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Kansas adopts voluntary program to control bovine Johne's disease

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On the recommendation of a group of producers and veterinarians across the state, Kansas has joined national efforts to control Johne's disease.

Mary Ann Kniebel from White City, representing the beef industry, and Warren Winter from Hillsboro, representing the dairy industry, co-chair the state committe. The program will be administered by the Veterinary Services Division of USDA/APHIS, Kansas State University and the Kansas Animal Health Department.

Key elements of the voluntary program include:

Education – informing producers about the cost of Johne's disease and providing information about management strategies to prevent, control, and eliminate it;

Management – working with producers to establish good on-farm management strategies; and

Herd testing and classification – helping to separate test-positive herds from test-negative herds.

K-State's College of Veterinary Medicine and K-State Research and Extension will lead educational efforts. Private-practice veterinarians will conduct the on-farm portion. These veterinarians will be trained and certified to perform risk assessments, recommend critical management practices for individual herds, and conduct testing to provide herd status in the program.

At the producer's request, certified veterinarians will provide all or some of these services, with the initial visit and a qualifying number of test samples paid for with funds administered by the Kansas Animal Health Department. Producers can participate at various levels. They can obtain herd status through testing and be recognized for putting approved management practices and plans into place and conducting herd tests to increase confidence that their herd is not infected.

Web-based training available

K-State offers an opportunity for accredited veterinarians to receive certification for the Johne's program through a Web-based training program. The first 150 veterinarians to certify through the online program can do so at no charge (a $200 value). As a bonus, they will receive a CD, titled "Johne's and Beyond," from the U.S. Animal Health Association. This is a great control, management and herd health education tool for livestock health professionals, a $50 value. For details on the training, e-mail David Gnad at *dgnad@vet.k-state.edu*.

Also in this issue

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

Lab aquires new Johne's culture system

Jerome Nietfeld
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Mycobacterium avium subsp. *paratuberculosis* (*M. paratuberculosis*), the cause of Johne's disease, is very fastidious and has a long generation time that requires many weeks to produce visible colonies on solid agar. The current "gold standard" culture methods using solid agar often require four months for growth and identification of *M. paratuberculosis*, which is a problem for veterinarians and producers implementing Johne's control programs.

The K-State Veterinary Diagnostic Laboratory recently acquired a new liquid medium system (ESP Culture System II, Trek Diagnostic Systems) that requires less time for identification. With the ESP Culture System, fecal samples are inoculated into small bottles that contain liquid medium and a sponge (*M. paratuberculosis* prefer to grow on a solid surface).

Antibiotics, egg yolk and supplement are then added, and the bottles are placed in a special incubator. Each bottle is sealed, and a needle sensor extends through the cap into the headspace inside the sample bottles. Changes in gas consumption or production caused by bacterial growth trigger the sensor that turns on a light to indicate a positive sample. Positive samples are then acid fast-stained and tested by polymerase chain reaction to confirm the presence of *M. paratuberculosis*. A recent study indicated that the ESP System is more sensitive than traditional culture, besides being much faster. The maximum incubation period with the new ESP System is six weeks. The old solid media methods samples are incubated for up to 16

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Isolating *Mycoplasma avium* subspecies *paratuberculosis* from commercially pasteurized milk

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On Aug. 10, 2004, the Johne's Information Center Web site (*http://johnes.org/*) at the University of Wisconsin School of Veterinary Medicine posted a short summary of a study headed by Jay Ellingson, Marshfield Clinic Laboratories in Marshfield, Wis. The Marshfield researchers purchased milk from stores in California, Wisconsin and Minnesota and tested it for *Mycobacterium avium* subspecies *paratuberculosis* (*M. paratuberculosis*), the cause of Johne's disease of ruminants. The methods used are not described, but they are reported to have been more sensitive than methods commonly used for identification of the organism in clinical samples. Of 702 milk samples, 2.8 percent were positive for viable (capable of multiplying) *M. paratuberculosis*. The incidence of *M. paratuberculosis* was similar in samples from all three states, but the incidence varied between seasons.

Background

Crohn's disease is a chronic inflammatory bowel disease of humans that most commonly affects the ileum and, to a lesser extent, the colon. Microscopically, there is granulomatous inflammation characterized by macrophages, multinucleated giant cells, neutrophils and eosinophils associated with a small central area of necrosis.

