy of responsible, productive and innovative agricultural practices. It would be unfortunate that a few deplorable situations could tarnish that reputation. Government, industry, veterinarians and producers have roles to play in education, prevention and investigation in the area of animal welfare. Together, we can do better.

What's inside

Improving biosecurity for beef cattle producers

By: Andy Acton, Client Service and Business Development Manager, PDS

The Saskatchewan Ministry of Agriculture is bringing forward a biosecurity initiative for beef cattle producers, and the interesting thing about this plan is the delivery system – the local veterinarian practitioner. After a series of "train the trainer" sessions, practicing veterinarians in Saskatchewan can provide a combination of seminars and farm consultations to educate their clients about biosecurity and how it could improve their operations.

It is generally accepted that biosecurity in beef cattle operations is not at as high a level when compared to intensive livestock enterprises. It is much easier to achieve a reasonable level of biocontainment with swine that spend all of their producing lives indoors but it is a logistical challenge for a beef operation. Having said this, there are a number of lessons to be learned about biosecurity that apply to beef cattle operations, and they can best be understood in the context of fixing that old favorite cattle containment system – the barbed wire fence.

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When discussing biosecurity and the concept of a "closed" herd with clients I've often used the phrase "barbed wire is a lousy wall". What we need to improve beef herd biosecurity are tools that can change that fence into a wall, figuratively at least. What are some of the components of practical biosecurity that can help us achieve that goal?

1) **Awareness**—Of the diseases of concern and how to establish their presence in the herd. What is the current status of the herd with respect to BVDV, or Johne's disease? Is the client aware of what diseases can be brought into a herd, and which ones can be screened for, prior to bringing new additions into a herd?

2) **Questions** - Lots and lots of questions, especially when sourcing new animals for a herd. What vaccinations have the new animals had? What health problems have been present on the operation in the recent past? Have visitors and suppliers been on other operations, especially ones which have current health issues such as calf scours? Where have those boots been?

3) **Sanitation** - Never an easy job for a beef cattle operation, but it is getting easier. Improved washing systems for vehicles transporting cattle, and improved facilities make keeping things clean easier than in the past. Knowing the details about various classes of disinfectants and which ones will be practical for your clients to use is a key here.

4) **Vaccination** - This is a big one for beef producers, and helps to fill in the spaces between the strands of barbed wire, especially with respect to BVDV and other viral diseases, and to a lesser extent for other pathogens. We all know breaches of biosecurity will occur, but a client who uses a proper and comprehensive vaccination strategy can usually come through introduction of a "new" pathogen with a lot less economic hardship.

5) **Monitoring–** Of current diseases present in the herd, and hopefully, screening new introductions for diseases of concern. Ensure the client knows what tests are available, and which are practical for a given disease. Testing virgin bulls for *T. foetus* may not make a lot of sense, but having an ear notch done for BVDV might. That same ear notch for BVDV may mislead a producer buying a bred heifer into thinking he is safe from that disease by testing her at herd entry and not testing the calf the next spring. I think disease monitoring will be an increasingly **important part of our future jobs** as bovine veterinarians. **The key point here is that your producer really needs you to negotiate the minefield that is herd health monitoring and biosecurity.** A blanket list of recommendations from a website or a consultant from afar really doesn't cut it for a large majority of beef cattle operations. They need the knowledge of veterinary professional that is familiar with their operation and is willing to help them make economical changes and improvements that will actually work on their operations.

I've purposefully left out "management" as a point as that term is often overused in the concept of disease prevention. I've heard and been taught that "proper management" be proposed as the way out of seemingly every disease entity. Then, I've seen many of these diseases occur because "management" was being used instead of using the appropriate vaccine. The other reason I left it out was that I'd like to bring you, the veterinary practitioner, into the conversation. There are, of course, lots of "management" ideas that are crucial for beef herd disease prevention. If you have a novel strategy or idea that would be a useful addition to the Beef Cattle Biosecurity Program, please e-mail it to me for possible inclusion in the program material (dsac@sasktel.net). Extra marks if it involves lots of large scale laboratory testing.

Kathryn Ross, Saskatchewan Ministry of Agriculture presenting the beef cattle bio-security program in Saskatoon.

