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

Spring 2003

Volume 6, Number 1

Welcome . . .

Dr. Larry Hollis is the new Beef Veterinarian and coeditor of *Kansas State Veterinary Quarterly* newsletter. He joined K-State in September, bringing extensive experience in food animal medicine, management and diagnostics.

A native of the Texas panhandle, he received his DVM degree from Texas A & M University. He completed a large animal clinical internship at Oklahoma State University and worked in private practice in Texas, specializing in feedlot and stocker health management.

Larry was head of diagnostic services for the Texas Veterinary Medical Diagnostic Laboratory in Amarillo for seven years. While there he earned a master's degree in beef management and nutrition from West Texas A & M University.

Larry has taught beef cattle/feedlot production medicine and worked in industry as a manager of field technical services for Syntex Animal Health and as senior technical service veterinarian for Pfizer Animal Health. We are glad to welcome him aboard.

Also In This Issue

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

Vet Quarterly to be Available Only Online

This is the final printed issue of the *Kansas State Veterinary Quarterly* newsletter. Beginning with the next issue, the publication will only be available online at *www.oznet.ksu.edu/dp_ansi/nletter/VetQuarterly.htm*. To subscribe to this newsletter electronically, send an e-mail message to: *mailserv@lists.oznet.ksu.edu*.

Leave the subject line blank. In the first line of the message, type: subscribe VetQuarterly. Then press the "enter" key twice to add a blank line following the message. Then press "Send". To unsubscribe, follow the same procedure except in the body, type: unsubscribe VetQuarterly. When a new issue is available you will receive an e-mail notice containing a Web link for you to access the newsletter. Pass this information along to other veterinarians who might benefit.

Accessing Vet Quarterly on the Web will allow more timely delivery and ensure availability despite an uncertain budget. If you don't have Web access, contact Linda Siebold at *lsiebold@oznet.ksu.edu* or 785-532-1281 for options.

If you have questions, contact Larry Hollis, 785-532-1246, *lhollis@oznet.ksu.edu* or George Kennedy, 785-532-4454, *kennedy@vet.ksu.edu*.

KSU Diagnostic Lab Now Testing for Chronic Wasting Disease of Deer, Elk

Chronic Wasting Disease (CWD) is a specific, infectious, neurological disease of deer and elk in the United States and Canada. The disease is one of a group of diseases called transmissible spongiform encephalopathies (TSEs). It is similar to, but not the same as, scrapie in sheep, bovine spongiform encephalopathy (mad cow disease), and to a disease in humans called new variant Creutzfeldt-Jakob Disease (nvCJD).

There is convincing evidence that the new variant form of Creutzfeldt-Jakob Disease is caused by the same agent that causes bovine spongiform encephalopathy and is the result of ingesting beef contaminated with brain or spinal cord tissue from affected cattle. Bovine spongiform encephalopathy (BSE) has never been found in the United States. Scientific evidence to date indicates CWD of deer and elk is a distinct disease from these other diseases.

The cause of CWD, and the other TSEs, is not known for sure, but has been associated with the accumulation of an abnormal, protease-resistant protein referred to as prion protein in the brain, and in some instances, eyes and lymphoid tissue of affected animals. Demonstration of this protein in tissue is the basis of current diagnostic tests.

Evidence suggests that deer and elk may become infected by ingesting feed or water contaminated by infected saliva, urine and/or feces.

Captive and free-ranging mule deer, white-tailed deer, and elk are all susceptible. Research suggests other ruminants such as wild and domestic sheep, goats, cattle,

See CWD, page 7

West Nile Virus in Kansas in 2002

After months of preparation and planning with Kansas Department of Health and Environment (KDHE) and Field Coordinator, Dr. Tom Jurasek, the Kansas State Diagnostic Laboratory (KSVDL) started testing birds and mosquitoes for West Nile virus in May. Dr. Jurasek and his crew collected mosquitoes, bled sentinel chickens and picked up dead birds from all over the state when notified by citizens of the presence of dead birds in their area. Surveillance emphasis was placed on corvids (in most of Kansas, that means crows and blue jays, but in western Kansas, magpies also qualify).

