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Precautions for Handling Heat Stress in Cattle

Larry C. Hollis, DVM, M.Ag.
Extension Beef Veterinarian

During the hot summer months, have a plan for how you will handle heat stress in your cattle. High daytime temperatures rarely cause problems alone, but high humidity combined with elevated temperatures can create problems for cattle. Primary factors of temperature and humidity are compounded by secondary environmental factors, including several consecutive days of high temperatures, lack of nighttime cooling below 75°F, lack of shade, lack of cloud cover, lack of wind, lack of air movement within pens, or grazing endophyte-infested fescue pastures. When primary and secondary factors are combined with animal-related factors such as dark hides, heavy body weights, or advanced pregnancy, the situation can rapidly become deadly.

Signs of heat-related distress in cattle during hot humid weather include going off feed, standing in ponds or with heads over the water trough, standing on the highest point in the pen or pasture trying to catch a breeze, panting, salivation, or open-mouth breathing. Working cattle so all handling is completed by mid-morning, or better yet, postponing all gathering or handling procedures until after the critical heat period has passed, are management procedures that will reduce heat stress. Ready access to abundant cold water is essential. Access to shade and the ability to move away from structures such as solid fences or barns that reduce air flow should also be considered. Sprinklers that provide large drops of cold water can provide relief, but they may only increase the humidity problem if they do not wet the cattle's skin thoroughly. Cattle should have the freedom to stand under the sprinklers as needed, and then move to a dry area where evaporation will help cool their bodies.

University of Nebraska researchers have developed a temperature-humidity index to help producers anticipate when heat stress will become a problem. When there is no daytime wind or nighttime temperatures do not drop below 75°, and conditions reach an index score of 75, producers should be on the alert for heat

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Blue-Green Algae in Farm Ponds

Kansas practitioners may have been asked about blue-green algae, especially over the past two summers in drought-stricken areas. A K-State Research and Extension fact sheet, *Identification and Management of Blue-Green Algae in Farm Ponds*, MF3065, offers practical information about the organisms and toxins involved, sampling to test for the organism, and management options to reduce disease or death of cattle, pets, and wildlife that have been exposed to the harmful bacteria.

The publication, co-authored by Deon van Der Merwe, veterinary toxicologist, Carol Blocksome, agronomist, and Larry Hollis, beef veterinarian, is available at http://www.ksre.ksu.edu/bookstore/pubs/MF3065.pdf

Blue-green algae rapidly reproduce and may form blooms, or large colonies, that are visible as scum on the water's surface.

Also in this issue

Vitamin A Deficiency Linked to Perinatal Calf Mortality

Jerome C. Nietfeld, DVM, Ph.D.,
Gordon Andrews, DVM, Ph.D.,
Bhupinder Bawa, DVM, Ph.D.
Veterinary Diagnostic Laboratory

This spring the diagnostic laboratory received an unusually large number of calves that were aborted late in the third trimester, stillborn, or born weak and died within 24 hours of birth. In most cases no evidence of infectious disease or cause of death was identified. One week the lab received four term or near-term calves and placentas from a group of 160 heifers. Of these, about 10 calves had been born dead or died within a few hours. Two had been previously submitted. All calves were small and thin, weighing less than 50 pounds. One had moderately severe internal hydrocephalus, but all test results were negative and there was no evidence of an infection. Livers of calves found to be negative for trace minerals were then submitted to the Michigan State Diagnostic Center for Population and Animal Health (MSU) for vitamin A and vitamin E analysis. Results showed vitamin E and trace mineral levels in samples from the first few calves to be adequate, so testing for them was discontinued. Vitamin A levels were shown to be below the MSU reference range (Table 1), leading to analysis of additional livers.

Hepatic Vitamin A Results

The MSU reference range for fetuses is 8-40 µg/g, and for neonates it is 50-200 µg/g. Carotene, the source of vitamin A in green feed, is poorly transported across the placenta. Vitamin A stores are normally low at birth, and neonatal animals rely on an adequate supply from colostrum. Consequently, levels in calves that have nursed should be much higher than in fetuses. Although the MSU reports did not give a specific value to differentiate marginal from critically low levels, vitamin A values of 4-8 µg/g from calves that had not nursed were interpreted as being "…marginal for fetuses" and values of less than 4 ug/g as "…critically low for fetuses. Values in this range are consistent with abortion due to Vitamin A deficiency." Other sources give similar reference values. The book *Vitamin Levels in Animal Health*[1] gives the adequate range for vitamin A in bovine fetuses as 5-15 µg/g and for neonatal calves as 50-125 µg/g, but does not specify critically low levels. According to *Veterinary Medicine, 9th edition*, the critical hepatic level at which clinical signs are likely to appear is 2 µg/g.[2]

Using the MSU values, of 21 livers from calves that had not nursed, one was adequate, nine were marginal, and 11 were critically low (Table 1). The vitamin A level for a 1-day-old calf that had nursed was 8.16 µg/g, which is adequate for a fetus or a calf that has not nursed, but if the interval between nursing and death was sufficient to allow absorption