The cause of Crohn's disease is unknown. Many causes have been suggested, including autoimmune, genetic and infectious. The similarities between Crohn's disease and Johne's disease of ruminants were noted as early as 1913, but no association between Crohn's disease and *M. paratuberculosis* could be identified, and *M. paratuberculosis* was largely forgotten as a possible cause. In recent years, as methods of identifying *Mycobacterium* species have improved, numerous researchers have begun to find a possible association between *M. paratuberculosis* and Crohn's disease. However, other investigators have not been able to demonstrate a relationship between the two. This situation still exists. I reviewed several studies published in 2004 and found that some studies find the organism present in a statistically significantly higher proportion of individuals with Crohn's disease than in individuals suffering from other enteric diseases or in healthy individuals. However, other studies found no such association. Early studies to evaluate the effectiveness of antibacterial agents against *Mycobacterium* species for treatment of Crohn's disease found that the drugs were ineffective. Although these compounds were ineffective against *M. paratuberculosis*, compounds that are effective against the organism do show promise in the treatment of Crohn's disease. Because of the conflicting results, a firm conclusion cannot be made as to whether or not Crohn's disease is caused by Johne's disease. (The Johne's Information Center Web site[1] contains an excellent review of the zoonotic potential of *M. paratuberculosis*.)

Milk as a possible source of *M. paratuberculosis* infection

Because *M. paratuberculosis* is often shed in milk from cows with Johne's disease, milk has been suggested as a possible source of infection for people who have no contact with cattle or other ruminants. Many assume that pasteurization destroys the organism and that milk is not a likely source of infection. However, recent research suggests that this might not be the case.

A 2002 Canadian study[2] found that *M. paratuberculosis* could be isolated from two of 11 milk samples spiked with 10^5 bacteria/ml and pasteurized by the high temperature short time (HTST) method. The investigators also tested 710 milk samples from retail stores in Ontario, Canada. Fifteen percent of the samples (n = 110) were positive by polymerase chain reaction (PCR), which detects viable and nonviable organisms for *M. paratuberculosis*, but none of 44 PCR positive and 200 PCR negative samples were culture positive. The authors concluded that the failure to isolate *M. paratuberculosis* from retail milk could have been due to the lack of viable organisms in the milk or that their culture methods were not sensitive enough to detect very low numbers of organisms in milk.

A similar study from the United Kingdom[3] found that 1.8 percent of 567 commercially pasteurized milk samples contained viable *M. paratuberculosis* organisms. The positive samples came from eight of the 241 plants from which the samples originated. Subsequent investigation indicated that the HTST method of pasteurization had been correctly carried out at all plants from which the culture positive samples came. Fingerprinting of the *M. paratuberculosis* isolates from commercial milk samples showed them to be distinct from laboratory strains. A review of the heat resistance of *M. paratuberculosis*[4] concluded that experiments on the subject have yielded differing results. The authors also stated that it is important to accurately determine the heat resistance of the organism and implement standards that will ensure that pasteurized milk does not contain viable *M. paratuberculosis*.

Regardless of whether or not *M. paratuberculosis* is involved in causation of Crohn's disease, it is becoming apparent that current pasteurization methods are probably not completely effective in destroying the organism. Based on published reports it is probable that the heat resistance of different strains of *M. paratuberculosis* varies. In order to maintain consumer confidence in milk and ensure public safety, it is important to find ways be certain that milk does not contain *M. paratuberculosis*. This could be by elimination of Johne's disease from cattle, pasteurization or both.

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Nationwide kennel cough outbreak in racing greyhounds

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A team of national researchers lead by Cynda Crawford, D.V.M., Ph.D., a veterinary immunologist at the University of Florida at Gainesville, believes an escape variant of equine influenza virus caused a January 2004 respiratory disease outbreak in racing greyhounds in Florida. The outbreak may have been the cause of a nationwide racing greyhound kennel cough epidemic last summer.