Enzootic Ataxia in Farmed Elk in Saskatchewan

By: Al Perry, Veterinary Pathologist, PDS; Henry McCarthy, McCarthy Veterinary Services, Wawota; Bonnie Brandt, Animal Hospital of Assiniboia

In June and October of 2007 enzootic ataxia was diagnosed in 4 adult cow elk on two farms in southern Saskatchewan. Enzootic ataxia is a degenerative disease of the spinal cord and brain, primarily described in sheep and goats, but also in other species, including farmed cervids. Although the cause is unknown, it is generally associated with copper deficiency and has been prevented by copper supplementation. Enzootic ataxia has been well documented in New Zealand, Australia, and Europe in farmed red deer. [1,2,3,4] North American elk are susceptible to copper deficiency; [1] however, we are not aware of previous reports of enzootic ataxia in farmed elk in Canada.

The clinical signs, nutrition, and management were similar on both farms, although only limited information was available for one of the farms (farm A). On farm A, over the previous year, 9 adult elk (sex wasn't indicated) in a herd of 150, and on farm B, over the previous 6 months, 7 adult cows in a herd of 40 cows and 50 bulls, developed ataxia slowly progressing to caudal paresis. Farm A elk were on pasture supplemented with alfalfa hay and whole oats, and were provided with a salt block and mineral tub. Farm B elk were on pasture supplemented with alfalfa/brome hay, whole oats, and salt and trace mineral blocks were provided; the source of water was a dugout.

Necropsies were performed on 4 mature adult females, 2 from each farm (cows 1 and 2 from farm A, cows 3 and 4 from farm B), ages 4-13 years, which had been euthanized and submitted to the laboratory for chronic wasting disease (CWD) testing and postmortem examinations. Cows 2 and 3 were in poor body condition with small deposits of fat; cows 1 and 4 were in good body condition; there were no significant grossly visible lesions. Significant microscopic lesions were evident only in the spinal cord and brain. Only a few sections were examined from the

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brain and spinal cord of cows 1 and 3, more complete examinations were made for cows 2 and 4. In all four cows the most severe lesions were evident in the spinal cord. Severe, bilaterally symmetrical myelopathy was evident in all sections from throughout the spinal cords. Lesions were mainly in ventral medial and dorsal lateral white matter tracts. Deficiency of myelin, spongiosis, axonal degeneration, and gliosis were evident. In dorsal and ventral horns there were moderate numbers of spheroids and there was degenerative change (chromatolysis) in a few neurons. In the medulla of the brain there were similar changes in white matter, and in the midbrain there were a few degenerative neurons and spheroids.

Hepatic copper levels were deficient in all 4 cows (farm A: cow #1 - 1.30 ppm, cow #2 - 2.30 ppm, farm B: cow #3 - 4.17 ppm, cow #4 - 4.89 ppm; wet weight, normal – 20-120 ppm [5,6]). Magnesium, manganese, iron, cobalt, zinc and selenium were normal except in cow #1 iron was high normal, in cow #2 iron and molybdenum (1.80 ppm) were high normal, and in cows #3 and #4 molybdenum was high normal (2.22 and 1.73 ppm).

In all 4 cows, testing of the brain obex for CWD (Biorad ELISA) was negative.

There was no follow-up information available for farm A, the herd was dispersed in the fall of 2008.

On farm B the herd was treated by the owner with Copasure© boluses (Animex, copper oxide). Affected animals didn't improve and there have been no new cases since October, 2007.

In addition to enzootic ataxia in adult elk, other herd health problems which have been associated with, or are suspected to be caused by copper deficiency include: osteochondrosis (causing enlarged joints and lameness) and unthriftiness in calves, and antler abnormalities and reduced fertility in adults.[1]

Because signs of copper deficiency are often nonspecific many differential diagnoses should be considered when investigating herd problems. **Differential diagnoses for enzootic ataxia include diseases affecting the CNS (e.g. CWD, viral, bacterial, and parasitic encephalomyelitis, and traumatic injury), as well as muscular diseases (e.g. nutritional myelopathy and exertional myopathy).**

In cases of enzootic ataxia microscopic lesions in the spinal cord are quite quickly obscured by postmortem change. Necropsy must be performed on fresh carcasses, preferably on elk euthanized the same day, to confirm or rule out the diagnosis. You can assess copper status by determining serum or hepatic copper. Hepatic copper assessment is more useful as copper is stored in the liver.