Preparations were made to test horses via two methods. One test was done serologically using the capture ELISA test for the presence of IgM. This test measures IgM, which is the first gamma globulin produced following an infection and is indicative of early or active infection. The second test was done via the PCR test (polymerase chain reaction) for viral RNA in tissue. A PCR test was also used for testing the mosquito pools.

In spite of bird surveillance and the expectation that the first cases would be in birds and in eastern Kansas, the first animal found with West Nile virus was a horse in Cowley County the second week of August.

Infection was confirmed in this case via serology, PCR testing for virus in the brain tissue, and histologic examination of formalin-fixed tissues for evidence of viral encephalitis. The horse was positive via all those methods.

Following that initial horse, positive birds, horses, and mosquito pools became a nearly everyday occurrence at the Diagnostic Laboratory. Through the end of October, 1,262 horse sera were submitted for serologic testing and 716 positive samples were found. These were horses showing neurological signs and with a positive IgM test and were presumed to be sick due to West Nile virus.

Keep in mind that a positive antibody test only means an animal has been exposed to the particular agent and has responded immunologically.

Tissues from 50 horses and mules were tested, either from necropsies performed at KSVDL or performed in the field by veterinarians, and 36 were positive for West Nile virus via the PCR test.

Increased rabies activity this year complicated the field diagnosis on neurologic horses. Preliminary estimates suggest that close to 30 percent of affected horses either died from the disease or were euthanized because of the disease.

Surveillance in birds was performed mainly using immunohistochemistry on formalin-fixed tissues. More than 350 birds were collected representing all counties in the state, and submitted to the Diagnostic Laboratory. One hundred fifty-seven birds were found positive via either immunohistochemistry on formalin-fixed brain tissue or PCR. Twenty-nine different species of birds were tested, with positive birds from 11 different species.

Nationwide, and since the virus was first found in New York, more than 100 different species of birds have been found positive for West Nile virus. Of concern was that large raptors also seemed to be quite susceptible. The KSVDL identified West Nile virus in great horned owls, hawks and golden eagles.

By the end of October, 103 of 105 counties had evidence of West Nile virus activity either from mosquito pools, positive birds and/or positive horses. At this time, 15 people with neurologic signs had been found positive, with several more cases pending at CDC. So far, there have been no human deaths reported from West Nile virus in Kansas.

The virus spread more quickly than expected, and widespread drought conditions did not appear to inhibit the spread of the virus. As predicted, the virus was mainly confined to birds, horses and people and of course its mosquito vectors, but the virus seemed to expand its host range this year.

A number of states, including Kansas, confirmed low numbers of West Nile virus infection in a variety of animals including squirrels, emus, mountain goats, dogs, cats, sheep and reindeer. In Kansas, cases were confirmed in squirrels, an emu and a mountain goat.

It is known that many animals will seroconvert, so without a necropsy, histologic lesions and demonstration of virus in tissues, actual disease due to West Nile virus is largely speculative. It would still appear that most mammals, other than horses, are highly resistant to the virus, but on occasion dogs and cats apparently can develop clinical disease.

New Regs for Shipping Samples

Effective February 14, 2003, the Code of Federal Regulations (CFR) changed some of its requirements for packaging and shipping of hazardous materials. Diagnostic specimens, any human or animal material not known or suspected of containing a pathogen, should now be shipped with the triple packaging system described below. These shipping conditions are defined in all transportation company policies and meet the International Air Transport Association (IATA) regulation for Packaging Instruction 650.

The basic triple packaging system consists of a waterproof primary container, a waterproof secondary container, absorbent material and a “sturdy” outer shipping container. The primary container can be a Vacutainer tube sealed with adhesive tape, screw-capped conical tubes, or other plastic screw-cap containers. The waterproof secondary container includes Ziplock plastic bags, conical 50 ml test tubes, and screw-cap containers.

The absorbent material should be placed between the primary and secondary container. The quantity of absorbent material should be sufficient to absorb all liquid contained in the primary container and can consist of items such as paper towels, cotton balls, or filter paper. The outer shipping container is most often cardboard. Styrofoam containers should be within an outer cardboard box. Plastic bags and paper envelopes are unacceptable outer containers for shipping biological materials.

