Beef Cattle Health Management I & II

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RECOGNIZING AND MANAGING COMMON HEALTH PROBLEMS OF BEEF CATTLE

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Many health problems in beef cattle can be managed successfully if they are detected early. Cattle owners can prevent or minimize losses by taking steps to keep the problems from recurring or spreading to the rest of the herd.

Below are common problems found in beef cattle as well as the probable causes of those conditions and suggested measures to prevent recurrence.

"MY COWS' EYES ARE CLOUDY AND RUNNY."

When cattle have cloudy, runny eyes, the inflamed and painful eyeballs and eyelids are probably infected with a virus or bacterium or damaged from sunlight or cancer. These conditions include pink eye, IBR virus eye, cancer eye or photo eye.

Specific diagnosis and proper treatment may require close observation, available history, laboratory testing and professional assistance.

Pink eye (infectious keratoconjunctivitis)

Although sporadic cases of eye diseases occur in all seasons of the year, this highly contagious bacterial disease is most common during the summer.

Observations:

The onset of pink eye is sudden, beginning with an excessive flow of tears. The animal holds the eye partially closed, rubs the eye and seeks shaded areas. Soon an ulcer develops in the central area of the cornea and an opaque ring develops around the ulcer. Within 48 hours of onset, the entire cornea becomes cloudy.

Next, the lining of the eyelids becomes red with mucus and pus. As the ulcer deepens and extends completely through the cornea, the eye ruptures and loses fluid, and the eyeball collapses. The infection may affect one or both eyes.

Management:

The infected cattle must be isolated and treated immediately by a veterinarian to eliminate the infection and prevent spread to other cattle.

IBR virus eye (infectious bovine rhinotracheitis)

The IBR virus is transmitted through the air and can spread rapidly through the herd. It causes upper respiratory infections, and it is most prevalent in the fall and winter.

Observations:

In the early acute stage, a few cattle may develop cloudy corneas, similar to pink eye. The opacity spreads inward from the outer edge of the cornea, and there is no ulceration.

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Management:

Isolate the affected animals until the viral infection runs its course, and vaccinate the whole herd and purchased replacements.

Cancer eye (squamous cell carcinoma)

Cancer often appears as smooth plaques on the eyeball and ulcers or horn lesions on the eyelids. It occurs more often in cattle with no eye pigment and those that are constantly exposed to bright sunlight.

Observations:

As in cases of pink eye, cancer eye causes an excessive flow of tears. This cancer can be identified by the appearance of the lesions on and near the eye. The cancerous growths develop on the third, upper and lower eyelids and eyeball, and they spread to internal lymph nodes and organs.

Management:

Early detection is necessary for heating or freezing therapies or for surgical removal of the tumor alone. In chronic cases with more extensive involvement, the entire eyeball and eyelids must be removed.

Photo eye (photosensitization)

This noninfectious condition is a hypersensitivity to sunlight after ingestion of various plants or administration of certain drugs.

Observations:

In addition to cloudiness of the cornea, signs of photo eye include sunburn of nonpigmented eyelids, nose, teats, vulva and areas of the head, body and legs. If the affected cattle are exposed to sunlight for prolonged periods, blindness and severe skin damage will result.

Management:

Protect the animal from sunlight until its eyes and skin have healed. Shelter it during the day and allow it to graze on pasture at night.

"MY CALVES HAVE AREAS OF HAIR LOSS WITH SKIN LESIONS."

Calves commonly become infected with ringworm fungus and wart virus. These two infectious, contagious conditions are easily recognized and differentiated by the appearance of localized hair loss with skin lesions.

In cases where there is generalized hair loss with skin lesions, possible causes other than ringworm or warts include photosensitization, dietary deficiencies, infections of worms and infestations of horn flies and lice.

Ringworm fungus (dermatophytosis)

In the early stages, a fungus infection of the skin often goes unnoticed because the affected areas are small and slightly raised with roughened hair. Infected cows often serve as sources of the fungus, which is transferred by direct contact to calves.

Observations:

After several weeks of the fungus infiltrating the hair follicles, the hair falls out, leaving distinct circumscribed, grayish lesions. The scaly lesions coalesce to form large patches of hair loss at least 3 inches in diameter. They are often located on the face and neck and are more common in young cattle.

Management:

Although the infection tends to clear up spontaneously after several months, separate and treat the affected calves with a prescribed medication to prevent transmission to the others.

Warts (papillomatosis)

Warts are fibrous tumors of the skin and mucous membranes and are caused by many strains of the papilloma virus. The virus is usually transmitted to calves by direct contact from infected cows. It also can be transmitted by contaminated instruments that puncture the skin and by biting flies such as horn flies and stable flies.

Observations:

The cauliflower-type growths occur primarily on the head, neck and shoulders, in the mouth and vagina, and on the teats, vulva and penis.

Management:

To prevent transmission to other calves, isolate those with warts. Over a period of 3 to 12 months, the affected calves build immunity against the virus in the warts and skin. Once the immunity kills the viruses, the warts dry and slough.

"EVERY WINTER, MY COWS RUB THEIR HEADS, NECKS AND SHOULDERS."

Even though lice are known in the winter to cause cattle to itch and rub on objects such as fences, posts, trees and barns, another common cause of itching and rubbing is the aftermath of the allergic dermatitis produced during the previous summer and fall by a horn fly infestation.

Horn fly allergy (allergic dermatitis)

During the horn fly season, cattle often develop a skin allergy to the saliva of the biting horn flies. After several weeks, an inflammatory reaction occurs in the skin, and many hair follicles are destroyed.

Observations:

Before the damaged hair falls out during the winter, the retained hair causes an itch sensation, and the cattle rub their faces, necks and shoulders from December through March. As a result of rubbing these areas, the hair coat becomes sparse, and irritated skin lesions develop.

Once the dead hair is removed by rain and rubbing, a normal hair coat returns. If no crawling lice are on the skin or lice eggs are glued to the hairs, the diagnosis is based on a history that the cows had a horn fly infestation the previous year.

Management:

To prevent recurrence of this cold-season problem, take steps to reduce the horn fly population during the warm seasons.

"I HAVE OCCASIONALLY A COW OR A BULL CRIPPLED ON ONE FOOT."

A cow or bull with a lame foot should be examined closely. Pick up the foot with a rope, and wash and examine between the toes carefully, looking for a foot crack, a corn, swelling, heat or a discharge. You will need professional assistance to differentiate some of the other abnormal conditions of the foot.

Unobservable problems inside the foot include bruises, abscesses, fractures and foot founder, or laminitis. The lameness may also be related to long toes as well as joint inflammation of the leg, including the hip on the rear and shoulder on the front.

Foot crack (web tear)

This condition often occurs after cattle walk on rough terrain or when a bull places its weight on the foot when mounting for breeding. These actions commonly spread the toes wide apart and cause the skin to tear. Also, long toes predispose to the likelihood of excessive spreading of toes.

Observations:

If the problem is not a corn or foot rot, check for signs of foot crack, along with swelling and heat of the foot. The web of skin between the toes is also likely to be cracked deeply into sensitive tissue.

Management:

The damaged tissue must heal from the inside out. To prevent further tearing, the cow or bull must be confined for a few weeks to limit walking and the toes trimmed and taped together.

Foot rot (necrotic pododermatitis)

If the problem is not foot crack, the likely problem is foot rot, a bacterial disease of the foot. During warm, wet weather, the bacteria in manure mixed with mud commonly gain entry through tiny cracks and abrasions of the skin between the toes and heel bulb, causing swelling and dead tissue.

Observations:

The signs of foot rot include a hot, swollen and painful foot with pus discharge and a dead odor, fever and loss of appetite and body weight. The infection may spread to the skin of the pastern and fetlock and to bone joints inside the foot.

Management:

Because the pus discharge contains bacteria and serves as a source of new infections, segregate the cow or bull from the rest of the herd for proper antibiotic treatment. To prevent occurrence of more cases, the unsanitary conditions leading to this condition must be corrected.

Corn (interdigital hyperplasia)

The development of scar tissue, or corns, in cattle is thought to be caused by stretched skin folds between the toes in heavy, splay-toed breeds.

Observations:

A painful and hard, tumor-like, vertical mass develops in the web of skin between the toes.

Management:

The mass must be removed surgically and the toes bandaged closely together.

"ONE OF MY COWS COUGHS, PROTRUDES HER TONGUE AND BREATHES WITH HER MOUTH OPEN."

The cow obviously has a lung disease in which inflammation elicits an irritated cough, and reduced air space encourages open-mouthed breathing. Because several infectious and noninfectious causes are possible, professional assistance will be needed to make a specific diagnosis by physical and laboratory examinations.

A common infectious lung disease is pneumonia; a common noninfectious condition is fog fever.

Infectious lung disease (pneumonia)

Pneumonia is a highly complex, contagious disease and may be caused by one of several viruses in concert with various bacteria. Pneumonia caused by bacteria is generally serious.

Observations:

Fever, coughing and labored breathing are caused by inflammation and swelling of the lungs and the accumulation of mucus, blood and pus that interfere with airflow in the air passages. The animal tries to get more air by stretching out its head and neck and protruding its tongue.

Management:

When you see signs of pneumonia, isolate the sick cow for antibiotic treatment. Laboratory tests are needed to identify the specific viruses or bacteria involved to develop an effective vaccination plan for the herd. The plan should include vaccinating the cows, nursing calves, bulls and replacements with the proper vaccines.

Because stress can contribute to the occurrence of this disease by lowering an animal's resistance, cattle owners need to minimize adverse conditions of cold or hot weather to prevent pneumonia in the herd.

Fog fever (pulmonary emphysema and edema)

Fog fever is caused by a toxic reaction in the lungs after the cow ingests a large quantity of an amino acid in lush, green grass in spring or fall. Diagnosis is based on a history of the cows being moved within the previous 10 days from a dry, brown pasture to a lush, green pasture.

Observations:

Fever is not present; coughing is minimal; and the onset of symptoms is sudden. Breathing is obviously difficult, with the animal breathing through its mouth, extending its tongue and drooling saliva.

Management:

The affected cow should be treated by a veterinarian and handled carefully to prevent death by suffocation brought about by exercise. Move the herd from the lush pasture and gradually return it over 3 weeks by feeding hay and limiting grazing time.

"MY CALVES HAVE RUNNY, SNOTTY NOSES."

Runny, snotty nose can be associated with pneumonia if the calves have fever, are coughing and have labored breathing. Otherwise, the calves may simply have an inflammation of the sinuses of the head, which is called sinusitis.

Runny, snotty nose (sinusitis)

Nasal drainage in calves may be the normal discharge of mucus from the sinuses of the head. On extremely hot, cold or windy days, inflamed sinuses can discharge excess drainage, even if there is no infection. Also, irritants and allergens in the environment such as dust, pollen and mold cause inflammation of the sinuses.

Observations:

When viruses and bacteria infect the sinuses, they produce a head cold and cause a nasal discharge that is a clear, mucus or pus type. Often the infection is limited to the head and does not involve the lungs.

Management:

Do not use antibiotics if there is no or only a low-grade fever; allow the condition to run its course. Respiratory vaccines may lack the specific antigens to prevent recurrence.

"SOME OF MY COWS GOT THE STAGGERS, WENT DOWN AND ARE UNABLE TO RISE."

Cows that cannot rise must be checked by a professional, who will conduct physical examinations and evaluate their diet and environment. Although the cause may be one of many poisonous plants, it is more often the result of grazing on Dallisgrass. In chemical poisoning cases, the cause is often the consumption of toxic amounts of lead or arsenic from batteries or lubricating grease of vehicles or machinery. If the cause is dietary, it is likely that the cattle have a common metabolic disorder such as polio, ketosis or grass tetany.

Polio (polioencephalomalacia)

Cows with polio are thin and usually have been on a diet high in sulfate and low in protein and roughage. They probably have been confined and fed a grain diet without roughage.

Observations:

As an affected downer cow attempts to stand, the ankles remain flexed or knuckled over.

Management:

Immediate treatment by a veterinarian to relieve swelling of the brain is necessary to prevent permanent brain damage. Adequate roughage must be fed with grain concentrates.

Range ketosis (acetonemia, hypoglycemia)

Cows with range ketosis are usually thin, on a low-carbohydrate, low-energy diet and likely are stressed from cold weather or calving and nursing.

Observations:

In addition to the incoordination before going down, the cows are observed pressing against walls, posts and trees, bellowing and tongue wallowing and licking.

Management:

Immediate treatment by a veterinarian is directed to raise the blood sugar and improve glucose metabolism.

Grass tetany (hypomagnesemia)

The affected cows are thin, grazing lush pasture high in nitrogen and potassium and likely are stressed from cold, cloudy weather or calving and nursing.

Observations:

In addition to staggers, signs in cattle include tossing the head, bellowing and galloping before going down with convulsions.

Management:

Immediate treatment by a veterinarian is directed to raise the blood magnesium.

"I HAVE OCCASIONALLY A THIN, DOWNER COW."

Dietary deficiencies are the most common cause of weakness and weight loss in cattle. Enteric bacteria and parasites may be contributing factors.

Observations:

Tipoffs to problems in the diet include weakness and loss of weight.

Management:

Evaluate the nutritional intake, comparing it to the protein and energy requirements of the herd. Make adjustments if necessary.

If the problem is limited to an individual cow instead of affected the entire herd, seek professional assistance to identify the cause, such as infections of body cavity linings (pleurisy, peritonitis) and abscesses and cancers of internal lymph glands and organs.

"I CONTINUE EVERY YEAR TO HAVE COWS PROLAPSE AND RETAIN AFTERBIRTH."

It is common for a cow that has difficulty in calving to bruise her uterus. A thin, weak cow may have a prolonged calving process that commonly causes a bruised uterus.

Observations:

The inflamed, swollen uterus quite often causes straining with prolapse of the vagina, cervix or uterus. If prolapse does not occur, the placenta may be retained because of bruising inflammation.

