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Animal Health Perspectives

Provincial roles in animal disease events

By: Dr. Betty Althouse, Chief Veterinary Officer, Saskatchewan Ministry of Agriculture

The four pillars of emergency management are prevention, preparedness, response and recovery.

The province has many roles in animal disease emergency management. We manage surveillance, detection and response for provincially named diseases. We play a role in foreign animal disease (FAD) events, with the province supporting federal response, and coordinating with and supporting the affected livestock sector.

Prevention activities are focused on enhanced biosecurity at all levels, from preventing disease entry into the country through border activities that restrict or impose conditions on imports of animals, animal products, feeds and feed ingredients; as well as travelers; through to biosecurity at animal events and preventing disease entry and spread at the farm level.

Preparedness activities include training on early disease recognition for producers and veterinarians; ensuring laboratory capacity to recognize and confirm the disease; and, developing disease response plans and exercising them. Saskatchewan has a Terrestrial Animal Disease Emergency Support (TADES) plan with the Canadian Food Inspection Agency (CFIA) that outlines a collaborative response to a terrestrial foreign animal disease (TAD) event, and clarifies responsibilities such as communications pathways and notifications.

Some of the provincial roles and responsibilities in emergency response include ensuring animal welfare, coordinating the involvement of industry, providing premises identification information and mapping, as well as providing animal health expertise in biosecurity, epidemiology, disease control and emergency management. In Saskatchewan, the Public Safety Agency coordinates provincial-level emergency responses such as evacuations, flood response, forest or grass fire response. In an animal disease event they can assist with general coordination, additional staff for roadblocks or security, accessing equipment like portable generators, or large excavation equipment for disposal.

Provincial staff work closely with livestock organizations to ensure communications are effective and reach all producers. For example, information is needed not only on the current outbreak but also on early disease recognition and prevention and biosecurity.

Current prevention and preparedness activities for African Swine Fever (ASF) provide a good example of integrated emergency management planning.

CFIA, working with the national swine industry, has developed response plans that include small containment zones around infected farms. Zoning agreements are in place with the United States, the European Union and are in development with Japan. But, we expect, and are planning for, border closures for at least a short time, until it can be shown that the disease is contained.

The Canadian pork industry is highly export dependent, so if border closures to swine or pork occurs, even for a short time, there are market concerns. Loss of sales can quickly lead to overcrowding and an immediate price drop, reducing or wiping out farm income while producers still have costs to feed and house animals. National work continues on alternatives or enhancements to existing insurance and emergency funding supports. Nationally, the Executive Management Board comprised of CFIA, industry, and the three largest pork producing provinces (QC, ON, MB) coordinates and prioritizes ASF prevention, preparedness, and response and recovery initiatives.

Because of the integration of the western swine industry, the western Chief Veterinary Officers, pork industries and CFIA have formed an ASF western steering committee, with 4 priority working groups. The groups are:

- Events management and communications
- Destruction and Disposal
- Zoning and permitting
- Market Interruption and financial assistance

ASF emergency management planning continues to be a work in progress, with a focus on prevention and preparedness. We hope we never need to implement the response and recovery phases. In an emergency event, there are always things that can't be anticipated or planned for, so having strong relationships and communications with producer organizations and across governments are invaluable.

WHAT'S INSIDE

Vitamin Status and its Association with Neonatal Beef Calf Losses

By: Dr. Barry Blakley, DVM, PhD, Veterinary Toxicologist, WCVM

Vitamins, in particular, vitamins A and E, play a crucial rule in the growth and survival of beef calves. Fetal development, abortion, stillbirths and postnatal survival are influenced by vitamin nutrition. The assessment of vitamin status and the risk of disease and mortality is influenced by a variety of factors. In general, as the vitamin status declines, the probability of disease and a lack of vigor increases. Interpretation of diagnostic data and the implementation of preventive measures and treatment of affected calves remains a challenge. Intervention using corrective strategies is often unsuccessful.

