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Individual animal identification: it's coming fast

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The discovery of the BSE-infected Holstein cow in Washington state reinforces what most veterinarians understand all too well – the United States does not have a uniform animal-disease tracking system. This fact was underscored by the inability to trace all 81 herd mates of the infected Canadian-born cow to determine their location since their arrival in this country.

For several years, organizations including the National Institute of Animal Agriculture (NIAA), the USDA, and the U.S. Animal Health Association (USAHA) have been working to create an animal identification plan that meets the needs of the U.S. livestock and animal industries – the United States Animal Identification Plan (USAIP). USAIP has the support of the National Cattlemen's Beef Association, most breed associations, the Kansas Livestock Association and the Kansas Animal Health Department.

The USAIP calls for a coordinated program to identify premises where animals are raised in each state, then individually identify the animals raised on each premise. Animals would be tagged with an electronic ear tag containing a unique 15-digit number encoded in the tag. Specific ear tag numbers would be assigned to each premise.

Once installed, tags would remain with the animal for life, similar to a citizen's social security card. Tags would use radio frequency indentification (RFID) technology so the tag numbers could be read, stored and transmitted electronically. Each time animals changed location, the tag would be scanned and the new location reported to a central data collection point. The use of RFID technology would be required so information could be transferred and traced quickly and accurately.

In a Dec. 30, 2003, address concerning new BSE control measures, U.S. Secretary of Agriculture Ann M. Veneman, said that implementing a plan to enable traceback of a foreign animal disease within 48 hours of discovery was a priority. As a result, details of the USAIP are being completed quickly.

The first phase of the plan (allocation of identification numbers by the individual states) is tentatively scheduled for July 2004. The second phase (electronic ID of all animals entering interstate commerce) is tentatively scheduled for July 2005. The third and final phase (electronic ID of all animals moving in intrastate commerce) is tentatively scheduled for July 2006.

Veterinarians may be called upon by their clients to provide one or more of the following services: help in procuring RFID tags, applying tags, scanning or reading tag numbers electronically, and forwarding premise relocation information to the central database. Veterinarians will likely receive questions about details of the plan and how it will affect livestock producers. For details on the USAIP, or to keep up with the development of the plan, log on to *www.usaip.info.*

Also in this issue

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

Watch for signs of deadly avian influenza

On Feb. 23, the USDA confirmed an outbreak of highly pathogenic avian influenza (HPAI) in a flock of chickens in Texas. This is the first outbreak of highly pathogenic avian influenza in the United States in 20 years. This strain has been typed as H5N2. Other recent avian influenza outbreaks have been of the low pathogenic strains. Some low pathogenic avian influenza (LPAI) strains can mutate to highly pathogenic strains.

While Kansas does not have a large poultry population, this development concerns everyone because avian influenza (AI) can infect most avian species and be easily transmitted. Migratory waterfowl are the main reservoir for AI.

Kansas practitioners should be alert for flocks of domestic poultry, game birds or pet birds that are affected by a disease with high morbidity and mortality and showing one or more of the following clinical signs:

- Sudden death
- Depression and anorexia

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Q & A: Bovine spongiform encephalopathy

Report from the American Association of Bovine Practitioners – Food Quality, Safety and Security Committee

By now, few have not heard about bovine spongiform encephalopathy (BSE), commonly known as mad cow disease. There is still a great deal we do not know about the disease, but this article should give a basic understanding.

Q. What is bovine spongiform encephalopathy?

A. The disease commonly known as mad cow disease is known in veterinary circles as bovine spongiform encephalopathy. This disease has only been found in one cow in the United States. Any suspected case must be reported to animal health authorities (state and federal veterinarians).

Q. What causes BSE?

A. Researchers have isolated a small protein that can infect other animals if it is injected or fed to them. The most accepted theory is that the protein is an altered form of a naturally occurring protein called a **prion**, which is smaller than a virus. The altered prion recruits other normal prions to act in the same manner, accumulating in brain and spinal tissue and interfering with normal function. Prions are more resistant to destruction than most viruses and not all rendering and sterilization processes will destroy this protein.

Q. What are the signs of BSE?

A. Signs include nervousness, aggression, increased startle response to stimuli (such as noises or contact), depression, severe muscle tremors, unusual gait or stance, reduced rumination, altered heart rhythm and weight loss. Although the animal will be infected with BSE for approximately two to eight years before showing any symptoms, once signs appear the disease progresses rapidly and generally the animal will die within six months. The signs of BSE are similar to many other diseases that affect the nervous system of cattle, including grass tetany, nervous ketosis, listeriosis (circling disease), polio and rabies.

