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Foot-and-mouth Disease: A Review of the Facts

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In light of the outbreak of foot-and-mouth disease around the world, readers may find information on foreign animal diseases beneficial. The most important case in a foreign animal disease outbreak is the first case. Prompt recognition, confirmation and containment are essential to avoiding the economic disaster that has occurred in England. Starting with this issue, we'll highlight one or two of the important foreign animal diseases in each issue.

Although information on foot-and-mouth disease has been widely disseminated, here's a review of the salient features of foot-and-mouth disease and vesicular stomatitis, one of the key differentials for foot-and-mouth disease and a disease present in the United States. We hope this will help food animal practitioners familiarize themselves with these potentially devastating diseases.

—The Editors

Foot-and-mouth disease (FMD), sometimes referred to as hoof-and-mouth disease, is a highly contagious viral disease of domestic and wild artiodactyls (cloven-hoofed animals including cattle, pigs, sheep, goats, water buffalo, deer, elk and others) characterized by fever and vesicles with subsequent erosions in the mouth, nares, muzzle, feet or teats. It is considered the most infectious disease known in medicine. The disease produces virtually 100 percent morbidity in a susceptible population of animals, but mortality in adult animals is low. Most infected animals recover from infection, but the disease is economically devastating because recovered animals fail to gain weight or produce milk or fiber. The disease is widely distrib-

uted throughout Africa, the Middle East, Asia and South America. Until the present outbreak, the British Isles and most European nations were free of the disease. North and Central America, Australia, New Zealand and Japan are presently free. There are reports of human infection that results in a mild flu-like illness, but the disease is not considered a human health threat, nor is the disease a food safety issue.

The Virus

The etiological agent is a virus in the genus Aphthovirus in the family Picornaviridae. There are seven serotypes and more than 60 subtypes of the virus, and new subtypes occasionally arise spontaneously. Some strains have a predilection for particular species for example, affecting pigs, but not cattle, but this is the exception rather than the rule.

The virus can remain viable in dry fecal matter for up to 14 days, in urine up to 39 days, soil surface in summer for three days, soil surface in autumn for 28 days and slurry in winter up to six months. The virus can remain viable in unpasteurized milk, other dairy products, semen, embryos, and meat products such as lymph node, bone marrow, blood clots and uncooked salted/cured meats.

Transmission

Transmission can occur by direct or indirect contact with infected animals, wind-borne transmission of aerosols from infected animals (10 kilometers or more), aerosol from bulk milk trucks, feeding of contaminated garbage, contact with contaminated objects, such as hands, footwear, and clothing, artificial insemination,

and contaminated biologicals such as hormones. A person in contact with infected animals can harbor sufficient virus in the respiratory tract to serve as a source of infection for susceptible animals. In an outbreak, the primary means of transmission is by aerosols, and direct and indirect contact.

Sheep act as maintenance hosts, pigs act as amplifier hosts, and cattle act as indicator hosts. Clinical signs in sheep and goats tend to be mild and may go undiagnosed. The virus is replicating. The animals are producing infectious aerosols, spreading the virus by contact and contaminating fomites. The disease in pigs spreads rapidly because they produce 30 to 100 times more virus in aerosols than sheep or cattle. Clinical signs and lesions in cattle usually develop more rapidly and severely than in sheep or pigs. Animals

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Thank you to the Pfizer Animal Health Group, Livestock Division, Cattle Products Group for financial assistance in publishing this newsletter.

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recovered from infection can become carriers.

Clinical Signs

Clinical signs in cattle include fever, lameness, recumbency, anorexia, fall in milk production, excessive salivation, drooling, serous nasal discharge and vesicle formation. Vesicles may form on the tongue, dental pad, gums, soft palate, nostrils, muzzle, interdigital space, coronary band and teats. Pregnant cows may abort, and calves may die without vesicle formation.