Equine influenza virus causes influenza in horses and is a group A influenzavirus, which is the group that causes influenza in people, birds and pigs. The disease is present throughout Europe, North America and parts of Asia. The disease produces severe symptoms, and horses typically develop a fever and a dry, hacking cough. Horses become ill and are reluctant to eat and drink for several days, but usually recover in two to three weeks. Several of the equine influenza virus isolates from the deceased greyhounds were sent to the Centers for Disease Control (CDC) for further evaluation. On the basis of genetic sequencing, the CDC's Ruben Donis, Ph.D., chief of molecular genetics, concluded that the virus found in the canine respiratory samples resembled a strain of equine influenza virus isolated from horses in Wisconsin last year. Using pre- and post-exposure serum samples, Crawford also found significant rises in antibody titers specific for the influenza-like virus in the infected greyhounds.

The January 2004 outbreak in several Florida racetracks killed eight greyhounds and resulted in a statewide quarantine that limited the spread within the track and the state. Initially 24 dogs were affected with symptoms that included cough, fever and some sneezing. Of those 24 dogs, eight died and the other 16 fully recovered. Three months after the outbreak Crawford released the preliminary findings of the investigative team involving virologists from Cornell University's College of Veterinary Medicine in Ithaca, N.Y., and the national Centers for Disease Control and Prevention. Doctors Brad Fenwick and William Fortney at Kansas State University also were involved in the diagnostic testing. The preliminary data also were presented at the National Greyhound Association's Spring Meet in Abilene, Kan., in April 2004.

This infectious tracheobronchitis agent appeared to be highly contagious. The vast majority of affected dogs were febrile (104°), had a cough and exhibited mild sneezing. A minority of dogs became depressed, developed acute fevers higher than 106°F and had blood-tinged nasal or oral discharge. Two dogs that were fine at bedtime were found dead, lying in a pool of blood, the next morning. Necropsies reveal a severe hemorrhagic pneumonia. These symptoms are similar to what has been reported in the canine Streptococci Toxic Shock Syndrome (STSS) seen in dogs of all breeds including greyhounds. Adding to the confusion, several of the affected dogs (both sick and deceased) cultured positive for *Streptococcus canis* or *Streptococcus zoo* and *Bordetella bronchiseptica*. Isolating *Bordetella bronchiseptica* from dogs that have recently been immunized with an attenuated intranasal vaccine is not unusual. However by using DNA fingerprinting of the Bordetella isolates, Fenwick's laboratory determined those isolates were a virulent street strain of Bordetella and not an incidental vaccine strain. *Mycoplasma* sp. was also found in several affected dogs leading to a conclusion that the outbreak was in fact multifactorial.

> Aggressive fluid therapy was the single most important therapy associated with recovery of critically ill patients.

While antibiotics with spectrums against *Streptococcus*, *Mycoplasma* and *Bordetella* may have been helpful in minimizing progression of the disease, aggressive fluid therapy was the single most important therapy associated with recovery of critically ill patients. Early symptom recognition and "scratching" (not racing) any possibly sick dog was also thought to be beneficial in decreasing the mortality rates. Thanks in part to a statewide quarantine, the outbreak was confined to several Florida tracks.

The June/July 2004 nationwide kennel cough outbreaks cost the greyhound racing industry millions of dollars in lost revenues because of track closures and quarantines on dog movement between tracks. This outbreak also appears to have started in Florida but rapidly spread to Texas, Kansas and East Coast tracks via transportation of infected/asymptomatic dogs. Unfortunately this particular kennel cough outbreak had much higher morbidity. Despite local quarantine efforts, approximately 70 to 80 percent of the greyhounds at each track were eventually affected. However, mortality was significantly less, perhaps because of early recognition of the disease, vigilant antibacterial and fluid therapy, or possibly a less virulent agent(s).

Although this kennel cough epidemic resembles the one in Florida last January, where the equine influenza virus was first isolated, researchers are uncertain about the etiological agent(s). Ongoing investigations of both outbreaks should shed more light on the exact cause(s) and preventative strategies.

To date, no dog has been experimentally challenged with the equine influenza-like strain to see what symptoms, if any, it may cause in dogs. There is no concrete evidence that dogs in the general population have been affected with this particular viral agent.

For more information on *Bordetella* DNA fingerprinting contact Selected Diagnostic Services, Kansas State University College of Veterinary Medicine, Manhattan, KS 66506, or 785-532-4421.

