

VETERINARY

FOR THE PRACTICING VETERINARIAN

Quarterly

Winter 2008
Volume 11, Number 1

Winter and holiday health hazards for animals

John C. Haliburton, D.V.M., Ph.D.
Texas Veterinary Medical Diagnostic Laboratory

With the arrival of the winter months and holiday season, there are additional health hazards which are of concern for animals; some are potentially fatal. A few of these health risks could be brought into the home inadvertently, thereby increasing a pet's possibility of exposure.

To keep the season safe, protect animals from contact with or ingestion of the following:

Antifreeze — this mixture commonly contains ethylene glycol, a product that can cause lethal kidney failure and metabolic acidosis (accumulation of acid in the blood and body tissues) if ingested. It has a sweet taste that attracts animals and can be toxic in small doses (i.e., 1-2 tablespoons can produce toxicity in a medium-

sized dog). Antifreeze can be toxic even when diluted in water. At least one brand of antifreeze is available that uses propylene glycol for the active component as an alternative to ethylene glycol. Larger quantities of the propylene glycol-based antifreeze usually have to be swallowed to produce toxicity as compared to ethylene glycol-based antifreeze. Additionally, propylene glycol-based antifreeze does not metabolize in the animal's system to form products that cause kidney damage; however, it can still cause illness and death via metabolic acidosis. An antidote is available for antifreeze poisoning, but early recognition of ingestion and immediate intensive treatment are imperative for the survival of the animal. The best medicine, though, is to prevent animals from being in contact with this toxic substance by having antifreeze changed by a professional who knows how to properly dispose of it. If individuals change their own antifreeze, they should not drain it into the sewer or leave it sitting out in a pan for any amount of time. All it takes is a few seconds for an animal to ingest it.

Baking chocolate — this form of chocolate contains a higher concentration of stimulant (theobromine) than regular chocolate. One-fourth pound can be toxic if eaten by a small dog, such as a poodle.

Mistletoe — the berry of this plant is the most toxic component, especially if it is chewed instead of swallowed whole. If the berry is ingested in sufficient quantity, it can cause gastrointestinal and neurological symptoms, including convulsions.

Poinsettia — whether or not this plant is toxic has been debated for years. The most recent findings are that it contains

no toxic chemical. However, as with any plant that an animal is not accustomed to eating, it can cause diarrhea and vomiting (a protective mechanism to eliminate the foreign substance). Animals tend to be attracted to poinsettias, so it is a good practice to keep these plants out of their reach.

Ivy — this plant is not acutely toxic, but it can cause gastrointestinal upset if ingested.

Christmas cactus — this plant is non-toxic, but it can cause vomiting and transient diarrhea if consumed.

Tinsel — cats in particular are attracted to playing with Christmas tree tinsel. If ingested, it can cause an intestinal blockage or intussusception (prolapse of one part of the intestine into the cavity of an immediately adjoining part). If indoor cats are present, it would be prudent to avoid using strands of tinsel. It would also be advisable to place breakable ornaments at the top of the tree. An investment in shatterproof ornaments might also be worthwhile.

Glow jewelry¹ — dibutyl phthalate is a chemical contained in glow-in-the-dark jewelry, which is a popular item at a variety of festivities. Although the chemical may have the potential to cause death via respiratory paralysis, cats generally will only ingest a minimal amount due to its unpleasant taste and the fact that only a small amount of the chemical is present in the jewelry. Cats that have bitten into the jewelry may exhibit heavy salivation, hyperactivity, and aggressive behavior, but they typically recover within minutes. Immediately after a cat ingests this chemi-

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

2007: Banner year for anaplasmosis

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

It is common for us to see a few cases of bovine anaplasmosis, but in 2007 we have had a large number of cases submitted to the K-State Veterinary Diagnostic Laboratory. It also seems that the numbers of dead cows and cows with clinical signs in affected herds has been higher than normal. For example, one herd of fewer than 100 head lost 12 cows before the first cow was submitted. The history for this herd was fairly typical. Most of the affected cows were found dead at pasture, but a few had gotten thin and quit eating. All of the affected animals had been mature cows, more than three years old. Histories of multiple thin, anemic, icteric, and dead cows have been common this fall.