Clinical signs in pigs include fever, anorexia, lameness, reluctance to move, pain when forced to move and recumbency. Vesicles develop on the coronary band, heels, interdigital space and snout. Pregnant sows may abort, and piglets may die without showing clinical signs. Clinical signs in sheep and goats tend to be mild and include fever, lameness, and development of vesicles in the interdigital space and coronary band, dental pad, lips, gums and tongue. Mild lameness may be the only sign. Pregnant animals may abort, and nursing lambs may die without showing clinical signs.

The characteristic gross lesions in all affected species are single or multiple vesicles that can range in size from 2 mm to 10 cm in diameter and can occur at all sites previously mentioned. Multiple

The virus can remain viable in unpasteurized milk, other dairy products, semen and embryos.

vesicles frequently coalesce to form large lesions. Following vesicle rupture, an erosion forms that is covered by a gray fibrinous coating that may become yellow, brown or green. The erosion heals and the epithelium is restored with a line of demarcation that gradually fades. Animals that die may have gray to yellow streaks of degeneration and necrosis in the myocardium ("tiger heart"), and erosions of the rumen pillars.

Foot-and-mouth disease should be suspected in cattle when there is simultaneous salivation and lameness, and a ve-

sicular lesion is seen or suspected, especially when multiple animals are affected. Examine the feet of any animal that shows excessive salivation, and examine the mouth and nostrils of any lame animal in order to avoid missing the diagnosis. In pigs, sheep and goats, foot-and-mouth should be suspected when animals have sore feet, and a vesicular lesion is seen or suspected, or both.

The differential diagnoses include the other vesicular diseases including vesicular stomatitis in cattle, sheep and goats, and swine vesicular disease and vesicular exanthema in pigs. In addition, rinderpest, malignant catarrhal fever, BVD-Mucosal Disease, IBR virus and bluetongue virus can cause oral erosions in cattle. The definitive diagnosis of foot-and-mouth disease is dependent upon laboratory confirmation. Samples to submit for diagnosis include epithelium from lesions, vesicular fluid, heparinized blood or EDTA blood, serum and esophageal-pharyngeal fluid collected with an instrument called a probang, and a good clinical and epidemiological history. The appropriate state and federal officials should always be contacted when a vesicular disease is suspected.

Practical On-farm Strategies Help Prevent FMD

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As of this writing, Canada, the United States and Mexico are free of foot-and-mouth disease (FMD). This unique status is shared with Australia and New Zealand in the Southern Hemisphere. Several other countries in the world are also free of FMD. However, many Asian countries, several in South America, Africa, and most recently the United Kingdom, the Netherlands, and France are experiencing outbreaks of the disease.

This article describes management strategies to prevent the introduction of disease to a farm with particular reference to foot-and-mouth disease. Although this piece refers specifically to cattle, these general strategies are applicable to other farm livestock.

On-farm Control

Kansas livestock producers prevent the introduction of FMD and other foreign animal diseases by common sense and

practical on-farm strategies. FMD virus is easily killed by cleaning or washing clothes with bleach or washing soda or by dry cleaning. People may harbor the FMD virus in their nasal cavity for up to two days. Knowledge confirms that persons who have been working with FMD animals must stay away from healthy animals for at least five days.

To prevent the introduction of foreign animal diseases from infected animals on farms in countries with the diseases, Kansas producers should follow these guidelines:

New Arrivals

- Maintain a closed herd. The first method is not to purchase cattle. A truly closed herd does not allow the introduction of outside animals. For practical reasons, there are few truly closed herds in Kansas. In the case of an FMD outbreak, owners should maintain closed status until the outbreak is contained.

- Do not allow the introduction of outside animals to the herd.

- Prevent fence-line contacts of stock with other cattle.
- Use artificial insemination for breeding, and do not bring in bulls.
- Do not exhibit at shows.
- Restrict visitors.
- Know the source of incoming animals.
- Require health certification on all incoming animals.
- Receive incoming animals during daylight, and inspect all animals as they are taken off the truck.

