

# VETERINARY

FOR THE PRACTICING VETERINARIAN

# Quarterly

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## Anderson named director of diagnostic laboratory

Dr. Gary Anderson has been named director of K-State's Veterinary Diagnostic Laboratory. Anderson earned a bachelor's degree in biology from South Dakota State University and a D.V.M. and master's degree in veterinary pathology from Kansas State. He continued his studies at the University of California – Davis, under Dr. Bennie Osborn, where he received a Ph.D. in comparative pathology with a minor in immunopathology/immunovirology in 1983.



Dr. Gary Anderson

Anderson worked in the Veterinary Science Department at the University of Nebraska – Lincoln from 1983 to 1988, first as an assistant professor and later as an associate professor. In 1988 he joined Sanofi Animal Health where he was director of Development and Regulatory Affairs – Biologics, and was promoted to vice president of biological and pharmaceutical research. In 1995 he formed ImmTech Biologics, where he was president, general manager and owner. In 2002, ImmTech was sold to Novartis Animal Health, and Anderson continued to serve as vice president/director of the Immtech Division until he accepted the director position at K-State.

Throughout the years Anderson has maintained close ties with many in the Diagnostic Medicine/Pathobiology Department and has been instrumental in the development of several innovative vaccines. His career has involved diagnostic medicine, team building and laboratory management. It is our privilege to have him return to K-State.

Please feel free to contact him, stop by the lab when you are in Manhattan, or introduce yourself at a veterinary meeting. We know you will enjoy getting to know Dr. Anderson as he assumes leadership of the diagnostic lab. His phone number is 785-532-4454 and e-mail address is [ganders@vet.k-state.edu](mailto:ganders@vet.k-state.edu).

## Update on the USDA's BSE testing program

*Jerome Nietfeld, D.V.M., Ph.D.*  
*Veterinary Diagnostic Laboratory*

In December 2003, the Animal and Plant Health Inspection Service (APHIS) of the United States Department of Agriculture (USDA) identified a 6.5 year-old dairy cow from Washington State that was infected with bovine spongiform encephalopathy (BSE). The cow was subsequently found to have been born in Canada and imported into the United States in 2001.

Even though APHIS was already testing at a level many times above that recommended by the World Organization for Animal Health (OIE), the agency decided to expand its BSE testing program. The enhanced program is intended to be a one-time effort that will provide a snapshot of the U.S. cattle population

to determine if BSE is present in the United States and, if so, at what level.

Nowhere in the world has BSE been diagnosed in cattle less than 20 months of age, and approximately 88 percent of the cattle slaughtered in the United States are less than 20 months old. Consequently, APHIS is concentrating its efforts on testing cattle that are most likely to be affected, rather than the general slaughter population. The goal of the program is to test as many high-risk cattle as possible in a 12- to 18-month period. Samples from high-risk cattle are being obtained from state and federally inspected slaughter plants, custom-exempt slaughter plants, veterinary diagnostic laboratories, veterinary teaching hospitals, veterinary clinics, farms, animal feed slaughter facilities (pet food plants),

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

## K-State researchers awarded \$805,000 to develop animal ID system

Mary Lou Peter

*K-State Research and Extension*

Kansas State University researchers will work with the Kansas Animal Health Department to develop a national animal identification system, thanks to an \$805,000 federal grant.

The grant, awarded by the U.S. Department of Agriculture, is part of \$11.64 million awarded in a competitive program to states and tribes for a national identification plan's premise identification network, according to K-State research veterinarian Mark Spire.

Spire, along with K-State Research and Extension beef cattle specialist Dale Blasi, has been working with KAHD on a project to evaluate the use of field-based, mobile technologies. Using global positioning satellites (GPS) that mark animal loading and unloading sites, the system will link state health

authorities' databases in the central United States with electronic individual cattle identification, premise identification and the location of the animals.

"Once completed, the system will use electronic RFID (radio frequency identification) technology placed on an animal to track it from the farm of origin through the marketing processes and all the way to slaughter," Spire said.