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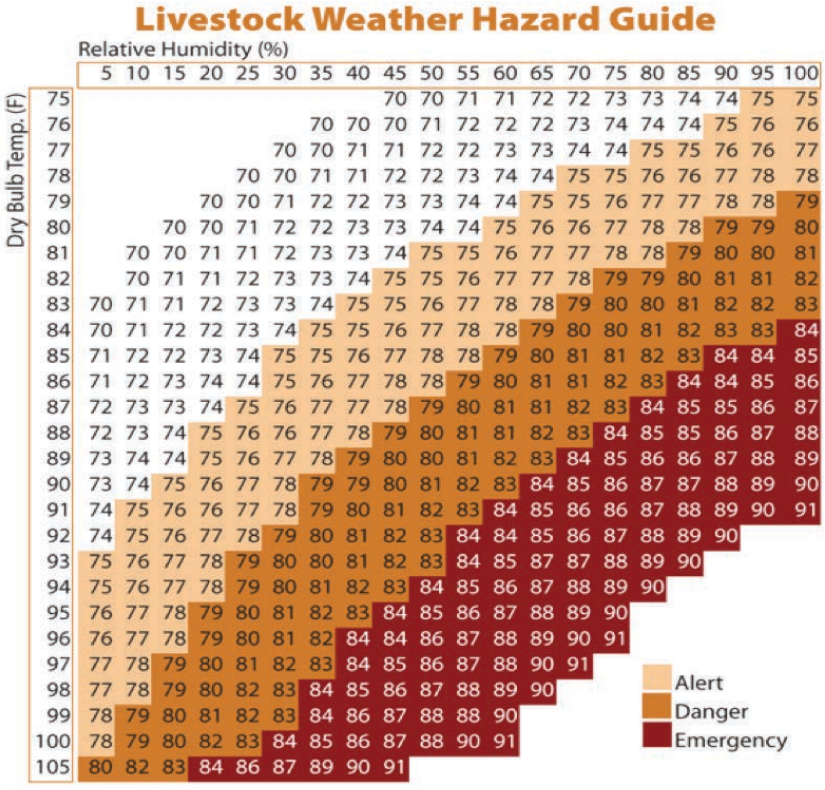
stress problems. When the index reaches 79, the situation is dangerous. When the index reaches 84, emergency conditions exist. If the index stays above 84 for three days in a row, deaths usually start to occur, especially if the wind suddenly stops blowing.

Panting scores probably give the best visual method to estimate the severity of heat stress on cattle: 80 to 120 breaths per minute = moderate; 120 to 160 = danger; and more than 160 = emergency. If signs of moderate heat stress are seen, producers may have a very short time to provide a mechanism for cooling the cattle before the situation becomes life threatening.

Listen to or watch radio, television, or electronic media programs that present heat index information for humans. When it is high for humans, it is also high for cattle. Both people and cattle should be taken into consideration.



Source: University of Nebraska-Lincoln Extension

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of vitamin A, the level is deficient. The values for three 6- to 7-day-old calves were 13.74, 15.88, and 86.9 µg/g. 86.9 µg/g is adequate and was from a calf with clinical signs of "bovine hysteria" or "bonkers," and whose mother was being fed ammoniated feed. Reports for the 6- to 7-day-old calves with values of 13.74 and 15.88 stated that with adequate intake of good quality colostrum, hepatic values should have been near or above the reference range for neonatal calves (50-200 µg/g). Also included in the table is a sample from a 650-pound bison with a hepatic vitamin A level of 171 µg/g. MSU did not have reference values for bison, but stated that reference values for adults of other species are greater than 300 µg/g.

In growing cattle, males are more likely to develop clinical signs of vitamin A deficiency than females. In our cases, 18 of the 20 calves for which the sex was specified were male, and in two herds where multiple calves had been affected, the history indicated that only bull calves were affected.

Is vitamin A deficiency a cause of perinatal calf mortality?

Yes. The literature indicates that vitamin A deficiency can cause abortions, stillbirths, weak calves, and increased neonatal deaths.[1,2] In utero vitamin A deficiency can restrict growth of the bones of the skull decreasing the size of the cranial cavity resulting in herniation of the cerebellum, decreased diameter of the optic foramens, leading to blindness, and increased cerebrospinal fluid pressure. It also decreases absorption of cerebrospinal fluid leading to hydrocephalus, both prenatal and postnatal. Vitamin A deficiency can reduce fertility in bulls and cows and increase retained placentas. In a 2009 report of congenital vitamin A deficiency from Queensland, Australia, 168 of 406 (41.4%) calves were born dead or, more commonly, died within 48 hours of birth.[3] The hepatic vitamin A levels in the calves that were analyzed

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Table 1: Hepatic Vitamin A Results from the Michigan State Diagnostic Center for Population and Animal Health