Plants may be hazardous to livestock this fall

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Material presented here was modified with permission from *A Guide to Plant Poisoning of Animals in North America* by A. P. Knight and R. G. Walter (2001), Teton NewMedia, Jackson, Wyoming. Check availability of the printed book at *www.veterinarywire.com*. The complete publication is also online with free registration at *www.ivis.org/special_books/Knight/toc.asp.*

Kochia, summer cypress, Mexican fireweed
Kochia scoparia

In many areas of southwestern North America, kochia weed is used as a forage for livestock and is often cut and baled as a winter feed. Kochia is a rapidly growing herbaceous annual with branching stem and simple, entire, hairy leaves that become particularly red in the fall. The size of the plant varies, attaining heights of 6 feet (2 meters) where the moisture and organic content of the soil are high. The seeds are small, brown and slightly ribbed. Kochia weed is often erroneously called tumbleweed because it breaks off at ground level in the winter and gets blown about.

Kochia

Kochia has a mixed reputation as a nutritious forage and a potential poison to cattle and sheep. A specific toxin has not been identified to account for syndromes encountered in livestock whose diet consists predominantly of kochia. Nitrates, oxalates, sulfates saponins and alkaloids have been identified in the plant, depending on the stage of growth, growing conditions and geographic region.

Kochia is used as forage and is made into hay for livestock in many arid regions of North America. If grazed or cut before it reaches maturity, kochia does not appear to be toxic, but it is hazardous to ruminants if it is high in nitrates. Its nutritive value is similar to that of alfalfa. However, livestock grazing kochia when it is mature or stressed by drought are prone to poisoning, at least in some regions. Poisoning is more likely if the animal's diet consists predominantly of kochia. The variable toxicity of kochia appears related to the plant's stage of maturity, and perhaps a combination of the effects of its various toxic components. The general toxic effect appears to be that of degenerative liver disease with secondary photosensitization and liver failure.

Cattle, sheep and occasionally horses appear to be the most commonly affected when mature kochia is the predominant constituent of their diet. The most common clinical signs observed in cattle include poor weight gains, depression, weakness, excessive tearing, incoordination and photosensitization. Other clinical signs associated with kochia poisoning include appetite loss, diarrhea, icterus and mouth ulcerations. Affected animals frequently have elevated serum liver enzymes and bilirubin levels. Some animals will be blind and walk in circles or follow fences endlessly. These neurologic signs may be due to the high levels of sulfate that – once reduced in the rumen to hydrogen sulfide – result in degenerative changes in the brain, leading to depression and blindness. Long-term consumption of mature kochia results in death. However, cattle and sheep will recover from kochia poisoning provided they are given a balanced ration where kochia weed is not the only food available.

Mature kochia

Yew, Taxus species

Several species of yew grow naturally or as ornamentals in North America, generally preferring more humid, moist environments. Western yew (*T. brevifoliat*) and American yew (*T. canadensis*) are two indigenous species. English yew (*T. baccata*) and Japanese yew (*T. cuspidata*) are commonly cultivated species in North America.

For many years, yews have been known to be toxic to humans and animals. Yews contain a group of 10 or more toxic alkaloids, the most toxic of which are taxine A and B, collectively referred to as taxine. Taxine inhibits normal sodium and calcium exchange across the myocardial cells, depressing cardiac depolarization and causing arrhythmias. Yews also contain nitriles, ephedrine and irritant oils. All parts of the plant, green or dried, except the fleshy part of the aril surrounding the seed are toxic. Livestock are frequently poisoned when fed prunings from cultivated yews. The highest concentration of the alkaloids is generally found in the leaves in winter. All domestic animals, including birds, are susceptible to the cardiotoxic effects of the alkaloid. Adult cattle and horses have been fatally poisoned with as little as 8 to 16 oz. of yew leaves or 0.1 to 0.5 percent of their body weight. Drying of the leaves does not appreciably decrease toxicity. Animals generally will not eat yew if they are fed a balanced diet. Interestingly, deer appear to be able to eat yew without problem.

Japanese yew leaves and fruit

Sudden onset of muscle trembling, incoordination, nervousness, difficulty breathing, slowed heart rate, vomiting, diarrhea, convulsions and death are characteristic of yew poisoning. Sudden death may be the only sign in many cases. Deaths may occur several days after the yew was eaten.

No postmortem lesions are diagnostic of yew poisoning. Diagnosis must be based on eliminating other causes of sudden death, evidence of access to yew, and the presence of yew leaves in the animal's rumen and stomach contents. Finely chewed leaves may have to be examined microscopically to positively identify them as yew. Identification of taxine from chewed plant material and rumen contents using mass spectrometry affords a more precise means of confirming yew poisoning.