Q. Is there a test for BSE?

A. Currently, testing in cattle occurs after death. The brain must be examined under a microscope to find characteristic lesions, which are holes in the brain tissue that give it a sponge-like appearance. There is not a test available to perform on live cattle to check for BSE. There is at least one company that is very close to marketing a live animal test. Even in humans, the diagnosis of similar diseases is usually confirmed only after death.

Q. Is this disease unique?

A. No. BSE belongs to a group of diseases known as transmissible spongiform encephalopathies (TSE). These diseases include scrapie in sheep and goats, chronic wasting disease (CWD) in elk and deer, transmissible mink encephalopathy in mink, and feline spongiform encephalopathy in wild and domestic cats (including large cats such as lions). Exotic and domestic ruminants have been diagnosed with BSE, which is associated with consumption of contaminated feed. There are a number of TSE diseases in humans as well, including Kuru, Gerstmann-Straussler-Scheinker Syndrome, fatal familial insomnia, Creutzfeldt-Jakob Disease (CJD) and variant Creutzfeldt-Jakob Disease (vCJD).

Examination of the brain tissue and proteins produced in infected animals shows that BSE, feline spongiform encephalopathy, transmissible mink encephalopathy and vCJD are indistinguishable and could possibly be transmitted between species if infected tissues are consumed. In fact, the apparent source of feline spongiform encephalopathy, transmissible mink enceophalopathy and vCJD was BSE-contaminated food.

Q. How is BSE transmitted?

A. Consumption of contaminated feedstuffs is the primary means of infection. British officials believe BSE may have been present, but undetected, in a very small number of cattle for years or that it is a mutated form of scrapie. The widespread use of ruminant-derived protein, including brain and spinal cord tissue, in cattle diets recycled the infection. Because less than one gram of infected material is necessary to cause infection, the disease was amplified in the cattle population by this feeding practice. Offspring of BSE infected cattle are more likely to develop BSE; however, it is not known why this occurs.

Q. Is BSE in the United States?

A. As of January 2004, BSE has been isolated in one dairy cow in the United States and one beef cow in Canada. Both animals originated in Alberta, Canada, which is of importance with respect to possible source of infection and bans on beef imports to other countries. Both animals were born before the ban of ruminant-derived protein feeding to other ruminants, which may be the source of their infection.

Not only has USDA-APHIS been examining brains, but they have directed efforts toward the brains of animals that have the highest risk. Cattle sent to a slaughter facility with signs of neurological disease have their brains submitted for testing. A subset of other non-ambulatory (downer) animals without signs of neurological disease is also tested. This is how the cow recently discovered in the United States was identified. She was unable to stand because of a calving injury. Because she did not have a neurological disease, her meat was allowed into the human food supply before testing was completed. USDA changes in the ability of packing plants to take downer cows will make this type of surveillance effort much more difficult.

Q. What are we doing to prevent BSE in our cattle population?

A. The risk of BSE occurring in the United States is minimal and decreasing because we have taken preventive steps to break the disease cycle.

The USDA and the FDA are reviewing management practices that place our cattle population at risk. When research shows that something is a potential risk, regulatory measures are enacted to prevent the risk. In 1985, the United States banned beef imports from the United Kingdom because of diseases other than BSE. In 1989, live animal imports from

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countries affected by BSE were banned. In 1996, a voluntary ban was announced by national livestock organizations on the feeding of ruminant-derived protein to ruminants. The FDA enacted an official ban on most mammalian protein in ruminant feed in 1997. This ban did not include blood, milk, gelatin or equine and porcine protein (species that do not have TSEs). In 1997, a ban on live ruminants and ruminant products from Europe was implemented because of the spread of the disease in Europe and the lack of what APHIS deemed appropriate BSE surveillance by those nations. APHIS further prohibited all animal protein imports from Europe, regardless of species, in December 2000.

Although the United States previously allowed the feeding of ruminant-derived protein to ruminants, the BSE risk has always been lower than in the United Kingdom. Cattle feeding practices are different between the two countries. In the United Kingdom, ruminant-derived protein served as an inexpensive protein source for calves and cattle. In the United States, producers have more options for protein supplementation because of a large supply of inexpensive, high-protein plant sources such as cottonseed meal or soybean meal.

