



Animal Health Perspectives

Changes to the PDS Board of Directors:

By: Yanyun Huang, Interim CEO, PDS

I am pleased to announce that Drs. Grant Maxie, William Murphy, Susan Cork, Nancy de With and Trent Wennkamp joined PDS's advisory Board, effective September, 2019. They will contribute valuable experience and expertise in the areas of diagnostic laboratory management, surveillance, clinical practice and marketing to the PDS Board

of Directors. The current PDS Board chair is Dr. Wayne Lees and vice chair, Dr. Julie de Moissac. With the Board's guidance, PDS will continue to provide client-focused veterinary diagnostic services and support veterinary teaching and applied research. PDS is actively developing new diagnostic platforms and tests

to meet the needs of animal health in western Canada. We are saddened to bid farewell to Drs. Craig Dorin (out-going PDS Board chair), Jan Bystrom and Chris Byra who are stepping down from the Board. They all dedicated a significant amount of time and effort to the governance and direction of PDS, and were vital in the success of

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PDS over the past several years. Their presence on the Board will be greatly missed.

The Impact of Trace Mineral Status on Prenatal and Postnatal Beef Calf Disease

By: Dr. Barry Blakley, Veterinary Toxicologist, WCVM

Trace mineral deficiencies have been associated with fetal, neonatal and postnatal disease in beef calves in Western Canada. In most herd investigations, substantial losses frequently cannot be attributed to a simple mineral deficiency. Essential trace mineral panels are available through diagnostic laboratories like Prairie Diagnostic Services. The majority of the investigations occur during the traditional calving season. Trace mineral evaluation of animal tissues is often part of the initial investigative strategy. The evaluation should focus on the

affected animal population and not on individual animal testing. With most minerals, liver analysis is preferred over blood analysis. In live animals, liver analysis is not a practical option. Multi-element analysis of the tissues enables the veterinarian to assess individual minerals plus metal-metal interactions or metal-vitamin interactions, which may be biologically and clinically relevant.

Interpretation of prenatal and postnatal data in calves is a challenge. Adult normal values are reasonably well defined, but trace mineral uptake,

prenatally and postnatally, plus corresponding metal metabolism often shift dramatically at birth and for the first few weeks of life. For example, copper metabolism and kinetics impacting on tissue distribution are immature at birth. Extrapolation from other livestock species is extremely unreliable and should not be attempted. The mineral requirements are highly variable with developmental age. Deficiencies ultimately impact on morphology, growth and function. Consequently, the disease manifestations over time relate to developmental

age for specific metals are variable. As a result, "normal values" for individual metals vary dramatically with developmental age. Normal values, consequently, may vary considerably from reference to reference. The selenium, copper, manganese, molybdenum, iron and magnesium status may vary with age. The variability is also influenced by the status of the cow, supplementation practices, season (most minerals increase during the summer months), water quality, metal interactions, colostrum

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consumption, dehydration, and infectious disease. In most instances, reliable data on these factors is unavailable, further compromising data interpretation. For some metals, these factors are extremely relevant clinically. For other metals, the factors have limited significance. Therefore, it is difficult to make broad generalizations that apply to all metals in every situation.

Two metals that are often problematic are copper and selenium. Normal reference values are highly variable. This variability compromises interpretation. The copper-molybdenum interaction is a prime factor influencing the variability. Age-related effects on metabolism and absorption are additional confounding factors. In general, neonates have a higher copper concentration as compared to earlier developmental stages or adults. The impact of adult supplementation on the developing fetus may require months to raise fetal concentrations. In the case of selenium, another important, often deficient element, similar factors impact on the variability. Supplementation of the pregnant cattle take many weeks, possibly 6 weeks, to

raise the fetal concentration to normal. Consequently, supplementation in late pregnancy may have little or no impact on the fetal viability. The late pregnancy supplementation may raise colostrum concentrations. Another major consideration is the vitamin E-selenium interaction. Both nutrients have antioxidant properties, but there is an absolute requirement for each nutrient. Consequently, a high vitamin E status may partially mask a selenium deficiency. It is important to evaluate concentrations of each nutrient. If selenium and vitamin E status are classified as marginal, white muscle disease or immune dysfunction may be present. In some instances, significant muscle degeneration may be detected histologically, yet the vitamin E and selenium status were distinctly in the normal range. In many of these cases, supplementation in late pregnancy restored the status to normal, but the muscle degeneration that occurred prior to supplementation remains. In general, the selenium status of the neonate is about 2-3 fold higher than adult cattle. Over supplementation of selenium is another concern. Excessive postnatal injections may also result in degenerative change and death.