Diagnosis of anaplasmosis in living cattle can be by identification of organisms on erythrocytes, serology, or preferably, both. Calves infected before 6 months of age rarely show clinical signs. Cattle between 6 months and 3 years are at increased risk of clinical illness, while cattle over 3 years commonly develop signs of acute anaplasmosis that can be

fatal. Because animals that recover become persistently infected carriers, a positive serologic test proves infection but not clinical disease. Of course, the combination of typical clinical signs and a positive serological test is pretty good evidence of anaplasmosis, but even better is the combination of finding infected erythrocytes and positive serology. The positive blood smear proves parasitemia and the serology confirms the cytologic results.

Diagnosis in a dead animal can be a little more problematic. Necropsy lesions are usually subtle and consist only of a thin, icteric cow with a moderately swollen spleen that has a jelly-like consistency on cut surface. To me the blood appears watery, but this is subjective, especially since the blood is what either did not clot or is from clots that have dissolved after formation. Histologic examination of tissues is not especially rewarding. With special stains *Anaplasma* organisms can be seen on erythrocytes in the tissues, but formalin fixation and processing shrinks everything, and the erythrocytes and *Anaplasma* organisms are better visualized with a blood smear. While doing the necropsy, I like to take a drop or two of

blood and make smears on a microscope slide and stain them as one would whole blood from a live cow. If the animal is not too decomposed, the *Anaplasma* organisms can be seen in the smear. If you feel uncomfortable interpreting the results, submit the slides along with other samples to the lab. It is better if the person doing the postmortem make the slides before mailing them, because there will be much less decomposition of the erythrocytes. Some books say to make impression smears of the spleen. Personally, I have not found them to be helpful. The splenic smears are always too thick to be able to interpret. You can also collect blood that flows from the incisions when you open the animals and submit them for ELISA for *Anaplasma marginale*.

Now that December is here and it is cold, biting fly season is over, and we should have less transmission and fewer new cases of anaplasmosis (unless herds have a tick problem). However, because of the persistent carrier state in clinical disease survivors, it will still be in these herds next year.

Cost of subclinical Johne's disease in dairy cows

*Jerome Nietfeld, DVM, PhD
K-State Veterinary Diagnostic Laboratory*

In late October I attended the 2007 meeting of the National Johne's Working Group at the annual meeting of the United States Animal Health Association. One of the most informative presentations was by Dr. Mario Villarino, a Texas A&M extension veterinarian. Dr. Villarino outlined his experiences with two large Texas dairies participating in the National Johne's Demonstration Herd project, which was initiated to identify control procedures that work. The dairies have been enrolled in the program for about 4 years and during that time have continued to grow by adding home-raised and purchased heifers. Both have several thousand cows, with the larger one scheduled to plateau in the next year or so at 14,000 cows. At the beginning of the project 7 to 10 percent of the

cows were testing positive by the Johne's ELISA. Neither dairy has a problem with cows developing clinical signs of Johne's disease. Both dairies cull cows when milk production falls below 30 lb milk/day, and Johne's positive cows are culled before the disease is clinically evident. However, they have found that the days in lactation for Johne's ELISA positive cows average 130 days less than for Johne's negative cows. They calculated that each Johne's ELISA positive cow costs them \$205.00 because of early replacement. The ELISA is the only Johne's disease test used by the dairies, and they do not use the information to make management decisions concerning culling of cows. The information is used only for colostrum management and calf segregation. ELISA positive cows are marked, and their colostrum is never used to feed newborn calves. Calves born

to Johne's positive cows are segregated from those from negative cows. I am not sure if heifer calves from positive cows are used as herd replacements, or if they are sold because of the possibility of in utero transmission. Since instituting this testing program, both dairies have significantly reduced the numbers of home raised replacements that become Johne's disease positive. When asked if these dairies would continue the program if the Federal government discontinued its financial support of the Johne's control program, Dr. Villarino said that both owners are very positive about the program and that they would definitely continue. They feel that costs for ELISA testing are low when compared to the money saved by decreasing Johne's in their herds.