For shipment of clinical specimens, United Parcel Service (UPS), Federal Express (FedEx), and Airborne Express all provide, free of charge, a plastic outer pouch labeled “Diagnostic Specimens”. Small boxes of properly packaged materials are slipped inside these lab packs, so that the plastic pouch is over the secondary container.

When shipping clinical specimens through the United States Postal Service (USPS), in addition to the triple packaging system, you are required to place a label on the outer package marked “CLINICAL SPECIMAN-BLOOD, URINE, TISSUE, etc. as applicable. The USPS Packaging Instruction 6C is available at your local post office for reference.

If you have questions, please call the Diagnostic Laboratory at 785-532-4349 for specific recommendations and guidelines.

Lingering Drought Effects: Vitamin A Deficiency, Nitrate Toxicity

Larry C. Hollis, D.V.M., M.Ag.
Beef Veterinarian, Kansas State University

Even though several of the drought-stricken areas of western Kansas have received moisture during the past weeks, residual drought effects may linger and cause problems if not taken into account.

Probably the most prominent and insidious effect may be vitamin A deficiency. Because pastures were so dry for so long during the summer, and many did not have the opportunity to green up sufficiently following any late rain and before cold weather set in, vitamin A stores in the liver of many cattle may have been depleted earlier in the fall. In cows, deficiency can produce rough hair coats, poor milking ability, poor reproduction, increased susceptibility to diseases such as pinkeye, pneumonia, etc. As the disease progresses, central nervous signs such as staggering gait, convulsions and acute death have been observed. At calving time, retained placentas are commonplace, and there is an increased incidence of uterine infection. In severe cases, cows may die from uterine infections caused by normally non-pathogenic bacteria.

Calves born to deficient cows are often born weak, slow to nurse, and may be born blind. These calves are highly susceptible to scours and pneumonia from a variety of bacteria, which are normally non-pathogenic. As calves grow, development of the optic nerves connecting the eyes to the brain is compromised, and the calves gradually go blind. Frequently, this is not noticed until calves are moved to unfamiliar surroundings, where they tend to panic, run into objects, and may go into convulsions.

Unless the vitamin A status is known to be good, cattle from drought-affected areas should be considered to be vitamin A deficient. An injection of vitamin A is the quickest way to correct the short-term situation. Incorporation of vitamin A in supplemental feeds or mineral mixes should be used to help ensure that the deficiency does not reoccur before grass greens up next spring.

Nitrate toxicity may continue to be a problem in both grazed crop residues and baled forages throughout the winter. Nitrates tend to accumulate in the lower one-third of the stalk and become a greater risk as cattle gradually clean up the fallen grain and leaves and finally begin eating the stalks. This risk is accentuated during periods of snow cover unless alternate desirable feedstuffs are also made available. Consumption of larger than normal quantities of marginally toxic feedstuffs may occur during periods of adverse weather, resulting in cumulative nitrate toxicity problems.

There is probably still some aflatoxin-contaminated corn around from last summer's drought. Some producers or grain dealers may try to offer this corn for sale now that the heightened aflatoxin awareness period has passed. Even though the legal limit for sale of corn for use in feeding cattle is 300 ppb, Diagnostic Labs at KSU and Texas A&M report dramatic reductions in performance anytime corn contains more than 100 ppb aflatoxin.

The bottom line is that producers need to know the potential toxicity problems associated with what is being fed. They should be reminded to beware of bargain feed, either as baled forage or whole corn. If in doubt, have the products tested for suspected toxins.

Weather Favors Mold Growth, Raises Mycotoxin Concerns

John A. Pickerell and Frederick W. Oehme,Veterinary Diagnostic Laboratory, Department of Diagnostic Medicine/ Pathobiology

Mycotoxins hazardous to food animal production are produced by the molds *Aspergillus, Penicillium* and *Fusarium* after growth on food grains. Moisture as well as fall or winter temperatures favor mold growth. Drought will damage grain envelopes making grain carbohydrates more accessible for mold growth. Last fall's combination of moisture following summer drought should alert us to the possibility of mold contamination in our food grains. Damaged grain makes available an energy source that favors the growth of molds.