Management:

Treatment by a veterinarian is directed to replace the prolapse and expel the retained placenta.

"I HAVE LOW CONCEPTION RATES, REPEAT BREEDERS AND ABORTIONS IN MY COWHERD."

Dietary deficiencies and stresses of hot weather and malnutrition in cows continue to be major causes of reproductive failures. Abnormal ovaries and uterus and starvation of the embryo or fetus are commonly associated with inadequate intake of protein, energy, minerals or vitamins. These reproductive problems occur in stressed cows on poor quality or short grazing without provisions of hay and nutrient supplements.

Observations:

The herd has an unusually high number of abortions, repeat breeders and low conception rates that cause a large percentage of open cows.

Management:

If the problem is caused by poor nutrition, evaluate the nutritional intake and take corrective measures.

Professional assistance is essential to diagnose infectious diseases, including testing of fetuses, placenta and blood samples.

"I HAD SEVERAL CALVES SUDDENLY DIE THAT BEFORE DYING WERE RAPIDLY BREATHING, WEAK AND FEVERISH."

Many infectious causes of rapid breathing, weakness and fever, followed by sudden death of calves are possible. Ask a veterinarian to perform a necropsy on one of the dead calves and make a specific diagnosis by physical and laboratory examinations. Two common diseases that cause sudden death in calves are lepto and blackleg.

Lepto (leptospirosis)

Lepto is caused by one of five strains of bacteria. The bacteria are shed with urine from infected animals, such as cattle, raccoons, skunks, opossums, rodents, deer, swine and dogs. The bacteria may be shed for many months.

For calves, the likely exposures are from the urine from carrier cows that were stressed at calving and from diseased and convalescent calves. Cows may have the disease but show no signs of it. Calves are infected with the bacteria when they ingest contaminated urine on teats, hair, grass and hay and in water. Newborn calves are the most susceptible to the acute disease.

Observations:

The acute form of the disease causes high fever, rapid and difficult breathing, depression, bloody urine, incoordination and death. Lepto calves are often mistakenly diagnosed and treated for pneumonia. Because the bacteria can kill unborn calves as well as nursing calves, it is suggested that cattle owners evaluate the cow herd's pregnancy rate and look for aborted fetuses.

Management:

For a closed herd, the most effective approach for control is annual vaccination of all cattle; for an open herd, vaccinate twice yearly. If you time the vaccination in the cow herd during the last trimester of pregnancy, it will provide immunity to the newborn calves through the colostrum.

Use polyvalent killed vaccines containing three or five common serovars. Different vaccines vary in effectiveness, and vaccine failures may occur.

Blackleg (clostridial disease)

When the cause of sudden death of a calf is blackleg bacterial toxins (poisons), the first point to make is that the calf swallowed blackleg spores from the soil. This means the ground is contaminated with the spores that never die. During rains, these spores are normally concentrated by surface water in various spots in the ground, and drought or rains will cause them to surface from the soil.

When ingested by a calf, the spores go to the muscles and remain dormant. A trigger breaks them out of dormancy, sometimes months or years later. Then the bacteria multiply rapidly and produce toxins in the muscles, killing the muscles (black dead muscles), causing blood poisoning and sudden death.

The most common trigger is fast growth. Another trigger is muscle exertion, such as that caused during working, weaning and hauling. Affected calves may be infected at an early age and die of blackleg at a later age. When blackleg occurs, the transmission was not necessarily recent, but possibly months ago.

Observations:

Sudden death and rapid, gaseous decomposition are the most common signs of blackleg.

Management:

The death is so rapid that treatment is normally ineffective. All dead calves should be burned with untreated wood products to keep from contaminating the ground.

Because other calves can have the bacteria in dormancy, guard against triggers such as stress and rapid growth. Vaccinate the remaining calves. If these calves die, they were already infected with the dormancy of blackleg bacteria before vaccination. Vaccination after exposure will not prevent the dormancy from breaking out.

The seven-way blackleg vaccine should be used because other strains in addition to blackleg that also cause sudden death can be present. The seven strains can be diagnosed only in a dead calf by necropsy and laboratory tests. In addition to blackleg, the other six clostridial diseases that cause sudden death are black neck, black liver, malignant edema, and B, C, D enterotoxemia.

A proper vaccination program includes annual vaccination of the entire herd (calves, cows, heifers, bulls), not just calves. Grown cattle die from four of the seven different blackleg-type bacteria. Cows should be vaccinated during last 3 months of pregnancy or twice a year.

"SOME OF MY CALVES ARE RAPIDLY BREATHING, WEAK, FEVERISH, SCOURING AND DYING."

Because several infectious causes are possible, professional assistance is required to make a specific diagnosis. Fresh feces from live calves must be submitted for laboratory testing, and one of the dead calves must be submitted for necropsy and physical and laboratory examinations. Results of these examinations commonly reveal the presence of tissue damage in the small intestine (enteritis) and large intestine (colitis) and bacteria in the blood (septicemia).

Scours (enteritis-colitis septicemia)

Nursing calves are at high risk to fatal diseases such as scours from the day they are born and continuing during the time of the year when one is calving cows and heifers, moving and mixing these cows and heifers, and bringing in bulls to them. At this time, the baby calves can have low immunity and be highly susceptible to diseases. They can die from scours by dehydration and from septicemia by systemic infections.

Scours are caused by bacteria (*E. coli* and *C. perfringens* B, C, D), viruses (rotovirus and coronovirus), and protozoa in the intestines (cryptosporidia and coccidia). Scours and dehydration worsen when affected calves nurse natural or artificial milk and receive oral antibiotics.

The sources of these deadly germs in the pasture include contaminated ground and fecal shedding from the cows, heifers and bulls. When a pasture trap is used year after year for close observation of calving cows and heifers, the ground becomes heavily contaminated with germs from manure. This contamination is long standing during cool, wet weather by a build up of manure from the calving cows and heifers and scouring calves.

Observations:

Calves infected with these germs breathe rapidly and are weak, feverish and scouring. Death also may result.

Management:

To correct the dehydration, the affected calves must be removed from nursing and given oral electrolytes until the scours have stopped.

Preventive measures include increasing the level of immunity in colostrums and having all calves nurse the first day of birth. Calf scours can be controlled by vaccines containing *E. coli,* rotavirus, coronavirus and *C. perfringens* B, C, D. Establish an annual vaccination program to provide immunity for the newborn calf though the cow's colostrum. The pregnant cows and heifers need to be vaccinated late in pregnancy to be in colostrums and provide the protective immunity against the fatal baby calf diseases.

Other preventive measures include reducing the level of exposures to infectious organisms during calving and breeding seasons. To reduce the calf mortality related to scours and septicemia in a cow herd calving over a period of several months, use more than one pasture trap to provide clean maternity areas.

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BIOSECURITY FOR BEEF CATTLE OPERATIONS

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For beef cattle, biosecurity involves a system of management practices that prevent diseases from infecting a herd. Although biosecurity is often associated with foreign animal diseases, the term also applies to common diseases that affect herds, such as blackleg and bovine viral diarrhea. Vaccines can help prevent disease, but other management practices can be even more important. By developing biosecurity protocols that protect cattle from the common diseases, producers are establishing a safety net against a possible outbreak of a foreign animal disease in the U.S.

HOW DISEASE IS SPREAD

Disease spreads directly—from an infected animal to a susceptible animal—or indirectly, from an infected animal to an object or equipment, and then to a susceptible animal. For example, feeding a calf with a bottle that has not been properly sterilized can be a way of indirect transmission.

Disease is transmitted in seven primary ways:

- Aerosol: Disease pathogens are carried in the air on moisture droplets from sneezing or coughing.
- Direct contact: Disease pathogen contacts an open wound, saliva, blood or mucous membranes, or is passed from nose to nose, by rubbing and biting.
- Oral: Susceptible animals consume disease-causing athogens in contaminated feed and water or lick or chew contaminated objects.
- Reproductive: Disease pathogens are spread during mating or gestation.
- Vehicles: Contaminated objects, such as needles, trailers, trucks or clothing, transfer the disease-causing pathogen from an infected animal to a susceptible animal.

- Vector-borne: A living insect, animal or human carries the disease from an infected animal to a susceptible animal.
- Fomites: Diseases are transmitted through contaminated soil, water and food.

IMMUNITY

Immunity allows the animal to resist a disease by preventing the pathogen's development or by counteracting the effects of its toxins. Immune animals have antibodies, which destroy a specific pathogen before it causes an illness. Immunity is natural, active or passive.

Natural immunity is provided by the body's natural defenses, such as the skin and nasal passages, which help keep disease pathogens out of the body. Some cells in the body also attack disease-causing foreign particles. Fetuses can acquire antibodies in utero through placental transfer.

Passive immunity comes through the transfer of antibodies from one animal to another, such as through

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colostrum in the mother's milk shortly after birth. Newborns must receive about 10 percent of their body weight in colostrum within the first 24 hours after birth to ensure some protection against diseases.

Active immunity is provided by protective vaccinations or by the body's fight against an infection. Both modified-live and killed vaccines cause the body to produce antibodies without actually acquiring the disease. Booster vaccinations may be necessary to maintain immunity.

VACCINATIONS

Total disease prevention is not possible; therefore, any ranch biosecurity plan requires a sound vaccination program that targets diseases the cattle may be exposed to.

Vaccines are only as effective as the animal's immune response; injecting cattle with vaccine does not guarantee the herd's immunity. Factors such as nutritional, shipping, social and weather stress can decrease the level of immune response. Minimizing animal stress will improve the disease protection within the herd. Handling and administering vaccines according to the manufacturer's label is important in maintaining the integrity of vaccine and providing protection against the targeted disease.

When handling and working with vaccines:

- Read the label and/or medication insert before vaccinating animals.
- Observe the expiration date and storage information.
- Keep refrigerators at the proper temperature to maintain vaccine effectiveness, usually between 36 degrees F and 46 degrees F.
- Protect vaccines from sunlight.
- Give the right vaccine to the right species. If the label indicates it is for use in swine, do not use it in cattle. This extra-label use is illegal unless done under the supervision and recommendation of a veterinarian.
- Give the proper dose in the appropriate area on the animal, using the recommended technique.
- Do not insert a used needle back into an open bottle. Always use a sterile needle.
- Use a transfer needle or a sterile needle to reconstitute modified-live vaccines.
- Use boiling water, not chemical sterilants, to disinfect syringes.
- Mix only the quantity of modified-live vaccine that will be used within 1 hour.
- Dispose of the remaining opened vaccine properly after completing the day's inoculations because the vaccine does not keep well once the bottle seal has been punctured.
- Give booster vaccinations when the label requires it.
- Keep a record of all vaccinations and treatments.
- Follow withdrawal periods.

Consult a veterinarian to ensure proper timing and implementation of a vaccination schedule. Even under ideal conditions, vaccinations are not 100 percent effective. Take extra care in handling and administering vaccines to achieve the highest possible level of immunity.

Evaluate the cost-benefit ratio of any biosecurity management practices. Do the benefits outweigh the costs? For example, if a weaned calf is worth about $550, the loss of that calf can cost the ranch $550 in lost revenue. If a vaccination routine that costs $1.50 per animal, including new needles for each, is implemented on a 40-cow herd, the total cost for this biosecurity practice may be as low as $60. If the result is one more calf, the net benefit is $490.

PROCEDURES FOR HANDLING INCOMING CATTLE

Almost every ranch eventually must add new breeding animals to the operation. Some stocker or feedlot operations continuously add new cattle. These new cattle can bring disease to the ranch. Minimize this risk by:

- Defining the level of disease risk for the new cattle. For example, yearling virgin bulls from a purebred breeder with a strict health protocol may be low risk, while cows from an unknown source may be high risk.
- Isolating new animals from the rest of the herd for at least 3 weeks, and possibly at a location off the ranch.
- Watching the isolated animals closely for symptoms of illness, such as elevated temperature and abnormal behavior.
- Consulting a local veterinarian to determine which diseases to test quarantined animals for.
- Vaccinating cattle according to ranch protocols.

LIMITING UNAUTHORIZED ACCESS TO PASTURES AND CATTLE

Unauthorized visitors may introduce diseases to the ranch, increase the risk of theft and cause liability issues. To help prevent this:

- Keep doors and gates locked at all times.
- Post "No Trespassing" signs.
- Conduct random security checks and look for signs of unauthorized activity or entry.
- Maintain good perimeter fences.
- Know your neighbors and set up a crime watch program.
- Secure pesticides, fertilizers, feed and nutrients.
- Secure water sources and identify alternative sources.

GENERAL BIOSECURITY PRACTICES

Consider these additional general management tips:

- Disinfect reusable equipment, including tattooers, implant guns, ear notchers, dehorners and castration knives, between animals. Sterilize equipment that has been used off the ranch before it is brought back to the ranch.
- Identify cattle and maintain current records.
- Watch cattle for adverse health symptoms or behavior; sudden and unexplained deaths; large numbers of sick animals; unusual ticks or maggots; blisters around an animal's nose, teats, mouth or hooves; difficulty rising and walking; a drop in milk production; and a large number of dead insects, rodents or wildlife. Contact a veterinarian immediately if these symptoms occur.
- Keep cattle away from exotic wildlife that may harbor disease.
- Develop a carcass disposal plan.
- Remove animals that are "reservoirs" for certain diseases such as Johne's, trichomoniasis or bovine viral diarrhea. These animals continue to shed the pathogen and infect other animals.
- Avoid fecal and urine contamination of feed and water sources.
- Control pest populations and limit access to feedstuffs.
- Create an emergency contact list of resource people within the community. Post copies near telephones and on bulletin boards. Have employees enter these numbers into their cell phones.

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BASICS OF CATTLE IMMUNITY

Tom Hairgrove[1] and Steve Hammock[2]

When establishing a vaccination program it is important to understand how animals naturally protect themselves from infection and how vaccination and other management practices enhance that protection.

There are three major ways the body defends itself against infectious organisms.