Bovine leukosis infection in Kansas

Larry C. Hollis, D.V.M., M.Ag.
Extension Beef Veterinarian

Bovine leukosis, caused by the bovine leukosis virus (BLV), occurs rarely in some Kansas herds, but has reached almost epidemic proportions in others. In “normal” herds, BLV will only take out an occasional animal. However, in one herd leukosis was the cause of death in 7 of 120 cows in less than a year’s time. The producer involved is currently considering liquidating his herd and starting over. In another herd with an unusual number of unexplained death losses annually, 111 of 179 cows were found to be seropositive for BLV. In yet another situation where a producer purchased six older cows at a special fall cow sale with the intent of getting one to two more calves from each cow before they were ultimately sold for harvest, five of the six cows were tested and found to be seropositive for BLV. None of the five had clinical signs of the disease, but their life expectancy was definitely shortened relative to the purchase price and expectations of their new owner.

Any time a producer has a single adult animal unexpectedly start to deteriorate in condition, BLV should be suspected as a possible cause. The lymphoma form with visible swollen lymph nodes occurs infrequently; thus, the lack of swollen nodes should not rule out BLV. Make sure not to overlook Johnes Disease and parasitism as potential differentials in these cases.

Because BLV infects white blood cells and is transmitted via the blood, any time blood is transferred animal-to-animal the potential is there to transmit the disease. The disease can be transmitted via multi-use needles used for vaccination or treatment, ear taggers, dehorners, tattoo pliers, implant guns, OB sleeves, and any other object that can carry blood from one animal to another. It may also be possible for the larger biting flies to transmit the disease. To a lesser extent the disease may be transmitted in utero or via nursing colostrum.

Biocontainment efforts to keep the disease from moving within a herd include testing herds and removing seropositive animals where only a small percentage of

animals are infected. In herds where BLV infection levels are higher, economics may dictate that the majority of the animals be retained and split into two herds – a positive herd where animals will be culled at the first sign of problems and from which no calves will be kept as replacements, and a negative herd which will serve as the only source of home-grown replacement animals. While testing the cow herd, be sure to test all bulls and replacement animals held back for future use. Use of disinfectants with all equipment that might transfer blood animal-to-animal should be instituted in the negative herds. The only exception to this is that disinfectants will kill modified live vaccines (Brucellosis vaccine and MLV respiratory and reproductive vaccines). With MLV vaccines, a new needle should be used for each animal. A good fly control program is also critical to keeping a negative herd free of the disease during biting fly season.

Biosecurity efforts to keep the disease out of a negative herd should be instituted once the herd is segmented or determined to be free of the disease. Producers should be encouraged to purchase only test-negative replacement bulls and females, or purchase from seedstock suppliers that maintain negative herds based upon the results of frequent testing. Testimonials such as “we’ve never seen BLV in our herd” don’t count. It is never recommended to buy a calf at auction to graft onto a wet cow that has lost her calf because of the potential of the calf to bring BLV or a host of other diseases into the herd.

Producers should be encouraged to be vigilant in their management and take steps to prevent introduction of the disease into their herds and transmission within herds where the disease already exists. Otherwise, this insidious disease may cause significant financial losses.

Watch for osteopetrosis in calves

Jerome Nietfeld, DVM, PhD
K-State Veterinary Diagnostic Laboratory

Recently we diagnosed osteopetrosis in an aborted red Angus fetus. This is the third consecutive year in which we have identified osteopetrosis in a red Angus fetus, with each of the three cases from a different herd. Osteopetrosis can be inherited in cattle, including the red and black Angus, Herford, Simmental, and Dutch Holstein-Friesian breeds. Osteopetrosis-like lesions have also been reported in calves infected in utero with bovine viral diarrhea (BVD) virus (for a more complete discussion of the two forms of osteopetrosis see *Kansas Veterinary Quarterly*, Spring 2006, volume 9, No. 2). In the red Angus cases, the affected calves were negative for BVD virus and the pathologic changes in the skulls and long bones were typical of inherited osteopetrosis and different from those reported in BVD virus-infected calves with osteopetrosis-like lesions.