Q. What is the current situation in Europe?

A. The outbreak of BSE in the United Kingdom peaked in 1992-93 at approximately 1,000 new cases per week. The rate of new cases has been declining rapidly since then, and fewer than 1,200 cases were discovered in 2002. Rapid increases in the number of cases in native cattle have occurred in France, Ireland and Spain. These increases may be due to the increased surveillance occurring in these countries, but concern for further spread is warranted. Austria, the Czech Republic, Finland, Greece, Switzerland, Belgium, the Netherlands, Liechtenstein, Denmark, Luxembourg, Germany, Slovakia, Slovenia, Israel, Italy, Japan, Poland, Portugal, Canada and the United States have all identified BSE-infected native cattle.

Q. What is "the human form of BSE"?

A. A disease that has occurred in the United Kingdom has been linked to the consumption of brain or spinal cord tissue from BSE-infected cattle. This disease is vCJD. While the signs of vCJD are similar to classical CJD, the diseases differ in distinct ways. In vCJD, the age of onset is much younger, the duration of the disease (once signs are apparent) is longer, and the microscopic appearance of the brain is different. Tests that can be performed after death show that proteins in the brain tissue of affected individuals are different between CJD and vCJD. No one knows yet how many people will be affected with vCJD; however, the annual number of cases in the United Kingdom has decreased over the past few years. It is important to note that vCJD has not been associated with the consumption of sheep proteins. If scrapie were transmissible to humans, then the consumption of sheep brain (considered a delicacy by some) would likely have caused the emergence of vCJD long ago.

Q. What is the risk of getting vCJD if I eat beef products?

A. Based on the media response to the diagnosis of BSE in the cow in Washington, you might think the chance of developing vCJD was extremely high. This is NOT the case. In the United Kingdom, which has the highest incidence of both BSE and vCJD, a total of 137 people died of vCJD from 1995-2003 out of a population of 60 million. The highest loss was in 2000, when 28 people died. Since 2000, the number of vCJD deaths has dropped, with 16 people affected in 2003. For 2003, that is 1 in every 3.75 million people – and this is in a country where they were diagnosing BSE in 1,000 animals per week at one time – not just one animal.

IF we assumed the United States had the same number of people die annually as the United Kingdom did in 2003, that would translate to approximately 80 people dying of vCJD in 2003 out of a population of 300 million. This is a totally invalid assumption because we do not have the level of infection of the United Kingdom. To add a different perspective, in 2002, more 16,000 people died because of AIDS-related illnesses. In 2000, another 16,000 people died in alcohol-related car accidents. From 1996-1998, more than 10,000 people died from fires or burns and almost 60,000 people were killed in homicides involving firearms. Approximately 700 people are killed annually in bicycle accidents. Many other foodborne illnesses claim lives of at least 10 times as many people as you could expect vCJD to take – even at the rate that has been seen in the United Kingdom.

Certainly there is a risk, but it is miniscule compared to many other problems. The food supply will always have some risks, not just the meat supply, but also vegetables (recall the 2003 hepatitis outbreak in Pennsylvania associated with green onions).

Q. What about chronic wasting disease in deer and elk?

A. CWD has been found in wild deer and elk populations in several Midwestern states and a few privately owned herds. There has been no evidence of the disease in the eastern United States. There has also been no evidence that CWD can mutate to cause vCJD in humans consuming brain or spinal cord tissue from infected animals. BSE and vCJD produce similar proteins in the brain tissue of infected animals. CWD produces proteins that are distinctly different from BSE and vCJD, which makes CWD a different disease. Unlike BSE, CWD can be spread by close contact between infected animals or exposure to contaminated areas.

Additional information

- USDA Web site for updates on the situation in the United States: *www.usda.gov*
- FDA Web site for food updates: *www.fda.gov*
- Canadian Food Inspection Agency for updates on the Canadian situation: *www.inspection.gc.ca*
- Department for Environment, Food and Rural Affairs for the United Kingdom: *www.defra.gov.uk*
- Office International Des Epizooties Web site for updated information on BSE throughout the world: *www.oie.int*
- The Centers for Disease Control for information on CJD and vCJD: *www.cdc.gov*
- AABP Web site for links to hot topics in the cattle health industry: *www.aabp.org*
- Center for Food Safety and Public Health, Iowa State University: *www.cfsph.iastate.edu*
- NCBA Web site for an updated review of the current research on TSE: *www.beef.org*
- Your state veterinarian and department of agriculture is also a source of information on BSE and other reportable diseases.