1. The first method is physical barriers, such as skin, normal microorganisms, and self-cleaning procedures such as coughing, sneezing, vomiting and diarrhea. Organisms that penetrate the body are often eliminated by these procedures. Animals must be adequately hydrated and nourished for these barriers to work effectively.
2. The second method of body defense is native or innate immunity. The native immune system controls invading organisms with chemicals and/or by ingesting them. The native immune system lacks memory, so each infection is treated in the same manner. The immune system needs adequate nutrition (including energy, protein and minerals) to function at a maximum level. Stress reduces the efficiency of the native immune system.
3. The third method is the acquired immune system, which responds to vaccines. This system can recognize and destroy specific invaders. With acquired immunity, the body remembers specific invaders and can respond more intensely if stimulated by those invaders later. While physical barriers and the native immune system respond rapidly, the acquired immune system takes days to weeks to become effective. When the acquired immune system is compromised, as in human AIDS patients and cattle with bovine viral diarrhea (BVD), other diseases can rapidly overcome the animal's defenses.

Acquired immunity may involve the production of a specific antibody (humoral immunity); or, it may involve the rapid recognition and destruction of specific foreign cells (cell-mediated immunity). The humoral immune response is relatively easy to measure and it is the most common way immune responses to vaccine and/or disease are detected. Cell-mediated response is much more difficult to quantify. The body reacts to specific diseases with either an antibody or a cell-mediated response. Organisms that attack the outsides of cells usually respond to antibodies. Organisms that invade the cell, such as all viruses and some bacteria (including brucellosis), often are better controlled with a cell-mediated immune response.

Vaccines made from modified live products are usually more efficient at protecting against diseases such as brucellosis or BVD that infect the insides of cells. Modified live vaccines replicate in the animal and usually do not require boosters. However, these vaccines are easily degraded and made ineffective by exposure to chemicals or extremes of light or temperature.

Vaccines made from killed products are usually more efficient at destroying organisms that attack the outsides of cell, such as those that cause blackleg or tetanus. Killed products do not replicate, so boosters are usually needed for good protection. Killed products can give undesirable results if shaken excessively or frozen.

All vaccines should be handled according to manufacturers' recommendations.

A vaccination program is simply a tool in a total health program. Animals must have adequate nutrition for their immune systems to work properly. Animals also should be protected from environmental and social stressors and parasites, which may decrease an animal's natural response to disease and the effectiveness of vaccines.

Administering too many vaccines, or vaccines that are not compatible, also can lower the immune response. Some vaccines should not be administered to pregnant animals because they may cause reproductive loss.

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Vaccines may not be effective when given to calves with high levels of maternal antibodies. All of these factors are reasons why you should consult with your veterinarian when designing vaccination programs.

Vaccines are not always effective under field conditions, so producers should have reasonable expectations of vaccine programs. A vaccine program to prevent unborn calves from becoming persistently infected with BVD might be quite different from one to control BVD in a group of stocker calves.

Always consult with your veterinarian, who is familiar with disease patterns in your area and can recommend the most effective vaccination program.

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Bovine Viral Diarrhea-Brief Overview

1. Bovine Viral Diarrhea Virus (BVDV) was first recognized in the United States in 1946 as a virus associated with diarrhea, hence the name. The most significant adverse economic impact of Bovine Viral Diarrhea (BVD) is due to reproductive loss. Many (70-90%) of BVD infections in a cow herd go unrecognized. BVD is caused by an RNA virus capable of infecting other ruminants and pigs. The virus can affect multiple systems, including reproduction, respiratory, nervous, circulation, immune function, skin, bone, and muscle.
2. Bovine Viral Diarrhea is often only recognized as a Bovine Respiratory Disease Complex (BRD) component and identified in the stocker-feeder portion of the cattle industry.
3. Bovine Viral Diarrhea is problematic because it is uniquely maintained in nature through persistent infection (PI). Persistently infected animals result when the fetus becomes infected during the first 125 days of pregnancy. Suppose the virus infects the fetus during this period. In that case, the fetus does not mount an immune response, and becomes immunotolerant to the virus, resulting in the PI shedding the virus throughout its lifetime. In North America, there are three major subtypes of BVD, BVDV 1-a, BVDV 1-b, and BVDV 2-a. Mutations in the BVD genome are common, leading to variations in the virus (termed a Quasi-Species). Currently, BVDV 1-b is most commonly isolated from affected cattle. There are two strains of BVDV virus, cytopathic (CP) and non-cytopathic (NCP). Only the NCP strain is related to persistently infected cattle (PI), and only approximately 1% f the cattle population is PI.
4. Cattle can also become infected later in gestation or after birth and become transiently infected, similar to a human contracting the influenza virus from a family, a coworker, etc. The source of the virus resulting in a transiently infected animal can be another transiently infected animal or a PI. If the transiently infected animal recovers, it will no longer be a shedder.
5. Producers should develop a biosecurity program to control BVD based on their risks. Unless one has personal knowledge of the herd of origin, they should not comingle purchased pregnant females with their herd until they have calved and their calves tested negative for BVD. As with many other cattle diseases, good fences are essential to biosecurity; however, the BVD virus can infect animals in close contact through fences.
6. A well-thought-out vaccine program is vital for a BVD biosecurity program. Consult your veterinarian for assistance in developing your biosecurity plan. Another helpful website, is available from the Beef Cattle Institute at Kansas State University. https://ksubci.org/bvdbovine-viral-diarrhea-control-consult/

BVD Control Program?

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Texas A&M AgriLife Extension

TEXAS A&M UNIVERSITY Animal Science

TEXAS A&M AGRILIFE EXTENSION

Producer's Perception of Disease Threat

- Blackleg 45%
- Leptospirosis 40%
- BVD 35%
- Trichomoniasis 30%
- Brucellosis 20%
- Anaplasmosis 11%

TEXAS A&M AGRILIFE EXTENSION

Bovine Viral Diarrhea

- Reproductive loss.
- Bovine Respiratory Disease.
- Intestinal Disease.

TEXAS A&M AGRILIFE EXTENSION

Consequences of BVD

- Immunosuppression.
- Reproductive Loss.
- Associated with Bovine Respiratory Disease Complex.

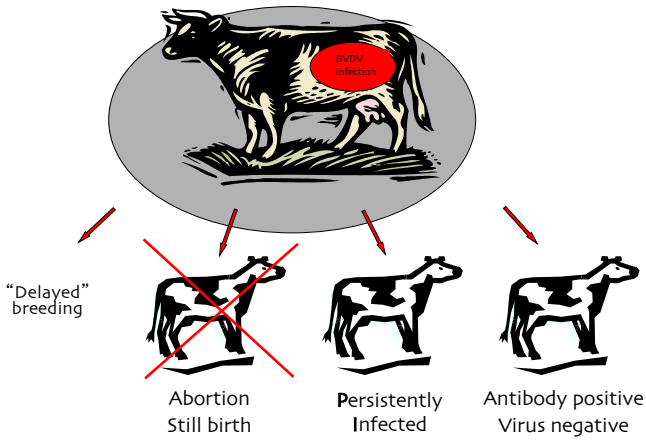
TEXAS A&M AGRILIFE EXTENSION

Types of Infection

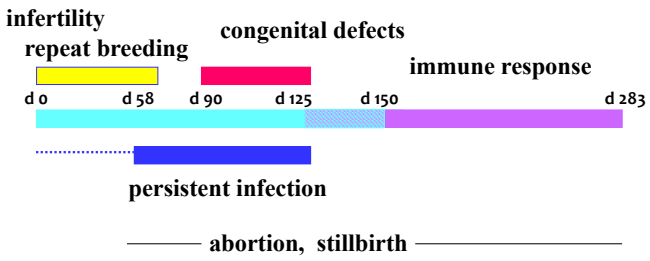
- Acute.
- Persistent infection.
- Chronic testicular.

TEXAS A&M AGRILIFE EXTENSION

Outcomes of *In utero* infections

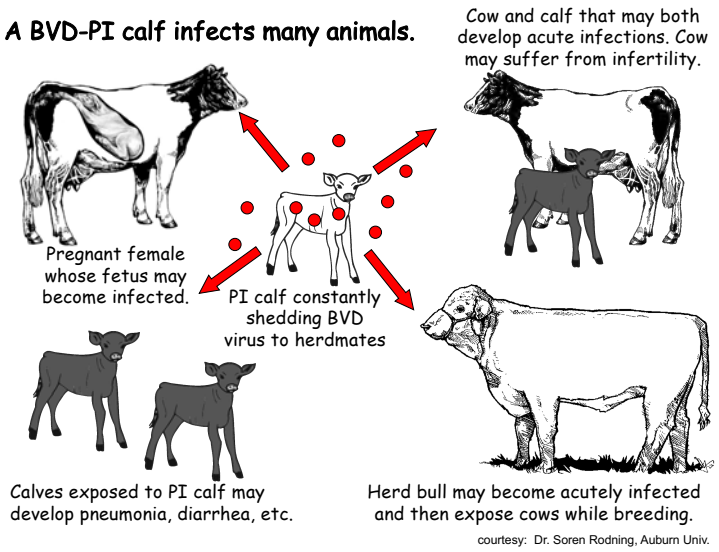


BVD INFECTION DURING GESTATION



Persistent Infection (PI)

- Immune system not adequately developed to recognize the virus as foreign.
- Immune system thinks the virus is part of self.
- Animal will never clear virus-will shed through out life.



courtesy: Dr. Soren Rodning, Auburn Univ.

Survival of Virus

- Ability to resist environmental degradation outside a host organism.
- If virus does not survive for long period outside of the host, persistent replication is necessary for continued survival.

Tests for PI

- May want to retest-value and use of animal.
- Timing is everything

What We Expect

EXTENSION

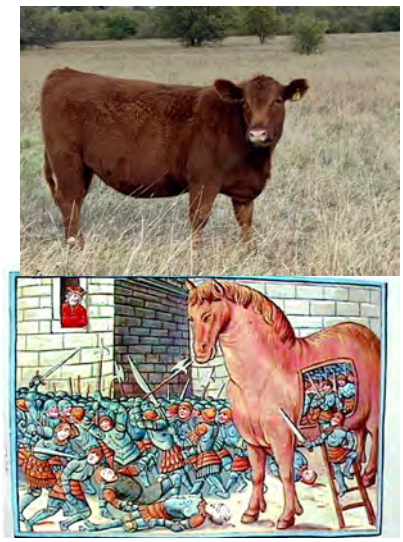
Hidden Danger

TEXAS A&M AGRILIFE EXTENSION

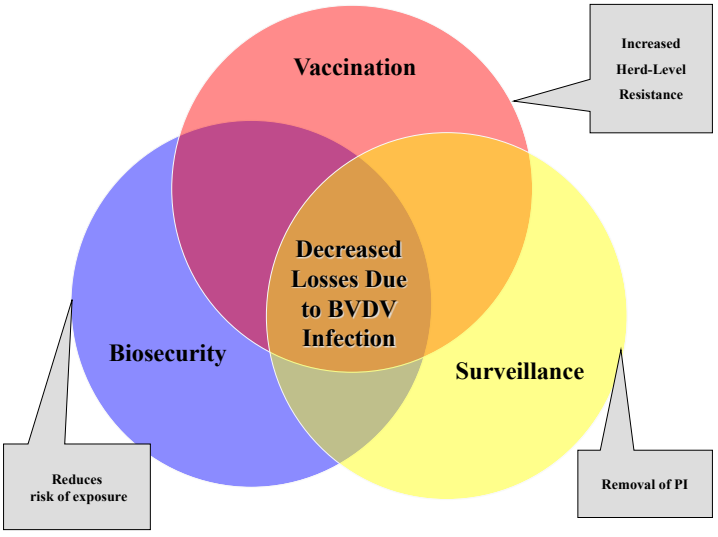
Cow calf Problem

- Control most efficient at the cow calf level.
- BVD biosecurity at stocker and feedlot level much less efficient.

TEXAS A&M AGRILIFE EXTENSION



Could this heifer be a "Trojan Horse"?



Biosecurity Plan Unique to Your Operation

- Your operation has risks and opportunities.
- Quarantine.
- Testing for diseases.
- Open or closed herd.
- Source of replacements.

Biosecurity

- Keep diseases you do not have from entering your herd.
- Manage diseases you already have in your herd.

Herds Without Disease

- Testing.
- Vaccination.
- Quarantine.

Herds With Disease Present

- Testing strategy.
- Vaccination program.
- Bio-containment and biosecurity.

Questions-Comments

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Texas A&M Beef Cattle Short Course 2023

BVD Cow/Calf Producer Impact Statement

There are defining moments in your life. Good ones. . . a marriage, a birth of a child, purchasing land and starting a homestead or ranch that will be a generational keepsake. There are bad ones . . . the loss of a loved one, a failed business venture, or the discovery of a BVD PI calf in a cow/calf operation. Many years ago, we were changing the direction of our ranch and it was very exciting. We had already converted to a grass-fed, rotationally grazed, holistically managed, regenerative cattle operation. We had just taken the plunge to convert from our commercial sale-barn bought herd to registered South Poll cattle. South Poll cattle are bred to thrive in grass-fed operations in hot and humid environments like we have here in East Texas. Things were going great for us and we were selling off the larger-framed cattle we had owned prior to getting the South Polls. We put together a group of seven F1 tiger stripes to sell as a lot. They were what ranchers called "fancy": pretty girls that were bred. They would be worth some money and would help us buy some registered South Polls as good quality registered South Polls were and still are not inexpensive. As one of the first breeders of South Poll cattle in Texas, we were on the forefront of a new breed and in two niche markets (South Polls and grass-fed). We were setting ourselves up for success. But having to travel cross-county to put together our new herd of expensive girls, we decided to take the F1s to a special replacement female sale a couple of hours away that only sold in lots. We thought we could maximize the sale price of those cattle there and use the funds to replace them with South Polls.