Farm Traffic

Minimize traffic between farms, ranches, and feedlot operations to prevent the spread of diseases. Farm visitors wearing boots or clothing freshly contaminated with infectious agents can spread cattle diseases among farms. Birds, rodents, pets, people, equipment, and vehicles contaminated with manure (or other bodily excretions) should be considered potential

See Strategy, page 4

Problem May Not Be FMD, But Vesicular Stomatitis

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Portions of the following article are from "Foreign Animal Diseases" by the Committee on Foreign Animal Diseases of the United States Animal Health Association, revised 1998, and used here with permission of the USAHA.

Vesicular stomatitis is a viral disease characterized by fever, vesicles and subsequent erosions in the mouth, teats, and feet. Horses, cattle and swine are naturally susceptible. Sheep, goats and South American camelids are less susceptible but can be infected. A variety of wildlife, including deer, feral pigs, rodents, raccoons, bobcats, monkeys, birds and insects can be infected but may not show clinical signs. Humans may develop mild flu-like signs and occasionally blisters in the mouth.

Region

The disease is limited to the Western Hemisphere and occurs in Mexico, Central America and several northern South American countries. There have been periodic outbreaks in the United States, particularly the southwestern United States. The disease is enzootic on some of the barrier islands off the coast of Georgia.

Serotypes

The vesicular stomatitis virus is an RNA virus. There are two serotypes, New Jersey and Indiana. The latter has three subtypes. Although the New Jersey and Indiana serotypes are serologically and immunologically distinct, the infections are clinically similar.

Transmission of this disease is still not entirely clear nor is the reservoir between outbreaks known, but the virus has been shown to be transmitted by the sand fly and the black fly. Transovarial transmission has been demonstrated in some species of flies, and this may be how the virus overwinters. The virus has been isolated from a number of other insects including mosquitoes and some non-biting insects.

The disease is generally seasonal, occurring during warm weather, which is consistent with an insect vector. In addition to insect vectors, the saliva and vesicular fluid from the lesions of clinically

affected animals is infective, and infection can also occur by direct contact or ingestion of contaminated feed or water.

Clinically, it is not possible to tell vesicular stomatitis from foot-and-mouth Disease.

Clinical signs

In cattle, after a short incubation period there is fever and the development of vesicles on the dorsum of the tongue, dental pad, lips and the buccal mucosa. This results in anorexia and profuse salivation. The vesicles are often not very prominent, and erosions and ulcers are often more evident on physical examination. Lesions can also be found around the coronary bands and on teats. Secondary mastitis is a common complication in lactating cattle. Recovery is usually rapid. Adults tend to be affected more than calves.

In horses, the signs are similar to those in cattle with fever, depression, anorexia, salivation and development of vesicles/ulcers in the oral cavity. The vesicles rupture to form shallow ulcers. In horses, lesions around the coronary band may also develop, with lameness and deformities of the hoof wall in recovered animals.

In pigs, lesions develop on the snout or around the coronary band. Lameness is more frequent than in other animals.

The morbidity rate tends to be less than with foot-and-mouth disease and is often given as 5 to 10 percent, but in more closely confined herds, such as dairies, morbidity may be considerably higher.

There usually is no mortality although the disease can cause considerable economic loss from disruption of lactation and due to the quarantinable status of this disease. The major importance of vesicular stomatitis in U.S. livestock is due to its similarity with foot-and-mouth disease.

Clinically, it is not possible to tell vesicular stomatitis from foot-and-mouth disease.

The differential diagnosis in cattle includes foot-and-mouth disease, foot rot, chemical and thermal burns, BVD, malignant catarrhal fever, bluetongue, IBR and rinderpest, cowpox, and pseudocowpox.

In pigs, the differential diagnosis for vesicular stomatitis includes foot-and-mouth disease, swine vesicular disease, vesicular exanthema, and possibly chemical or thermal burns.

In sheep the differential diagnosis includes bluetongue, contagious exanthema, lip and leg ulceration and foot rot.