Information in this article was taken from many sources including those listed above. Population estimates used in calculations were from the U.S. Census Bureau, the Centers for Disease Control, the Office International Des Epizooties and the United Kingdom Department of Health Web sites.

Scrapie eradication program summary and update

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Diagnostic Laboratory

In 2001, the USDA announced a new program to eradicate scrapie from the United States by 2007 and seek official recognition from the international community as being "scrapie free" by 2017. In 2001, the USDA mailed information explaining the program to veterinarians and sheep producers. The majority of sheep and goat producers and their veterinarians are already participating in the program and are well aware of it. This article provides an update of the program, sources for additional information, and briefly reviews scrapie and the eradication program for veterinarians not involved in the program who may receive questions.

Scrapie

Scrapie is a fatal disease of the central nervous system of sheep and goats that has been known in Europe and the United Kingdom for at least 250 years and in the United States since 1947. Australia and New Zealand are the only countries recognized by the United States as scrapie free. As of August 2001 in the United States, scrapie has been diagnosed in more than 1,600 sheep in approximately 1,000 flocks, and in seven goats.

Scrapie is believed to spread from infected ewes to their offspring and to other sheep by contact with the placenta and placental fluids that contain large quantities of the infective agent. The disease progresses slowly, and clinical signs are not evident before two years after exposure. After clinical signs are evident, the duration of illness is typically one to six months. The first clinical symptoms consist of subtle behavioral changes. Affected animals become nervous or aggressive and often separate from the flock. Animals may appear demented, star gaze, or head press, and many sheep develop subtle to severe pruritis and chew or rub off large patches of wool, which is where the disease earned the name scrapie.

Motor abnormalities such as incoordination, high-stepping with the front limbs and "bunny hopping" with the rear limbs are common. Animals may become hypersensitive to stimuli, have fine tremors and fall down in a convulsive-like state. After clinical signs appear, death will follow.

Why eradicate scrapie?

It is estimated that scrapie costs U.S. producers $20 to $25 million annually, and this does not count the loss of export opportunities. Because of scrapie, packers and sheep producers have difficulty disposing of sheep offal and dead sheep. Other countries have expressed concerns about importing ruminant products, which has affected the market for meat and bone meal. However, the driving force behind scrapie eradication is that it is a transmissible spongiform encephalopathy (TSE). This group of diseases is caused by prions and includes bovine spongiform encephalopathy (BSE); transmissible mink encephalopathy; chronic wasting disease of deer and elk; feline spongiform encephalopathy; sporadic and variant Creutzfeldt-Jakob diseases of humans; Gerstmann-Straussler-Scheinker syndrome of humans; and fatal familial insomnia of humans. There is no evidence that scrapie is a human health hazard, but many feel that BSE originated by feeding cattle meat and bone meal that contained rendered scrapie-infected sheep.

> Scrapie costs U.S. producers $20 to $25 million annually

Evidence indicates that variant Creutzfeldt-Jakob diseases (vCJD), which has killed 137 people in Great Britain, is caused by the same agent that causes BSE, and that people acquire vCJD by consuming beef from BSE infected cattle. Because of BSE and vCJD, there is a worldwide push to eradicate TSE diseases.

USDA Scrapie Eradication Program

Features of the new program include:

1. Implementation of a new identification system for interstate movement of certain classes of sheep and goats that will allow tracing of infected animals to their herd of origin.
2. Active surveillance of animals at slaughter to detect infected herds and to estimate the national and regional incidence of scrapie in the United States. Live animal tests will be used to detect scrapie in animals that do not show clinical signs.
3. Provide cleanup strategies that will allow producers with infected flocks to stay in business, preserve breeding stock and remain economically stable. This will be accomplished through the use of genetic testing to identify genetically resistant sheep that will be retained, and genetically susceptible sheep that must be removed or their movements restricted. The program also provides indemnity payments to producers to remove infected and genetically susceptible exposed animals.

Animal Identification

Producers must determine if their sheep or goats need to be identified for interstate movement. If so, they should contact the local APHIS Veterinary Services office to obtain a premise ID number. In most cases, the identification will consist of USDA-approved ear tags from APHIS or private companies. In some instances, tattoos, ear notches or paint brands can be used. For tag orders and tag information, call 1-866-USDA-TAG and ask to speak to the tag clerk.