When we got to the sale, they separated one F1 heifer from the group. She was a yellow tiger-stripe color. The rest of the group were red-tiger stripes. Because the barn sold in lots, we knew she wouldn't bring as much selling as a single cow and we liked her, so we brought her back home. Little did we know that this one choice to preserve our capital investment in our cattle would just about ruin us. It would teach us about things we had never heard of before or, if we had, we hadn't paid much attention to: things like, for example, biosecurity; bovine viral diarrhea disease (BVD); snap tests; and persistently infected (PI).

BVD is a tricky disease. Remember in school the story of the Trojan horse? It was such a ubiquitous and interesting idea that the concept of a "Trojan horse" became part of our every-day vernacular. BVD PI is a Trojan horse. The only way to have a BVD PI calf is for it to be infected in its mom's uterus. All other animals exposed to BVD will have what they call a "transient" infection. Think of having a transient infection like having the flu. You have it. You are noticeably sick, but most people get over it and continue on with life just fine. Think of a "permanent infection" (PI) as something like an immune deficiency disease. You always have it and you probably won't die from it, but it can destroy your immune system and (prior to current treatments) you will probably die of something else (not the original disease). Depending upon the strain, BVD can be highly contagious. Something as simple as nose-to-nose contact or drinking from the same water trough can transmit the disease.

If you have a bred cow or heifer, there is no way to test for BVD PI in the calf that is in her uterus. You can buy a bred animal, test the cow/heifer for BVD, and assume all is well with the world on your ranch and continue on. But the calf inside of her, that cutesy little, sweet little thing that you will be so excited to have and will take pictures of and share with your friends and family, could be infected with BVD PI. Unless you test that calf, you will never know. And at that point, that sneaky BVD disease . . . that Trojan horse of a disease . . . could have infected more animals on your ranch as they grow in their momma's uterus. That's what happened to us. One calf in the first year turned into nineteen calves the next year, despite removing the first BVD PI calf the night of the discovery (unfortunately, that meant a bullet and a hole in the ground). That calf was already nine months old and had infected the next year's calf crop. If one led to nineteen, how many would nineteen lead to the next year? The thought made us shudder and threatened to destroy the ranch. If we didn't get smart quick about BVD PI and real quick, it could ruin our reputation as registered seed-stock producers and decimate our ranch income. Although at the time there was no law in Texas (like there is now) that makes it illegal to sell BVD PI animals to others without disclosing their status, doing so was just something we could not live with. We wanted no one else to be in the situation we were facing. Overnight, a calf that was worth $500 suddenly became worth $15 – $25. Times that by nineteen calves and then skip an entire breeding season (no calves) the next year. That was a tough financial pill to swallow. But disclosing and selling saved us the mental anguish of nineteen bullets and a gigantic hole in the ground.

It's hard to prove where we picked up BVD definitively, but it is our hunch that we picked it up at that sale where we took the group of F1s. Like preschools, sale barns are not bad, but they can be where our cattle and children are first exposed to things that will challenge their immune systems. Our F1 first-calf heifer was born on our place. She was short-bred, about three months along. We would later learn that she was most susceptible to being infected with a BVD transient infection that would cause her to have a BVD PI calf. The F1 heifer would later test negative, and her calf would test BVD PI positive. At the time of discovering the BVD PI positive calf, no other calf on our ranch tested positive. We tested them all. (And soon, we would test every bovine on our ranch – cow, heifer, calf, and bull along with all our sheep – and every one would also test negative). That calf that was positive was the largest, the healthiest looking, and the earliest born on our ranch that year. It was also the last calf we tested. Wrapping up for the night, we thought we were in the clear. We were running the SNAP BVDV Antigen Tests from IDEXX chute-side on the ranch. Each test required a triangle-shaped ear notch from each calf. We placed each notch in a test tube along with the testing solution. A few swirls later and the tube sat in a rack for ten minutes. Then we poured the solution into a cartridge. When the color moved across the screen to a window, we would snap the one side of the test down and set another timer for ten minutes. It was kind of like checking a woman's pregnancy test. One dot and the result was negative; two dots and the result was positive.

That night in the cold of November, we were almost done and all the tests so far had one dot. We had portable heaters running and were ready to get inside with family visiting for

Thanksgiving. There were just a few tests left to read and it would be all over. The testing would be done. We could declare the herd BVD PI free. But in the fluorescence of the overhead light that hung above the squeeze chute, my eyes watered as they strained to see . . . was that a second dot on the very last test? I checked with the others. It was faint, but it was there. I checked the instruction booklet for the test. What if it was really faint? Did it count? According to the instructions, it did. But what if we made a mistake in running the test? It was the biggest, healthiest calf . . . the last animal tested in the entire herd. How could it be? We frantically retested and sat on pins and needles through the twenty minutes required to test – ten for the solution and ten for the snap cartridge. And there it was again, a bright blue check dot and a faint positive dot. A dot forever ingrained in our memories and that foretold the almost destruction of our small ranching operation. We had kept that yellow-tiger-striped F1 that was short bred because we wanted a few more dollars for her. It was her calf that was the BVD PI. We thought we had the best interests of our ranching operation at heart. But that decision almost ruined us. There is a thing called ranch biosecurity that we have since learned a lot about.

So why were we testing our entire herd in the cold of November? It was a wild hunch. We kept good calving records in Cattle Max, a software program we love and first learned about the Texas A&M Beef Cattle Short Course many, many years ago. We had pregnancy tested our cows and first-calf heifers and noticed that our conception rate was lower than usual. About seventy percent instead of the usual ninety-five-plus percent. We did our own pregnancy testing. We would run the cow in the chute, take a blood sample from her tail, and mail it off to the lab. It wasn't hard to do and at the time only cost $3.00 per sample. It was a good thing to do for managing our herd and keeping records and let us know what to expect in calving season. But in the fall of 2018, our pregnancy rates were lower. Not catastrophically lower, but enough to be noticeably lower. Thirty percent of our cows were not bred. We racked our brains . . . had it been a dry year (no, not that should create this issue). . . had we pulled the bulls out too soon (no, we had a defined ninety-day calving season) . . . was a bull infertile (no, after pulling bulls each year, we get a breeding soundness exam on each one to make sure). What was it? It wasn't trichomoniasis. We test the bulls each year before and after our breeding season. Perhaps we didn't send big enough blood samples to the lab for pregnancy testing. If your sample isn't big enough, then you could have false negatives. There we a few cows we remember it was hard to get enough blood from. Perhaps that was it. We pulled out the submission form. On our copy, we had marked for our own reference, which ones were low on blood for a case just like this. Unfortunately, the theory didn't hold. The open ones were not the ones with smaller blood samples submitted. However, looking at that submission form again, we noticed there was a place to test that same blood sample for two other things . . . BVD PI and Johnes. Until then, we had ignored these two other columns.

We had heard of Johnes. It was something that caused weight loss and diarrhea in adult cattle . . . a wasting disease. That wasn't something we seemed to have. But what was BVD PI? We hadn't remembered hearing of it before and the acronym didn't seem to give any clue as to

what it might be. It was on the same sheet as our pregnancy testing, so we hypothesized it could be a disease affecting breeding rates. A quick internet search came up with bovine viral diarrhea disease. That didn't sound like a breeding disease . . . until the next paragraph. Oh my. This was a Pandora's box. This could be bad. This could be what we had. So a call to our veterinarian to enquire about BVD and the comment on the phone is, "You don't have it. You don't have sick calves. You only have to doctor 1 – 2% of your calves a year. Don't worry about it. You would know if you have it." It turns out in hindsight we just weren't far enough into our BVD journey for the veterinarian to suspect it without testing.

This is where divine intervention or perhaps just trusting a gut feeling came into play. We didn't take our veterinarian's advice and forget about BVD. We decided that we would test the calves. Doing so would eliminate one possibility and allow us to advertise a BVD PI free herd. Then we would move on to figure out the real reason for our low conception rates that year. We figured it was probably some management decision we had made, not some disease. So we ordered one hundred twenty-five snap tests and got to testing the calves. And that, my friends, is the beginning of the lifelong relationship we will now have with BVD. Please don't get me wrong. Our herd is now free of BVD. We have no permanent and no transient infections. One of the great things about the ranching and cattle world is that people will help people; it's a small connected world. We came to the BVD seminar at the Texas A&M Beef Cattle Short course and approached Dr. Hairgrove at Texas A&M and Dr. Falkenberg, who was with the USDA. They offered to help us. Our experiences hopefully provided researchers with additional information on how BVD PI works and how to test for it. With lots of blood, sweat, and tears . . and a skipped calving season (no income that year) we eradicated BVD from our herd and closed our herd (a part of our biosecurity measures). We now help others learn about BVD PI and hopefully prevent it from happening to others. We advocate for doing the right thing, even when it hurts. For us, that meant not selling those BVD PI calves into channels where they would infect others and perpetuate the disease.

BVD and the resulting BVD PI is a devastating disease. BVD is talked about a lot in feed yards. There it produces economic losses ranging from sick calves needing doctoring, to calves that won't grow, to death losses. It isn't being talked about as much in cow/calf operations, but it should be. BVD PIs are created in the cow/calf operation. Let me repeat that. BVD PIs are created in the cow/calf operation. BVD PIs are created in utero. The momma gets a transient infection, and when conditions are right, the growing calf in her uterus becomes permanently infected. We have been free of BVD now for many years, but we still test every calf every time to make sure BVD – that Trojan horse – never sneaks back into our herd. It only takes a transient infected animal across the fence to rub noses with our cattle to bring it back into the herd. We practice biosecurity measures. When neighbors put cattle in pastures bordering our ranch, they give us advanced notice and we move ours away. We no longer buy bred heifers or cows from ranches that are not BVD PI free. We won't ever let a short bred animal that leaves our place to come back into the herd.

Not only is BVD created on the cow/calf producer's ranch, but the financial impact it causes there, I would argue, is even larger than in the feedlot. The loss in conception rates (or perhaps, conversely, the increase in early embryonic losses) coupled with sick calves and death losses are not only financially, but also emotionally, devastating. No one wants to have calves that can't walk right, can't see right, and that die no matter what you do. But just because you don't have these, doesn't mean you don't have BVD. And that's the tricky part. Other than the decreased conception rates, our first year of being infected with BVD PI, there were no other indications. Our infected calf was the largest and the healthiest. That's one of the sneaky ways BVD works. It wants to propagate and spread and so some of the infected calves will appear healthy and thrive. If they all died, it wouldn't spread to other calves in utero.

In our situation, we went from one calf being infected to the second year with nineteen (or twenty percent of our calf crop) infected. Some of these infected calves were bull and replacement heifer quality. We never would have suspected anything was wrong with these big, healthy, beautiful calves without testing. If we hadn't had detected BVD, what would the third year's calf crop infected percentage have been?

We decided we would skip an entire breeding season to eradicate the disease while we tested, eliminated PIs, and vaccinated. Oh, vaccinated you say . . . you just decided to vaccinate your herd so your calves won't get BVD, am I right? Even the drug companies that make the vaccinations will tell you that you can't vaccinate your way out of BVD if you have a BVD PI animal. It can help. You can reduce the number of animals that will get a transient infection. You might, depending upon the vaccination, prevent some PI infections. But you cannot vaccinate your way out of the situation without testing and eliminating BVD PI calves from your herd. Now this isn't a head in the sand kind a thing. I can hear you saying, "But I don't want to find it and have to skip a calving season. It would be better to leave well enough alone." I really looked up to my grandfather. He seemed to know everything. I remember him telling me as a child that it is always better to know the facts, even when you are facing something bad, because then you can decide what to do. Not knowing takes away your opportunity to make those decisions.

You don't have to make the same decisions we did. You can vaccinate each year, remove the PIs, and reduce the herd's infection over several years. For us, emotionally, we could not face this dragging on year after year. The stress of not knowing how many calves would be BVD positive each year was much worse than taking one big hit and skipping a breeding season. We had been trying to shift our calving season from May, June & July to January and February by breeding one month earlier each year. This was a chance to do it in one fell swoop.

Dr. Hairgrove tells the story of a smart, successful producer who eradicated BVD from his herd, but got it again when he bought a group of bred heifers. I am grateful each time I hear it. It is my reminder to be vigilant with our own herd and our practices. We have to help and educate others around us. It is only through helping one another and being honest with our customers and each other that we will stop this disease in its tracks.

As a new or inexperienced producer, there are a lot of things to learn. Even as experienced producers, ones who have taken care of cows their entire life, we can't be experts in everything. But the ranching world is a small world and a friendly world. . . one where neighbors help neighbors and one where everyone is a "neighbor" even if they are states or continents away. If we can help you, please reach out to us. Many thanks to Dr. Hairgrove for all his help and support over the years and for asking us to share our story with you here today.

JR & Kara Jones

Hoof & Hide LLC

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UNDERSTANDING BOVINE VIRAL DIARRHEA IN BEEF HERDS

Tom Hairgrove[1], Tammy Beckham[2], and Jason Banta[3]

Bovine viral diarrhea (BVD) is a disease of cattle that may affect the respiratory, immune, nervous, blood or circulatory, and reproductive systems. Scientists first believed this disease was related to diarrhea and damage to the intestinal tract, hence the name bovine viral diarrhea.

About 70 to 90 percent of all BVD infections go undetected. The most economically important consequence of this disease is reproductive loss, which is increasing in the United States.

A virus propagates in one of two ways, "hit and run" or "infect and persist." In the "hit and run" scenario, when an animal is infected it will either recover or die. An animal infected with BVD usually recovers or dies within about 1 week. BVD would behave this way in a group of stocker or feeder animals. If the infected animal passes the BVD virus along to another animal before it recovers or dies, the virus survives; if it does not, the virus dies out. Animals infected in this way are said to be transiently infected, or temporarily infected for a short time.