Molds grow in spots that have ideal temperatures and moisture for their growth, producing mycotoxins optimum to those growth conditions. Mycotoxin production rates are associated with the bins or feeders holding the food grain. If non-moldy grain is put in the same structure, within 2 to 4 weeks similar molds will once again grow. These molds will produce mycotoxins that rise to the same levels as were in the previously stored grain. Thus, molds growing on damaged grains with optimal moisture will release mycotoxins when they reach optimum numbers.

Moisture and fall temperatures assist mold growth, generating mycotoxins and causing deleterious effects in livestock that consume them. For example, aflatoxin, a known human carcinogen can contaminate cows milk, resulting in condemnation of that milk. In addition, aflatoxin can reduce growth and immune function in food animals. It is secreted by either *Aspergillus* or *Penicillium* growing on corn or milo at 16 to 28 percent moisture and 55 to 90 F, with the greatest mold growth at 78 to 90 F.

Zearalenone affects reproductive function in gilts, sows, boars, heifers and cows in Kansas. It is generated by *Fusarium* growing on corn, milo, barley or wheat at 22 to 25 percent moisture, with alternating high and low temperatures (45 to 70 F). This growth is recognized as scab or pink ear rot.

Vomitoxin at low concentrations is associated with feed refusal and reduced immune function in cattle and swine. It is produced by *Fusarium* growing on corn, milo, rye, barley or wheat at alternating cool and warm temperatures from 40 to 60°F.

Aflatoxin, zearalenone and vomitoxin can be assayed from grains sampled from trucks, bins or feeders containing suspect feed. Samples are collected in much the same way as probes used to obtain grain moisture samples. The individual collections are combined and about 1 pound of the grain mixture is submitted for analysis. The samples should be kept under cold conditions so that the mycotoxins are measured reflect those already present at the grain holding facilities. Mycotoxins are extracted from pulverized grains, separated on thin-layer chromatograms and detected by colors when sprayed with specific reagents. The

See MYCOTOXIN, page 7

Dogs and Cats with Repeat Low Rabies Antibody Levels Studied

Kristen Schweitzer, Susan Moore, Lisa Bausch and Rolan Davis; Kansas State University Rabies Laboratory

Abstract

Dogs and cats with Fluorescent Antibody Virus Neutralization (FAVN) results repeatedly below the 0.50 International Units per milliliter (IU/mL) level required for export to rabies free areas were evaluated. Veterinarians were asked to complete a questionnaire. Data collected included age, breed, medical conditions, medications, rabies vaccination and route of administration. Frequently reported data included subcutaneous route of vaccination, the use of Purevax vaccine in cats, and a majority of dogs were from a few large breeds. Senior age may be a factor in cats with repeatedly low rabies antibody titers as well.

Introduction

Rabies antibody testing has become increasingly popular as a method of verifying rabies vaccination and immune response in dogs and cats that are being imported to rabies-free areas. This is in part due to increasing public pressure for a reduction in lengthy quarantines. Kansas State University has been conducting rabies antibody titers via the FAVN (Fluorescent Antibody Virus Neutralization)[1] method for the purpose of animal export since 1997. A titer sufficient for exporting a domestic dog or cat to many rabies free areas is >/= 0.50 IU/mL. This standard was set by the World Health Organization[2] as the level that assures specific rabies antibody as distinguished from non-specific neutralization. There is no established *protective* rabies antibody level for animals. Occasionally, an animal's rabies antibody titer may not reach this level even though it has been currently vaccinated and a two to three week interval has passed before the serum sample was drawn. Alternatively, there are animals that reach a 0.50 IU/ml titer, but fail to hold that level for more than a few months. For the purpose of this study, only animals that had at least two non-passing FAVN results (titers below 0.50 IU/mL) were examined. The database was searched retrospectively through the year 2000, (a total of 14,820 cases). A total of 34 animals were identified as repeat failures. Each submitting veterinarian was contacted and asked to complete a form containing information about the animal's identity, age, breed, sex, spay/neuter status, rabies vaccinations (type, dates, lot numbers, and route of administration), medical conditions, additional vaccinations or medications given at or near the time of rabies vaccination, and comments about stress or other conditions the animal may have had. A total of 28 responses were obtained.