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Maxillary osteoma in a juvenile gelding

By: Chris Wojnarowicz,Veterinary Pathologist, PDS

Osteomas are benign bone masses occasionally encountered in young animals. [1, 2]They are associated with bones that form by intramembranous ossification such as, the cranium or pelvis.[1, 2] This brief report describes a case of maxillary osteoma in a 2 year-old Percheron gelding.

The maxillary mass was noted by the owner about six months prior to presentation. The mass grew steadily and was now interfering with prehension and mastication. The mass distorted the left side of the face and could no longer be covered by the lips leading to mucosal drying and ulceration. The mass was excised and submitted for histological examination. The excised mass (Fig.1) was hard, smooth, ovoid and was 10 cm in the rostro-caudal plane, 7 cm in latero-medial plane and 6 cm in dorso-ventral plane. The mass grew out of the left incisive bone. Rostral to the mass there was a single incisor tooth. Caudal to the mass there was normal incisive bone.

Fig. 1: Maxillary osteoma, side view. Note the size of the tumor (each "step" in WCVM tag denotes 1 cm). Partially engulfed incisor is located at the rostral edge of the mass.

On cut section (Fig.2) the mass had a dark red core surrounded by light red mantle. The width of the mantle was uneven, wider at the latero-ventral edge and narrower along the medial border. Other than the single incisor, no additional teeth were embedded in or engulfed by the mass.

Fig. 2: Maxillary osteoma, fixed tissue cross section. The outer mantle consists of variably cellular fibrous connective tissue while the core contains the spicules of well-differentiated mature bone.

Histologically, the mass was covered by a multifocally ulcerated oral epithelium. The epithelium was moderately hyperplastic with numerous, short, anastomosing rete pegs. Below the epithelium there was a wide zone of fibrous connective tissue with centripetally increasing degrees of cellularity. Spindloid cells were arranged in interlacing fascicles, which were loosely arranged and relatively sparse below the epithelium but became progressively denser and more collagen-rich centrally. Gradually the

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fibrous tissue merged with bony trabeculae that formed the core of the mass. In general, the trabeculae were oriented perpendicular to the surface of the tumor. Each consisted of lamellar bone with minimal patchy foci of woven bone. All trabeculae were rimmed by osteoblasts and rare osteoclasts. Two weeks following surgical excision the horse was reported to be doing fine.

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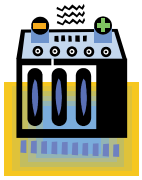
1. Meuten DJ, ed. Tumors in Domestic Animals, Iowa State Press, Ames, Iowa. 2002; 4th Edition
2. Miller MA,Towle HM, Heng HG, Greenberg CB and Pool RR. Mandibular ossifying fibroma in a dog, Vet. Pathology 2008; 45:203-206

Lead toxicity in cattle and the potential for food residues



By: LeeAnn Forsythe, Surveillance Veterinarian, Saskatchewan Ministry of Agriculture and Barry Blakley, Veterinary Biomedical Science Department Head, WCVM

Lead poisoning continues to be the most predominant toxicity encountered in cattle. In 2009, Prairie Diagnostic Services identified 114 herds in Saskatchewan with high blood or tissue lead levels (as of December 17, 2009, as compared to 48 herds in 2008). Currently, the primary source of lead on the prairies is discarded batteries, while lead poisoning from oil has been reduced dramatically due to the ban of tetra-ethyl lead in gasoline. Lead poisoning is both a food safety issue and a cause of significant economic loss for beef and dairy producers.

Clinical signs of lead poisoning in cattle are characterized by neurological signs which include depression, ataxia, blindness and seizures. The most severely affected animals die within 24 hours of initial onset of clinical signs, but some animals may die up to two weeks post-exposure.