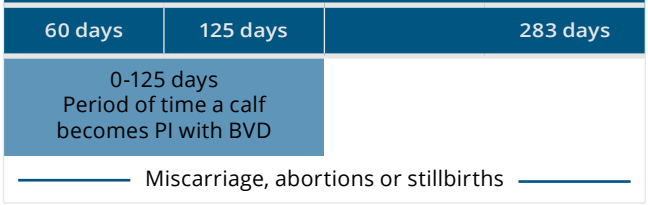
BVD also behaves in the "infect and persist" mode because it can be passed from cows to their unborn calves. If they live, these calves will remain infected all their lives and are said to be persistently infected (PI). Understanding BVD persistence is essential to designing an adequate disease control program.

There are two biotypes of the BVD virus, cytopathic (CP) and non-cytopathic (NCP). Non-cytopathic virus predominates in the cattle population and is the biotype responsible for persistent infection.

INFECTION DURING PREGNANCY

Figure 1 depicts the gestation timeline of a cow. During the first 4 months of pregnancy a cow that becomes infected with non-cytopathic BVD may pass the virus to her unborn calf. The calf 's developing body fails to recognize the BVD virus as foreign, so the calf remains persistently infected for life ("infect and persist").

Fig. 1. Gestation timeline of cow.



The major reservoir and source of BVD virus is persistently infected cattle, and this is primarily how the BVD virus remains alive in nature. The PI animal sheds the virus continually and infects other animals. If the infected animals are not pregnant females, they usually have a relatively short sickness and either recover or die. However, if a pregnant female encounters a PI animal during the first 125 days of gestation, her calf may become persistently infected. If a pregnant female is herself PI, she will always produce PI calves.

Pregnant cattle infected at any time during gestation can abort. Many, but not all, cows infected from 9 days before breeding to 45 days of gestation will miscarry. The virus may infect the reproductive tract and prevent conception or hamper development of the embryo or fetus.

Cows infected between day 100 and day 150 of gestation that do not abort may produce calves with deformities, primarily of the nervous system. These calves often have problems walking, sometimes have problems with eye development, and occasionally lack normal hair development. Growth retardation occurs more rarely.

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Cows infected between day 125 and day 150 of gestation may produce normal calves, but the calves are twice as likely as noninfected calves to experience severe illness in the first 10 months of life.

THE SPREAD OF BVD FROM FARM TO FARM

BVD usually spreads between farms when new cattle are introduced that are persistently infected or are carrying persistently infected calves. BVD is often introduced when infected stocker cattle have contact with pregnant females, either when the groups are comingled or when they have contact across a fence. Bulls infected with BVD can shed virus in their semen. All bulls purchased should be tested to ensure that they are not infected.

The role of wildlife in the spread of BVD is not certain at this time. BVD does replicate in wild ruminant species such as camels, deer, elk and bison, but it is difficult to predict the importance of wildlife in spreading BVD to cattle populations.

SYMPTOMS OF BVD

In beef herds, several cows in a herd usually will abort a short time before calving season. At the beginning of calving season, premature births and stillbirths occur. Weak calves are generally born during the first 2 to 4 weeks of calving season. Some calves are born alive but they die quickly. Even with intensive care most infected calves die within a few hours.

Losses tend to be epidemic when BVD is first introduced into a non-immune pregnant herd. Once PI animals establish infection in the herd, losses continue but not as dramatically.

CONTROLLING BVD

1. Quarantine all replacement animals for at least 21 days to ensure that they are not temporarily infected with BVD. Test all replacement breeding stock, and any animals that may have contact with breeding stock, for PI status.
2. Isolate all new cows that are pregnant until they have calved and all their calves have been tested for PI status and are found negative. It is important to test these calves before the dams are rebred to eliminate the possibility of producing more PI cattle.
3. Dispose of all PI cattle in an ethical manner. Placing these cattle back in the livestock marketing system, where they may infect other cattle, is irresponsible. Instead, infected cattle should be euthanized, sent directly to slaughter, or fed in isolated pens.
4. Vaccination can prevent or slow the “hit and run” version of the BVD virus that produces temporary disease. Vaccinating cows to prevent the infection of calves, and thus the birth of PI cattle, is helpful but not 100 percent effective.
5. Establish a biosecurity and vaccination program that is tailored to your operation, with advice from your veterinarian. The timing of vaccination and the choice of vaccine (modified live vs. killed) are management decisions that will vary with individual operations. Always follow label instructions and Beef Quality Assurance Guidelines when using any vaccine.

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Body Condition Scoring Beef Cattle

Body Condition Scores

1 to 9 system:

1= very thin

9 = OBESE

Priority of Nutrient Use

Cow's perspective

1. Maintenance
2a. Growth
2b. Lactation
4. Reproduction

Priority of Nutrient Use

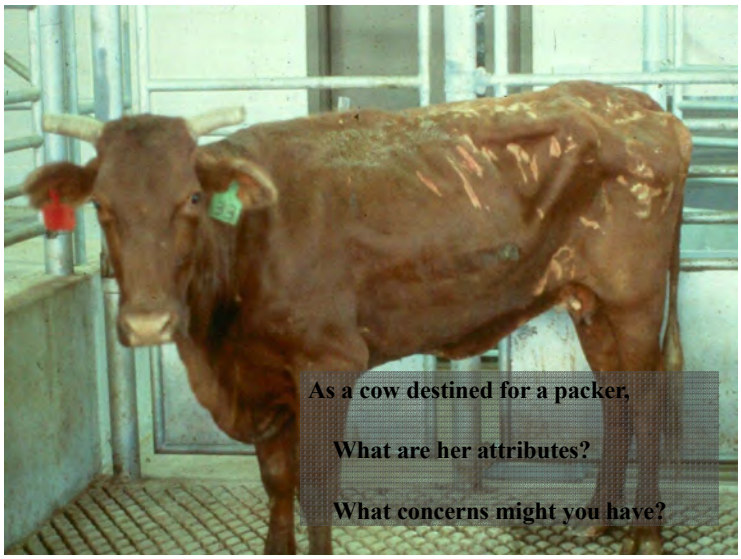
Cattleman's perspective

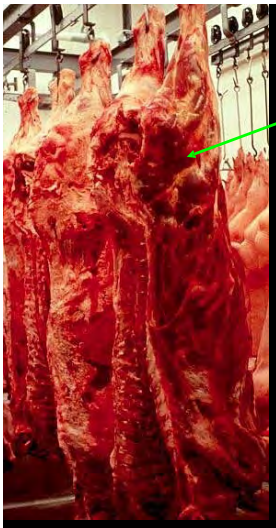
1. Reproduction
2. Lactation 3a. Maintenance
3b. Growth

Priority of Nutrient Use

Dairyman's perspective

1. Lactation
2. Lactation
3. Lactation
4. Lactation





Dark color

Attributes

✓ Lean yield

Value

Concerns

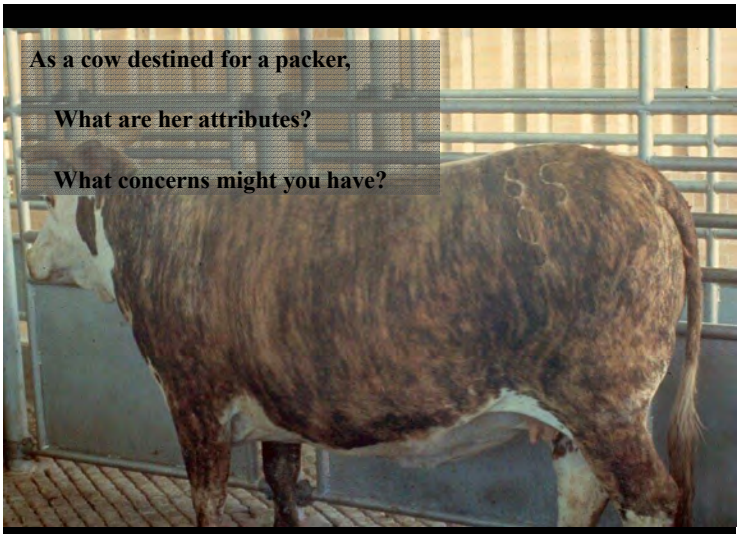
x Bruising

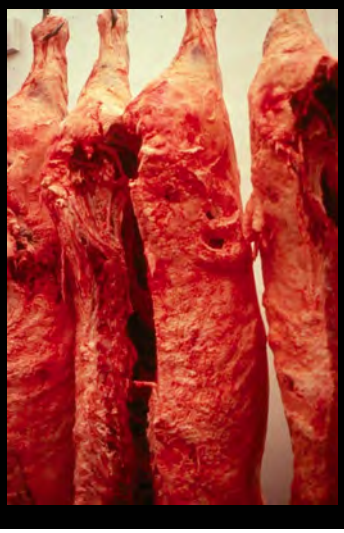
x Trim loss

Emerging Issues Most Frequently Cited

Packers	Affiliated Organizations
1. Bruises	1. Pricing & Prompt Payment
2. Antibiotic Residues	2. Antibiotic Residues
3. Birdshot/Buckshot	3. National ID & Verification
4. Arthritic Joints	4. Pathogen Control
5. Yield (DP & product)	5. Injection-site Lesions
6. Condition (leanness)	6. Birdshot/Buckshot
7. Condemnation Rate	7. Anti-microbial Resistance

National Market Cow and Bull Beef Quality Audit (NCBA/CSU, 1999)





Fat cows are Wasty

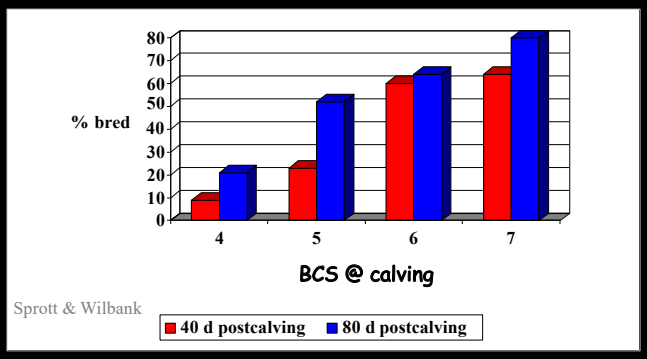
Concern

x Trim loss

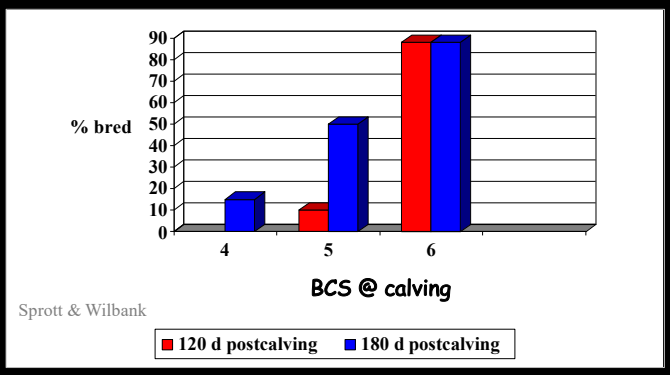
Remember, the primary product harvested from cull cows/bulls is lean red meat.

Relationship between Body Condition Score and Reproduction

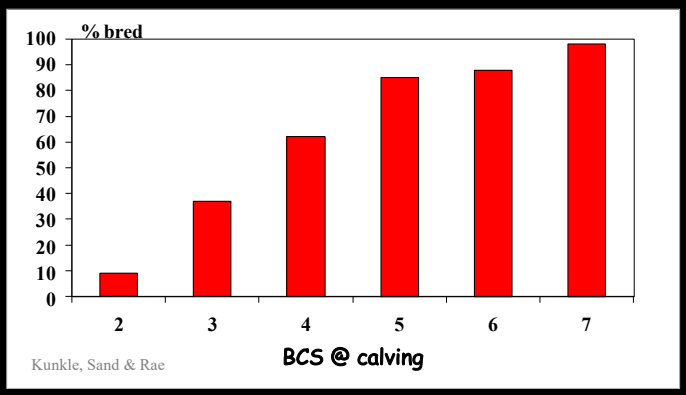
Effect of Body Condition on Rebreeding by 1st Calf Santa Gertrudis Heifers



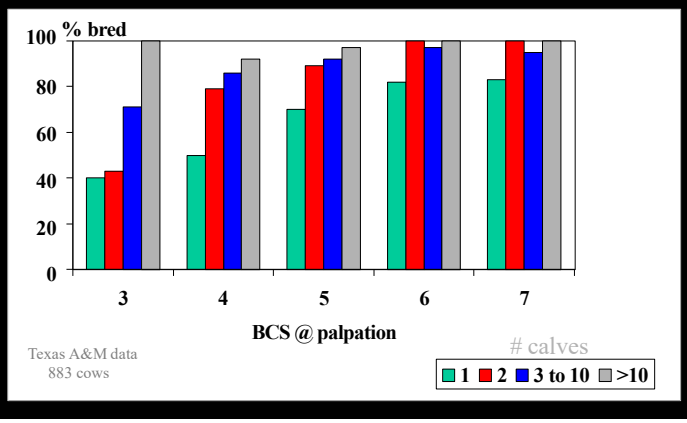
Effect of Body Condition on Rebreeding by 1st Calf Hereford Heifers



Body Condition Score vs. Conception Rate (Florida study, mature cows)



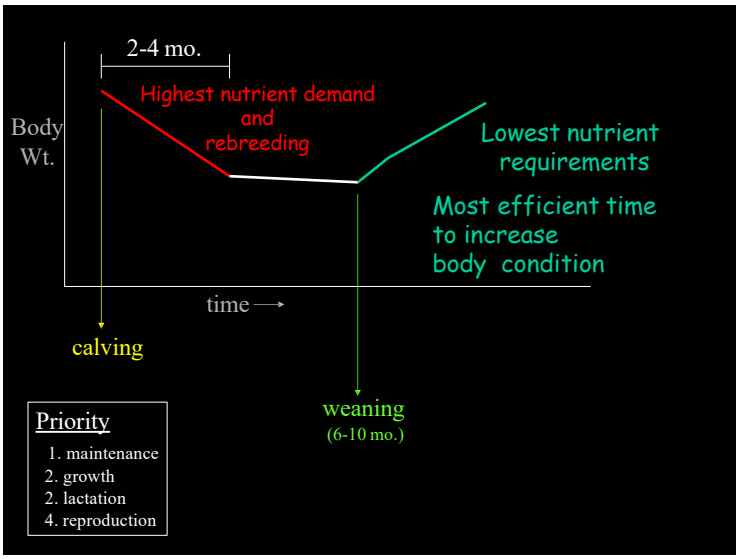
Body Condition Score vs. Conception Rate