Results

The information from the 28 completed questionnaires was compiled and examined for correlations.

Breed

The distribution of the 28 animals included 20 dogs of nine different breeds, seven cats, and one ferret. The ferret was included as some rabies-free areas include ferrets in their reduced quarantine policy. Certainly, dogs composed the largest percentage of repeat failures (70%) and of those, large breed dogs comprised the majority. Certain breeds such as the Labrador Retriever, German Shepherd, Australian Shepherd and the Golden Retriever appeared frequently, but it is unclear if this may be due to those breeds' popularity as pets or if there may be a genetic correlation. Seventeen of the dogs were purebred. The cats were primarily domestic shorthairs, one Siamese mix and one Persian mix.

Age

The age of animals with repeat low rabies antibody titers varied from one year to 13 years in range. Four of the seven cats (57%) were 12 years or older. Twelve of the dogs (60%) were four years of age or younger. The ferret was three years old.

Vaccine type and route of administration

The rabies product used and frequency among all study animals is demonstrated in Figure 1. Most of the major manufacturers of rabies vaccines marketed in the United States were represented. Purevax is a non-adjuvented canary-pox vector vaccine licensed for use in cats only. The route of vaccinations reported was 100% subcutaneous (SQ). There were no failures involving intramuscular (IM) vaccinations reported. Eight of 28 animals (31%) reported receiving another vaccination at the same time as a rabies vaccination.

Medical Conditions

Data was collected concerning any medical conditions the animal may have had, medications and vaccinations they were given, and general comments on the condition of the animal. The relevant data is displayed in Figure 2. Some animals reported more than one condition, but the majority (57%) reported no medical condition or concerns. Thyroid condition included both hypothyroidism and hyperthyroidism

Discussion

While there appears to be no single reason why some dogs, cats and ferrets occasionally may not reach or maintain a rabies antibody level of at least 0.50 IU/mL, there do appear to be several factors worth considering. Five of seven felines reported use of Purevax. The route of immune stimulation may be different than that of other inactivated adjuvanted vaccines due to the live canary pox vector of Purevax vaccine .The

See RABIES, p. 5

Possible Reasons for a Low Response

Physiological

- Blood drawn same day as vaccinated
- Underlying immuno-suppressed condition
- Only one vaccination in lifetime
- No recent vaccination
- On medication that may inhibit a response.
- Breed of animal?

Mechanical

- Vaccine administered improperly
- Vaccine expired or stored improperly
- Questionable vaccination history (no vaccinations or unknown history)
- Route of administration
- Type of vaccine

immune response stimulated, while sufficient to allow the animal to survive challenge, may not yield as long-lasting titer. However, it may also appear in this study more often due to current popularity of this vaccine. Senior age may also play a factor in immune response to rabies vaccine in cats. This could be due to physical changes in the immune system of the elderly cat.

Certain dog breeds such as Golden Retrievers, Labrador Retrievers, German Shepherds, and Australian Shepherds, may be genetically predisposed to immune problems and therefore to lower antibody titers. However, these breeds are also very popular pets and could be submitted much more frequently than others. It appears as if large breed dogs in general do have a higher frequency of repeat low rabies antibody titers (90%). The dose of rabies vaccine is the same regardless of animal size. Large dogs may not generate as high a titer as compared to small dogs with the same dose.

Stress has been demonstrated to suppress the immune system in some instances[3]. Working dogs such as certified guide dogs and show dogs may be under additional stress. The fact that at least 20% of the dogs in this group were also coincidentally guide dogs or show dogs seems significant. A quantitative study of guide dog rabies antibody titers via FAVN is currently being compiled at Kansas State University.

The only universal factor among all twenty-six cases in this group is the subcutaneous route of vaccination (100%). The ferret was vaccinated subcutaneously and appears to have no other commonalities. A certain amount of antigen is likely absorbed into the lipid layer when vaccine is delivered subcutaneously that would not be when given intramuscularly. This may in part explain reports of some decreased titers via the subcutaneous route compared to the intramuscular route[4,5]. However, with no intramuscular route data in this study to compare to, it is inconclusive.

