Kansas K-STATE Kansas State University
VETERINARY Quarterly
FOR THE PRACTICING VETERINARIAN

Winter 2002

Volume 5, Number 1

With Symptoms Similar to BVD-mucosal Disease, Rinderpest May Pose Threat to the State's Cattle Industry

The following article on rinderpest is one of a continuing series of updates on foreign animal diseases that pose a potential threat to animal agriculture.

Rinderpest is a serious disease of cattle that has existed for centuries, ravaged herds and affected the course of wars and invasions. Its similarity to BVD-mucosal disease could make it a threat to our cattle industry.

Rinderpest, also known as cattle plague, is a peracute to acute, highly contagious viral disease of cattle. All cloven-hoofed animals are susceptible to infection, but the severity of disease varies considerably between species. It is characterized by fever, erosive or hemorrhagic lesions on most mucus membranes, diarrhea, lymphoid necrosis, and high mortality. In sheep, goats, and pigs the infection is generally mild or subclinical, but these species can transmit the virus.

Etiology. The rinderpest virus is a morbillivirus immunologically related to canine distemper, human measles virus, marine mammal morbilivirus, and a disease of sheep and goats known as peste des petits ruminants.

Geographic distribution. Currently, rinderpest is present in the Indian sub-continent, Near East, and sub Saharan Africa, but historically it has made forays into West Africa, South Africa, Europe, Asia, and South America.

Transmission. Secretions and excretions, particularly nasal-ocular discharges and feces, are infective for one to two days before clinical signs and eight to nine days or longer after onset. Spread is by direct and indirect contact, contaminated water, feed, clothes, or equipment. Aerosol apparently does not play an important role in transmission.

Clinical Signs. Following an incubation period of three to 10 days, cattle initially show depression, anorexia, fever, nasal and lacrimal discharge followed by watery to hemorrhagic diarrhea.

The disease progresses to dehydration and frequently death within six to 12 days. Mortality in a naive population is very high. In young animals, highly susceptible populations, or with highly virulent strains, many animals are found dead without obvious premonitory signs.

Gross Lesions. The most important lesions occur in the mucus membranes, particularly those of the digestive tract. In this regard, the gross lesions can mimic BVD-mucosal disease. Oral lesions are variable, from none to extensive, and consist of grey, necrotic foci that coalesce and erode leaving punched-out raw, red areas. The oral lesions tend to be on the inner surfaces of the lower lip, gingiva, sides and ventral surface of the tongue and soft palate.

Lesions can occur in the esophagus, but apparently are generally milder than those of mucosal disease. The abomasum tends to be intensely congested, with edema of the mucosa and occasional ulcers. Peyer's Patches are usually very obvious as hemorrhagic and necrotic linear foci throughout the small intestine, especially the distal small intestine. Intestinal lesions are most severe in the cecum and colon where the intestinal wall becomes intensely congested and edematous and may contain frank blood and blood clots.

In general, lymph nodes tend to be enlarged and edematous on cut surface.

Differential diagnosis. This disease could easily be initially missed in this country

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Also in this issue

Editor's Note: This is the first edition of the Kansas Veterinary Quarterly Newsletter without co-editor, Dr. Jerry Stokka. Dr. Stokka has joined Pfizer Animal Health. We would like to thank Dr. Stokka for his role in helping to start and publish the Veterinary Quarterly and also for his many contributions to Kansas State and the Kansas livestock industry. –George Kennedy, Editor

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

Survey Examines Effect of Management Practices on Mycoplasma Infection in Kansas Cattle

Mark Spire, Jan Sargeant, Dale Blasi, Kansas State University; Ricardo Rosenbusch, Iowa State University

In the spring of 2001 a survey was mailed to more than 880 stocker/backgrounder operations in Kansas. The purpose of the survey was to gather information from producers on receiving-program management practices and whether or not they have had loads of cattle affected with nonresponsive pneumonia with or without an accompanying arthritis. Of the 300 surveys returned, 232 were used in the analysis presented in this report. The unused surveys either contained incomplete data or were received after data analysis had begun. A total of about 264,000 head of cattle are represented in the analysis.

Why did we do a survey?