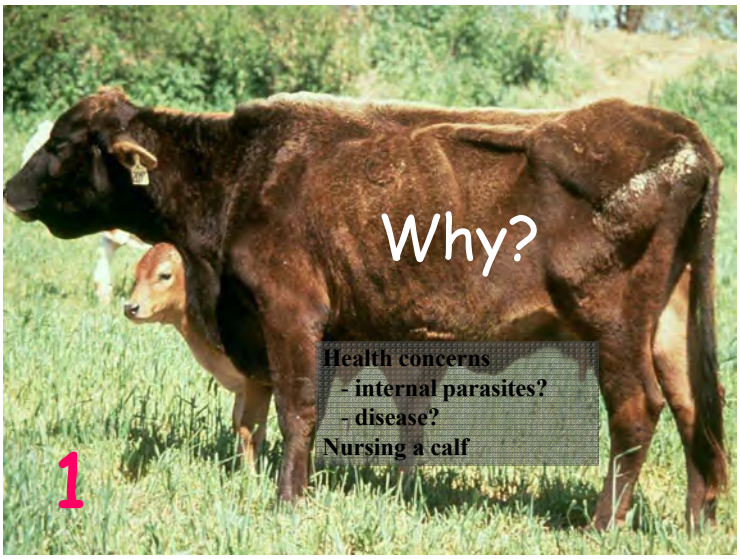


Body Condition Score

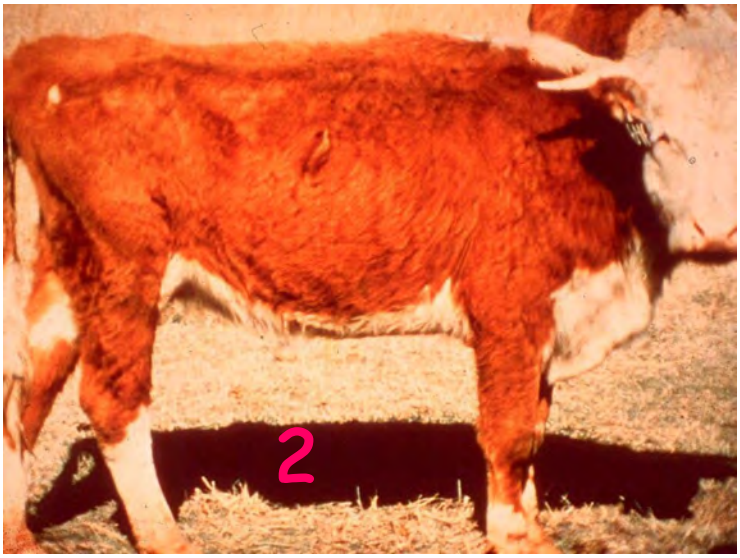
Critical time:

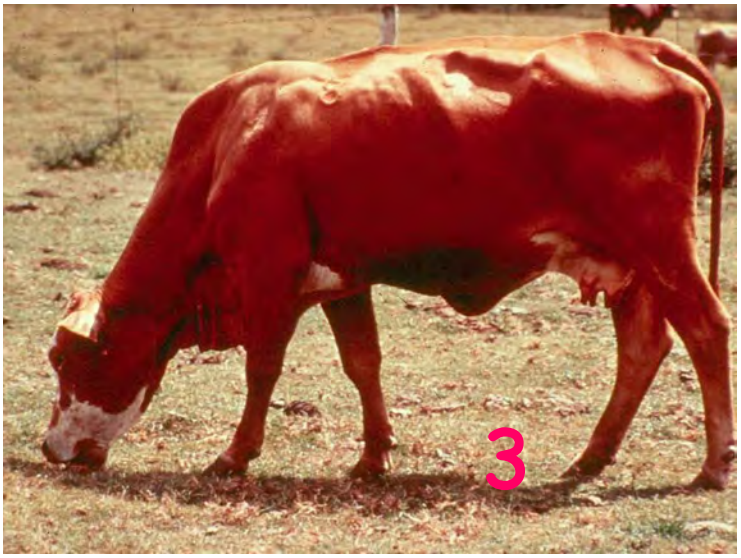
at calving






Why?
Forage Availability
- not enough to eat
Nursing a big calf
1


2

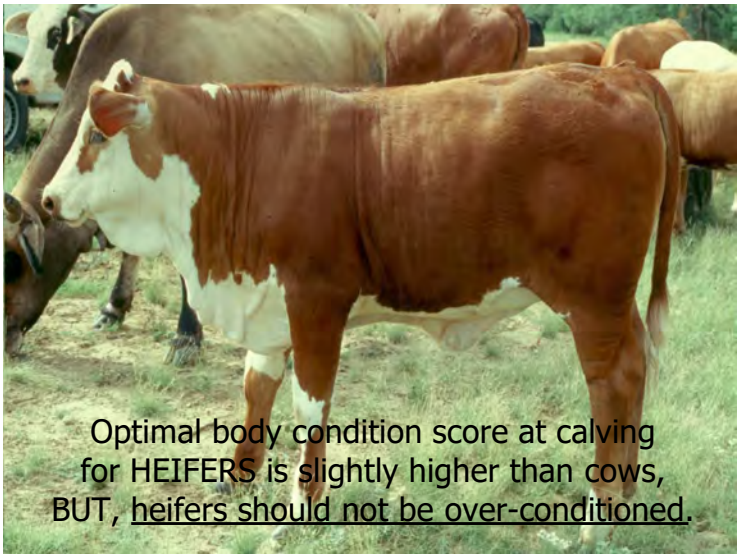

3


4

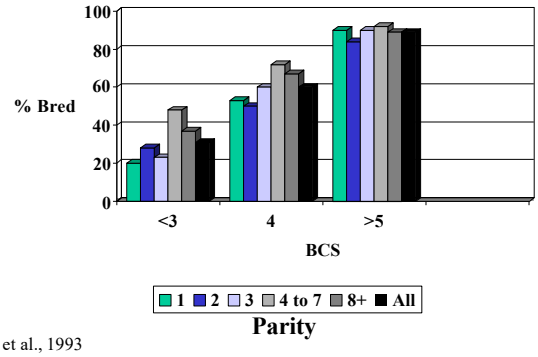

5


6

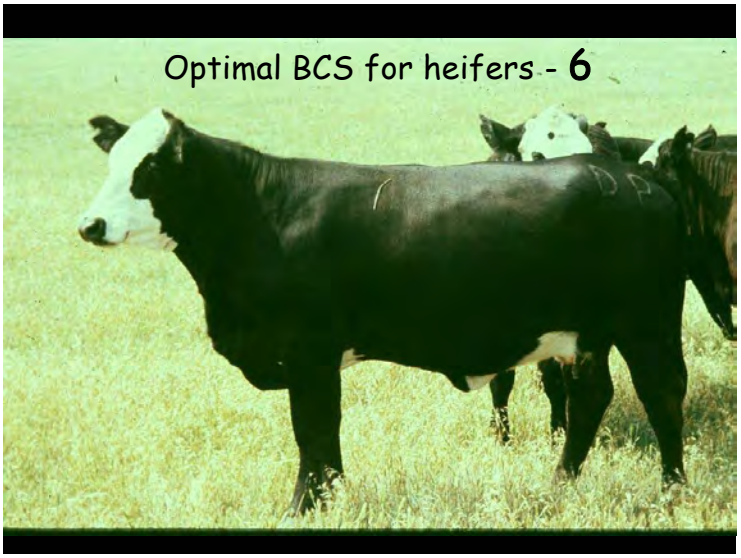
Exceptions to the rules



Relationship of Parity and BCS to Pregnancy Rate

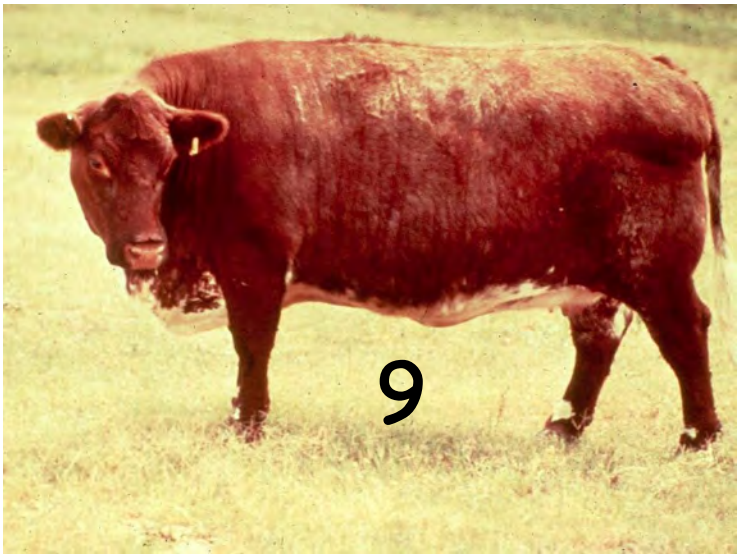


Rae et al., 1993

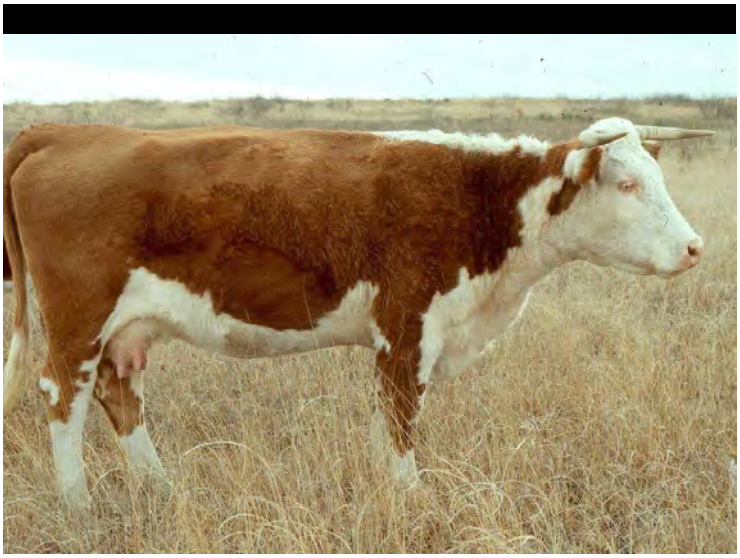


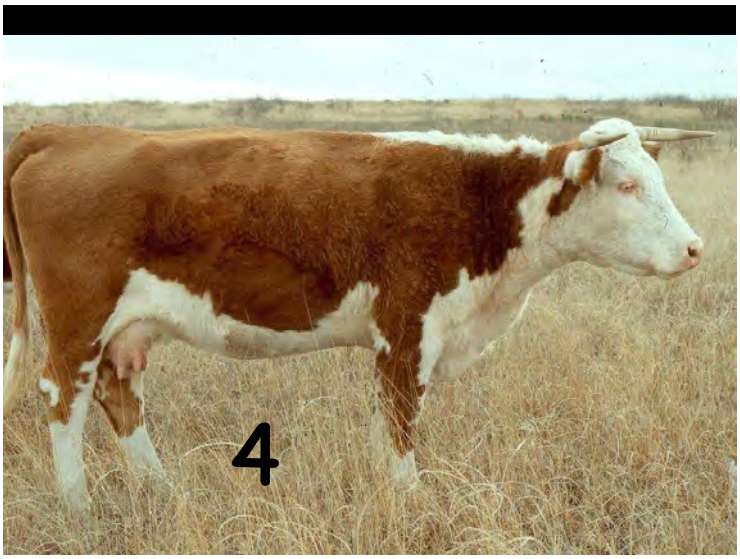
Exam Time

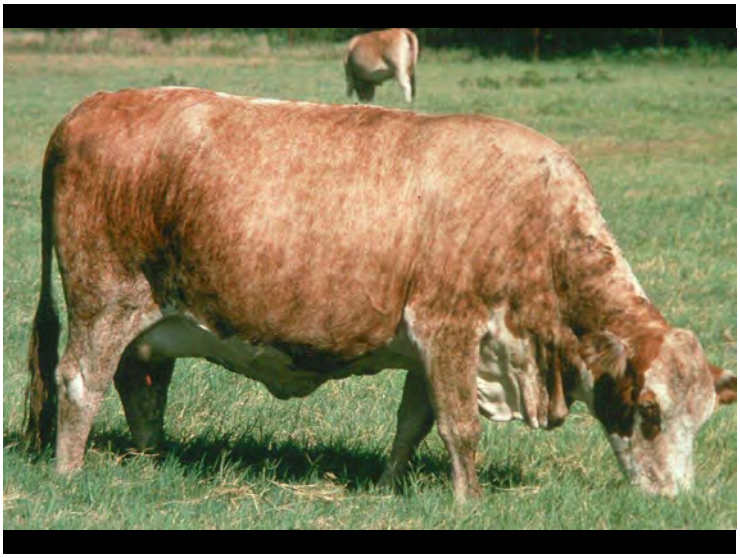
Look at the following slides and make your assessment of the cow's body condition. Then left-click to reveal the condition score.