"This project fits well with established research within the Colleges of Veterinary Medicine and Agriculture (Department of Animal Sciences and Industry), linking animal surveillance to central databases using mobile wireless information technologies,"

Blasi said. "Moreover, this project will address cattle movement – one of the critical issues faced by the national animal identification system. It fits well with the USDA's desires to have 48-hour trace-back capabilities in the event of a national animal disease emergency or as part of ongoing disease surveillance programs."

Several companies are working cooperatively with K-State and the KAHD on the project.

More information about the national animal identification system (NAIS) is available at [www.aphis.usda.gov](http://www.aphis.usda.gov).

**This project will address cattle movement – one of the critical issues faced by the national animal identification system.**

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livestock auctions and public health laboratories. The USDA considers the following to be high risk:

- nonambulatory cattle
- cattle exhibiting signs of a central nervous system disorder
- cattle exhibiting other signs that can be associated with BSE, such as emaciation or injury
- dead cattle

In addition to high-risk cattle, the USDA is randomly collecting and testing samples from apparently normal aged cattle at slaughter plants. The carcasses from these animals are held and not allowed to enter the food chain until the samples test negative for BSE. The goal of APHIS is to test at least 268,500 cattle, which will detect one case of BSE in 10 million adult cattle at a 99 percent confidence level. If there are only five BSE positive animals in the targeted population in the entire US, this program is capable of detecting it.

Before May 1, 2004 the USDA had tested more than 72,500 samples from high-risk animals. The Washington state dairy cow identified in December 2003 was the only positive animal. From June 1, 2004 through Mar. 31, 2005, APHIS tested samples from 294,593 cattle and all have been negative.

This information is from the United States Department of Agriculture's Animal and Plant Health Inspection Service website, which is available at: [www.aphis.usda.gov/lpa/issues/bse\\_testing/index.html](http://www.aphis.usda.gov/lpa/issues/bse_testing/index.html)

## Serology for *Leptospira hardjo-bovis* in cattle

*This is an e-mail written by Carole Bolin, D.V.M., Ph.D., from Michigan State University, Diagnostic Center for Population and Animal Health. It was posted on the LIST-SERV maintained by the American Association of Veterinary Laboratory Diagnosticians.*

There has been considerable confusion regarding testing cattle sera for antibodies against Hardjo. Producers and veterinarians are being educated that the type of Hardjo that we have in the United States is Hardjo-bovis (which is true) and that the labs doing leptoserology use Hardjo-prajitno as antigen (which is also true). However, veterinarians and others often conclude that their lab needs to switch to using Hardjo-bovis in their serology panel for

lepto to pick up antibodies against Hardjo-bovis. This is not true. Hardjo-bovis and Hardjo-prajitno are essentially serologically identical and Hardjo-prajitno makes a great antigen to pick up antibodies against either hardjo type. This statement is backed up by mountains of data from my lab (now and when I worked at USDA) and testing done by NVSL this past year. There is no need to switch antigens for the MAT (microscopic agglutination test).

Also, cattle given the new Hardjo vaccine, Spirovac (either alone or in combinations) may have very high titers to Hardjo. I have seen field titers in excess of 6,400 on many occasions. So, when interpreting leptotiters in cows, it is important to know what vaccine was used.

# Kinyoun acid-fast procedure makes diagnosis of Cryptosporidiosis quick, easy, reliable

Jerome C. Nietfeld, D.V.M., Ph.D.  
Veterinary Diagnostic Laboratory

Diarrhea is the most common cause of illness and mortality in newborn calves. From 5 to 30 days old, *Cryptosporidium parvum* by itself or in association with other infectious agents, is an important cause of diarrhea. Because *C. parvum* is zoonotic and can cause diarrhea in all ages of people who might ingest the organism, it is important to accurately and rapidly identify calves shedding the organism in their feces so extra precautions can be taken to prevent human infection.