The vitamin status is controlled by many factors which impact on survival and resistance to infectious disease. Primary sources of vitamin A nutrition are variable from region to region and influenced by feed type, drought, storage after harvesting, season, and susceptibility to degradation. In green feeds consumed during the summer months carotene is the predominant form. Little vitamin A (retinol) is present. Carotene is converted to vitamin A. In the cow, storage of most forms of vitamin A may last up to four months. In most beef cattle operations, pregnant cattle are approaching mid-gestation when vitamin intake begins to decline. The vitamin requirements to maintain pregnancy, and provide sufficient amounts for lactation and colostrum increase substantially. During this time period, cattle are no longer consuming fresh vitamin-enriched green feed. Vitamin A is light sensitive and moisture sensitive. Consequently, up to 95% of the vitamin A content in the feed may be lost prior to consumption. In addition, the transfer of carotene and vitamin A across the placenta is extremely limited. The stability and kinetic impact is evident in the fetus or neonate. The liver vitamin A content is extremely low. This deficient status results in altered embryonic development which is manifested by abortion, stillbirth, abnormal nervous tissue development and weak calves postnatally. Weak calves respond to varying degrees to vitamin A injections at birth. Since vitamin A affects epithelial tissue development, placental degeneration and retained placentas may also be evident.

In order to minimize the impact of vitamin A deficiency in the calf, supplementation of vitamin A by injection or in the feed is essential by mild gestation. The full impact will become evident in calves consuming colostrum. Carotene and all forms of vitamin A, particularly retinol, are present at high concentrations in the newborn calf following colostrum ingestion. The vitamin content in the newborn may increase 5-10-fold. Diagnostically, it is often possible to determine whether the newborn ingested colostrum based on serum vitamin A concentrations. The impact on growth and resistance to infectious disease is clinically significant.

Analytically most laboratories measure only retinol. In the liver, retinyl palmitate is the predominant storage form (approximately 90%). In the blood retinol is the predominant form. Fortunately most analytical methods convert all other forms of vitamin A to retinol to assess the total vitamin status. Feed analysis for vitamin content may be misleading. Stability and multiple forms of the vitamin compromise interpretation in the feed. Associated with tissue analysis, the most significant confounding variable is age. The "normal" vitamin A status as detected in the liver or blood is distinctly age-dependent. From fetus to adult the variability may exceed 100-fold. It is critical to estimate age for all submissions. In the first month of life, dramatic differences are observed. Investigative strategies should focus on population rather than individual animals. As the overall herd status declines, the risk of disease and neonatal mortality increase. Serum and liver evaluations are informative and useful from a dose-response perspective. It should be emphasized that vitamin A is highly teratogenic. Over supplementation will result in abnormal developmental problems depending upon the dose and time of supplementation during pregnancy. This highly teratogenic consequence is not associated with over supplementation with vitamin E.

Many of the factors associated vitamin A status also apply to vitamin E. Deficiencies of vitamin E are associated with muscle disease and immune dysfunction. Deficiency is manifested by weak calves, stillbirths and abortion. Again, the status is age-dependent and influences the interpretation of diagnostic data. Liver and serum are routinely analyzed for vitamin E (alpha-tocopherol) the predominant tissue form. Serum concentrations are highly age-dependent. The fetal liver concentrations are much lower as compared to other ages. Liver concentrations, postnatally, exhibit less age–dependent variability. By approximately 6 months of age the vitamin concentrations attain adult values. Limited placental vitamin E transfer accounts for the distinctly lower liver concentrations in the fetus. Once colostrum is administered, a major source of vitamin E, the tissue concentrations increase substantially and the muscle degeneration (white muscle disease) and immune dysfunction are less likely to occur. From a kinetic prospective, vitamin E is not stored in the liver to any great extent. Liver concentrations reflect recent uptake or supplementation. Recent injections with a day or so can be identified and often classified as high-normal. Age, colostrum and supplementation status are important historical facts impacting interpretation. In live calves, collect serum samples prior to supplementation.