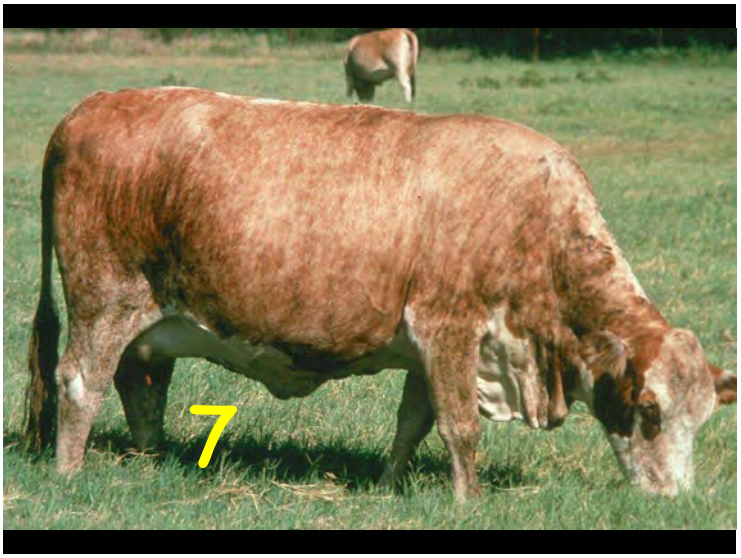

9


1




4





Body Condition Score

Summary

1. Related to reproduction
2. Reflects nutrient intake
3. Indicator of forage availability qu

Rick Machen, Ph.D.
Extension Livestock Specialist
Texas Cooperative Extension

for
more
information

http://animalscience.tamu.edu

Trace Mineral Supplementation of Grazing Beef Cattle

John D. Arthington, Professor and Center Director

Range Cattle Research and Education Center
University of Florida / IFAS

Introduction

The nutrient quality of forage, particularly perennial warm-season grasses, is often lacking in trace mineral content to supply the requirements of most all classes of grazing cattle. Since forage is the most significant contributor to the trace mineral nutrition of grazing beef cattle, it is important to consider these deficits and how they may impact the performance of the cattle consuming them. Focusing on grazing beef cattle, this proceedings article will provide an overview of (1) individual essential trace minerals, (2) trace mineral antagonists, (3) methods of supplementation, and (4) assessment of herd trace mineral status.

Review of Individual Trace Minerals Essential to Grazing Beef Cattle

Copper

Copper is one of the most common trace nutrient deficiencies in grazing cattle. Copper is an important cofactor in approximately 30 enzyme systems. Deficiencies occur through the prolonged consumption of forages low in Cu and/or the consumption of forages containing elevated concentrations of the natural Cu antagonist, Mo. As well, dietary S is an important catalyst in the Cu / Mo interaction. Dietary S levels greater than 0.30 % are often considered suspect in their potential for initiating Cu deficiency. Blood Cu concentrations are elevated during instances of stress, suggesting that stressed cattle may have a greater Cu requirement. Copper oxide is poorly absorbed and should not be used as a source of Cu in cattle supplements. Some signs of Cu deficiency include, (1) immune suppression (failure to respond to vaccination), (2) rough, dull hair coat, and (3) anemia.

Zinc

Like Cu, Zn is also an important cofactor in many enzyme systems. In ruminant diets, Zn deficiency has been shown to be an important contributor to male fertility. As well, diets fortified with adequate available Zn have been shown to improve hoof structural soundness in beef heifers. Copper and Zn are absorbed through similar pathways indicating a competition for absorption pathways. Therefore, mineral supplements should be formulated with a Cu:Zn ratio of around 1:2 or 1:3. Some signs of Zn deficiency include, (1) connective tissue degeneration (hoof integrity), (2) bull reproductive failure (especially young developing bulls), and (3) anorexia and weight loss (notably in calves).

Selenium

Selenium deficiency in grazing cattle is widely recognized throughout the world. Unlike most other essential trace nutrients, Se supplementation offers a narrow range between deficiency and toxicity. In fact, many regions in the world are concerned with Se toxicity in pasture forages. Selenium is essential for the maintenance of tissue integrity. Widely recognized deficiency

symptoms include the degeneration of tissue resulting in a condition known to as “white muscle disease”. Selenium supplementation is commonly addressed via the inclusion of sodium selenite in cattle supplements. Selenium inclusion is federally regulated in the United States at a maximum inclusion level not to exceed 3 mg of supplemental Se daily. If adequate mineral intake is achieved, Se deficiency is rarely a problem when adequately supplemented via sodium selenite. Some signs of Se deficiency include, (1) muscle degeneration (white muscle disease), (2) reproductive failure, and (3) immune suppression. Selenium is closely related to vitamin E. In fact, consideration to Se nutrition cannot be made without also considering vitamin E. A lack in either may be compensated by the other.

Manganese

Manganese has been shown to be an important trace nutrient for proper bone formation in young animals and optimization of fertility in female cattle. Although dietary Mn absorption and retention in cattle is low, Mn deficiency in grazing cattle is uncommon. Considering the importance of Mn on cow fertility and young calf development, it is most important to focus on optimal Mn nutrition prior to and following calving. Manganese sulfate is the most available form of Mn, but it is often difficult to find commercially. As an alternative, Mn oxide is an acceptable and widely used source of Mn supplementation. Some signs of Mn deficiency include, (1) bone abnormalities, (2) reduced growth rate, and (3) reduced fertility.

Iodine

Iodine is critical for the maintenance of proper thyroid function. This occurs through the essential role of I in the regulation and synthesis of thyroid hormone production. The influence of thyroid hormones affects nearly every physiological process in mammals. Ethylenediamine dihydroiodide (EDDI), often provided in trace mineral supplements as a foot rot preventative, provides a quality source of available I. As well, the inclusion of iodized salt in the base mineral mix may provide adequate I supplementation in most cases. Some signs of I deficiency include, (1) reduced fertility, (2) enlarged thyroid (goiter), and (3) stillborn, weak, and/or hairless calves.

Iron

Iron deficiency is seldom a problem in cattle consuming forages. In contrast, the antagonistic impact of dietary Fe on Cu absorption is often more of an important issue when attempting to balance trace mineral nutrients. Further, many ingredient sources of other trace nutrients are naturally contaminated with Fe. Taken together, additional supplementation of Fe to grazing cattle is probably not a concern. Iron deficiency is occasionally an issue in young calves or in adult cattle suffering blood loss usually as a result of parasite infestation. Iron is provided in most all trace mineral supplements in the form of Fe oxide. This inclusion is provided only as a coloring agent, providing the classic dark red appearance of many salt-based mineral supplements. Iron oxide is basically *unavailable* to the animal. If supplemental Fe is needed, Fe sulfate should be considered. Some signs of Fe deficiency include, (1) anemia, (2) immune suppression, and (3) decreased calf weight gain.

Cobalt

Cobalt is essential to ruminants through its participation in the ruminal synthesis of vitamin B12. This metabolic process, unique to ruminants, allows us to virtually ignore the dietary supplementation of B-vitamins in cattle. In fact, since Co is poorly stored in body tissues, Co status in ruminants is commonly assessed via measurements of blood vitamin B12 concentrations. Multiple Co sources are utilized in mineral formulations, including carbonate, chloride, and sulfate. Some signs of Co deficiency include, (1) loss of appetite leading to weight loss, (2) listlessness and diarrhea, and (3) anemia.

Trace Mineral Antagonists (Iron, Molybdenum, and Sulfur)

The requirement for supplemental trace minerals can also be greatly impacted by the presence of mineral antagonists. These antagonists cause trace mineral deficiencies to be grouped into two broad categories depending on the characteristics of their development; 1) Primary deficiency, and 2) Secondary deficiency.

1. Primary mineral deficiencies are the result of the consumption of feeds that are naturally low in one or more trace minerals. These deficiencies usually require an extended period of time for their development, often a year or more. The lack of supplemental mineral is a common characteristic of primary mineral deficiencies, as they are rare under normal well-managed cattle production systems.

2. Secondary mineral deficiencies are the most common. Secondary deficiencies are derived from the consumption of one or more mineral antagonists that interfere with the normal metabolism of another mineral. A simple mineral evaluation of a feedstuff may suggest adequate trace mineral concentrations are present; however, the presence of a mineral antagonist will decrease the availability of the mineral, potentially leading to a deficiency.

Iron Antagonism

Iron is the second most common trace metal in the earth. Iron is found in nearly all sources of cattle feed, including water. As well, a considerable amount of Fe may also be digested through the intake of soil during grazing, as well as the soil contamination of harvested forages. Indeed, with the exception of young animals, Fe deficiency is rare in healthy cattle reared under modern agricultural conditions. The more likely contribution of Fe to cattle is its ability to antagonize other trace minerals, notably Cu and Zn. The maximum tolerable concentration for Fe in cattle diets is 1000 ppm; however, dietary concentrations of 250 to 500 ppm have been linked to Cu deficiency. The antagonistic role of Fe in Cu nutrition is not well understood. One explanation relates to the potential disassociation of ferrous sulfide complexes in the low pH of the abomasum. Under this scenario, sulfide may be able to react with Cu, forming insoluble Cu-sulfide complexes. Reductions in the performance of dairy cattle in New Zealand have been linked to Cu deficiency as a result of the consumption of high-Fe forages.

Molybdenum Antagonism

Molybdenum is an essential trace element required by all animals; nevertheless, reports of Mo deficiency are rarely recorded. In contrast, the antagonistic impact of Mo on Cu metabolism has been recognized for many years. Typically, Mo exerts its influence on Cu through the association with S in the formation of ruminal thiomolybdates. However, additional evidence exists which shows that decreased animal performance can be related to Mo toxicity independent of decreased Cu availability. Heifers consuming supplemental Mo (dietary concentration = 5 ppm) have been shown to exhibit signs of Cu deficiency, whereas, heifers supplemented with Fe and at the same Cu status had no signs of Cu deficiency. In these studies, the signs of Cu deficiency included reduced growth and feed efficiency and infertility (Phillippo et al., 1987a,b). More recently, infertility responses have been further linked to the direct impact of Mo and have been shown to be reversible with supplemental Cu (Kendall et al., 2006). In another study (Gengelbach et al., 1997), calves provided diets with supplemental Mo had a lower rate of gain compared to Fe supplemented calves. Both groups of calves had an equivalent extent of Cu depletion compared to Cu-supplemented control calves. These results suggest that some conditions, which are linked to Cu deficiency, might be more accurately described as a toxicity from the antagonist (i.e. Mo toxicity).

Sulfur Antagonism

Sulfur is found naturally in nearly all feedstuffs. The form of S varies widely from inorganic salt to organic S-containing amino acids. Recently, more evidence has been derived from commercial cow/calf production systems suggesting that S may be a primary contributor to secondary Cu deficiencies. Although Mo is an essential component in this antagonism, it will seldom affect tissue Cu stores when S levels are limiting. Sulfur, on the other hand, can impact both Cu and Se metabolism by forming insoluble sulfide complexes – independent of Mo (Suttle, 1974). We have found that a dietary concentration of S of 0.30% (total S) is sufficient for this antagonism to become a concern. The beef cattle NRC suggests a maximum tolerable concentration of dietary S of 0.40 %. Grazing cattle can achieve S from multiple sources including, (1) S-containing fertilizers (Arthington et al., 2002), (2) high-S byproducts (i.e. distillers grains, sugarcane molasses, and feathermeal; Arthington and Pate, 2002), (3) high-sulfate water sources, and (4) atmospheric deposition (i.e. acid rain).

Trace Mineral Supplementation

Supplementation of trace minerals may occur through a variety of means, including free-choice loose mineral mixes, trace mineral blocks, fortified energy and/or protein supplements, injections, boluses, and forage biofortification.

Free-Choice Loose Mineral Supplements

Free-choice mineral supplements are offered with the anticipation of adequate intake to offset nutrient deficiencies. Variation in free-choice intake, however, is a common problem impacting the efficacy of this management system. Although many contributing factors exist (Bowman and Sowell, 1997), variation due to changing seasons of the year is one common factor. When

supplementing free-choice minerals it is important to realize that cattle ***do not*** have the nutritional wisdom to consume trace minerals as needed. We have all heard the statements, "My cattle are not consuming mineral, so they must not need it" or, "My cows are eating four times their normal level, I guess they really need it". Cattle only possess the ability to consume salt at the level of their requirement. Consequently, by altering the salt inclusion in mineral mixes, we can both encourage and discourage mineral intake. Remember that the majority of trace mineral intake beyond that nutritionally required by the animal is excreted in urine and feces. Over consumption of trace mineral may be an important inefficiency in beef cattle production systems.

Despite challenges with intake variation, free-choice mineral supplementation is the most common supplementation strategy in grazing beef herds. In nearly all cases, it is an effective, cost-efficient means of delivering adequate mineral supplementation. Although formulations vary greatly, the common base mix should contain approximately 20 to 30% salt. Intake is often targeted at two to four ounces per head per day. Unfortunately, achieving this target intake by all animals does not occur. Several animals within a herd will consume very little to no mineral at all. However, on the average, mineral consumption usually meets the desired intake levels. It is this averaging effect, over time, which allows free-choice mineral supplements to be the most practical choice for most cattlemen. Seasonal variation is evident. During the wetter summer months, cattle readily consume salt-based mineral supplements. In contrast, during the dryer winter months free-choice intake may be reduced by 15% or more. Generally, as moisture content of forages increase, intake of free-choice supplements also increases. In one demonstration study, the voluntary intake of a salt-based free-choice supplement among grazing beef cows was surveyed over two consecutive years. Voluntary intake was correlated (R^2 = 0.39) with precipitation events in the two preceding months (data courtesy of Vigortone Animal Nutrition; unpublished data). Similarly, we reported a large seasonally-impacted reduction in voluntary intake of salt-based, free-choice mineral supplements among grazing beef cows in southern Florida (Arthington and Swenson, 2004). In that study, cows were offered supplement in amounts to provide their targeted (assumed) intake on a weekly basis. All unconsumed supplement was measured weekly and the results were calculated as a percent refusal. During the dry season, when forage moisture was low, the percent refusal was high (i.e. voluntary intake was low); however, during the wet season, when forage moisture was high, voluntary intake was at or above the targeted amount. This annual variation in intake is explained by changes in a cow's craving for salt. Annual variation in salt craving differs throughout the world, but is an important consideration with supplementing grazing cows via salt-based, free-choice mineral products.