In horses, the differential diagnoses would include oral ulcerations due to grass awns or other mechanical trauma or chemical and blister and beetle poisoning. Oral vesicular lesions of unknown cause but not due to vesicular stomatitis virus have been recently described in the United States (JAVMA, May 1, 2000):1399).

Key Differences

A few differences between foot-and-mouth and vesicular stomatitis include:

- Horses are affected by vesicular stomatitis, but not foot-and-mouth disease.
- The morbidity within a herd is usually considerably less with vesicular stomatitis than foot-and-mouth disease.
 - In a given animal, lesions of vesicular stomatitis do not tend to be as widespread as with foot-and-mouth disease, i.e., lesions may be just in oral cavity or just on feet, or just oral and udder, etc.
 - There are no rumen or other internal lesions at necropsy with vesicular stomatitis.
 - Mortality and morbidity in young animals is considerably less than with foot-and-mouth disease, and there are no heart lesions caused by vesicular stomatitis as there can be with foot-and-mouth disease.
 - Stabled animals are often not affected, presumably because of less exposure to biting insects.

As with foot-and-mouth disease, if clinical signs suggest this vesicular disease, contact the nearest state or federal animal health officer immediately and stop move-

See Stomatitis, page 5

Diagnostic Lab to Assist in BSE Detection

Reprinted from *Kansas Animal Health News*
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The European community's scientific steering committee has placed countries in one of four categories of risk assessment for bovine spongiform encephalopathy (BSE).

Risk Level I — Highly unlikely that BSE could be present

Risk Level II — Unlikely, but cannot be excluded that BSE exists

Risk Level III — Highly likely that BSE could be present

Risk Level IV — Countries have reported cases of BSE

The United States has been placed in Risk Level II and has protested its rating.

Surveillance

Surveillance in the United States is targeted at those animals most likely to be affected. They must be 20 months of age or older and meet at least one of the following conditions:

1. Reported to be exhibiting signs of a progressive neurological disease on farm.
2. Negative rabies suspects at a veterinary diagnostic laboratory.
3. Condemned at slaughter because of a CNS condition.
4. Presented for slaughter as a "downer cow."

The Kansas Animal Health Department and USDA, APHIS, Veterinary Services has implemented a program for BSE surveillance. Practitioners should report cattle more than 20 months old exhibiting signs of central nervous system disease to the Kansas Animal Health Department 785-296-2326, Veterinary Services 785-235-2365 or their local state or federal veterinarian. Program personnel will obtain brain samples upon the death or euthanasia of the affected animal, have the sample tested for rabies and obtain a histopathologic examination including the BSE screening tests at no expense to the reporting veterinarian or owner. The K-State Diagnostic Lab is assisting in BSE surveillance by histopathologically screening bovine brains for BSE that have been submitted by practitioners for rabies testing.

On a recent national television program one person interviewed was critical that the new tests for BSE now used in Europe were not being used in this country. In a report issued in December 2000, the European Commission for Health and Consumer Protection admitted that these BSE tests cannot be used to reassure the public of the safety of continental beef. A spokeswoman for the commission said that a negative test (using the new tests)

did not mean the beef is negative. None of the three approved tests have been validated on animals appearing healthy but incubating the disease, so it is not possible to estimate the reliability of a negative test.

The facts concerning the Texas feedlot reported to have fed prohibited ruminant material are as follows:

It was fed once. At most each 600-pound animal received 5 1/2 grams. The prohibited material was of domestic origin. The feed company responsible purchased all 1,222 animals. Meat from these animals will not enter the human food supply. Safeguards now in place work. The industry acted in a responsible manner.

When submitting bovine brains for rabies testing, be sure to include the brain stem down to the cranial spinal cord. The medulla at the obex is the location needed for BSE surveillance.

Strategy, from page 2

disease carriers. Specifically, control birds, rats and mice. Pigeons, sparrows, starlings, and rats and mice may act as carriers of infectious agents on their feet and within their digestive systems.