Recently, there has been a trend away from intramuscular rabies vaccination because of the reported risk of vaccine-associated sarcomas[6] and increased risk of adverse reactions. This would seem to justify additional studies into the efficiency of the route of immunization.

The existence of even a small percentage of repeatedly low rabies antibody titers among the export animal population is frustrating to the pet's owners and veterinarians. Any helpful data generated is surely welcome. Documented cases such as these also serve to authenticate the animal import process of vaccination and rabies antibody titration before entry to rabies-free areas.

LITERATURE CITED

1. Cliquet F, Aubert M, Sagne L. 1998. Development of a fluorescent antibody virus neutralization test (FAVN test) for the quantitation of rabies-neutralising antibodies. J Immun Meth 212, 79-87.

2. Briggs, DJ etal. 1998. A comparison of two serological methods for detecting the immune response after rabies vaccination in dogs and cats being exported to rabies-free areas. Biologicals 26, 347-355.

3. Cohen S, Miller GE, Rabin BS. 2001. Psychological stress and antibody response to immunization: a critical review of the literature. Psychosom Med 63, 7-18.

4. Sikes, RK, Peacock GV, Acha P, Arko RJ, Dierks R. 1971. Rabies vaccines: Duration of immunity study in dogs. J Am Vet Med Assoc 159, 1491-1499.

5. Aubert M. 1992. Practical significance of rabies antibodies in cats and dogs. Rev Sci Tech Off Int Epiz 11, 735-760.

6. Morrison, WB, Starr RM. 2001.Vaccine-associated feline sarcomas. J Am Vet Med Assoc 218, 697-701.

Check the Kansas State University Rabies Laboratory Web site for information and serology submission forms:
http://www.vet.ksu.edu/rabies

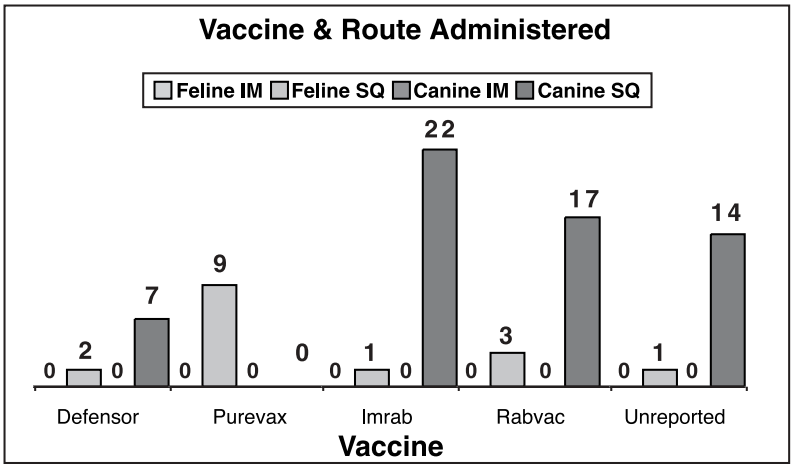

Figure 1. The number of vaccinations and product type reported for all animals. A majority (71%) of the cats reported receiving Purevax exclusively or at least once. One cat had unreported vaccine type and one reported not receiving Purevax. One hundred percent of all vaccines reported before failing titers were administered subcutaneously.

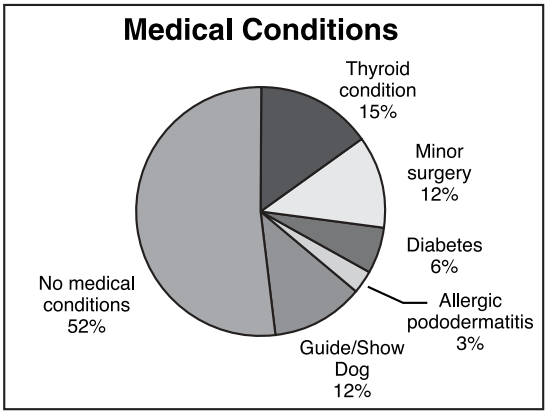

Figure 2. Medical conditions and general health reported for all animals included in the study. Three guide dogs and one show dog were included due to the frequency of inclusion in the "comments" section of the questionnaire, but were not considered a medical condition.