	Case #	Sex	Vit Aµg/g	Interpretation	Comments
1	103264	M	11.66	Critically low	20 calves from 160 heifers. Small (< 50 lbs), 2-4 weeks early to term. Some stillborn, some breathed but not seen alive or die within 1 day. Mild hydrocephalus in one
	103378	F	5.31	Marginal	
	103965a	M	4.43	Marginal	
	103965b	?	1.66	Critically low	
2.	106256	?	2.19	Critically low	4-6 calves 3-4 weeks premature. Neutrophilic pneumonia
3	106329	M	2.26	Critically low	Premature
4	106412	F	7.65	Marginal	1 day old. Large calf born to heifer. Ok at birth, comatose next day. No milk in abomasum.
5	106818	M	0.89	Critically low	Multiple stillborn/weak calves. Both—lungs inflated. Sera from 5 cows in adequate range
	107003	M	3.82	Critically low	
6	107017	M	4.33	Marginal	Died at birth. Did not nurse
		M	8.16	??	1 day old. Nursed. Interpretation depends on colostrum intake and degree of absorption
7	107272	?	11.37	Adequate	Term, found dead. Third in 3 days.
8	107274	M	7.23	Marginal	8 stillborn in 1 pasture; none in other pastures
9	107382	M	3.95	marginal	Found dead, lungs inflated. 5th of 25 calves stillborn/perinatal death. **All male**.
10	108000	M	1.22	Critically low	Found dead, lungs inflated. 4th stillborn/perinatal death.
11	108014	M	3.38	Critically low	Weak/stillborn calves. Second submitted
12	108019	?	<0.67	Critically low	Found dead, lungs inflated. 6th in herd
13	108023	M	6.02	Marginal	3 abortions, 3 born alive and die in a few hours
14	108226	M	<0.86	Critically low	About 400 cows, 25-30 calves born early and died
15	108647	M	4.90	Marginal	Stillborn
16	109122	M	4.27	Marginal	1 day old, ~20/200 calves stillborn or die in 1-2 days
17	109759	M	86.93	Adequate	1 week old. Bovine hysteria (bonkers)
18	109641a	M	6.20	Marginal	Stillborn
	109641b	M	13.74	Deficient	7 days old
					~20% stillborn or weak and perinatal deaths. All male.
19	111672	?	15.88	Deficient	6 days old. Fed milk replacer. 6/80 calves small, weak, die.
20	107275	?	171.4	Deficient	650 lb bison. Abscesses in lung, trachea. Pleuritis, pericarditis, Mycoplasma bovis positive

MSU reference ranges: fetus = 8–40 µg/g; neonate = 50–200 µg/g; adult = 300–1000 µg/g

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ranged from undetectable in a calf dying at 12 hours of age, to 8.6 µg/g in one dying at 48 hours of age.

Was vitamin A deficiency responsible for late-term abortions, stillbirths, and neonatal deaths this past spring?

The honest answer is "We do not know with certainty." Vitamin A is the vitamin deficiency most likely to occur in cattle, and the conditions in 2012 and 2013 were conducive for deficiency to occur.[2] Cattle at pasture normally receive adequate vitamin A because green forage is a good source of carotene. However, when the plant dies or begins to dry up, sunlight begins to oxidize and degrade carotene. Similarly, carotene is degraded during storage, with the level decreasing faster in hay than silage. Except for yellow corn, grains are not a good source. Consequently, cattle maintained on dry pasture during a drought, fed stored forage for prolonged periods, and/or fed high concentrate diets are subject to deficiency unless supplemented. [2,3,4] It is common for cattle to not receive adequate vitamin A during winter months, but normally they have sufficient hepatic stores to maintain them without problems. Almost all of Kansas was extremely dry during 2012, and large parts of the state were in drought conditions for 2011-12, increasing the time cattle were on dry pasture and consuming stored feeds.

Warnings that drought conditions can lead to increased incidence of vitamin A deficiency and perinatal calf loss were issued by extension personnel at the University of Arkansas[4] and Oklahoma State.[5] In the spring of 2013, Iowa State Extension and Outreach released a bulletin saying that because of the drought they were seeing increased stillborn and weak calves due to vitamin A deficiency and producers should be supplementing cows and newborn calves.[6] As early results began coming in, Dr. Gordon Andrews and a submitting veterinarian recalled that following the last drought the number of aborted/stillborn calves shot up.

The MSU results indicate that most calves were vitamin A deficient with about half of them critically low, so it would be easy to say vitamin A deficiency was the culprit. However, we did not hear reports of signs of vitamin A deficiency in the live calves in affected herds, as is usually the case.[2,3] In the case from Australia, many calves displayed central nervous system signs and/or were blind. Microscopically there were degenerative changes in the stalk of the optic nerves where they entered the brain, but not the eye, and there was squamous metaplasia of the epithelium lining interlobular ducts in the parotid salivary gland, which is reported to be specific for vitamin A deficiency. We did not find any of these lesions, but only a few calves were examined for these changes so their absence does not rule anything out. Finally, the veterinarian for a herd with two calves with critically low vitamin A bled five cows, one of which had a dead calf, and all serum vitamin A levels were normal. Taken on an individual basis, serum is not a reliable indicator of vitamin A status, but if multiple samples are analyzed they should provide an accurate picture of a herd's status[1]. It seems reasonable that if vitamin A deficiency was a real problem at least one cow should have been deficient.

Vitamin A deficiency may not have been the sole cause of the perinatal deaths, but it could be a warning sign for general under nutrition, which is recognized as a cause of decreased birth weight and viability in humans and animals, especially if it occurs in the third trimester.[7,8] Vitamin A is also important for the health of neonates. In a study of the effects of different levels of vitamin A in milk replacer, calves with decreasing vitamin A levels experienced significantly more days of fever and diarrhea. In parts of the world, vitamin A deficiency is an important problem in human infants, and preventing it significantly decreases infant mortality.