After the second osteopetrosis case, the Red Angus Association implemented a program to identify affected calves. They also contracted with a researcher to identify the mutation responsible for osteopetrosis and to develop a test to identify clinically normal carriers of the osteopetrosis mutation. Such a test will also allow identification of offspring of carrier cattle who do not carry the osteopetrosis mutation. It is estimated that tissues from approximately 10 calves with osteopetrosis will be required for identification of the defective gene. If anyone suspects osteopetrosis in a red Angus calf osteopetrosis, please contact Larry Keenan of the Red Angus Association at 940-387-3502 or Larry@redangus.org. Alternately, you can contact Jerome Nietfeld at the K-State Veterinary Diagnostic Laboratory at 785-532-4460 or nietfeld@vet.k-state.edu. If you suspect osteopetrosis in other breeds, please contact me.

Novel H2N3 influenza virus isolated from U.S. pigs

Jerome Nietfeld, DVM, PhD

K-State Veterinary Diagnostic Laboratory

At the 2007 annual meeting of the American Association of Veterinary Laboratory Diagnosticians there were reports of isolation and characterization of novel H2N3 influenza viruses from pigs with acute respiratory disease. The viruses were isolated by the Minnesota Veterinary Diagnostic Laboratory from samples submitted in April and September 2006 from two separate swine farms. The two farms were 4 miles apart, but did not share pigs, personnel, feed, or transportation. The viruses could not be typed using serologic or molecular methods designed for swine influenza viruses. Therefore, they were sent to the National Animal Disease Center for sequencing and to St. Jude Children's Research Hospital for serotyping. All eight RNA segments were sequenced and the viruses identified as H2N3 influenza viruses. This is the first report of H2N3 influenza virus being isolated from pigs in the U. S. The hemagglutinin (HA), neuraminidase (NA), and RNA polymerase A (PA) genes were derived from waterfowl influenza viruses, and the other five gene segments were from triple reassortant influenza viruses (genes from swine, avian, and human viruses) currently circulating in U. S. swine. The hemagglutinin had a mutation that allowed the viruses to preferentially bind mammalian viral receptors. Experimentally, the viruses were pathogenic for swine, mice, and ferrets, and they spread to in-contact animals. It is thought that the avian viruses were transmitted to the pigs by the use

of water from ponds that are frequented by migrating waterfowl in the spring and fall. At the time of the presentations in October 2007, there had been no known transmission to humans.

Why should people who do not own or work around pigs care? Because influenza viruses that cause human pandemics are derived from waterfowl viruses, and pigs are susceptible to both avian and mammalian viruses. Pigs are considered to be possible "mixing pots" for creation of new influenza viruses. The hemagglutinin (HA) of influenza viruses is responsible for binding influenza viruses to cells, and antibodies to a homologous HA prevent infection. There are 16 different HAs that occur in avian influenza viruses that circulate in waterfowl. Since 1900, only H1, H2, and H3 influenza viruses have circulated in humans and since 1968, only H1 and H3 viruses have circulated. Normally, humans are not susceptible to avian influenza viruses because we lack the receptor to which the viruses bind. Conversely, birds are not susceptible to mammalian (human and swine) influenza viruses because they lack the receptor to which mammalian viruses bind. That is one reason (there are also others) that the H5N1 avian influenza viruses that have been receiving worldwide notoriety for several years for killing tens of millions of birds and more than 100 people have not spread in the human population.

Pigs are susceptible to both mammalian and avian influenza viruses. Because influenza viruses have a segmented genome, in pigs simultaneously infected

with multiple influenza viruses, new viruses with genes from different parent viruses can be produced. Once in a new host, influenza viruses are unstable and undergo frequent mutation, with the HA gene having the highest frequency.

Since 1998, reassortant H1N1, H3N2, and H1N2 viruses with genes from avian, swine, and human influenza viruses have become common in U. S. swine. (For example, we recently isolated influenza virus from two pigs from the same finisher building, and one was H1N2 and the other was H3N2.) While these viruses are a concern because they can be transmitted to humans, they are not a concern to cause a pandemic because H1 and H3 influenza viruses have co-circulated in humans since 1977, and everyone has some immunity.