One method for diagnosing cryptosporidiosis is by concentration and floatation of oocysts in fresh feces with Sheather's sugar solution followed by bright field light microscopic examination.<sup>1</sup> Because the oocysts are small (4 to 5  $\mu\text{m}$ ) it is easy to focus in the wrong plane, making it is easy for inexperienced examiners to miss the organisms when using the floatation procedure. In addition, it is easy to mistake cryptosporidia for yeasts, which are approximately the same size and shape.

Another method used to distinguish between cryptosporidia and yeast is based on the fact that cryptosporidia are acid fast while yeasts are not.<sup>1</sup> Thin fecal smears made on glass slides can be stained in less than 10 minutes utilizing the Kinyoun acid-fast procedure. The cryptosporidia are small spheres against a blue-green background. The oocysts can easily be seen with a 20X objective, and with experience can be observed with the 10X objective.

In addition to being quick and easy, the method is reliable. Several veterinary diagnostic laboratories routinely use this method rather than fecal floatation. When I was at the veterinary diagnostic laboratory at South Dakota State University, not only did our lab use the method for cryptosporidiosis diagnosis, so did a number of veterinary practices in the surrounding area.

If you decide to perform an acid-fast staining procedure in your practice, use the Kinyoun method rather than the Ziehl-Neelsen method. The Kinyoun method is done at room temperature, while the Ziehl-Neelsen procedure requires that slides covered with stain be heated almost to boiling. Kinyoun acid-fast stain kits are made by several companies: Becton Dickinson Microbiology Systems (BBL), Difco and Remel are some of the com-

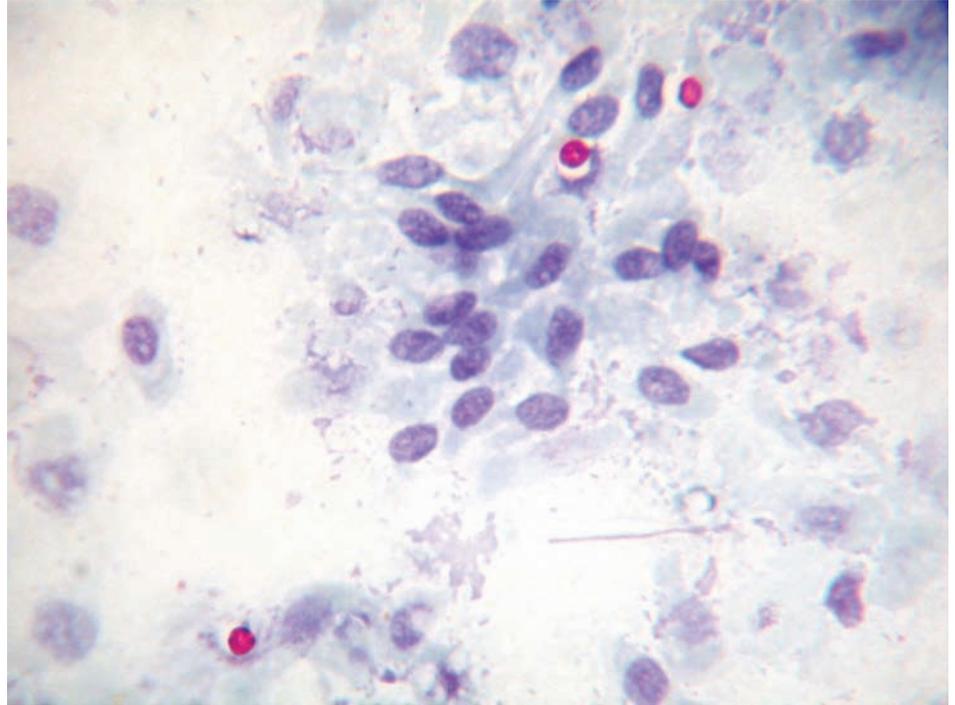


Figure 1. Three cryptosporidia stained by the Kinyoun acid-fast technique. Photographed at 400X.