Even with normal reproductive performance, supplementation of vitamin A is important in times of drought and during the winter. When supplementing keep in mind that heat, sunlight, high humidity, salt, and trace minerals all degrade vitamin A.[1,2] In one study 50 to 90 percent of the vitamin A in commercial supplements was destroyed in a week by exposure to high humidity, high temperature and trace minerals. Supplements containing trace mineral should be put out fresh every week or two, especially in the summer because heat and high humidity are the most important factors in degradation of vitamin A.

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Studies Suggest Mastitis is Not Just a Dairy Issue

Cathy Bandyk, Nutritionist,
Quality Liquid Feeds

As beef producers, we don't spend a lot of time talking about mastitis. But maybe we should. Milk production can explain more than 60 % of the variation seen in pre-weaning calf gains, and research has shown that each 1-pound increment in daily milk production correlates to more than 7 pounds of weaning weight. Mastitis infections reduce milk yield, and, in turn, can knock weaning weights 7–12%, or even more.

The Basics

Mastitis is, by definition, inflammation of the mammary gland. While these infections can be the result of an injury, virtually all cases are due to infectious agents such as staphylococcus and streptococcus bacteria. Mastitis is classified by severity into four classes:

- Peracute – swollen, hot, red udder; fever, depression, weight loss, depressed appetite;
- Acute – severe udder inflammation; some fever, mild depression;
- Subacute – less pronounced udder symptoms; cows do not appear sick;
- Subclinical – no visible signs or symptoms, but infectious agents present.

The bacteria responsible for mastitis are widely distributed in the environment. Cows can come in contact with them through bedding, on pastures, in dirt lots, from other cows through cross-suckling by calves, and from flies. Research has shown that problem bacteria, such as *Staphylococcus aureus*, can already be present in the teat canals of heifers prior to their first lactation.

Factors often associated with mastitis include wet and muddy conditions, confinement settings, nutritional stress (and resulting impacts on immune function), teats that are wide and flat-tipped, older cows (since pendulous udders are more prone to physical injury and contamination), and significant fly populations. Biting flies, such as horn flies, can actually drive infectious bacteria into mammary tissue when feeding. Open sores on udders, due to multiple fly bites, are obvious sites for establishment of infections. And virtually all flies can physically carry disease organisms onto an animal.

A common indicator of mastitis is somatic cell count (SCC). White blood cells known as leukocytes constitute the majority of the somatic cells in question. The number of somatic cells increases in response to pathogenic bacteria like *S. aureus*. Typically, values of less than 100,000 cells/ml would be considered uninfected, while cows with greater than 300,000 cells/ml are infected with significant pathogens.

How Big is the Problem?

Several research projects have evaluated the prevalence and significance of mastitis in beef cows and heifers, reporting infections in 7–54% of the animals involved. In a study at Louisiana State University, mastitis in heifers reduced weaning weights 23 pounds. This matches closely with a couple published veterinary case studies, which reported decreases of 31 ½ and 26 ½ pounds. Another study (Watts et al.) specifically tied *S. aureus* infections to a 42-pound depression in pre-weaning gains. In this group of cows, 37% of the animals (and 18% of all quarters) were infected with the organism.

Work at North Carolina State evaluated mastitis in a group of Simmental heifers, collecting and analyzing milk samples six times during their initial lactation. They found mastitis-affiliated bacteria in milk from about ⅓ of the animals. Using a SCC of 292,000 cells/ml as the dividing line, were grouped into "high" and "low" SCC groups. Elevated SCC levels were associated with a 16% reduction in milk production. At Oklahoma State, scientists working with Hereford and Hereford cross females also showed that mastitis negatively affects the nutritional composition of milk, reducing concentrations of butterfat, lactose, and protein. In this study, they found that 62% of the heifers were infected, and that this value jumped to to nearly 67% in cows that were nursing their fifth to ninth calf.

Mastitis also impacts reproduction. Israeli research showed that mammary infections can double the length of time it takes a cow to return to heat after calving. This is accompanied by lower levels of reproductive hormones, and reduced follicle function and oocyte competence.

Management Options

Researchers at Penn State tested the efficacy of an intramammary infusion of cephaprinbenzathine, given at weaning in hopes of reducing mastitis during the following lactation. While this protocol did eliminate existing infections in a majority of animals, it apparently did nothing to prevent new infections. This was somewhat surprising, since this is a proven practice with dairy cows, but the authors suggested the differing response was due to the extended length of the "dry period" in beef vs. dairy production systems. Regardless, this would probably not be a practical option in most beef cowherds.

In another Oklahoma study, intramuscular injection of antibiotics (oxytetracycline) at weaning and/or calving was evaluated as a mitigation tool for mastitis. Unfortunately, the drug was not effective. Regardless of treatment, 53.7% of cows were infected at weaning, and 43.4% were infected at their subsequent calving date. If one or two quarters were impacted, weaning weights dropped 22 pounds; if three or four quarters were infected, the resulting reduction was 56 pounds. The predominant bacteria was *S. aureus*, and its concentration relative to other infectious species increased with cow age.