The appearance of an H2 virus in pigs should be a concern because H2 influenza virus has not circulated in the human population since 1968, when the H3N2 "Hong Kong flu" appeared and the H2N2 virus that had circulated since 1957 disappeared. Thus, people born after 1968 have had no exposure to H2 influenza virus and are completely naive.

Those of us born before 1968 have been exposed, but obviously our immunity has declined a little in the last 40 years. Vaccination will not help, because there is no reason to include an H2 virus in the vaccines. The jump from pigs to people is not nearly as great as the one from birds to people. That is why the changing nature of influenza in pigs continues to be a concern and to be monitored by both animal and human health researchers.

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cal, it helps to feed it small quantities of milk, canned food, or tuna juice to dilute the chemical in its mouth. Wash off any drops of the chemical that might be on the cat's coat and flush the cat's eyes with water if there has been ocular exposure. There is no known antidote for dibutyl phthalate; cats that have ingested large quantities should be closely monitored and given supportive treatment if warranted.

Cold — the U.S. Animal and Plant Health Inspection Service's Animal Wel-

fare Act recommends that ambient temperature should not drop below 50 degrees Fahrenheit, especially when sick, aged, or young animals are present. Additionally, animals should always be provided with adequate protection and shelter from the direct effect of wind, rain, or snow. Remember, animals (in many areas) are not acclimated to cold weather, so they must be protected from extreme weather conditions accordingly.

If you know or suspect that an animal has ingested any of the above items (1-8), immediately consult a veterinarian, animal emergency clinic, or poison control center. The ASPCA National Animal Poison Control Center can be reached at 1-800-548-2423.

1Rosendale, ME. Veterinary Medicine 1999; August:703.

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Viable MAP found in pasteurized retail milk

Jerome C. Nietfeld, DVM, PhD

K-State Veterinary Diagnostic Laboratory

There are clinical similarities between Johne's disease of ruminants and Crohn's disease of humans. For many years there has been speculation that *Mycobacterium avium* subsp. *paratuberculosis* (MAP), the cause of Johne's disease, is the cause of Crohn's disease. Viable MAP and MAP genetic material have been found in samples from Crohn's disease patients, but MAP has not been proven to cause Crohn's disease. Also, there is no documentation of transmission of MAP from ruminants to humans. If MAP is the cause of Crohn's disease, it is important to identify possible methods of transmission from ruminants to humans.

Cattle with Johne's disease sometimes shed MAP in their milk. A 1996 survey by the National Animal Health Monitoring System (NAHMS) found that, based on ELISA, 21.6% of U. S. dairy herds have a prevalence of MAP infected cows of greater than or equal to 10%.¹ At the 2007 annual meeting of the U. S. Animal Health Association it was reported that a 2007 NAHMS survey found that 65.6% of the 507 dairy herds for which testing was completed were positive for MAP. The results of the latter study are based on MAP culture of six environmental samples per herd. University of Minnesota research estimates the sensitivity of culturing environmental samples is approximately 80%. For several years there has been controversy concerning the ability of pasteurization to completely kill MAP in milk. Obviously, if pasteurization does not completely kill MAP, retail milk represents a significant source of possible exposure of humans to MAP. A study in the United Kingdom found that 1.8% of the retail pasteurized milk samples that they cultured contained viable MAP, which increased concerns that pasteurization might not completely inactivate MAP. The remainder of this article summarizes the findings of the only survey of retail pasteurized milk in the United States for viable MAP.²

Methods: Researchers from the Marshfield Clinical Laboratories in Marshfield, Wisconsin collected 20 pints of retail

pasteurized milk from California, Wisconsin, and Minnesota (3 of the 5 leading dairy production states in the United States) per month for a year. Milk samples that arrived at the laboratory damaged, expired, or at a temperature above 10 C were discarded. The remaining samples were processed and cultured on solid agar at the Marshfield laboratory and tested by two PCR techniques for MAP DNA. The samples were then sent to TREK Diagnostics for liquid culture for MAP. For MAP culture the bacteria in 80 ml of milk were concentrated by centrifugation into 1.5 ml aliquots. Two hundred microliters of each sample were used for solid culture and 1.0 ml was used for liquid culture. Positive cultures were confirmed by both PCR techniques and by morphologic characteristics and nutrient requirements on solid agar.