Possible Causes of Weak Calf Problems

T*wig Marston, Ph.D., Beef Specialist, Kansas State University*

Weak calves can be a frustrating problem for cow/calf producers. They normally won't stand to nurse, don't get colostrum unless force fed (often too late for adequate transfer of passive immunity) and often have an increased susceptibility to a variety of secondary diseases.

Economic losses can range from nonexistent to catastrophic depending on the percentage of newborns that are affected. Several factors can individually or in combination cause weak-calf syndrome, making it a tough problem to correct once it appears.

Dietary protein levels pre-calving can have a major effect on calf vigor and survivability. Research indicates that cows consuming two or more pounds of crude protein daily have very few "weak-syndrome calves". In the same study dams that were consuming less than a pound and a half of crude protein daily had nearly 10 percent weak calves.

Forage testing is the easiest and best way to determine the crude protein content of feedstuffs. Forage test results will indicate the percent of protein within the sample, and by simply multiplying that percentage by the amount of feed consumed daily per head, producers will have calculated the protein intake of their cows. Another study conducted by Colorado State University researchers showed that restricting protein intake before calving will significantly extend the time it takes for calves to stand and nurse.

Vitamin A should be another big concern to cattle producers. The drought has caused them to use many feedstuffs that are naturally deficient or completely devoid of vitamin A. Dark green forage or hay color is usually a telltale sign of vitamin A concentration when it comes to visual appraisal. During this past fall and winter it seems this color of feeds have been in sparse supply.

Common characteristics of vitamin A deficiency are increased abortions, more retained placentas, and possibly the birth of weakened, blind, or dead calves. Supplementation through ingestion or injection of vitamin A will quickly remedy any deficient status. Under normal circumstances beef cows in early lactation will require 30 to 50,000 IU of vitamin A daily. A common and inexpensive route of administration is to include vitamin A via a mineral/trace mineral supplement. If the situation is critical, injection with the 2,000,000 IU of vitamin A (normally 4 mL) is recommended.

When trying to find the cause of weak calves one must consider all possibilities. Unfortunately, the disease known as leptospirosis can be the culprit. Several strains of leptospirosis have been isolated making vaccine selection and timing very important management decisions. Lepto can silently sneak up on a cowherd because the initial signs often pass unnoticed making the first symptoms of lepto an abortion storm or a series of stillborn and/or weak, nonresponsive calves. In some areas or herds with high exposure levels, a single annual vaccination given at pregnancy checking time may not be sufficient to stop reproductive losses. Submitting a calf for necropsy will help rule out this disease as a possibility.

Finally, calving difficulty can have a direct effect on calf vigor. The longer and slower the birthing process, the greater the tendency for the calf to become oxygen-deprived and the greater the amount of trauma experienced by the dam and her calf. As birthing trauma increases, so does swelling and soreness. Studies have shown that when oxygen deprivation of up to 8 minutes occurs during the calving process, calves are frequently born alive but die within 10 to 15 minutes after birth.

Weak, newborn calves are a frustrating problem to confront. Because the various factors discussed can work independently or in combinations, they make weak calf syndrome difficult to conquer. Closer observation, timely intervention and diagnostic support in herds experiencing weak calves is critical. A total management program, which includes teamwork between cow/calf producers, their veterinarians, extension, and other supporters, will be most effective in providing the best animal husbandry.

CWD, from page 1

pronghorn antelope, bison, and moose are either resistant or less susceptible.

Affected animals have been found in farmed and/or free-ranging deer and elk in Colorado, Wyoming, Nebraska, Montana, Minnesota, South Dakota, Kansas, Wisconsin, Illinois and Oklahoma. As far as we know, people are not 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.