This persistence of *S. aureus* infections was observed in both Angus and Brahman sired heifers in research done at the University of Arkansas. Bacterial infection was measured in teat secretions/milk collected during pregnancy and early lactation. If *S. aureus* was present in an early sample, it was almost always detected in milk samples taken later from the same quarter. They also showed that if a quarter was clean during pregnancy, there was a high likelihood it would remain so going into lactation. The authors made a point of comment-

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Diagnostic Tests Available for Porcine Epidemic Diarrhea Virus

Editor's note: In May, 2013, porcine epidemic diarrhea (PED) virus was diagnosed for the first time in the United States by the Iowa State University Veterinary (ISU) Diagnostic Laboratory and the National Veterinary Service Laboratory. Sequencing demonstrates that the virus is 99.4% homologous to a PED virus isolated in China in 2012, but no one knows how the virus entered the United States. The following is a PED fact sheet compiled by Dr. Kelli Almes of the Kansas State Veterinary Diagnostic Laboratory (KSVDL).

General Disease Facts

The presence of porcine epidemic diarrhea virus (PEDV) was confirmed in the United States on May 17, 2013. Porcine epidemic diarrhea (PED) is caused by a porcine coronavirus and results in vomiting and occasionally diarrhea in sows and gilts and severe diarrhea and vomiting in nursing and recently weaned pigs.

Clinical signs of PED are indistinguishable from the epidemic form of the disease caused by a different porcine coronavirus, transmissible gastroenteritis virus (TGE). There is no cross-protection between these two coronaviruses.

PED is a pig-only disease which does not affect other species or humans and is not a food safety concern. PED has been present in Europe and Asia since 1971 but has never previously been reported in the United States.

Within our naïve pig populations, morbidity will likely near 100 percent, and mortality may range anywhere from 50 to 100 percent in nursing pigs, but is reported to be 1 to 3 percent in fattening pigs and negligible in adults.

Incubation time is typically less than 36 hours and virus is shed in feces for up to 11 days. Laboratory diagnosis is required for definitive confirmation.

Producers should work closely with their veterinarian for sample submission and diagnosis.

Control and Prevention

Stringent biosecurity and disinfection procedures are the most effective for prevention and control. PEDV is susceptible to chlorine bleach, Virkon S, Tek-Trol, and 1 Stroke Environ. Thorough cleaning followed by disinfection and drying is recommended. Infection is mainly via the fecal-oral route, but short distance aerosol transmission is possible. The virus can remain stable in the environment and travel easily on boots, tires, vehicles, and other fomites.

Diagnostic Testing

KSVDL is performing virus isolation ($35.50) and/or PCR ($28.50) testing on feces and fresh intestinal samples. However, to date all laboratories are reporting that virus isolation is difficult and often yields false negative results, so PCR is recommended for initial diagnosis. An additional testing method, in situ hybridization, is in development and will soon be available. This test will be performed on formalin fixed intestine along with histopathologic examination.

Confirmed cases

ISU has been collating information from the diagnostic laboratories at Iowa State University, Kansas State University, South Dakota State University and the University of Minnesota and publishing it on the American Association of Swine Veterinarians' website. As of June 19, 2013, the four diagnostic laboratories have identified PED virus in samples from 197 farms in 12 states, including Kansas.

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ing on the widespread presence of dry scabs on the udders of these heifers, largely due to horn flies.

All of this highlights the value of mastitis prevention, starting with developing heifers.

Critical control points include:

- Adequate and balanced nutrition to support immune function;
- Good hygiene in facilities and pastures to minimize opportunity for udder contamination and to limit fly breeding sites;
- An integrated fly control program that targets all problem flies.

In today's market, all of these represent cost-effective investments in the health and productivity of the cowherd.

Use of a BVD Management Tool: BVD CONSULT

Bovine viral diarrhea virus (BVD) infection is responsible for a variety of economically important syndromes in beef herds. The economic losses from BVD infection will vary between herds based on herd immunity and stage of gestation at the time of exposure, the virulence of the BVD strain, and other factors. The virus is known to cause immune suppression, respiratory disease, infertility, and fetal infection. Fetal infection (infection of the fetus during pregnancy) can lead to early embryonic death, abortion, birth defects, stunting, or the birth of persistently infected (PI) calves.

Persistently infected cattle can result when susceptible pregnant cows are exposed to BVD virus during the first half of gestation and the virus passes from the dam to the fetus. Many times infected fetuses are aborted, but if a PI fetus survives to term, it will always have a tremendous amount of the virus in its body and cannot mount an immune response to clear the virus. A PI animal will secrete BVD virus throughout its life; in contrast to animals that become infected after birth that secrete the virus and are contagious for a few days to two weeks. These PI calves constitute the main reservoir and source of BVD virus for spread within the herd and to other herds of cattle.

Cattle persistently infected with BVD virus can be identified by a number of laboratory tests. Based on the NAHMS 2007-08 Beef Cow-Calf study, only 8.8 percent of U.S. cow-calf ranches had one or more PI animals identified; this means that one in every 11 to 12 herds have PI calves and most are not aware of their presence.

Vaccination programs can provide fairly good protection against BVD-induced disease when the exposure is from non-PI animals that are transiently infected with BVD. Vaccination programs offer some (but decreased) protection against BVD-induced disease when the exposure is from PI animals because of the tremendous amount of virus excreted by PI animals. Vaccination programs are an important component in BVD control, but will only offer a high level of protection if herd contact with PI animals is eliminated.