Manganese concentrations in fetal and neonatal tissues are typically about 0.6-0.7 fold less than corresponding adult animals. Manganese is not stored to any great extent in the fetal liver. Supplementation of the cow may have limited impact on the fetus. The risk of low manganese concentrations in the fetus is not well defined. Low manganese concentrations in the fetus or neonate are frequent observations in clinically "normal" animals.

The iron status related to developmental age is highly variable. Interpretation again remains a challenge. A low iron status does impact on immune function and potentially on in utero development. Frequently, in an abortion case or neonatal death, the iron status is considered to be high to normal. For many, this may be considered to be over supplementation. This in fact is not the case. The animal senses an infectious organism in the body. The physiologic cytokine response is to "hide" the iron in the liver and "starve" the microorganism to enable the immune system to destroy the invader. This same physiologic response is frequently observed with liver zinc status. If you observe high normal iron and zinc concentrations, infectious agents are likely a factor on the

clinical syndrome. Examination of blood samples from live calves will demonstrate "deficient" iron and zinc concentrations. The metals have been sequestered physiologically in the liver. The degree of "deficiency" in live animals may be a useful indicator of prognosis.

Tissue concentrations of zinc are easily measured, but are highly variable for physiological and kinetic reasons. Animals under stress or subjected to limited food intake for only 1-2 days may appear distinctly zinc deficient. Tissue analysis is a poor indicator of chronic zinc status. Interpretation of the values should be made with considerable caution. True zinc deficiency in cattle is a rare event.

Occasionally, the analysis of a fetal or neonate liver for trace metals indicates that virtually all metals are deficient. The simple interpretation is the maternal diet is inadequate for all metals. Practically, this is highly unlikely. The probable interpretation is placental insufficiency. All of the nutrients have a severely compromised ability to cross the placenta. Growth retardation may also be evident. The placental damage is often associated with an infectious agent or mycotoxin exposure.

In summary, age-related effects on mineral metabolism, absorption, excretion through nutrient interactions, physiologic disturbances or placental insufficiency compromise interpretation of diagnostic data. Consequently, there is wide variation in normal values. Investigations should emphasize population assessment and not individual animal testing. As the population concentrations decline, the risk of disease and mortality increase. **There is no distinct concentration for each metal that is considered to be a diagnostic certainty from an interpretation perspective.**



Do you know who to call for animal-related human health support?

Complaint	Contact
Animal noise	Animal control/RM office
	Local police agency
Dangerous dog/bite injury	HealthLine 811
	Rabies Hotline (1-844-772-2437)

saskatchewan.ca/livestock



Perinatal Beef Calf Morbidity and Mortality

By: Dr. Barry Blakley, Veterinary Toxicologist, WCVM

During the late winter and early spring months in Western Canada many beef calves fail to thrive. In spite of intervention by producers and veterinarians, this multifactorial disease problem remains a major source of frustration and economic loss. From year to year, the extent of the problem may be highly variable. Factors including trace mineral and vitamin status, adequate nutrition, infectious disease, management and environmental conditions are potential causes. Many of these factors play a major role well before parturition. Diagnostically, it is often impossible to identify a single cause. Simple analysis of animal feed or tissue to assess health concerns may not be useful in many instances, the insult may have occurred during gestation. At the time of the birth, the window of opportunity to confirm the suspected etiology may have passed.