panies that manufacture kits that contain the necessary components. Kinyoun staining kits should be available from any distributor that carries microbiological supplies. The staining procedure is as follows<sup>1</sup> (this procedure is the same as the one furnished with my kit):

1. Prepare a thin smear by placing a very small amount of feces on a glass slide using a tooth pick, wood splinter, the point of a knife, etc., and mixing it with a small amount of tap water. Spread the mixture over the surface of the slide. The smear should be thin enough to easily see through it.
2. Allow the smear to air dry and then heat fix the slide over an open flame.
3. Stain with carbol-fuchsin solution for three to five minutes.
4. Gently wash with running water until no more stain is removed by the water.
5. Rinse with decolorizer and wash with running water. Decolorize until no more stain is removed, but be careful not to over decolorize because it is possible to decolor the cryptosporidia.
6. Apply the counter stain (usually brilliant green) for 30 to 60 seconds and wash with running water.

7. Allow the slide to air dry.

8. Examine with a microscope. Microscopes are designed for examination of slides with a number one coverslip on the slide. A coverslip can be applied directly to the slide or by placing a drop of immersion oil on the slide and placing the coverslip over the oil. Let the slide sit a few minutes for the oil to spread over the slide beneath the coverslip, and examine.

9. Cryptosporidia appear as small red spheres against a blue-green background. (Figure 1).

<sup>1</sup>Ridley RK, Olsen RM: 1991. Rapid diagnosis of bovine cryptosporidiosis with a modified commercial acid-fast staining procedure. *J Vet Diagn Invest* 3:182-183.

# Plants may be hazardous to livestock this spring

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This information 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](http://www.veterinarywire.com). The complete publication is also online with free registration at [www.avis.org/special\\_books/Knight/toc.asp](http://www.avis.org/special_books/Knight/toc.asp).

## Senna, Coffee Weed, Coffee Senna

*Cassia occidentalis*, *Senna occidentalis*

## Sickle Pod, Coffee Weed, Coffee Bean

*Cassia obtusifolia*, *C. tora*, *Senna obtusifolia*

*Cassia spp.* have several compounds that bind strongly to cell membranes, but the specific toxin responsible for muscle degeneration has not been identified. The toxin induces acute muscle and liver degeneration that can be rapidly fatal in most animals. The greatest concentration of the toxin is in the seeds. *C. occidentalis* and *C. obtusifolia* are more toxic than other species, but all *Cassia spp.* should be considered toxic unless proven otherwise.

Cattle, sheep, goats, horses, pigs, rabbits and chickens are susceptible to poisoning by



Senna

*Cassia spp.* All parts of the plant are toxic, although most poisoning occurs when animals eat the pods and beans or are fed green-chop containing cassia. Ground coffee senna beans fed to cattle at the rate of 0.5 percent of their body weight induce severe muscle degeneration. Roasting of the beans partially reduces their toxicity. At low doses, *Cassia spp.* can cause diarrhea and decreased weight gain. As the amount of Cassia in the animal's diet increases, muscle degeneration increases and causes the clinical signs of poisoning.

In cattle, a moderate to severe diarrhea develops shortly after consumption. Abdominal pain, straining (tenesmus) and diarrhea are caused by the irritant effects of anthraquinones in *Cassia spp.* Affected animals remain afebrile. Depending on the amount of plant or seeds consumed, muscle degeneration begins after several days, causing weakness and recumbency. The urine may be coffee colored due to myoglobinuria from acute muscle degeneration. The levels of serum enzymes creatine kinase and aspartate transaminase are usually elevated, reflecting acute muscle degeneration. Renal failure may develop secondarily to the myoglobinuria. In severe cases, hepatic failure may be the cause of death. In more chronic cases, cardiomyopathy and hyperkalemia from muscle degeneration cause cardiac irregularities and contribute to the death of the animal. Respiratory difficulty is caused by the degeneration of the intercostal and diaphragm muscles.