The cattle industry has made significant efforts in recent years to control BVD based on research that has provided a more complete understanding of the epidemiology of BVDV, enhanced availability of diagnostic tests for detecting PI cattle, and a better idea of the economic impact of BVD on cattle herds. Current knowledge of the epidemiology of BVDV, the availability of efficacious vaccines, and the improvement in diagnostic tools has made the control of BVD feasible.

BVD CONSULT is an internet-based tool, designed to aid in the development of BVD control programs for cow-calf herds. CONSULT is an acronym for collaborative, online, novel, science-based, user-friendly, learning, tool. The tool is the result of a multi-organization (AVC, AABP, National Cattlemen's Beef Association) effort to combine available BVD research into a user-friendly and organized format. The purpose is to develop BVD prevention and control programs for individual herds that emphasize key management decisions that impact the success of these programs.

BVD CONSULT provides bovine veterinarians with an opportunity to consult with their clients and develop BVD prevention and control programs for any cow-calf herd. For herds that currently have PI cattle present, the tool will help to create a plan to identify and remove the PI cattle and to establish a strategy to reduce the likelihood of the herd becoming infected again. For herds that are currently BVD-free, BVD CONSULT can be used while a producer and herd veterinarian decide how to minimize the likelihood of BVD virus entering the herd and to reduce the impact if the herd is exposed.

The questions used in BVD CONSULT include:

1. Do you have active BVD in your herd?
2. Will you institute a testing strategy that identifies all BVD persistently infected (PI) cattle and remove them from your herd? (This question is seen by positive herds only. Producers of positive herds who choose not to test for PI cattle skip to question 6.)
3. Will you quarantine and test all new cattle coming into your breeding herd?
4. Can you prevent fenceline and direct contact of your pregnant herd with other cattle?

   If they cannot prevent contact: Are the cattle that your pregnant herd will have contact with likely to be infected with BVD (high risk or low risk contacts)?
5. Will all cows calving in the same pasture calve in 90 days or less? (Can you prevent contact of newborn calves with cows that are less than 150 days of gestation?)
6. Will you use an appropriate BVD vaccination strategy on heifers?
7. Have the mature cows in your herd been appropriately vaccinated for BVD?
8. Will you use an appropriate BVD vaccination strategy on mature cows? (Appropriate BVD vaccination strategies vary depending on the previous vaccination status of the mature cows.)
9. Will you apply appropriate surveillance methods?

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BVD CONSULT is set up as a series of questions with responses and was designed to mimic a conversation between a veterinarian and a producer who is concerned about BVD. The tool works through a decision tree in order to provide recommendations that are specific to individual operations. BVD CONSULT asks if the producer is willing and able to perform specific management practices that will aid in prevention or control and eradication of BVD. More information is available in the tool to help with the decision making process. After clicking on "yes" or "no" to each question, an appropriate response is given based on the choices that have been made, followed by another question. The questions that are asked, and the responses given, vary depending on the previous answers. There are 6 to 10 questions in total depending on the choices made. A printable report is available at the end of the tool which records the choices that were made and the responses that were given.

BVD CONSULT was created for the beef cattle industry to enhance the control of BVD in beef cow-calf herds. This project was funded by an educational grant from Pfizer Animal Health and was produced by Brad White, D.V.M., M.S. (Kansas State University), Bob Larson, D.V.M., Ph.D. (Kansas State University), David Smith, D.V.M., Ph.D. (Mississippi State University), Daniel Givens, D.V.M., Ph.D. (Auburn University), Dale Grotelueschen, D.V.M., M.S. (Zoetis Animal Health), Richard Randle, D.V.M., MS (University of Nebraska-Lincoln), and Sherri Merrill, D.V.M. (Allen, Kan.).

BVD CONSULT, as well as many other BVD management resources, can now be found at *www.BVDinfo.org*.

"Bovine Bonkers" in Nursing Calves

Jerome C. Nietfeld, DVM., P.h.D.,
Veterinary Diagnostic Laboratory

Bovine bonkers, bovine hysteria, and ammoniated feed syndrome are terms for a hyperexcitability syndrome of cattle consuming ammoniated feed. [1,2] Clinical signs can include nervousness, involuntary ear twitching, rapid blinking, ataxia, salivation, polypnea, stampeding and running blindly, bellowing, and convulsions.[1,2]

Affected animals frequently run blindly into objects resulting in broken bones and even death. The onset is acute, and severity of clinical signs can decrease rapidly. After only a short period, affected cattle are sometimes calm, eating, and appear normal. Clinical signs usually disappear soon after removal of the ammoniated feed.

In some cases the condition develops in neonatal calves nursing cows consuming ammoniated feed. In these cases, the nursing cows are not clinically affected and it is believed that the toxin is transmitted from the feed through the milk. Mortality is typically higher in nursing calves than in adult or juvenile cattle. There is some evidence that the toxin in the feed is concentrated and excreted in milk, but it is also possible that calves are more susceptible to the effects of the toxin. This spring the lab has been presented with three cases of hyperexcitability in nursing calves whose mothers were consuming ammoniated forage. In two cases, a calf was submitted to the diagnostic laboratory and tests for other likely causes were negative. In the third case, a calf was not submitted, but bovine bonkers was very likely based on the clinical signs and history.