Control people and pets. People spread contaminated material directly on footwear, hands, and clothing. To decrease the spread of contaminants:

- Inform herd workers, visitors, and truckers of your farm protection methods and insist upon cooperation from these individuals.
- Keep visiting vehicles out of areas accessible to livestock.
- Insist visitors wash/disinfect their boots before entering and leaving.
- Supply rubber boots and clean coveralls for visitors.
- Provide a footbath containing an effective disinfectant.

- Control the movement of dogs and cats between farms.

- Wash farm clothing with detergents and bleach or washing soda.

- Ask foreign visitors about their visits to farms in their country of origin.

- Restrict visitors from the farm if the visitor has been on a farm with a contagious animal disease within the previous five days.

Equipment

Disease can spread from farm-to-farm indirectly by small and large equipment. To reduce this spread:

- Use your own equipment, halters, nose tongs, clippers etc., rather than borrowing them.
- Thoroughly wash and disinfect the inside, outside, and tires of equipment shared with neighbors.

Use disinfectants

Information about disinfectants is available on the product label or from farm supply dealers, veterinarians, the United States Department of Agriculture, and the product manufacturers. For best results disinfectants should be applied to cleaned surfaces.

Monitoring and Reporting

Provide training to employees on recognition of signs of FMD. Monitor livestock and promptly report any symptoms of FMD to your local veterinarian. FMD may cause high fevers, blisters around the mouth or on the feet, reduced appetite, and lameness. FMD can be confused with several other less harmful, diseases, such as vesicular stomatitis or swine vesicular disease. Failure to promptly report a case of FMD will endanger your neighbors and the entire U.S. livestock industry.

Treating Vitamin A Deficiency in Feedlot Cattle

Brad DeBey and John Ragsdale, Veterinary Diagnostic Laboratory Kansas State University

Last fall a small Kansas feedlot experienced morbidity and mortality in heavy-weight cattle as a result of vitamin A deficiency. A large percentage of the steers but very few heifers had clinical signs of weight loss, ataxia, excessive lacrimation, blindness and generalized edema. Carcasses at a slaughter plant had been condemned because of generalized edema. The cattle were being fed a diet of alfalfa hay, soybean meal, and either shelled corn or high moisture milo. Block salt was the only supplement added, with no vitamins or minerals added to the ration.

Two steers from the feedlot were submitted to the Kansas State University Veterinary Diagnostic Laboratory for diagnostic testing. Gross lesions were limited to generalized subcutaneous edema and intramuscular edema. Squamous metaplasia of salivary ducts, a pathognomonic lesion of vitamin A deficiency, was identified in one of the steers. Analysis of serum revealed 7.19 ng/ml of vitamin A (deficient if levels are below 150 ng/ml), and no vitamin A could be detected in the liver.

Symptoms

In young animals, the signs of vitamin A deficiency are manifestations of compression of the brain and spinal cord. In adult cattle, vitamin A deficiency is characterized by night blindness, keratinization of the cornea, hoof defects, weight loss, and infertility. Signs of vitamin A deficiency in growing cattle include edema of

the brisket and limbs, irreversible blindness, night blindness, and neurologic effects of increased intracranial pressure. The nervous signs in growing animals with vitamin A deficiency are related to increased cerebrospinal fluid pressure caused by decreased absorption of cerebrospinal fluid. Blindness in growing animals can result from compression of optic nerves secondary to abnormalities of modeling of bone that is associated with vitamin A deficiency. Vitamin A deficiency can also cause blindness as a result of loss of photoreceptors in the retina. Vitamin A-deficient dams may give birth to stillborn calves, or calves with congenital blindness, incoordination, thickened carpal joints, or hydrocephalic calves.

Vitamin A-deficient dams may give birth to stillborn calves or calves with congenital blindness, incoordination, thickened carpal joints or hydrocephalic calves.