From our Beef Stocker 2000 report on stocker demographics in Kansas, producers frequently commented on increasing numbers of loads of cattle with pneumonia and lameness that were nonresponsive to treatment. Over the last three to four years, producers, veterinarians and diagnostic laboratory personnel have raised similar concerns across Texas, Oklahoma and Kansas. While most reports have been in lighter weight stocker cattle, feedlots reported a similar problem in heavier cattle. We wanted to find out about management practices or other conditions that may be associated with groups of cattle developing a syndrome characterized by nonresponsive pneumonia, with or without arthritis.

Is there a cause for non-responsive pneumonia and arthritis?

Many producers and health professionals feel this disease syndrome may be due to a species of organisms called *Mycoplasma*. This disease syndrome can be referred to as Mycoplasmosis.

Why would they think Mycoplasma is a problem?

Routine culture of the lungs and joints of cattle with nonresponsive pneumonia and/or lameness frequently found *Mycoplasma spp*. Case reports from the Kansas State Diagnostic Laboratory in 2000/2001 have isolated the organism in 86%, 69% and more than 90% of lungs only, joints only or when both lungs and joints on the same case, respectively were submitted for microbiological culture. *Mycoplasma bovis* was most frequently cultured but other *Mycoplasma spp.* have been isolated.

What is Mycoplasma?

This species of organism is common in cattle. A unique feature is that it lacks a cell wall. This is important as many of our currently marketed antibiotics attack the cell wall of organisms. That makes mycoplasma difficult to treat and hinders the ability to develop an effective vaccine against it. It is frequently found in the nose and upper throat of cattle of all ages and in the reproductive tracts of both females and bulls. *Mycoplasma bovis* likes to inhabit the cells of the lower respiratory tract. In normal, healthy cattle, they are able to fight off infection by most organisms invading lung tissue, but if the opportunity arises during times of stress or concurrent illness, *Mycoplasma bovis* will move from the upper airways to the lungs and create pneumonia.

When *mycoplasma*-infected calves are placed in a group of non-infected calves, the organism can be isolated from the noses of noninfected calves within 24 hours and most calves in a group by seven days. Clinical signs of pneumonia can develop within two to seven days. If arthritis occurs, cases generally happen about two to three weeks after initial infection. *Mycoplasma bovis* has been found to survive up to 6 months at about 40°F in laboratory conditions, 20 days in straw and over 2 weeks in water. Even in cold weather the organism can survive 1 to 2 weeks on bedding and in water.

Is this a new or emerging organism?

It has been around a while. In a 1975 report on arthritic cattle in Iowa and Nebraska, *Mycoplasma bovis* was isolated from their joints. In a report on pneumonia in California feedlot cattle, *Mycoplasma spp.* were found in 86% of the lungs cultured. It has also been reported that increases in *Mycoplasma* blood titers after arrival in the feedlot are associated with increased sickness in the first month of the feeding period. These titeral changes are also associated with lower weight gain during the receiving period.

Does the organism cause pneumonia and arthritis by itself or does it need help?

That's the magic question. Most researchers feel that the organism is an opportunist that leaves the nose and throat of calves during periods of stress, nutritional deficiency and/or while an animal has a suppressed immune system caused by an infection of another organism. Reports from the Texas Diagnostic Laboratory in Amarillo found *Pasteurella spp.*, the organism most commonly associated with bovine respiratory disease, in 49.7% of respiratory isolates and *Mycoplasma spp.* in a third of the cases. Pollock and others found Bovine Virus Diarrhea (BVD) and *Mycoplasma bovis* in a high percentage of calves with chronic pneumonia and polyarthritis.

How was the survey developed?

There are no signs specific to *Mycoplasma spp.* that could be used to immediately differentiate them from other causes of pneumonia or multiple joint infections. Dr. Rosenbusch, an international expert on mycoplasmosis in cattle has worked with numerous producers experiencing pneumonia and arthritis in groups of young cattle. Through extensive laboratory testing, mycoplasmosis was deemed the most probable cause of many of the outbreaks. Drawing upon his experience with these outbreaks, we developed a clinical syndrome description generally considered to be typical of a mycoplasmosis outbreak. We asked producers if they have had one or more loads of cattle during the past year that matched the following clinical syndrome description:

About 2 weeks after arrival, calves pulled for treatment of pneumonia don't respond to treatment (no improvement after trying two different antibiotics). Calves are often eating well but those being pulled are depressed, have clear nasal discharge and often seek shade. About 3 weeks after arrival, arthritic calves are being pulled. Lameness may not always appear in a group, but if it does, the calves exhibited lameness and joint swelling in the knee, elbow, hip or fetlock joints and several joints may have been involved at one time. The conditions are progressive with affected calves ending up thin, dehydrated and depressed. Most death losses are occurring between 3 and 6 weeks

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after arrival. By about 6-7 weeks after arrival, the outbreak stops with little additional sickness and death loss.

Then how do you know it was *Mycoplasma spp.* causing those clinical signs?

We don't! Other organisms or mixed infections causing respiratory or arthritic conditions in stocker cattle may show clinical signs similar to the clinical description. We did not do laboratory testing in any producer's herd who had responded to the survey to confirm the presence or absence of *Mycoplasma spp.* in their cattle. We designed our survey questions to compare management practices in those operations that stated they had cattle matching the clinical syndrome description to those producers who stated they didn't have cattle matching the description.

Survey Demographics

The survey was mailed to producers in 92 counties. The report covers cattle raised in 87 counties. On the average, reporting stocker/background operations handled 1,140 head last year. They received nearly 15 loads, mostly steers, with an average weight of 493 pounds. Cattle were received year round with nearly 63% arriving in the spring and fall. About 45% of the cattle were described as native Kansas cattle, but 20%, 19% and 12% of the operations buy cattle exclusively from the Southeast, Midwest (excluding Kansas) and the Southwest, respectively. Thirty-one percent of the operations buy only Kansas cattle. On a state of purchase basis, 41.1% of the operations report purchasing cattle from only one state, while 24.8% purchase from two states, 33.6% purchase from three or more states and 0.5% reported other options.

One hundred and five of the operations reported having loads of cattle matching the clinical description. Overall these operations had about 34% of the loads they received affected with the syndrome. Affected herds had twice the number of calves treated for pneumonia than unaffected herds. Within affected herds 5.5% and 1.5% of the cattle were reported as having non-responsive pneumonia or arthritis,.

Key Findings Comparing Affected to Nonaffected herds

1) The syndrome was reported across all sizes of operations, but as operations get bigger, they were more likely to have a problem.

2) The syndrome occurred in all weight classes of cattle, but is more likely in lighter weight cattle. Steers and heifers were affected about the same.

3) As the number of loads received during the winter increased, the more likely an outbreak was to occur.

4) The syndrome was reported in loads of cattle from all regions of the country, but loads from the western region of the United States (Wyoming, Colorado, Utah, Nevada and California) seemed less likely to be affected. Home-raised calves or those procured in Kansas were less likely to have a problem.

5) The likelihood of having a problem increased as cattle were received from an increasing number of states. If an operator buys cattle from a single source, regardless of the region of the country (southeast, southwest, northeast), they are less likely to have reported a problem than if they buy from multiple sources across several states.

6) Affected herds were more than twice as likely to use metaphylaxis (mass treatment of cattle before clinical signs appear). The type of antibiotic used in either the metaphylaxis or treatment program did not appear to be a reason for having the syndrome.

7) The use of modified live viral vaccines did not appear to be the reason for a problem occurring.

8) Increasing stress by castrating or dehorning may increase the likelihood of having a problem regardless of whether the procedures were done on arrival or delayed.

9) Those operations feeding native grass hay as a primary ration ingredient had more reported problems than operations using other feedstuffs.

Are there some general recommendations to aid in controlling Mycoplasmosis?

Currently, there are no approved drugs in the United States to specifically treat mycoplasma nor are there any fully licensed vaccines available. Field experience with drugs commonly used in the treatment of bovine respiratory disease on cattle suspected with mycoplasmosis show a poor treatment response rate. Seventeen percent of the producers indicated they were using a mycoplasma vaccine in their receiving program. There wasn't any statistical difference between those herds with affected loads and those without who were using some type of mycoplasma vaccine, either autogenous or limited licensure products. While some experimental vaccines have shown merit, extensive controlled field experiments to prove effectiveness remain to be done.