Animals that do not need identification and have no movement restrictions

- Sheep less than 18 months old and all goats moving into slaughter channels.
- Low-risk commercial goats defined as those:
  - raised for fiber and/or meat
  - not registered or exhibited
  - have not been in contact with sheep
  - not scrapie positive, high-risk, exposed, or from infected herds
- Wethers for exhibition
- Animals moving for grazing where there is no change in ownership

Animals that require identification

- All breeding sheep regardless of age
- All sheep more than 18 months old
- All sheep and goats for exhibition, except castrated males
- All scrapie-exposed, suspect, test-positive, and high-risk animals
- Breeding goats, except low-risk commercial goats
- Sheep less than 18 months old in slaughter channels that are pregnant or have aborted, and sexually intact animals from a scrapie infected herd

Scrapie Surveillance

USDA-APHIS has been collecting data on the incidence of scrapie by testing culled ewes and by targeted slaughter surveillance. They have validated a live animal test that uses biopsies of lymphoid tissue from the third eyelid. Before these studies, the national incidence of scrapie was estimated to be 0.07 percent. Based on 12,508 mature sheep tested at slaugh-

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ter in the new program, the national incidence has been estimated to be 0.20 percent and the incidence in the central region, which includes Kansas, to be 0.21 percent.

Genetic Testing

It has been known for several decades that susceptibility is inherited, and scrapie can be eliminated through the right breeding program. It was thought that resistant sheep were actually susceptible, but the prions replicated much more slowly in their tissues. Thus, people considered exposed resistant sheep possible sources of infection for other sheep. Newer research indicates that prions probably do not replicate in resistant sheep, and these animals do not become silent carriers. The role of genetics in susceptibility of goats to scrapie is unknown, and all goats are considered to be susceptible.

Beginning in 2003, the USDA allowed owners of infected sheep flocks in all states to test their sheep to determine which scrapie-exposed animals can be moved interstate. In most instances, only sheep that are homozygous for scrapie susceptibility need to be removed. This will allow producers to keep a much larger proportion of their flocks than in the past. Testing requires a blood sample collected and submitted with the appropriate veterinary services form to an approved laboratory by an accredited veterinarian.

The major determinant of scrapie susceptibility or resistance is the prion protein (PrP) gene, which codes for production of prion protein and experimentally is necessary for development of scrapie in mice. In the United States, scrapie resistance is determined by the amino acids coded at codons 171 and 136. Codon 171 can code for glutamine (Q), arginine (R), lysine (K) or histidine (H). The presence of R confers resistance and the presence of Q confers susceptibility, and H and K are considered equivalent to Q. Codon 136 codes for either valine (V) or alanine (A). Codon 171 plays the major role in susceptibility. Sheep with an RR genotype are resistant (there has been only one case in a sheep in Japan), do not transmit scrapie, and their movement is not restricted. Those with a QQ genotype are susceptible, transmit the disease, and must be removed or their movement restricted. Codon 136 plays a minor role and is important only in sheep that are QR at codon 171. QR sheep that are AA at codon 136 rarely develop scrapie and probably do not transmit the disease. Scrapie has never been diagnosed in the United States in AA QR sheep, and there are no restrictions on this genotype. QR sheep that are AV at codon 136 also rarely develop scrapie and probably do not transmit the disease. There have been two cases of scrapie in AV QR sheep in the United States, and there are restrictions on this genotype only if scrapie has been diagnosed in an AV QR sheep in the flock.

Additional Information

The two best sources of information concerning scrapie and the national eradication program are Web sites maintained by USDA-APHIS and National Institute of Animal Agriculture. They have more complete information for veterinarians and producers on all of the subjects covered in this article and much more, including the addresses and phone numbers for the USDA-APHIS office in every state. The addresses for these Web sites are: *www.aphis.usda.gov/vs/nahps/scrapie/* and *www.animalagriculture.org/scrapie/Scrapie.htm*

In Kansas, the state scrapie epidemiologist is Dr. Donald Evans, USDA-APHIS VS, 1947 NW Topeka Blvd. Suite F, Topeka, KS 66608 Phone: 785-235-2365; FAX: 785-235-1464; e-mail: *Donald.E.Evans@aphis.usda.gov*