Horses may not exhibit the digestive and muscle degenerative signs of poisoning seen in cattle. Myoglobinuria may not develop in horses because they succumb to liver degeneration sooner than to muscle degeneration.

Poisoned horses are generally afebrile and severely ataxic and may die without showing other clinical signs. Serum liver enzymes may be elevated, reflecting acute liver degeneration. Gross lesions at postmortem examination consist of pale skeletal muscles similar to those seen in white muscle disease associated with selenium and vitamin E deficiency.

Skeletal muscle necrosis and renal tubular and hepatic centrallobular necrosis are characteristic histologic findings that differentiate Cassia poisoning from vitamin E and selenium deficiency. Diagnosis should be based on access to and consumption of *Cassia spp.* along with the presence of degenerative lesions in the muscles, heart and liver.

There is no specific treatment for Cassia poisoning. Affected animals should be

removed from the source of the plants as quickly as possible and fed a nutritious diet. Supportive care of the recumbent animal will help prevent further muscle degeneration due to pressure necrosis. Recovery depends on the severity of muscle and liver degeneration. Rarely does an animal recover once it has become recumbent. It is important to differentiate white muscle disease due to selenium and vitamin E deficiency from Cassia poisoning because the use of selenium and vitamin E in Cassia poisoning is contraindicated. Increased myodegeneration and higher mortality occur when selenium and vitamin E are used to treat Cassia poisoning.

## White Snakeroot

*Eupatorium rugosum*

White snakeroot is found in low, moist areas or bordering streams, often on rich or basic soils of open woodlands from eastern Canada, west to the Dakotas, south to Geor-



White snakeroot

gia and west to Texas. Synonyms for white snakeroot include *Eupatorium urticaefolium* and *Ageratina altissima*.

The toxic component of white snakeroot is tremetone (tremetol) and requires microsomal activation before it becomes toxic to mammalian cells. All animals as well as humans are susceptible to poisoning. The toxin has its highest content in the green plant and remains toxic when dried in hay. The tremetone is cumulative in animals and is secreted in milk from cows that have eaten snakeroot.

Humans drinking the milk develop a severe nervous syndrome known as milk sickness. In animals, the toxicity is known as "trembles" because of the muscle tremors induced by the toxin. Poisoning develops after an animal has eaten from 0.5 to 1.5 percent of its body weight in green plant. Feeding horses 1 to 2 percent of their body weight of the plant induced poisoning in one to two weeks. Approximately 20 lb of green plant consumed

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over several days induces poisoning in horses. Foals may develop white snakeroot poisoning when they drink the milk from their dam that has been eating the plant. Various fungi growing on plants produce tremorgenic toxins that cause muscle tremors in livestock that can resemble those seen in white snakeroot poisoning.

Horses, cattle, sheep and goats poisoned with white snakeroot are initially listless, depressed, lethargic and reluctant to move. Cattle, in particular, develop muscle tremors, especially after exercise, and may show signs of colic, constipation, blood in the feces and a peculiar acetone-like breath odor.

Once muscle tremors begin, animals are reluctant to move, showing marked stiffness and eventual recumbency. Horses may show signs of choking due to the paralysis of the pharyngeal muscles. Nursing animals may have milk run out the nostrils. Patchy sweating may be evident. The urine may become dark brown due to myoglobinuria. A rapid and irregular heart rate and signs of congestive right heart failure may precede death. Mortality is usually high in livestock showing trembles. Severe skeletal and myocardial degeneration cause death. Fatty degeneration of the liver and kidney is a prominent necropsy finding.

There is no diagnostic test for tremetone that does not give false-positive results due to normally occurring miscellaneous ketones secreted in milk. Diagnosis is based on clinical signs and the presence of white snakeroot in the hay or pasture where the animal has been eating.

There is no specific antidote for white snakeroot poisoning. Laxatives and activated charcoal improve chances of recovery if administered early. Horses that exhibit difficulty in swallowing should be given water, electrolytes and appropriate nutrition via nasogastric tube. Recumbent animals should be placed in well-bedded stalls to prevent the development of pressure sores.