The lack of effective treatment and vaccine products on the market severely limits what can be done in a receiving program. Chances are a producer is going to buy cattle carrying the organism. As mycoplasma appears to be an opportunist occurring most frequently during times of stress or when a calf's immune system is weakened, management programs should focus on those procedures that can get calves started out in the right direction.

Watch your cattle-buying practices. Are you going to buy large numbers of cattle and find cattle free of mycoplasma? Probably not. The organism is too wide spread. As a simple recommendation, know your order buyer. Cattle represented as "cheap and too good to be true" probably aren't in the long run. Buying stale, stressed calves increases the likelihood of having cattle that respond poorly to treatment. A significant finding from the survey was that cattle-buying practices do increase the risk of having cattle with nonresponsive pneumonia and arthritis. Lightweight versus heavy weight cattle? You still have to buy what fits your program and pocketbook, but lightweight cattle are at greater risk. Minimizing the number of states you buy cattle from or at least sourcing cattle from a single order buying-facility regardless of the state or region of origin appears to help in reducing loads of affected cattle. This appears particularly important for cattle brought in during winter months.

Buy what you can handle. It takes a pretty good work day for one or two people to feed, check pens for sick calves and pull and treat those calves. Add into the mix days when you process a load or two, and it's not hard to see why everything begins to stack up. Cattle should be fed and observed for sickness first thing in the morning. Watching how calves rise and come to the bunk goes a long way in picking up sick animals. Waiting until later in the day is a problem, particularly if there is a wide difference in temperature from morning to afternoon as most calves will have increased respiratory rates that can mask signs of early pneumonia. Additionally, cattle appear to handle the stress of handling for treatment and

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processing earlier in the day than later in the afternoon or evening. Leaving sick calves for treatment until everything else is done just prolongs the time from when a calf actually gets sick and when the drugs begin to work. Because mycoplasma is an opportunist, extensive lung damage resulting from delayed or ineffective treatment of common pneumonia-causing organisms may increase the likelihood of mycoplasma invading the lungs.

Vaccinate for common respiratory pathogens. Again, mycoplasma is an opportunist. Doing all you can to minimize common respiratory viruses such as Infectious Bovine Rhinotracheitis (IBR), BVD, Parainfluenza – 3 (PI3) and Bovine Respiratory Syncytial Virus (BRSV) from occurring will decrease the likelihood of damage to the respiratory tract and debilitation. As clinical cases of BVD have been associated with increased risk to mycoplasma infection, a BVD vaccine component should be used in the receiving program. Based on survey results, whether a modified live or killed BVD vaccine was used, no particular vaccine program appeared to have an advantage over another. Based on the survey results, Pasteurella vaccines are currently being used in a large number of stocker operations. There was no statistical difference in the number of operations with affected loads of cattle using this type of vaccine and those that don't.

Minimize contact between arriving cattle and sick pen cattle. Large numbers of mycoplasma organisms are shed from nasal secretions of sick calves. Exposing new cattle to the unnecessary risk of contact with the organism should be avoided. Separate sick pens and receiving or holding pens. Clean and disinfect hospital pen waterers daily. Water fountains are a source of infection for calves that are sick from other causes besides mycoplasma and for incoming cattle being exposed to the organism through these and common handling facilities. The organism can stay viable in water for extended periods of time therefore, drain, clean, sanitize and rinse waterers daily. Disinfectant solutions of peracetic acid and iodophores have been shown to be effective against mycoplasma. These products are commercially available in the United States Hypochlorides tend to be ineffective because of the prolonged contact time needed to kill the organism.

Don't feed poor quality hay or hay in a form that is not easy for incoming cattle to eat. The relationship between poor nutrition and increased susceptibility to disease has long been recognized. Feed intake during the receiving period is typically low, which potentiates the stress effects of shipment, processing and illness. Calves need a high quality, palatable diet on arrival. A high percentage of survey respondents were using native grass hay in receiving diets. Not all native grass hay is created equal. In Kansas, forage quality deteriorates monthly from peak protein values in May and June until September with crude protein values declining from a peak of around 9% to 4% . The best way to know what you are feeding is to get your hay tested before the cattle start arriving. That way you can build a receiving ration that will match the needs of stressed cattle and still use a readily available hay commodity. Protein concentrations in the entire receiving diet should be in the 13.5-14.0% range. Limiting dietary protein can decrease immune function and increase susceptibility to respiratory pathogens. Calves already sick have decreased appetites and need additional protein in their diets to offset lowered intakes. If you are using native hay in receiving diets, feed the hay in a form that minimizes the amount of time a calf has to work at eating. Unbroken, large round bales require a lot more effort to eat and may actually limit the number of calves eating at one time. Breaking hay out into bunk line feeders and top dressing the protein and energy portion of the ration or using a complete ration during the first two weeks will increase consumption.