Ammoniation of forage helps improve palatability while increasing digestibility of protein and fiber, increasing nonprotein nitrogen content of roughage, and helping to prevent spoilage of high-moisture forage.[1] Because of a high incidence of hyperexcitability, initial attempts at ammoniation in the 1950s were discontinued.[1] Since the 1980s as investigators learned how to ammoniate forage without making it toxic, ammoniation of roughage has become increasingly popular.

The first rule is to only ammoniate poor quality roughage. Reducing sugars, such as glucose and fructose, can react with the ammonia to produce a number of imidazoles, one or more of which are thought to be responsible for the clinical signs. For that reason forages that have a high content of reducing sugars (such as green grasses or alfalfa hay, green cereal grain, mature cereal grain with the seed, or other high quality forage) should not be ammoniated. The toxin producing reactions are temperature dependent and ammoniation should only be done during cool weather.[1,2] In some studies, bovine hysteria only occurred in cattle fed ammoniated hay bales that had reached a temperature of 70 C.[2] The chemical reactions that occur during ammoniation are exothermic and that heat, plus heat from the sun shining on the plastic used to cover the forage, can increase the forage temperature up to 40 C,[2] so it would be easy to have the temperature in some bales reach 70 C during summer months.

It is possible, even likely, that more than one imidazole is involved in bovine bonkers. The one most often incriminated is 4-methylimidazole,[1,3] and it has been used to reproduce the disease by adding it to normal colostrum and milk fed to neonatal calves.[3] However, there are field cases where neither 4-methylimidazole nor other imidazoles were detected in the feed.[2] The authors[2] reference other case studies of ammoniated forage toxicosis where 4-methylimidazole could not be identified in feed or milk raising the possibility that other compounds might also be involved.

There is no specific treatment other than removing the ammoniated feed from the cows' diet and possibly milking out the cows before allowing their calves to nurse. There is a report in which calves treated with acepromazine and thiamine hydrochloride did not die, possibly because the sedation prevented self trauma.[1] In another case, seizures could not be controlled by intravenous diazepam.[2] Probably the best advice is for producers to realize that ammoniated feed fed to lactating cows can cause hyperexcitability in calves and to change feed if there is any hint of hyperexcitability in the calves.

Shipping Veterinary Laboratory Specimens: Do Your Shipping Practices Meet Federal Guidelines?

Jerome C. Nietfeld, DVM, P.h.D., Diplomate ACVP

An important question for veterinarians to ask themselves is "Do my practice's procedures for handling, packaging, and shipping laboratory specimens meet federal guidelines?" The importance of this question is illustrated on the American Veterinary Medical Association (AVMA) website in an article, "Guidance Developed on Shipping Laboratory Specimens," which tells of an Illinois veterinarian who was "facing tens of thousands of dollars in possible fines" because of her practice's procedures for shipping laboratory specimens after being audited by the Federal Aviation Administration (FAA).[1] At least one additional veterinarian was also told to correct shipping practices and provide required training to avoid fines. The AVMA provides an excellent overview of essential information regarding shipping laboratory specimens.[2] Just as important, the website provides contact information for shipping questions.

To meet international standards, in 2006 the United States revised standards for packaging and shipping materials that are infectious or are likely to be infectious for humans or animals. Primary enforcement of these standards is the responsibility of the U.S. Department of Transportation (DOT) Pipeline and Hazardous Materials Safety Administration (PHMSA), but other federal agencies such as the FAA, are also involved.

Most veterinarians are not used to thinking of laboratory specimens as hazardous materials, but anything that is known to contain or is likely to contain an infectious agent capable of causing disease in humans or animals is classified as a Class 6 hazardous material. PHMSA requires that anyone involved in classifying, handling, packaging, or shipping hazardous materials be properly trained and the training documented. It is the responsibility of the employer (including self-employed individuals) to provide training and to maintain records of the training for three years, after which retraining is required. PHMSA has published two brochures, T*ransporting Infectious Substances Safely* and *What You Should Know: A Guide to Developing a Hazardous Materials Training Program*, to help shippers understand the requirements and their responsibilities.[3,4] Both brochures and the PHMSA website include contact information for questions.

Shipping Laboratory Specimens

When preparing to ship laboratory specimens the first question is "Are my specimens likely to contain an agent that can cause disease in humans or animals?" If the answer is yes, the next question is whether the agent is a Category A or B infectious substance. Category A infectious substances are more highly pathogenic than Category B agents, have stricter shipping requirements, and are defined as "An infectious substance in a form capable of causing permanent disability or life threatening or fatal disease in otherwise healthy humans or animals when exposure to it occurs."[3] Category A agents are subdivided into those that cause disease in humans (includes zoonotic agents) and those that only affect animals. The publication, *Transporting Infectious Substances Safely*, lists examples of each category. The important thing for U. S. veterinarians to know is that, unless they are shipping pure cultures, everything they ship will be Category B. For example, cultures of *Bacillus anthrasis*, *Brucella abortus*, *Chlamydia psittaci* (bird strains), *Coxiella burnetti*, *Francisella tularensis*, rabies virus, eastern equine encephalitis virus, and multiple other zoonotic agents are category A. However, diagnostic specimens (carcasses, tissues, blood, swabs, etc.) from animals suspected to be infected with the same agents are Category B. If you are shipping cultures of infectious agents, especially zoonotic ones, it is best to contact the facility receiving the culture to find out proper shipping procedures. Packages containing Category B infectious substances are required to be identified by a UN 3373 label (Figure 1). Other labeling is not acceptable.