Vitamin A deficiency in cattle is very uncommon, as most diets are supplemented, or cattle are fed forages that contain adequate vitamin A activity (carotene or vitamin A). Green pasture is very rich in carotene, and in most pastured cattle, liver

reserves are sufficient to prevent clinical signs of deficiency for at least 6 months or longer. Yellow corn, new hay, and fresh silage are usually adequate sources of carotene, but potency decreases with storage, and drought-stressed or water-damaged crops may contain inadequate carotene. Heat, light, and mineral mixes are known to increase the rate of destruction of vitamin A supplements in commercial rations. Dietary carotene is converted to vitamin A by the intestinal epithelium, therefore diseases causing prolonged dysfunction of intestinal epithelium may result in vitamin A deficiency.

Steers are predisposed to vitamin A deficiency as compared to heifers. This sex difference is believed to be due to synthesis of beta-carotene in the corpus luteum and its conversion to retinol. The increased susceptibility of the steers compared to the heifers in this feedlot was profound, and can be an important clue in the diagnosis of vitamin A deficiency.

Treatment

Treatment of affected animals with parenteral or dietary vitamin A often is successful if the clinical signs are not severe or prolonged. Animals with neurologic dysfunction are unlikely to recover in spite of treatment.

In this case, it is believed that the cattle developed vitamin A deficiency because of the combination of lack of supplementation of the diet with vitamin A, and feeding of feedstuffs from drought-stressed crops. Parenteral treatment with vitamin A and dietary supplementation of the affected cattle resulted in response in those without severe clinical involvement.

Stomatitis, from page 3

ment of animals off the premises.

Rapid laboratory tests are available at the USDA's Plum Island Disease Center. In suspected cases, the federal or state animal health veterinarian will collect specimens for shipment to Plum Island.

Control of this disease is basically by restriction of movement of infected or exposed animals except for slaughter, along with disinfection and insect control. Commercial vaccines have been developed, but efficacy is apparently largely unknown.

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Research Roundup

Harlan Ritchie, Dan Buskirk and Steven Rust, Ph.D's Michigan State University Beef Cattle Specialists

Diet Modification May Reduce *E. coli* 0157:H7 in Finishing Steers

Scientists at the University of Manitoba reported significant differences in shedding of the bacterial pathogen *E. coli* 0157:H7 strain 3081 among yearling steers inoculated with the bacteria and fed three different grain diets for 10 weeks

Diets consisted of: (1) 85% cracked corn; (2) 85% barley; or (3) 70% barley and 15% whole cottonseed. Number of animals positive for *E. coli* during the 10-week period was significantly higher for the 85% barley-fed group than for the other two groups. Fecal pH of steers fed the corn diet was significantly lower than fecal pH of the steers fed the barley-based diets, likely resulting in a less favorable environment for *E. coli* in the large intestine of the corn-fed cattle. The authors concluded that minimizing environmental dissemination of *E. coli* together with diet modification may reduce numbers of *E. coli* positive cattle [Buchko and Holley, 2000. *J. Food Prot.* 63(11): 1467-1474].

Comparison of Urea Levels for Newly Arrived Feeder Calves

There is some controversy in the feedlot industry on whether urea should be included as a protein source for newly received feeder cattle. Using non-protein nitrogen sources such as urea can reduce supplemental protein costs. USDA researcher Andy Cole has suggested that urea intake be limited to less than 30 grams per day during the first 2 weeks after arrival (Cole, 1996), which computes to less than 1% urea in the receiving diet. To help shed light on this subject, New Mexico State and Texas Tech University researchers allocated 197 feeder calves (437 lb) to one of three diets for a 28-day receiving trial: (1) 0% urea; (2) 0.5% urea; or (3) 1.0% urea. Calves were offered sorghum-sudangrass hay (1 week only) and a 70% concentrate receiving diet for 14 days and then switched to a 75% concentrate diet from 14 to

28 days. No differences were noted among treatments for percentage of calves treated for Bovine Respiratory Disease or for average daily gain during the 28-day period. However, calves on the 0.5% urea level had an improved feed conversion ratio from days 14 to 28 and for the total 28-day trial. The authors concluded the results suggest that the optimum level of urea to feed newly arrived cattle fed a high-concentrate processed grain receiving diet is approximately 0.5% of the dry matter for maximum feed efficiency. They went on to say that these results may differ with higher protein levels than those fed in this trial (14%) and with higher roughage receiving diets (Duff et al. 2000. Clayton Livestock Res. Center Prog. Rep. No. 105, Clayton, NM 88415).