Provide a trace mineral program that meets or exceeds recommended allowances for the weight of calf purchased. A nationwide sampling of zinc content in forage samples found only 2.5% to have adequate levels of more than 40 PPM . It appears that most pasture management programs require some form of mineral supplementation program. Several trace minerals including zinc are critical for proper immune system function. If the likelihood of having receiving cattle from an area where forage zinc is low isn't risky enough, zinc serum levels will also decrease during transportation and stress. In a recent survey of feeder cattle by the authors, serum zinc levels on arrival were found to be deficient in 35% of incoming cattle sampled. In the same operation, 30% and 55% of cattle sampled at first treatment or at re-pull for treatment, respectively were found deficient. Cattle did not appear to have serum zinc levels return to normal until more than 60 days in the feeding program, even though ration levels were adequate. Pasture mineral supplementation programs will carry-over into the feed yard program. In a Nebraska mineral supplementation study, cattle receiving supplemental trace minerals (zinc, copper, manganese and cobalt) during the summer grazing period had significantly fewer sick calves and fewer treatments per episode than unsupplemented cattle.

Get control of a respiratory disease early. Metaphylaxis is the group treatment of high-risk cattle with antibiotics before clinical signs of illness are present. The survey indicated a significant difference in the frequently of use on affected operations as compared to operations not receiving affected loads. The question begs to be asked, "did it cause the problem" or "did they use metaphylaxis in an effort to prevent affected loads because they had had affected loads before"? We were not able to answer either of those questions. In the final analysis, metaphylaxis did not appear to play a significant role as data suggests that within operations using metaphylaxis there didn't appear to be any relationship between affected loads and unaffected loads receiving the procedure. Metaphylaxis is a proven management strategy to help reduce sickness, chronics and death loss rates in high-risk cattle. Its usefulness has been shown over many research trials, and it remains as a practical management tool for targeted loads of cattle.

Minimize additional stresses at processing. If you can't buy steers and clean-headed cattle, delay those procedures for about 30 days post arrival. Cramming them on top of every thing else at arrival just adds to the stress load.

These general management recommendations are designed to minimize stress, enhance the immune system, decrease contamination between groups of cattle, and control common respiratory pathogens.

A complete set of references is available on request.

Q&A: Chronic Wasting Disease of Deer and Elk

What is chronic wasting disease?

Chronic wasting disease (CWD) is a specific infectious, neurological disease of deer and elk in the United States. The disease is one of a group of diseases called transmissible spongiform encephalopathies (TSE). It is similar to, but not the same as, diseases such as scrapie in sheep, bovine spongiform encephalopathy (mad cow disease), and a disease in humans called new variant Creutzfeldt-Jakob (vCJD). The latter has been linked with bovine spongiform encephalopathy in people in Great Britain. Scientific evidence to date indicates CWD is a distinct disease from these other diseases.

What is the cause of chronic wasting disease?

The cause of CWD and the other TSE's is not known for sure, but thought to involve a novel protein, called a prion, in the brain that when present can transform other proteins and result in degeneration of brain tissue.

What are the clinical signs of chronic wasting disease?

The clinical signs of CWD include excessive salivation, emaciation or wasting, behavioral changes and weakness. It generally affects older animals and appears to always be ultimately fatal.

How is the disease transmitted?

Current evidence suggests oral exposure, or ingestion, is the primary natural route of transmission. Close contact seems to be necessary, or at least increases the chances for transmission.

Does it occur only in deer and elk?