Poisoning in humans today is infrequent due to the practice of pooling milk from many cows, which dilutes any tremetone that may have been present. The individual family in rural areas that drinks raw milk from their cow have a greater potential for poisoning if there is white snakeroot in the animal's pasture. Pasteurization does not detoxify tremetone in milk.

## Lily of the Valley

### *Convallaria majalis*

Although this plant is not indigenous to North America, it is commonly planted as ground cover in shady gardens. It is a hardy plant and can establish large stands when abandoned. The plant is potentially toxic to animals if they are allowed to graze on it or are fed garden clippings.

The cardiac glycosides (cardenolides) convallerin and convallamarin and at least 15 others are found throughout the plant and have similar cardiac effects to digitalis glycosides. The seeds have the highest concentration of cardenolides, but the flesh of the fruit is minimally toxic. The skin of the fruit and the flowers also contain saponins that cause abdominal pain and diarrhea. Cardiac glycosides are found especially the leaves. Generally, only very small quantities of the plants must be ingested to produce poisoning. Poisoning has been reported in dogs. Drought and freezing temperatures may cause animals to consume more of the toxic plant. Although reduced, toxicity is retained in the dried plants.

Animals consuming plants containing these cardiac glycosides develop heart and digestive disturbances before death. The glycosides act directly on the gastrointestinal tract causing hemorrhagic enteritis, abdominal pain and diarrhea. The cardiac glycosides induce a progressive decrease in electrical conductivity through the heart, causing irregular heart activity and eventually blocking cardiac activity.

In low doses, the glycosides have a beneficial therapeutic effect on the heart by increasing the force of contraction, slowing the heart rate and increasing cardiac output. Toxic doses of the glycosides cause a variety of severe dysrhythmias and conduction disturbances through the myocardium that results in decreased cardiac output and death. Animals consuming cardiac glycoside-containing plants often die because of profound cardiac effects. A variety of cardiac arrhythmias and heart block, including ventricular tachycardia and first- and second-degree heart block, may be encountered with cardiac glycoside poisoning.

Abdominal pain (colic) and diarrhea are also signs commonly seen in animals poisoned with cardiac glycosides. If observed early in the course of poisoning, animals will exhibit rapid breathing, cold extremities, and a rapid, weak, and irregular pulse. The duration of symptoms rarely exceeds 24 hours before death occurs. Convulsions before death are not common. In



*Lily of the Valley* fruits



*Lily of the Valley*

acute poisoning from cardiac glycosides, the postmortem findings include hemorrhages, congestion, edema, and cell degeneration of the organs of the thoracic and abdominal cavities. In less acute but fatal poisoning, multifocal myocardial degeneration and necrosis is often present.

No specific treatment is available for counteracting the effects of the cardiac glycosides. Gastric lavage or emptying should be induced as soon as possible. Adsorbents such as activated charcoal (2 to 5 g/kg body weight) should be given orally to prevent further toxin absorption. The cardiac irregularities may be treated using antiarrhythmic drugs such as potassium chloride, procainamide, lidocaine, dipotassium EDTA or atropine sulfate. The use of potassium in intravenous fluids should be avoided and serum potassium levels should be monitored closely. Intravenous fluids containing calcium should not be given because

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calcium augments the effects of the cardiac glycosides. Poisoned animals should be kept as quiet as possible to avoid further stress on the heart. The use of digoxin-specific antibodies to treat digoxin toxicity, used widely in humans, has not yet found application in animal poisoning.

A diagnosis of cardiac glycoside poisoning may be made if an animal is found dead and evidence indicates that the animal had access to plants known to contain cardiac glycosides. Detection of cardiac dysrhythmias, heart block and ventricular escape rhythms suggests cardiac glycoside or grayanotoxin poisoning. Detection of cardiac glycosides in the serum, urine, tissues and stomach contents is possible using high-performance liquid chromatography.