Clinical signs alone are not a good indicator to identify cattle with abnormal levels of lead in tissues intended for human consumption. The concentration of lead in the blood does not correlate well with clinical signs. Therefore, asymptomatic cattle with lead poisoning may inadvertently enter the food chain.

Oral absorption of lead is slow and incomplete. Lead is deposited in the kidneys, liver and bone and excreted in the milk, urine and feces. Changes in lead blood levels over time can be highly variable which could suggest absorption of lead from the lead particles in the rumen or reticulum or ongoing lead exposure. The reporting of lead half-life has been variable and difficult to predict, ranging from 48 hours to 2,507 days.

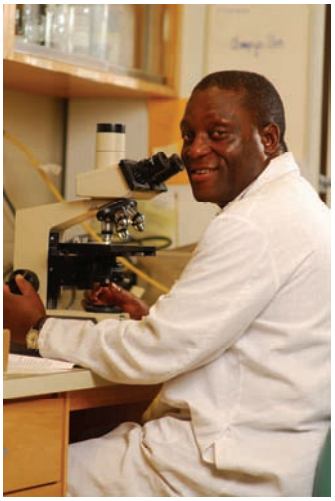
There is a significant public food safety concern over consumption of animal products derived from lead-poisoned cattle. It is not known how long exposed cattle should be held before they can be safely marketed or if they should ever be marketed for human consumption. Livestock producers need to ensure that cattle do not have access to lead by removing discarded batteries, oil, paint, shingles and other sources of lead. It is important to remember that asymptomatic exposed cattle may contain a sufficient amount of lead in their tissues, making them unsuitable for entry into the food chain.

References:

1. Waldner C, Checkley S, Blakley B, Pollock C, Mitchell B, Managing lead exposure and toxicity in cow-calf herds to minimize the potential for food residues. Journal of Veterinary Diagnostic Investigation. 2002; 14: 481-486.
2. Rumbeiha WK, Braselton W, Donch D, A retrospective study on the disappearance of blood lead in cattle with accidental lead toxicosis. Journal of Veterinary Diagnostic Investigation. 2001; 13: 373-378.

Quote: "Learning is not compulsory…….neither is survival."
-W. Edwards Deming

PDS Microbiologist Receives Diplomate Status



Congratulations go out to Dr. Musangu Ngeleka for achieving his Board Certification in Microbiology through the American College of Veterinary Microbiologists! Dr. Ngeleka recently wrote the College's general exam covering bacteriology, mycology, virology, immunology and molecular diagnostics as well as a subspecialty examination focused on bacteriology and mycology, both of which he passed successfully. Dr. Ngeleka manages the Microbiology Laboratory at PDS and provides technical supervision for the Bacteriology, Mycology, Parasitology and PCR services. He received his Diploma of Veterinary Medicine from the University of Congo (formerly Zaire) and a Master of Sciences (MSc) from the Department of Medical Microbiology at the University of Laval in Quebec-City, Quebec. He received his PhD from the Department of Veterinary Microbiology and Pathology, Faculty of Veterinary Medicine, University of Montreal, Saint-Hyacinthe, Quebec, while pursuing a part time internship and teaching assistant in Veterinary Clinical Bacteriology and Mycology at the same veterinary institution. The main focus of his PhD program was pathogenesis of *E. coli* infections in pigs.

Dr. Ngeleka spent two years as a post-doctoral fellow with the Vaccine and Infectious Disease Organization (VIDO) at the University of Saskatchewan, in Saskatoon, working on a genetically-engineered live attenuated *E. coli* vaccine for poultry.

He moved to Trinidad and Tobago and spent three years as an Assistant Professor of Clinical Bacteriology and Mycology at the School of Veterinary Medicine, The University of the West Indies prior to joining PDS.

The PDS Board of Directors, management and staff congratulate Dr. Ngeleka on this very significant achievement!

Readers' Feedback

The **Animal Health Perspectives** editorial team (Drs Mary Vanderkop and Moira Kerr, and Crystal Wagner) invite readers' comment on any material published in the newsletter or questions on material submitted by contributors.

Submit your comments or concerns to Dr. Moira Kerr (email: moira.kerr@pds.usask.ca) and they will be forwarded appropriately.

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