Approved laboratories for scrapie susceptibility and resistance testing (each of the labs have Web sites where more information is available):

GenMark
1825 Infinity Drive
DeForest, WI 53532
1-877-776-3446

GeneCheck Inc
1629 Blue Spruce Drive, Suite 106
Ft. Collins, CO 80524
1-800-822-6740

GeneSeek, Inc
4711 Innovation Drive
Lincoln, NE 68521
402-435-0665

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- Markedly decreased egg production
- Soft or misshapen eggs
- Swelling of the head, eyelids, comb, waddles and/or hocks
- Purplish discoloration of the comb, waddles or legs
- Nasal discharges
- Coughing or sneezing
- Incoordination
- Diarrhea

Lesions on postmortem include subcutaneous edema of the head and neck, and widespread hemorrhages including hemorrhagic tracheitis. Avian influenza can easily be confused with exotic Newcastle disease.

Avian influenza is usually spread from bird to bird via droppings or body secretions and can be spread from flock to flock by foot or vehicular traffic and any variety of other contaminated fomites.

Producers should be urged to practice good biosecurity and be alert for unusually high death losses in their birds.

If avian influenza is suspected, contact state or federal veterinarians immediately.

- Topeka USDA office 785-235-2365
- Kansas State Animal Health Department 785-296-2326

Diagnostic samples, tests for calf diarrhea

John Ragsdale
Diagnostic Laboratory

Calf diarrhea can be caused by a variety of pathogens including viral, bacterial and parasitic agents. Viral agents include bovine coronavirus (BCV), bovine rotavirus and bovine viral diarrhea virus (BVD). Bacterial agents include, but are not limited to, *Escherichia coli*, *Salmonella* sp. and *Clostridium perfringens.* Parasites include, but are not limited to, *Cryptosporidium parvum, Eimeria* sp. (coccidia), and occasionally *Giardia* sp.

This article includes what information to provide, postmortem samples to collect and submit for testing, and the diagnostic tests performed on each sample.

History

A complete history indicating the age of the affected animal(s) and any gross lesions is important for the diagnostician. Neonatal calf diarrhea caused by *Cryptosporidium parvum* usually only affects calves from one to three weeks of age. Conversely, calf diarrhea caused by enterotoxigenic *Escherichia coli* affects calves less than one week of age and usually less than five days of age. Providing the age of the affected calves helps the diagnostician decide which tests to perform, which provides an accurate diagnosis and reduces the cost to the producer.

Histopathology

Intestinal samples should be approximately 1 inch long and flushed with formalin or partially opened to allow adequate fixation of the villi. Samples should be taken of the duodenum, mid-jejunum, distal jejunum, ileum, colon, abomasum, spleen, mesenteric lymph node, liver and other tissues as indicated. *The distal jejunum, ileum and colon are the most important intestinal samples.*

Bacteriology

Sections of the middle to distal jejunum, ileum, colon and mesenteric lymph node (for *Salmonella*) should be submitted for aerobic culture. The intestinal sections should be 5 to 8 centimeters long. Other tissues can be submitted depending on the history or lesions. Small intestine can be submitted for anaerobic culture in cases of sudden death or postmortem findings that suggest clostridial enteritis.

Fluorescent Antibody

A 5-centimeter long section of jejunum and ileum should be submitted to test for BVD, BCV and rotavirus. A similar length of colon should also be submitted to test for BCV.

ELISA

One milliliter of colonic contents should be submitted for fecal ELISA testing for BCV and rotavirus. The FA and ELISA tests are both used to compensate for inadequacies in each test because of differences in the stage of disease and the degree of postmortem autolysis. The ELISA test is a more sensitive test.

Parasitology

Fecal flotation can be performed in office or 5 milliliters of colonic contents can be submitted for *Cryptosporidium,* coccidia and *Giardia*.

Virus Isolation

If desired, pooled samples of ileum, mesenteric lymph node and spleen can be submitted for virus isolation for BVD.

Bacterial cultures, ELISA testing for BCV and rotavirus, and examination for parasitic ova and oocysts can be performed on a fecal sample from a live calf.

Continuing Education

March 6
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Opthalmology Conference and Wet Lab on Preserving Animal Visual Health

March 7
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May 1
Bovine Conference on Current Topics in Pharmacology

June 6-9
66th Annual Conference for Veterinarians and Centennial Celebration Kick-Off

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*

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