Broadly speaking, etiologies may be considered under four major categories. These include: genetic, environmental, infectious and nutritional. The investigative strategy from a herd perspective often include: define the abnormality (abortion, stillbirth, malformation, and weak calves), recognize the patterns involved related to temporal, geographical, age, spectrum of the abnormality, pathology, clinical disease etc. Finally, the suspected diagnosis should be confirmed with diagnostic

testing. The nature and extent of the testing can often be time consuming, expensive and unrewarding. **In many instances, in spite of the most extensive testing, the etiologic agent may only be identified in 30% of the herd investigations.**

In most instances, genetic causes in cross-bred populations are unlikely. The typical strategies focus on other causes. Environmental factors need to be considered broadly. The environment may be divided into external and internal (maternal) factors. During early gestation or prior to conception maternal nutritional status is important. Vitamin, mineral, energy and protein components are critical for optimal fetal development. Often at birth, tissue concentrations of nutrients or biochemical alterations have returned to normal, but the tissue abnormalities from the previous nutritional state persist. Depending upon the dose and duration of exposure, the manifestation may be variable. The spectrum of abnormalities may be diverse/broad in nature. Growth retardation is a frequent observation. Since the majority of fetal growth occurs in the last trimester, small calves are often associated with late gestational causes. Placental insufficiency or damage, lack of proper nutrition or exposure to toxins such as mycotoxins are potential causes. Functional disturbances such as immune dysfunction

may be related to immune maturation in late pregnancy or organ development during the first trimester. Micronutrient deficiencies related to minerals or vitamins impact on normal immune system development or maturation. Micronutrient uptake and metabolism in the neonatal calf shift dramatically at birth often compromising interpretation of diagnostic data associated with nutrient states well before parturition occurs.

Many producers elect to provide minerals or vitamins to pregnant cattle just prior to calving. If the cows have nutrient deficiencies early in pregnancy, late supplementation may have little impact on the calf viability. It is well known that colostrum ingestion at birth has a major impact on infectious disease. In addition, the colostrum contains clinically significant concentrations of other nutrients such as the fat soluble vitamins. Neonatal calves have a limited ability to respond to vaccines at birth. Consequently, it is essential that adult cows be adequately vaccinated. The impact on antibody concentrations in the colostrum is an obvious benefit.

Management and external environmental factors in the broadest sense are also important. At the time of birth, the contribution of these factors in terms of risk may be difficult to assess. Overall poor nutrition related

to drought or poor water quality are examples. Under drought conditions, vitamin and micronutrient content of maternal diets may be suboptimal, ultimately impacting the developing fetus. In recent years, poor water quality, particularly sulfate concentrations impacts the bioavailability of essential metals such as copper or selenium. Altered rumen function manifested by vitamin deficiency (thiamine) or impaired calorie/protein metabolism or reduced feed consumption will impact the developing calf. Overcrowding in winters pens and untimely extreme environmental conditions are other potential factors. Interactions among multiple factors are difficult to quantify, but abnormal calf health increases as the number of varied risk factors increase.

To minimize the occurrence of perinatal calf disease producers should be encouraged to assess the nutritional status of the dam well before parturition, adequately vaccinate and maintain optimal overall nutrition and good quality water. Dams with a suboptimal nutritional status must be supplemented accordingly. The impact on calf morbidity at birth related to colostrum intake and overall nutrition and immune function will be evident. A multifaceted strategy prior to pregnancy, during pregnancy and after parturition are critical to minimize this complex disease syndrome.

CFIA Brucellosis Pilot Project announcement



In February 2020 the Canadian Food Inspection Agency (CFIA) will initiate a pilot project to strengthen Canada's brucellosis surveillance program in support of Canada's claim of freedom from brucellosis. This pilot project is being conducted collaboratively by the CFIA and participating Canadian Animal Health Surveillance Network

(CAHSN) laboratories including Prairie Diagnostic Services. The project will involve testing samples from select bovine abortion cases submitted to the CAHSN labs.

Please find a description of the pilot project from CFIA on PDS's website, <http://www.pdsinc.ca/Home.aspx>:

Bovine Brucellosis Abortion Screening Pilot Project.

Information about the project will also be communicated to regional veterinary associations (e.g., WCABP), surveillance network groups (e.g., WeCAHN), and producer organizations.



Congratulations:

Please join us in congratulating our newest Diplomate of the American College of Veterinary Microbiologists, Dr. Kazal Krishna Ghosh. Dr. Gosh achieved board certification in Bacteriology/ Mycology. Kudos to Kazal for all his hard work and commitment!

This is an important achievement in Kazal's career as well as a significant contribution to PDS in fulfilling our mission to deliver world-class, client-focused veterinary diagnostic services to animal owners, their veterinarians, educators, and researchers.

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.