Trace Mineral Blocks

In most grazing situations, trace mineral-fortified salt blocks cannot provide sufficient trace mineral intake to meet nutritional needs. Formulated as a hard, salt-based block, cattle are unable to consume enough product to achieve their necessary level of trace mineral supplementation. Nevertheless, some grazing situations dictate the need for this type of supplementation. When cattlemen are physically unable to provide loose mineral or fortified supplements on a regular basis, trace mineral fortified salt blocks provide an opportunity to offer long-term mineral supplementation, therefore lessening the potential for trace mineral deficiency.

Fortified Energy / Protein Supplements

One of the most effective management strategies for addressing trace mineral nutrition in beef cows involves the mineral fortification of energy and/or protein supplements. This is a simple approach, which ensures that trace mineral is offered to all animals on a regular basis. This may be achieved by fortifying traditional supplements with your current free-choice trace mineral supplement. Some producers simply fortify their winter supplement and return cows to free-choice product during months when supplement is not offered. This strategy is effective in decreasing the variability in free-choice trace mineral intake and in bolstering trace mineral tissue stores during the winter supplementation period. As well, mineral fortified winter supplements lessen the concern of poor winter mineral intake sometimes realized with free-choice, salt-based mineral supplements.

Injectable Trace Minerals

Injectable trace minerals have been available for many years, but the technology, targeted application, and scientific assessment of efficacy has more recently been a subject of attention. An advantage of injectable trace minerals, compared with traditional oral supplementation methods is the targeted delivery of a known amount of trace minerals to individual animals. This removes the variability associated with annual fluctuations in voluntary intake, which is common among cattle provided free-choice mineral formulations. In addition, injectable trace minerals can be used within production environments that might experience difficulty managing the routine delivery of free-choice mineral mixes, such as extensive rangeland systems, seasonal grazing of mountain meadows, and seasonally flooded pastures. Further, the contribution of wildlife to the overall consumption and disappearance of free-choice mineral mixes also can cause complications in these production environments and add further value to the use of injectable trace minerals.

Most cattle producers and veterinarians associate trace mineral injections with two product formulations that were common during the 1970's and 80's. These products were, 1) combined formulation containing Se and vitamin E, and 2) Cu glycinate. Prior to 2005, most of the research involving injectable trace minerals has focused on one of these two applications. These studies have shown enhanced humoral responses to antigens such as *E. coli* and *Mannheimia haemolytica* (Droke and Loerch, 1989; Panousis et al., 2001) and increased or maintained serum Se among Se-injected calves (Reffett-Stabel et al., 1989).

A notable and widely recognized problem with injectable Cu is injection-site reactions. Previous studies have reported variability in injection-site reactions among different preparations of injectable Cu supplements, with CuCa-EDTA causing the least and Cu glycinate causing the greatest tissue inflammation (Boila et al., 1984), and the s.c. injection route causing less tissue irritation compared to the i.m. injection route (Allcroft and Uvarov, 1959). Chirase et al. (1994) investigated injectable Cu glycinate (36 mg Cu) in BHV-1 challenged beef steers. Their results revealed a negative impact of injectable Cu on BW gain and feed DMI. The authors suggested that this response may have been due to the development of abscesses in 25 % of the Cu-injected

calves, which supports the importance of using injectable trace mineral products which cause as little injection site reaction as possible.

Today, there are injectable trace mineral formulations available that offer several elements in a single product, namely Cu, Zn, Se, and Mn. Reports of injection-site reactions are not as prevalent with these newer formulations. In addition, many studies have reported positive findings such as increased mineral status (Pogge et al., 2012), increased feed efficiency (Clark et al., 2006), improved humoral immune responsiveness (Arthington and Havenga, 2012; Arthington et al., 2014), reduced treatments for illness (Berry et al., 2000), and reduced morbidity treatment costs (Richeson and Kegley, 2011) in stressed feeder calves.

Trace Mineral Boluses

This form of supplementation involves the oral administration of a capsule (bolus) containing specific trace minerals in the form of highly compressed powders, soluble glass materials, or metal needles contained within a gelatin capsule. Administered using a balling gun, these boluses presumably drop into the reticulum-rumen where they dissolve slowly over time. Cobalt supplementation, for example, has been successfully applied to bolus applications for many years. Particularly for Co, bolus supplementation strategies are useful since the rumen microbes will receive a continuous supply of Co for the production of vitamin B12.

Availability of commercial sources of trace mineral boluses varies throughout the world. Reasons depend on, 1) the degree of extensive range utilized for grazing, which favor the benefits of bolus technologies, 2) the prevalence of trace mineral deficiencies in the grazed forages, and 3) local laws that limit the use of certain bolus technologies due to toxicity concerns (i.e. Se in the USA) or food safety concerns (i.e. glass boluses that remain in the rumen throughout the animal's life). Although there are certainly known benefits to the use of intra-ruminal boluses for the delivery of trace minerals, the technology is not without problems. Cows can regurgitate the boluses and the presence of hardware disease in individual animals can cause variation in the liberation rate of mineral from the bolus due to physical scratching of ingested metal against the ruminal bolus.

One of the most widely used forms of trace mineral boluses are Cu oxide needles packaged within gelatin boluses. Copper oxide boluses are effective in rapidly increasing liver Cu stores in cattle (Yost et al., 2002) and are likely more effective than injectable Cu for providing longer-term tissue Cu reserves in cattle (Rogers and Poole, 1988). In some studies, Cu toxicity was diagnosed in calves from Nebraska, Wyoming, and North Dakota beef herds receiving Cu-oxide boluses (Hamar et al, 1997; Steffen et al, 1997). Although toxicity conditions have been reported, particularly in calves, Cu-oxide boluses have continued to be considered among cattle producers as a potential tool for addressing Cu imbalances in grazing cattle.

We previously evaluated the effects of Cu-oxide boluses in two cowherds in southwest Kansas (Arthington et al., 1995). Copper bolus administration decreased calf ADG in Herd 1 (ADG = 0.81 versus 0.96 kg/d for bloused and control caves, respectively) and calf weaning weight in Herd 2 (14.1 and 27.9 kg lighter for bloused bulls and heifers, respectively, compared to non-bolused control calves).

One explanation for the negative impact of Cu-oxide bolus administration on calf gain relates to the potential antimicrobial effect of Cu in the rumen. Copper may be altering the ruminal microflora in such as manner as to negatively impact forage digestion. To investigate this, we examined the effect of Cu-oxide bolus administration on forage nutrient digestion in yearling crossbred steers (Arthington, 2005). In this study, intake of the forage fiber (NDF and ADF) and CP did not differ between treatments, however, digestibility of NDF and CP were greater and digestibility of ADF tended to be greater for Control steers vs. steers receiving Cu oxide boluses. One explanation for the decrease in apparent digestibility of forage nutrients in bolused steers relates to the potential toxicity of Cu to the ruminal microorganisms. Hubbert et al. (1958) conducted *in vitro* studies aimed at determining the mineral requirements of ruminal microorganisms. In their study, Cu was found to be particularly toxic to ruminal microorganisms. Depression in forage digestion was noted with fermentation media containing 1.5 mg Cu/L. Similarly, the ability of ruminal microorganisms to convert non-protein nitrogen into protein has been shown to be significantly reduced when ruminal liquid contained 10 mg Cu/L (McNaught et al., 1950).

Pasture Se Biofortification

One potential method for addressing Se nutrition in grazing cattle is the implementation of pasture Se applications with the intent of increasing plant Se content (biofortification) and thus the Se status of cattle grazing these forages. In Florida, spraying bermudagrass with Na selenate at Se application ranges of 120 to 480 g/ha resulted in substantial increases in forage Se content by 2 wk after application, decreasing rapidly by 12 wk post-application (Valle et al., 1993). Feeding forages grown on Se-fertilized hay fields impacts both Se status and performance of grazing cattle. In one study (Hall et al., 2013), weaned Angus-type calves were fed Se-fertilized alfalfa hay over a 7-week period. Alfalfa hay was grown on fields receiving applications of Na selenate in amounts providing 0, 23, 45, or 90 g Se/ha. These application rates resulted in a linear ($R^2 = 0.997$) response for Se application rate and subsequent Se content of alfalfa hay harvested 40 d after Se application. In addition, calves consuming these hay treatments (approximately 2.5% BW daily) experienced a linear ($R^2 = 0.979$) increase in whole blood Se concentrations as Se application rate (and Se content of hay) increased.

In a recent Florida study (Ranches et al., 2017), we produced a high-Se hay crop by spraying a Jiggs bermudagrass hayfield with Na selenate at a rate of 257 g Se/ha. Selenium content of hay, harvested 8 wk after Na selenate application, was greater for Se-treated vs. control pastures (7.73 ± 1.81 vs. 0.07 ± 0.04 mg/kg DM; $P < 0.001$). This hay crop was fed to weaned calves and Se status was evaluated over a 42-d study. A pair-feeding design was utilized, whereas each pen of high-Se hay calves was paired to a pen of Na selenite - supplemented calves. Liver Se concentrations remained unchanged for the negative control calves receiving no supplemental Se over the 42-d feeding period, but they were increased ($P < 0.001$) in calves receiving both high-Se hay and Na selenite treatments. Calves receiving high-Se hay had greater ($P < 0.05$) liver Se concentrations on d 21 and 42 than calves receiving Na selenite. Interestingly, this difference was attributed only to the paired pens consuming < 3 mg Se daily.

Analysis of Herd Trace Mineral Status

If a trace mineral deficiency is suspected, a producer may wish to conduct an evaluation of herd trace mineral status. With today's technologies, this task is fairly simple and cost efficient. The following steps should be considered with attempting to evaluate herd trace mineral status and effectiveness of the trace mineral supplementation program.

A. Rule out other influential factors

The first step in identifying trace mineral deficiencies is to attempt to rule out other more directly contributing factors. For instance, if average cow body condition score is less than 4 ½, chances are far greater that decreases in reproductive performance and/or immune function are a result of energy/protein deficiency versus trace mineral deficiency. Also, be sure to evaluate the basics of your current supplementation program. Does the product provide a balanced mineral profile using quality ingredients? Are the cattle being provided with a consistent supply of fresh, dry mineral? Are the cattle consuming the mineral at an appropriate level?

B. Forage trace mineral concentrations

Grazing cattle selectively consume forage with 25 to 30 % more crude protein than hand-clippings of the same pasture. In a field study, we attempted to collect the same forage being consumed by grazing steers. Prior to grazing controlled areas, we emptied the ruminal contents from four rumen-cannulated steers. During the grazing periods, we attempted to clip that forage which the steers were consuming. Later, the rumen of each animal was again emptied and the consumed forage rinsed with water. Even though we made attempted to clip exactly the forage being consumed, the steers selected forage higher in crude protein (30.0%), calcium (52.6%), and phosphorus (36.8%), compared to hand-clipped samples. However, no differences occurred in the trace mineral content of steer selected vs. clipped forage, suggesting that hand-clipped forage samples are a good reflection of the trace mineral concentration of animal-selected forage.

When collecting forage samples for trace mineral analysis it is important to collect the sample from areas where animals are grazing (selecting). Do not collect from non-selected forage areas and be careful to not contaminate your sample with weeds or dirt. Prior to collection, find a laboratory that will test forage for trace mineral levels. Many commercial laboratories offer an analysis package containing a group of trace minerals for $25 to $50 per sample. The laboratory will provide directions for collection, handling, and shipping. It is important to test for Cu, Zn, Se, Co, and Mn. It is also important to consider including antagonistic trace minerals, which may interfere with the normal absorption of other minerals. Three commonly recognized antagonists in forages are Mo, Fe, and S.

C. Herd trace mineral status

Often, it is possible to establish a reasonable plan of action by addressing points 1 and 2. However, in some instances it may be important to further explore a potential trace mineral deficiency by examining animal blood and/or liver mineral status. For two of the most commonly deficient trace minerals, Cu and Se, liver samples provide the most reliable indicator of actual animal stores. Blood samples are an unreliable approach for the measurement of these elements unless the cattle are severely deficient. Modern laboratory technology allows for the use of very small tissue samples for the analysis of multiple trace elements. Today's liver biopsy collection technique is simple, inferring very little stress to the animal. A summary of common indicators of trace mineral status for cattle is provided. Actual values are not provided. These will vary depending on the laboratory technique, moisture content of sample, and sample preparation processes. It is important to visit with your diagnostic laboratory prior to sample collection for information on how to handle and ship the sample. This laboratory should also be able to share with you their ranges of deficiency to sufficiency for the samples and minerals being tested.

Indicators of trace mineral status

Mineral	Indicator
Copper	Liver is the best indicator. Blood is a very poor indicator and should not be used. Ceruloplasmin enzyme activity can be used, but will be misleading in stressed animals.
Zinc	Zinc status is difficult to assess in living animals. Liver is a relatively poor indicator. Plasma or serum is the most commonly used indicator, but is reliable only for very recent dietary intake. Reduced feed intake is a common indicator of Zn status.
Selenium	Liver is the best indicator. Whole blood is a good indicator and better than plasma or serum. Glutathione peroxidase enzyme activity in red blood cells is fair to good indicator.
Manganese	Similar to Zn, Mn is also difficult to assess in living animals. Blood plasma or serum are poor indicators, both representing the Mn concentration of the most recent meal. Liver and hair Mn concentrations are fair indicators of deficiency and toxicity, respectively.
Iodine	Presence of goiter is a primary indicator of severe I deficiency. Diagnosis of subclinical I deficiency is difficult. Some reports indicate that milk I concentrations may be of some benefit.
Iron	Blood hemoglobin concentration is a good indicator of Fe status. Liver Fe concentration is also a good indicator.
Cobalt	In ruminants, functional Co appears in the form of vitamin B12. Therefore, blood and tissue vitamin B12 concentrations are a good indictor of Co status in ruminants. Liver Co concentrations are fair indicators of Co status.

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