There is no specific treatment or antidote for acute yew poisoning. If an animal is observed eating yew, immediate veterinary attention is indicated. Activated charcoal (2 g/kg body weight) and magnesium sulfate (2 g/kg body weight) as a cathartic should be given via stomach tube to decontaminate the rumen. A rumenotomy to remove the yew leaves from the rumen of cattle in early confirmed cases of yew consumption may be lifesaving. Atropine sulfate is reportedly effective in counteracting the slow heartbeat and heart failure, but should be used with caution. When possible, intravenous fluid therapy and other supportive measures should be used to support the cardiovascular system.

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Jimson Weed, Thornapple, Peru Apple, Stinkweed *Datura stramonium (D. tatula)*

Introduced from the tropics, jimson weed has become a naturalized weed throughout North America, established in disturbed soils, disused corrals, roadsides, and edges of cereal grain fields. Seeds are probably dispersed in cereal grains at harvest time. The name jimson weed or "Jamestown weed" was given to the plant in 1676, when a large number of soldiers were poisoned after eating the plant in Jamestown, Va.

Jimson weed seed capsule and seeds

A variety of steroidal (tropane) glycoalkaloids are found in the green parts of the plant and the unripe fruits. The more common alkaloids including solanine, hyoscine (scopolamine) and hyoscyamine (atropine) act similarly on the parasympathetic autonomic nervous system by competitively antagonizing the effects of acetylcholine at its receptor sites. This results in the accumulation of the neurotransmitter acetylcholine, and consequently inhibition of the parasympathetic nervous system, causing decreased salivation and intestinal motility, dilated pupils and tachycardia. The alkaloids also have a direct irritant effect on the digestive system, causing colic, constipation or hemorrhagic diarrhea.

Animals will eat members of this family when other forages are scarce or when crop residue products are fed to them. Signs of poisoning can be expected when 0.1 to 0.3 percent of an animal's body weight in green plant is eaten. Grain contaminated with seeds of jimson weed (*Datura stramonium*) can be a significant source of poisoning. Compared to other animals, cattle may be more susceptible to the toxic effects of solanine alkaloids. Initially there may be central nervous system excitement, but depression follows with increased heart and respiratory rate, muscle weakness, dilated pupils, colic and watery diarrhea. Rupture of the stomach and paralysis of the digestive system in horses can be a sequel to the effects of the tropane alkaloids. If large amounts of the tropane alkaloids are consumed over a short period of time, cardiac arrest may lead to death before digestive signs develop.

Animals showing severe anticholinergic signs consisting of muscle tremors, hyperesthesia, dilated pupils, intestinal stasis and depressed respiratory rate may be treated with physostigmine. Many animals, however, recover if treated symptomatically. Oral administration of activated charcoal as an adsorbent may be effective if given soon after the plants have been eaten.

Elderberry (*Sambucus Canadensis)*

Elderberry plants generally prefer open areas in the rich, moist soils along ditches and streams throughout North America. Elderberries contain a cyanogenic glycoside, sambunigrin, and an irritant alkaloid that acts as a cathartic. All parts of the elderberry plant are potentially toxic to animals, although animal poisoning is rarely encountered. Cultivated varieties of elderberry should be considered potentially toxic and probably should not be planted in or around animal enclosures. The purple to black ripe berries are edible if well cooked and are often used for making jams and wine.

The hydrogen cyanide (HCN) is highly lethal by rapidly inactivating cellular respiration. The characteristic cherry-red venous blood results from the failure of the oxygen-saturated hemoglobin to release its oxygen at the tissues; sudden death is the usual presenting effect. Successful treatment of acute cyanide poisoning depends on the rapid inactivation and removal of cyanide by injecting sodium nitrite and sodium thiosulfate intravenously.

Elderberry

Castor bean (*Ricinus communis*)

A lectin, ricin, is the principal toxin. All parts of the plant are toxic with the seeds containing the highest concentration of ricin, a heat-labile glycoprotein (toxalbumin). Other compounds in the seeds are responsible for agglutination and hemolysis. Ricinoleic acid present in castor oil is primarily responsible for its purgative action.