Other risk factors such as drought, have little impact for vitamin E. Often heifers have an overall lower vitamin status and often produce calves with a greater occurrence of disease. If vitamin E supplementation of the pregnant heifer is delayed, muscle degeneration may occur. Supplementation at a later time may restore the status to normal, but the degenerative muscle remains. The vitamin E-selenium interaction also compromises interpretation. Both nutrients prevent oxidative damage, but there is an absolute requirement for both agents. Many therapeutic products contain a mixture of both agents. These products often contain limited amounts of vitamin E by design or the vitamin may have degraded during storage. Excessive administration of the combined product to enhance the vitamin E status may result in over supplementation of selenium, producing toxicity with normal to subnormal vitamin E stores.

In summary, the interpretation of vitamin E and vitamin A status is influenced by age, nutritional status of the cow, kinetic and placental factors, vitamin stability in feed and vitamin form and interactions with nutrients such as selenium. The lack of well-established, age–dependent normal values remains a major challenge for veterinarians and producers from diagnostic, treatment and preventative perspectives.

Two cases of testicular blastomycosis in male dogs

By: Steve Mills, Veterinary Pathologist, PDS

CASE #1

A 5-year-old male English Cocker Spaniel presented to a primary veterinary clinic in rural south-eastern Saskatchewan in mid-June 2019 for a routine wellness visit and vaccination. Incidentally upon physical exam, a firm lump was palpated on the cranial aspect the left testicle. Neuter was recommended by the examining veterinarian and performed a short time later. Both testicles were submitted to PDS for histopathologic examination.

Histopathologic examination revealed marked, mixed inflammation with multi-focal to coalescing granulomas and variable necrosis effacing the normal testicular tissue. In numerous locations, round, yeast-like organisms were observed, characterized by a thick, double-layered refractile capsule and a heterogeneous magenta protoplasm (see Fig 1).

Figure 1: Blastomyces sp yeast (Hematoxylin and Eosin stain, 100X)

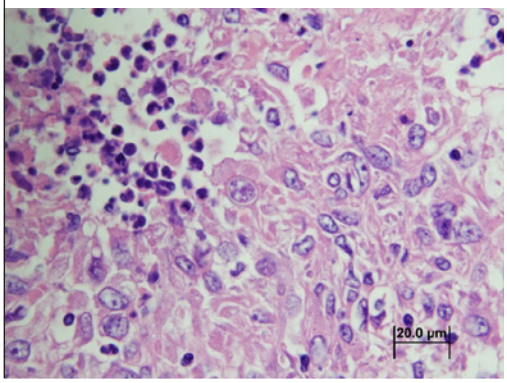
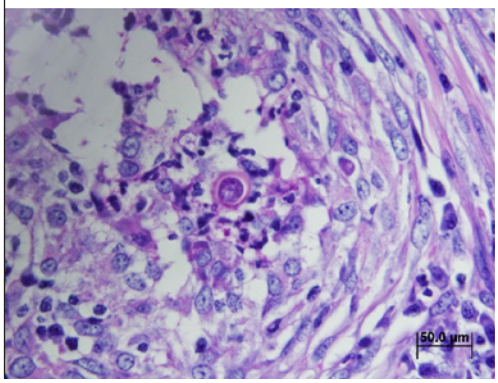


Figure 2: Blastomyces sp yeast (Periodic acid Schiff, 100X)



The morphological diagnosis was marked bilateral granulomatous orchitis consistent with systemic infection with *Blastomyces dermatitidis*. Histochemical stains for fungal organisms were also supportive of this diagnosis (see Fig 2).