If the samples are unlikely to contain an infectious agent they are exempt and need not be identified with a UN3373 label. Instead write or print "Exempt Animal Specimens" on the outside of the package. This lets the carrier know that

Figure 1. UN3373 label which is required to be displayed on the outer surface of packages containing Category B infectious substances. The width of the line must be at least 2 mm and the letters and numbers at least 6 mm high. No side of the diamond should be less than 50 mm in length.

the package is not hazardous. Except for labeling, packaging of exempt specimens is the same as for Category B infectious substances. Examples of exempt samples are blood or sera for blood chemistries, CBCs, serologic tests, etc., cytology slides, biopsies, and other samples unlikely to be infectious.

Packaging Guidelines

Laboratory specimens must be triple packaged. Samples are placed into a leak proof primary container, which is placed into a leak-proof secondary container along with sufficient absorbent material to absorb all liquid in the primary container should it leak. The secondary container is placed in a rigid outer container with enough packing material to keep the contents from shifting and to prevent breakage or leakage if dropped from a height of 1.2 m. A maximum of 1 liter of liquid or 1 kg of solid material are allowed per primary container and four primary containers are allowed in one outer container. The exception is if an entire body or organ is being shipped. Ziploc® bags are not considered acceptable primary containers (believe me they frequently leak when used to transport liquids), but they are acceptable as secondary containers. Whirl-Pak® bags are acceptable as primary containers (Whirl-Pak® also leak if overfilled with liquid and not properly closed. Taping Whirl-Pak bags and other primary containers shut is a good practice). Glass containers, such as blood tubes, should not touch one another. Place the paperwork in a sealed plastic bag (Ziploc® bags work well), and place in the outer container. The outer container is required to be rigid, so it is not acceptable to ship laboratory specimens in padded envelopes. Carriers differ in what they will accept. As an example, the FedEx Express brochure Pointers on Shipping: Clinical Samples, Biological Substance Category B (UN3373) and Environmental Test Samples[5] indicates that Styrofoam containers are not acceptable unless they are inside a cardboard box, but we receive Styrofoam containers not in cardboard boxes from other carriers, such as the U.S. Post Office. The FedEx Express publication[5] is an excellent source of information concerning packaging of Category B samples. It gives pointers as how to meet shipping requirements and contains specific examples of acceptable container and absorbent materials, unacceptable packaging, and proper labeling of specimens.

Training Requirements

According to the AVMA, specific formal training and documentation are required for anyone who packages or transports Category A materials.[2] Documented training is also required for anyone involved in shipping Category B materials. According to the AVMA, individuals who ship waste cultures or stock of a Category B infectious substance must be hazmat trained. People who ship Category B materials for routine testing must also be trained and the training can be "informal and in-house" and it must be documented.[2] It is not clear as to what constitutes formal and informal training. According to the PHMSA brochure, *What You Should Know: A Guide to Developing a Hazardous Materials Training Program*, hazmat training may be provided directly by the employer or by other public or private sources.[4] It must include general awareness and safety training as well as job- specific training, meaning that individuals who ship infectious material should be trained in that area, and not in how to ship explosives or flammable materials, for example.

Training records must include the following:[4]

- the hazmat employee's name
- the most recent training completion date
- a description of, copy of, or reference to training materials used to meet the training requirements
- the name and address of the person providing the training
- certification that the person has been trained and tested as required

Proper packaging and shipping of potentially infectious substances is important and something that should be taken seriously. The shipping requirements are clearly explained in the reference materials. They are reasonable and not difficult to follow. Training and documentation of training requirements are less clear. Questions concerning any facet of shipping infectious agents, especially the training, are best directed to the AVMA because they have asked specific questions of the DOT-PHMSA regarding this topic and to the DOT-PHMSA. The PHMSA website also has a long list of frequently asked questions and answers that are helpful.

Resources for Shipping Information:

1. American Veterinary Medical Association website. Guidance Developed on Shipping Laboratory Specimens, available at: https://www.avma.org/News/JAVMANews/Pages/100415b.aspx

2. American Veterinary Medical Association website. Required Training for Packaging and Shipping Lab Specimens, available at https://www.avma.org/PracticeManagement/Administration/Pages/Required-Training-for-Packaging-and-Shipping-Lab-Specimens.aspx

3. Pipeline and Hazardous Materials Safety Administration website in the Publications and Training Modules section. Transporting Infectious Substances Safely, available at http://phmsa.dot.gov/hazmat/training/publications

4. Pipeline and Hazardous Materials Safety Administration website in the Publications and Training Modules section. What You Should Know: A Guide to Developing a Hazardous Materials Training Program, available at: http://phmsa.dot.gov/hazmat/training/publications

5. FedEx Express website. Pointers on Shipping: Clinical Samples, Biological Substance Category B (UN3373) and Environmental Test Samples, available at www.fedex.com/downloads/shared/packagingtips/pointers.pdf

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Newsletter Coordinators

Larry C. Hollis, Extension Beef Veterinarian
785-532-1246 • lhollis@oznet.ksu.edu

Jerome C. Nietfeld
785-532-4460 • nietfeld@vet.ksu.edu

K-State Research and Extension
137 Call Hall
Manhattan, KS 66506