Captive and free-ranging mule deer, white-tailed deer, and elk are all susceptible. Limited experimental work has suggested other ruminants, such as wild and domestic sheep and goats, cattle, pronghorn antelope, bison, and moose are either resistant or less susceptible. It is thought that mule deer may be the primary host.

Affected animals have been found in commercial, or farmed, elk and deer in Colorado, South Dakota, Nebraska, Oklahoma, Montana, Saskatchewan, Canada, and most recently in Kansas.

Diseased animals have been found in free-ranging deer and elk only in a relatively limited area of northeast Colorado, southeast Wyoming, and recently in a small area of southwestern Nebraska. Surveillance of numerous animals in many states, including Kansas, has failed to find diseased animals anywhere else.

Is this disease a concern in Kansas?

This disease is of concern in Kansas because of our proximity to states affected with free ranging animals (Colorado, Wyoming, and Nebraska) and because it was recently identified in an elk in a captive herd in south central Kansas.

Is there a relationship between CWD and mad cow disease?

Both CWD and mad cow disease (bovine spongiform encephalopathy) belong to the same group of diseases, the transmissible spongiform encephalopathies (TSE), but ap-

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Mechanisms of Antibiotic Resistance

By Dr. Loren Shultz, formerly of the Department of Clinical Sciences, KSU College of Veterinary Medicine. Dr. Schultz is now with the Department of Clinical Sciences,University of Missouri.

Bacteria employ many different techniques to survive in environments in which antimicrobials are present. For antimicrobials to work they must gain access to the bacteria to interfere with critical cell functions. Different antibiotic classes target different cell functions. For example, beta-lactams, bacitracin and vancomycin inhibit cell wall synthesis. Polymixins affect cell membrane function. Aminoglycosides, chloramphenicol, lincosamides, macrolides, pleuromutilins, and tetracyclines inhibit protein synthesis. Nitroimidazoles, nitrofurans, quinolones, and rifampin alter nucleic acid metabolism. Finally, sulfonamides and trimethoprim interrupt intermediate metabolic pathways. The techniques that bacteria employ include antibiotic inactivating enzymes, decreasing antibiotic access, and altering the antibiotic's target.

Antibiotic inactivating enzymes are one of the most common mechanisms of resistance. The bacterium produces an enzyme that either destroys the antibiotic or alters it to a form in which it is no longer functional. Examples of inactivating enzymes include beta lactamases, aminoglycoside modifying enzymes, and chloramphenicol acetyltransferase. Beta-lactamases hydrolyze the beta-lactam ring found in penicillins and cephalosporins, converting it to penicilloic acid, which is incapable of inhibiting cell wall synthesis. There are numerous aminoglycoside-modifying enzymes. They function by changing different side chains on the antibiotic. These enzymes do not inactivate aminoglycosides, but alter them in a way that decreases transport into the bacteria and decreases binding to ribosomes.

Resistance can be achieved by decreasing antibiotic access to their designed targets. This can happen by decreasing the outer membrane permeability and having the ability to actively efflux the antibiotic from the cell. Gram-negative bacteria are commonly resistant to multiple antibiotics due to their complicated membrane structure that prevents some antibiotics from gaining access to the cell. The cell wall of some gram-positive organisms also provides protection to some antibiotics by deterring their entry. Antibiotic efflux pumps (proteins in the outer membrane that use cellular energy to pump antibiotics out of the cell) have been described for tetracyclines, chloramphenicol and fluoroquinolones.

The target that antibiotics are designed to attack can be altered in a way that makes either them or the bacteria resistant to the effects of the antibiotic. This can happen in one of three ways: the target itself can become resistant to the antibiotic, the bacteria can produce a new metabolic pathway that is unaffected by the antibiotic, or the bacteria can overproduce the target thereby diffusing the effect of the antibiotic.

It is important that we understand these mechanisms of resistance to aid us in the proper antibiotic selection. Also knowledge of resistance mechanisms are needed for the design of new antimicrobials.

Submitting Specimens to the Rabies Diagnostic Laboratory

1. Remove the head from body between skull and first vertebrae. Submission of whole animals will result in a $5 disposal fee. Exceptions to this fee are bats and small rodents. New national guidelines state that all specimens for rabies diagnostic testing must include brain stem. Submissions from livestock can be partial brains but must include brain stem, cerebellum and hippocampus.We prefer that you do not send live or frozen specimens, as both will delay testing.