Postmortem findings in may include focal pale areas and hemorrhages in the myocardium. Multifocal areas of necrosis and hemorrhage may be seen microscopically. The clinical cardiac abnormalities, sudden deaths and the lesions present in the heart typical of cardiac glycoside toxicity mimic poisoning due to the cattle feed additive monensin.

## Onions: Domesticated onion, Wild onion

*Allium spp.* (Lily family)

Onions are herbaceous plants with bulbs and narrowly linear leaves that smell of “onion.” The leaves are sheathing, usually basal and hollow. Onions are found throughout North America.

The alkaloid N-propyl disulphide is present in cultivated and wild onions, chives and garlic, and affects the enzyme, glucose-6-



*Wild onion*

phosphate dehydrogenase in red blood cells. Oxidation of hemoglobin results because there is insufficient phosphate dehydrogenase or glutathione to protect the red blood cells from oxidative injury. The oxidized hemoglobin in the red cells precipitates to form Heinz bodies. The cells containing Heinz bodies are removed by the spleen with the resulting anemia being proportional to the number of Heinz bodies formed and the rate at which the spleen removes the damaged cells. Cattle are the most susceptible to onion poisoning, horses and dogs are intermediate, and sheep and goats are the most resistant. Dog breeds such as Akitas and Shibas with high red blood cell levels of reduced glutathione and potassium are especially susceptible to the hemolytic effects of the oxidants in onions, and can be poisoned even after eating cooked onions.

The severity of Heinz body anemia that develops will vary with the quantity and the rate at which onions are consumed and animal species. Diets containing more than 25 percent dry matter of onion have the potential to cause clinical signs of anemia. The formation of Heinz bodies in sufficient numbers to cause anemia and hemoglobinuria (dark red-brown urine) may occur within one to three weeks of eating onions. Small numbers of Heinz bodies may be formed even though the amount of onion consumed may be less than that necessary to induce anemia.

The first noticeable sign of onion poisoning is often the presence of hemoglobinuria. Affected animals have pale mucous membranes and a fast, weak pulse. They may stagger and collapse as a result of anemia. There is frequently a distinct odor of onion on the breath, feces, urine and milk of poisoned animals. The onion flavor disappears from the milk after the lactating animal has been off of onions for 24 hours. In severely anemic animals, additional stress and heavy parasite infestations may be sufficient to cause death of the animal. Whole blood transfusions may be necessary in severely anemic animals.

## Pokeweed, Pokeberry

*Phytolacca americana*

All parts of the pokeweed plant contain saponins, oxalate and the alkaloid phytolaccine, with greatest concentrations in the roots and seeds. Pokeberry also contains a protein lectin (a mitogen) that can have a variety effects on the immune system. Pokeweed mitogen affects cell division and stimulates B- and T-cell lymphocyte proliferation. The plant should be handled with gloves because the mitogen can be absorbed through cuts and abrasions on the skin.



*Pokeweed*

Depending on the amount of the plant consumed, animals may show mild to severe colic and diarrhea. Fatalities are rare unless large quantities of the plant are consumed. Humans appear more severely poisoned by pokeweed and develop mouth irritation, stomach cramps, vomiting and diarrhea. Death may occur in children eating large amounts of the plant or berries. Sheep, cattle, goats, horses, pigs, poultry and dogs are susceptible to the toxic effects of pokeweed. The signs of poisoning are varied and include oral irritation, excessive salivation, vomiting, colic, bloody diarrhea, depression, prostration and death. Mild to severe gastroenteritis with ulceration of the gastric mucosa are common nonspecific findings on postmortem examination.

Intestinal protectants and other supportive treatments should be given as appropriate. Because there is no specific treatment, severely affected animals should be given intestinal protectants until the toxic plant material is eliminated from the digestive tract.

## Continuing Education

May 3, 5, 9, 11, 13

Spring VetBytes Continuing Education Audio Conference Series  
– Peter Chenoweth, Bull Problems: Common and Uncommon

June 5 – 8

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