Sickness During the Receiving Period Had a Marked Effect on Carcass Traits and Net Value

Reports continue to mount regarding that detrimental effect of sickness of incoming feeder cattle on their ultimate carcass value. In this Oklahoma State University study, 406 sale barn purchased heifers (465 lb) were placed in commercial feedlots following a 42-day receiving period. Heifers were categorized by severity of Bovine Respiratory Diseases (BRD): (1) never treated; (2) treated once; or (3) treated more than once. During the receiving period, heifers treated more than once gained significantly less weight than the other two groups, but they tended to compensate during the finishing period, resulting in no significant difference in final harvest weight. Nonetheless, there were important post-harvest differences, as shown in the following summary:

- Heifers treated for BRD during the receiving period tended to have lower (leaner) yield grades (2.53 vs. 2.42 vs. 2.36 for the three groups, respectively).
- Heifers that had multiple treatments for BRD had markedly marbling scores and a 25% reduction in percentage of Choice carcasses (66.2 vs. 59.4 vs. 41.1 for the three groups, respectively).

- The 25% decrease in marbling score reduced carcass value by \$2.31 per 100 lb. of carcass weight.

- Gross value per carcass was reduced by \$4.00 for heifers with one treatment and \$19.29 for multiple-treated heifers. Medical costs for these two groups averaged \$7.48 and \$18.00, respectively.

- When medical costs were combined with gross carcass value, the two treated groups netted \$71.48 and \$37.34 per head less than untreated heifers.

The authors concluded that the impact of BRD can extend far beyond medication cost, mortality and reduced performance, emphasizing the importance of prevention as early as possible (Stovall et al., 2000. Oklahoma State University Res. Rept. P-980).

Early Weaning and Creep Feeding Strategies to Produce Quality Beef

A review of early weaning and creep feeding strategies to enhance quality grade in calf-feds was presented at the 2001 Midwestern Section Meeting of the American Society of Animal Science. Here is a brief summary.

- Calves need to be on a starch-based creep feed diet for about 80 days in order to increase quality grade.

- Little difference has been observed in feedlot performance between different creep feeding strategies vs. no creep feed.

- Early weaning at 90 to 150 days of age increases quality grade dramatically, and improves feed efficiency in the feedlot.

- Management of early-weaned calves to get them on a high-energy diet as soon as possible is critical for success.

- Weaning calves at approximately 90 days has resulted in an 18% increase in pregnancy rates of dams, with a range of 8 to 26%.

Using a combination of early weaning, high-energy diets and cattle with the genetic potential to marble, it may be possible to produce cattle that will grade 50% prime at 15 months of age with feed:gain ratios of 6:1 or less (Faulkner et al., Berger et al., Day et al., and Anderson et al.)



Stokka joins Pfizer, bids K-State farewell

Dear Colleagues,

This issue of the *Kansas Vet Quarterly* will be my last as co-editor with Dr. George Kennedy. I have accepted a position with Pfizer Animal Health and will be relocating to North Dakota. (Yes, this was my first choice.) I have enjoyed my tenure at Kansas State University and it is with a certain amount of sadness that I leave one of the premier land grant universities in the world. I consider myself very blessed to have been associated with K-State and hope to have contributed in some small way. The positive aspect for me will be the fact that I will be continue to have contact with many of you both professionally and personally.

Thank you,

A handwritten signature in cursive script that reads "Gerald L. Stokka". The signature is written in dark ink and has a fluid, elegant style.

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