2. Double bag specimen in two plastic bags to prevent leakage. Close securely.

3. Place the specimen into a leak-proof container. Place frozen gel packs around the specimen. During the warm months, please include extra refrigeration as necessary. Do not use wet ice, as it may leak and cause contamination.

4. Complete the Request for Rabies Examination submission form. Use a separate form for each specimen. Place this form in a sealed plastic bag. If specimen is negative and histopathology for other disease is desired, please indicate this on a separate histopathology submission form.

5. Place the leak proof container along with the form into another box and seal the box thoroughly.

6. Write the address on the inside and outside boxes clearly, and be sure to include a return address.

7. It is recommended that a reliable overnight or two-day delivery service be used when mailing the specimen to the lab for testing. In emergencies, driving the specimen in is suggested. The Kansas State University Emergency Desk accepts packages 24 hours a day, 7 days a week and will promptly refrigerate the specimen. Do not ship by bus, as arrival time cannot be guaranteed.

8. The Kansas or Nebraska State Health Department, as well as the submitting veterinarian, will be contacted if a positive specimen is confirmed, or if specimen is unsuitable for testing.The Diagnostic Laboratory does not routinely telephone results if they are negative. An exception will be made if the submitting veterinarian requests a telephone call regardless of whether the specimen is positive or negative.

9. Cost for testing is $25 per specimen.

10. Laboratory hours are Monday through Friday, 8 a.m. to 5 p.m., excluding state holidays. Specimens must be received by noon to be tested that day.

11. Weekend testing is not routinely performed. Emergency testing requests will be evaluated on a case-by-case basis. In the event of an emergency, please call lab personnel as soon as possible. The lab can be reached at 785-532-4483 during lab hours, or after hours call 785-532-4100 and ask the emergency desk receptionist to contact someone for you.

12. Submit specimen to:

    Rabies Diagnostic Laboratory, College of Veterinary Medicine Kansas State University - Mosier Hall 1800 N. Denison Avenue Manhattan, KS 66506-5705.

Emergency Telephone Numbers

Kansas
Dr. Gail Hansen
State Public Health Veterinarian
KDHE
900 SW Jackson, Room 1051
Topeka, KS 66601
Phone: 785-296-1127

Nebraska
Roger Murray
Rabies Surveillance Coordinator
301 Centennial Mail South
P.O. Box 95007
Lincoln, NE 68509-5007
Phone: 402-471-2937

For more information regarding rabies, please check our Web site at:
http://www.vet.ksu.edu/depts/rabies/index.htm

K-State Lab offers PCR Test For Leptospirosis

Dick Oberst, Ph.D.
Diagnostic Medicine/Pathobiology

The diagnosis of leptospirosis is typically confirmed by finding a single high titer on serology or a fourfold increase on a paired titer. Several retrospective studies have documented that dogs initially may have negative acute titers, and that convalescent titers are necessary to confirm the diagnosis. In some dogs, a fourfold increase may not be seen until four weeks postinfection, resulting in a delayed diagnosis. Serology may lead to a missed diagnosis when the acute titers are low or negative and convalescent titers are not performed in the mistaken belief that the dog does not have leptospirosis.

Kansas State University Diagnostic Laboratory now offers a polymerase chain reaction test (PCR) to identify the presence of pathogenic leptospires in the dog urine. This test offers the advantage of a rapid diagnosis, with results returned usually in 48 hours. We have identified cases of canine leptospirosis in which the initial serologic test was negative and in cases that failed to ever develop a positive titer. In these cases, the diagnosis by PCR resulted in appropriate therapy that resulted in resolution of clinical signs.

The PCR can be used in conjunction with serologic testing, and we currently recommend that both tests be performed in dogs with suspected leptospirosis. Reasons to perform a leptospirosis PCR include:

1. Acute renal failure
2. Chronic renal failure (mild acute leptospirosis can mimic CRF in laboratory findings)
3. Polyuria/polydipsia with normal laboratory work
4. Evaluation of the zoonotic risk of a dog after recovery from leptospirosis
5. Unexplained fever in dogs
6. Chronic or acute liver disease

The spectrum of diseases that can be seen in dogs with leptospirosis is gradually expanding and additional disease syndromes may also warrant a submission for leptospirosis PCR.