There is no known threat to the human food supply from CWD. But because of Britain's experience with nvCJD in humans, which has been linked to BSE, and the fact that there is still a lot to learn about CWD, public health and animal health officials suggest a few precautions to hunters:

- Do not shoot, handle or consume any 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 field dressing a harvested deer or elk.
- When boning out deer or elk meat, do not include brain or spinal cord and discard brain, spinal cord, eyes, spleen and lymph nodes. Infective material has not been found in skeletal muscle. Normal field dressing coupled with boning out of carcasses will remove most, if not all, of these body parts. Cutting away all fatty tissue should remove remaining lymph nodes.
- Wash hands and instruments thoroughly after field dressing is completed.
- Avoid consuming brain, spinal cord, eyes, spleen, tonsils, and lymph nodes of harvested animals. Avoid consuming the meat from any animal that tests positive for the disease.
- To test a harvested deer or elk for CWD, the head should be removed between the first cervical vertebrae and the skull and a segment of the brain stem immediately behind the cerebellum submitted in formalin to a laboratory performing the test.
- Bury the unused parts of the carcass.
- Request your animal be processed individually, without meat from other animals being added to the meat from your animal.

Research indicates that this protease-resistant prion protein does not accumulate in muscle tissue, but prudence suggests that meat from positive animals not be consumed even though, as mentioned above, there is no known evidence that humans are susceptible to CWD.

The KSU Diagnostic Laboratory is now performing CWD testing on formalin-fixed brain tissue from deer and elk. This is a sensitive test, but very dependent on examining the proper area of the brain. The area required is the obex, which is the portion of the brain stem just behind the cerebellum where the fourth ventricle goes into the spinal canal. Without this portion of the brain, the test can not be properly interpreted and will have to be reported as unsuitable.

The KSU Diagnostic Laboratory is not able to accept whole heads or carcasses, as we have no way of disposing of positive heads or carcasses.

If planning to submit specimens for CWD testing, please call the lab in advance to make arrangements for testing details and fee information. Call 785-532-5650. Web address is *http://www.vet.ksu.edu/depts/dmp/service/index.htm*.

MYCOTOXIN, from page 3

color intensity is compared to standards to quantitate the presence of the various food grain mycotoxins.

The Comparative Toxicology Laboratories at Kansas State University's Veterinary Diagnostic Laboratory can analyze aflatoxin or use "mini-profile" assay (aflatoxin, zearalenone, vomitoxin) employing thin-layer chromatograms within 1 day after receiving samples. The laboratory's 24-hour, 7 days a week toxicity hotline, 785-532-5679, is also available to discuss mycotoxin concerns, results of feed analysis or offer consultation on potential poisonings.

Measurement of mycotoxins levels is the most useful way of predicting potential deleterious effects on food animal production, growth, immunity and reproduction after consumption of moldy food grains. Mold culture will give numbers of fungi (*Aspergillus, Penicillium, Fusarium and Cladosporium*) that indicate the potential to produce mycotoxins. But fungi numbers do not determine the actual presence of mycotoxins. Only direct measurement of mycotoxin concentrations in feed allow veterinarians and animal managers to predict potential toxicity to their animals and any undesirable conditions that will result from feeding moldy grains.

COOPERATIVE EXTENSION SERVICE
U.S. DEPARTMENT OF AGRICULTURE
KANSAS STATE UNIVERSITY
MANHATTAN, KANSAS 66506

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE $300

Continuing Education

April 5, 2003
Veterinary Technicians Conference

April 6, 2003
Small Animal Medicine Lecture Series
on Snow White and the Seven Dwarves

May 3, 2003
Investigating Pregnancy Wastage in Cattle Herds

June 1-4, 2003
65th Annual Conference for Veterinarians
Highlights from 2002 Heritage evening

For the most complete, up-to-date, conference information visit our Web site at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Linda M. Johnson, Ph.D. at 785-532-5696 or *Johnson@vet.ksu.edu*

The Kansas State University Diagnostic Laboratory and Department of Animal Sciences and Industry at Kansas State University greatly appreciates the sponsorship of the Kansas Veterinary Quarterly Newsletter by Pfizer Animal Health. These sponsorships in no way imply the Departments' endorsement of the products and services offered by the sponsors. The Departments welcome inquiries from other individuals, associations and firms that may be interested in cosponsoring this publication.

Kansas KSTATE VETERINARY FOR THE PRACTICING VETERINARIAN Quarterly

Newsletter Coordinators

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

G.A. Kennedy
785-532-4454 • kennedy@vet.ksu.edu

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