Results: Of the 702 pints of milk tested, MAP DNA was identified by PCR in 452 (64%) samples. Twenty samples (2.8%) were culture positive for MAP. There were no significant differences in the number of positive samples from any of the three states, but samples collected during July, August, and September were significantly more likely to be positive ($P = 0.05$). The researchers felt that because of the stringent requirements used to confirm that isolates were MAP, their results might underestimate the prevalence of viable MAP in milk.

Discussion: The fact that 64% of the samples contained MAP DNA indicates that MAP contamination of milk is common. Because MAP could be grown from only 2.8% of the samples, pasteurization greatly reduces the numbers of viable MAP in milk, but it does not kill all organisms. Based on these results it is possible that approximately three of every 100 cartons of milk purchased in the United States contain viable MAP. Thus, retail pasteurized milk represents an important possible source of MAP infection for humans. It also stands to reason that unpasteurized milk, unless from herds consistently test negative for Johne's disease, would be even more likely to contain viable MAP.

References

1. National Animal Health Monitoring System (NAHMS). 1997. Johne's disease on U.S. dairy operations. USDA: APHIS:VS, CEAH, NAHMS, Fort Collins, Colorado. No N245.1097.
2. Ellingson JLE, Anderson JL, Kozickowski JJ, et al. 2005. Detection of viable *Mycobacterium avium* subsp. *paratuberculosis* in retail pasteurized whole milk by two culture methods and PCR. *Journal of Food Protection* 68:966-972.

Continuing Education

January 11 Bull Evaluation and Management Conference	March 1-2, 2008 Equine Reproduction Conference for Veterinarians
January 26, 2008 Canine Care Workshop	March 8, 2008 Advanced Conference for Horse Owners
February 10, 2008 16th Annual Small Animal Conference on Clinical Hematology	April 6, 2008 25th Annual Frank W. Jordan Seminar on Fielding a Winning Team
February 23-24, 2008 Artificial Insemination Course for Horse Owners	June 1-4, 2008 70th Annual Conference for Veterinarians and KVMA Veterinary Trade Show
March 1, 2008 Veterinary Technicians Conference	

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

Upcoming Events

December 7, 2007 Beef Expo Judging Contest, Hutchinson	December 15, 2007 Junior Beef Producer Day, Manhattan
December 11, 2007 KSU Dairy Days, Seneca	December 18, 2007 PQA Plus Training, Manhattan
December 11-13, 2007 Range Beef Cow Symposium XX, Fort Collins, CO	December 18, 2007 Beef and Ethanol Meeting, Hoxie and Norton
December 12, 2007 By-Products Nutrition Conference, Garden City	December 19, 2007 Beef and Ethanol Meeting, Quinter
December 13, 2007 KSU Dairy Days, Whiteside	January 9, 2008 4-State Beef Conference, Washington
	February 5, 2008 KSU Swine Profitability Conference,

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Newsletter Coordinators

Larry C. Hollis

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

Jerome C. Nietfeld

Jerome C. Nietfeld
785-532-4460 • nietfeld@vet.ksu.edu

Contributors — K-State Research and Extension

Dale Blasi	Ron Hale	Twig Marston
Scott Beyer	Mike Brouk	Sandy Johnson
Joel DeRouchey	Mike Tokach	John Smith
Jim Nelssen	Bob Goodband	Cliff Spaeth

Contributors — Veterinary Diagnostic Laboratory

G.A. Andrews	R. Ganta	R. Pannbacker
M.M. Chengappa	S. Kapil	J.A. Pickrell
B. DeBey	K.S. Keeton	S.S. Dritz
D.A. Mosier	M.F. Spire	M.W. Dryden
T.G. Nagaraja	S. Stockham	B.W. Fenwick
M.J. Wilkerson	F.W. Oehme	

K-State Research and Extension

137 Call Hall
Manhattan, KS 66506

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