The principal toxin in castor beans is the glycoprotein ricin. Castor oil derived from the beans contains ricinoleic acid, an irritant that can cause severe intestinal irritation with profound purgation. Various other alkaloids and proteins are present in the seeds but generally are not toxic and have been used for a multitude of medicinal purposes. Some of the proteins act as agglutinins and are capable of causing red blood cell agglutination in vitro. Agglutination is not a feature of orally ingested castor beans, and ricin, when injected systemically, does not cause hemolysis, indicating that compounds other than ricin are involved.

Castor beans

Castor bean plant with flowers and seed capsules

The lectins, comprising two peptide chains joined by sulfide bonds, are capable of binding to certain cell receptor sites and inhibit cellular protein synthesis in the ribosomes. Because this process takes time, clinical signs of poisoning may not occur for several days after ingestion of the lectins. Lectin proteins also induce antibody formation when injected into animals, a factor that has been explored in an attempt to develop antitoxins to ricin. The allergic reaction encountered in humans exposed to dust from castor beans is not a reported problem in animals. Ricin is also capable of inhibiting the growth of tumor cells.

Ricin is one of the most poisonous plant compounds known, especially when injected. It has been used in assassination efforts and has the potential to be used in bioterrorism. As little as 1 mg ricin is lethal to humans. Reports of castor bean poisoning have varied considerably as to the number of beans that will induce poisoning and death. This may be accounted for by variations in the quantity of ricin present in some castor beans and the degree to which

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the beans had been chewed before they were swallowed. Human poisoning has occurred when 2.5 to 20 castor beans have been eaten, and animals may be fatally poisoned with four to 11 beans. A dose of 2 g/kg body weight of ground castor beans is reported as lethal in cattle. Horses are fatally poisoned by as few as 60 seeds; ruminants appear to be less susceptible because the toxin is broken down in the rumen. Domestic ducks are poisoned by three to four seeds. Studies in dogs in which pure ricin was injected experimentally demonstrated the minimum lethal dose was 2.7 mg/kg body weight.

Plant parts of castor beans other than the seeds are rarely reported as a cause for poisoning in animals, but the leaves are toxic. Cattle fed castor bean leaves developed signs distinct from those associated with ricin: neuromuscular impairment characterized by muscle weakness, tremors, salivation and excessive eructation. Recovery may occur after a short period or the animal may die, presumably as a result of the quantity of leaves consumed.

The signs of castor bean poisoning are primarily associated with severe gastrointestinal irritation, and begin several days after the consumption of a toxic dose. Affected animals stop eating and develop a severe hemorrhagic diarrhea. Lactation stops abruptly. Abdominal pain is often severe. Rapid loss of water and electrolytes through the diarrhea results in dehydration and hypovolemic shock. Increases in serum liver enzymes, creatinine, urea nitrogen, and sodium and potassium levels, and a decrease in serum total protein reflect the loss of fluid and electrolytes and the effects of the toxins on organ function. Untreated animals die from hypovolemic shock. Postmortem findings include severe pulmonary congestion, ulceration of the stomach and intestines, and fatty degeneration and necrosis of the liver and kidneys.

The diagnosis of castor bean poisoning can be difficult to confirm unless animals are observed eating the seeds or the intact seeds or parts are identifiable in the digestive tract at postmortem examination. Because it resembles sunflower and cottonseed cake, castor bean meal can be recognized in animal feeds by microscopic examination for the characteristic seed hull fragments.

Treatment involves quick removal of the seeds from the stomach and digestive tract. Vomiting can be induced in dogs and cats, or endoscopy can be used to remove the seeds from the stomach. Orally administered activated charcoal is beneficial. Purgatives, such as magnesium hydroxide, may help remove the toxin from the digestive system. Animals with diarrhea and resulting dehydration and hypovolemic shock should be given intravenous fluids and electrolytes. Such treatment has been the main reason that human fatalities have been almost eliminated. The use of immune serum to treat ricin poisoning is rarely necessary.

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weeks. Most culture-positive samples can be identified in less than four weeks. Recently, the bacteriology lab has confirmed clinical samples as positive in as little as 12 to 14 days. This is a big step forward for the lab in Johne's culturing and should be helpful to veterinarians and producers. The cost for the new procedure is the same as for the old method. Tanya Purvis is in charge of culturing at the bacteriology lab. Contact her with questions about the new procedure at 785-532-5650.

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