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PCR Test, from page 6

The steps for submitting a urine sample are as follows:

1. Collect 10-20 ml of urine in a sterile container (red top tubes work well); the urine can be collected by free catch, cystocentesis, or catheterization. In acute renal failure, it is preferable to collect a pretreatment urine sample, however many are still positive even after one-two days of therapy.

2. Cost is $20 per sample.

3. Ship urine by overnight/next day courier, preferably with an ice pack. We have shown no loss of DNA recovery in urine that sat at room temperature for 72 hours, so urine samples may be saved for one-two days in the refrigerator pending the decision to submit for PCR.

4.Submit to: Kansas State University Veterinary Diagnostic Laboratory c/o Dr. Richard Oberst, Molecular Diagnostic Lab, College of Veterinary Medicine, Kansas State University - Mosier Hall, 1800 N. Denison Avenue, Manhattan, KS 66506-5705

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because of its clinical and pathologic similarities to other diseases, particularly BVD-mucosal disease. This could delay recognition. A disease outbreak mimicking mucosal disease but in cattle of all ages with high mortality, and particularly if they have been vaccinated for BVD, should raise an index of suspicion.

Other differential diagnoses would include foot-and-mouth disease because of the oral lesions, but rinderpest should not be as explosively contagious as foot-and-mouth disease, malignant catarrhal fever, acute coccidiosis, salmonellosis, arsenic toxicity or ingestion of caustic chemicals.

Diagnosis. As with other foreign animal diseases, suspicion should prompt a call to the state or federal animal health authorities. Specimens that are requested for diagnosis include whole blood in EDTA or heparin, serum, swabs of lacrimal fluid, unfixed pieces of necrotic tissue from the oral cavity, spleen, lymphoid tissue including Peyer's Patches. Recommended formalin fixed tissues include oral lesions, liver, spleen, kidney, lymph nodes, and multiple levels of small and large intestines.

Control. An effective vaccine is available for this disease.

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pear to be specific to each species. In different species, the disease may be caused by different strains of the same or a similar agent.

What is the threat to farm livestock and humans?

As far as is currently known, neither people nor our common farm livestock (cattle, sheep and pigs) are susceptible to CWD.

This disease has been known since the late 1960s, and no cases have been discovered linking any disease in humans or livestock to CWD. Even where wild, free-ranging deer and elk share common pastures with domestic livestock, there has been no evidence of natural transmission to livestock.

Is the disease a threat to the food supply?

There is no known threat to the food supply from CWD. However, because of Great Britain's experience with vCJD in people, which has been linked to BSE, and the fact that there is still a lot to learn about CWD, experts are suggesting a few common sense precautions to hunters:

- Don't shoot an animal that is acting abnormally or looks sick or emaciated.
- If you see a deer or elk that fits that description, immediately contact the nearest Kansas Department of Wildlife and Parks conservation officer or district wildlife biologist.
- Wear rubber or latex gloves when you field dress a harvested deer or elk.
- In areas where CWD has been reported, minimize contact with a dead deer or elk's brain and spinal cord, and wash your hands after contact.
- When boning out deer or elk meat, do not include brain or spinal cord and, discard the brain, spinal cord, eyes, spleen, and lymph nodes.
- Bury the unused parts of the carcass.

How is chronic wasting disease diagnosed?

Research into the exact cause and transmission of CWD and the other TSE has been slow, in part because diagnosis has depended on microscopic examination of brain tissue from dead animals. Several new live animal tests are currently being evaluated that should facilitate diagnosis and further our knowledge of CWD and the other TSE and enable eventual eradication.

New regulations to protect Kansas deer and elk.

Because of the threat of CWD, the Kansas Animal Health Department has recently promulgated new regulations to help protect the health of Kansas deer and elk. These regulations require certain stringent qualifications for all animals of the cervidae family (deer and elk) imported into Kansas.

For farmed deer and elk in Kansas, a monitoring program is being developed for producers to follow to be able to determine the status of their herds and to work toward certification of being free of CWD.

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Kansas VETERINARY Quarterly
FOR THE PRACTICING VETERINARIAN

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