CASE #2

A 1-year-old male Cane Corso presented to a primary veterinary clinic in south-central Saskatchewan in early July 2019 for examination of an enlarged right teste. The testicle was firm and sore; a small scab was present on the scrotum. Neuter was recommended by the examining veterinarian and performed promptly. The epididymis was reported to be thickened, with purulent fluid and a draining tract. The abnormal testicle and attached spermatic cord were submitted to PDS for histopathologic examination.

On histopathologic examination, the testicular tissue and epididymis exhibited multi-focal to locally extensive pyogranulomatous inflammation and necrosis. In rare fields, round, yeast-like organisms were present, characterized by a thick, double-layered, refractile capsule and a poorly defined heterogeneous basophilic protoplasm. The morphological diagnosis was marked pyogranulomatous orchitis and epididymitis consistent with infection with *Blastomyces dermatitidis*. Histochemical stains for fungal organisms were also supportive of this diagnosis. *Blastomyces dermatitidis* is an environmental fungus belonging to a group that also includes *Histoplasma capsulatum*, *Coccidioides immitis*, and *Sporothrix schenckii*. In Canada it is historically acknowledged to be endemic in specific regions of southern Quebec, Ontario, and Manitoba. However, a recent retrospective report concluded that *B. dermatitidis* should be considered endemic in southern Saskatchewan, particularly along the Qu'Appelle and Assiniboine river systems.[1] It is not uniformly distributed however, instead found in widely dispersed 'hot spots'.

B. dermatitidis is a dimorphic species. The *mycelial* form grows optimally at 22°C - 25°C within wooded areas with sandy acidic soils that are in close proximity to water sources. They produce infectious spores (also called conidia) that once inhaled, are rapidly taken up by lung macrophages.[2] In the lung they transform into larger round *yeast* that are resistant to destruction, and instead proliferate asexually at body temperature (38°C) to cause pneumonia. The organisms can then spread to other organs via blood and lymphatic vessels. Lymph nodes, skin, eyes, and bone are reported to be the most common extra-pulmonary sites of infection, but numerous other tissues can be involved. Opportunistic infection with *B. dermatitidis* is most prevalent in young immunocompetent dogs, but it has been documented in cats, humans, and numerous other terrestrial mammalian species. The incubation period may vary from weeks to months. Clinical signs are usually acute or subacute, non-specific, and variable depending on the extent of organ/tissue involvement. Peripheral lymphadenopathy is common, and a large majority of dogs present with lung lesions that may or may not be manifest clinically. Hematology and biochemistry results are also non-specific but non-regenerative anemia, leukocytosis (with or without a left shift), and hyperglobulinemia are common. Hypercalcemia of granulomatous disease is an inconsistent finding.

Pre-mortem diagnosis of *B. dermatitidis* infection via serology can be very frustrating due to a lack of sensitivity. A more recently developed urine immunoassay for fungal cell wall antigen is highly sensitive for active disease but cross reaction with similar fungal agents such as *Histoplasma sp.* can occur.[4] PCR can be performed on fixed or unfixed samples to confirm. Cytology and histology of affected tissues is recommended where possible, and often diagnostic. Histology can

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be particularly fruitful when ancillary stains specific for fungal organisms are applied (Periodic acid–Schiff and/or Gomori Methenamine-Silver Nitrate). Culture is very helpful for a definitive diagnosis but rarely pursued due to significant risk of human (zoonotic) infection. Indeed, it bears repeating that although the yeast form of *B. dermatitidis* is not considered transmissible (and thus infected animals are not considered contagious), the mycelial form that grows in culture is highly infectious.

For intact male dogs, the testes and epididymis are important and often overlooked as potential nidi of disseminated *B. dermatitidis* infection. In one retrospective study, testicular lesions were observed in 17% of male dogs with blastomycosis.[5] Clinical signs may or may not be present. Testicles can be swollen, painful, or misshapen with or without accompanying scrotal lesions. Complete urogenital evaluation is recommended for intact male dogs with *B. dermatitidis* infection. Importantly, the testes and/or epididymis can become a source of persistent clinical infection that may confound attempts at antifungal treatment.[6] **Blastomycosis should be a differential diagnosis in any dog with testicular or epididymal lesions in geographic locations where the fungus is endemic.**

References:

1. Davies JL, Epp T, and HJ Burgess. 2013. Prevalence and geographic distribution of canine and feline blastomycosis in the Canadian prairies. Can Vet J 54: 753-760.
2. Caswell JL and KJ Williams. 2016. Respiratory system IN Maxie MG (Ed.), Jubb, Kennedy, and Palmer's Pathology of Domestic Animals, 6th Ed: Volume 2 (pp. 580-581). St. Louis, Missouri: Elsevier.
3. Legendre AM. 2012. Blastomycosis IN Greene Infectious Diseases of the Dog and Cat, 4th Ed. (pp. 606). St. Louis, Missouri: Elsevier.
4. Foy DS, Trepanier LA, Kirsch EJ, and Wheat LJ. 2014. Serum and urine Blastomyces antigen concentrations as markers of clinical remission in dogs treated for systemic blastomycosis. J Vet Intern Med 28: 305-310.
5. Arceneaux KA, Taboada J, and G Hosgood. 1998. Blastomycosis in dogs: 115 cases (1980-1995). J Am Vet Med Assoc 213(5): 658-664.
6. Totten AK, Ridgeway MD, and DS Sauberli. 2011. *Blastomyces dermatitidis* Prostatic and Testicular Infection in Eight Dogs (1992-2005). J Am Anim Hosp Assoc 47: 413-418.

Vitamin A Results in Bovine Fetuses and Stillborn Calves at PDS from June 1, 2014 – June 1, 2019

By: Shelagh Copeland, Veterinary Pathologist, PDS

During what we commonly call "abortion season" earlier this year I was surprised at the number of calves that were deficient to marginal for hepatic Vitamin A. The calves did not have the squamous metaplasia of the parotid salivary gland ducts however, which is reported to be specific for vitamin A deficiency. How then to interpret the result for the referring veterinarian? Do we have adequate normal value ranges for this age group?

To help understand the issue, the Vitamin A results from our laboratory were compiled on bovine fetuses and stillborns. Results were only used on those animals we felt reasonably confident were true abortions or stillborns. This is important as colostrum is very rich in Vitamin A and any ingestion would skew results. Those that were near term and had breathed or with unclear histories were not included. This lowers the total numbers but it supports ~ 38% of fetuses and stillborns can reach what has been considered normal values.

We still need more research in this area but there is a concern low Vitamin A can be a factor in abortions, stillbirths and weak calves or a warning sign there is something lacking in the ration that underlies the problem. **Now is the time of year to assess body condition in the herd, sort animals and ensure the winter feed program will produce strong healthy calves.** Feeds can be analyzed for protein and energy. Vitamin A levels could be assessed by taking serum from a few representative cows (3-5) and having it analyzed. One could also consider other analyses including Vitamin E. Provincial government livestock specialists are often available to help formulate adequate least cost rations. Injectable products may be available but they should only be used when necessary and in consultation with a veterinarian. If losses do occur and laboratory submissions are made please try to provide information on estimated gestational age and if colostrum was given. This will help us in interpreting the results on the animal submitted and on reviews such as this.

Bovine Fetus and Stillborn Hepatic Vitamin A Results
June 1, 2014 – June 1, 2019

YEAR	DEFICIENT	MARGINAL	NORMAL	HIGH NORMAL	TOTAL
2014	0	1	1	0	2
2015	15	12	18	0	45
2016	23	22	35	0	80
2017	16	12	36	0	64
2018	38	23	22	0	83
2019	20	15	13	0	48
Grand Total	**112**	**85**	**125**	**0**	**322**

READERS' FEEDBACK

The **Animal Health Perspectives** editorial team (Dr. Moira Kerr, Brian Zwaan and Kathryn Tonita) invite readers' comment on 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.