

VETERINARY

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Tips for keeping cattle cool, avoiding summer heat stress

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With the arrival of hot weather, and the fact that we actually have had rain in many parts of the state, memories of the summers of 1995 and 1999 may cross the minds of Kansas veterinarians and cattle producers. During brief periods of those summers, several conditions combined to create a deadly situation. Daytime temperatures exceeded 100°F for several days in a row. Humidity hovered near the 50 percent mark or higher. Nighttime temperatures rarely dropped below 80°F. There was no wind or cloud cover.

These conditions resulted in numerous cattle dying of hyperthermia.

The upper end of the thermoneutral zone for summer-adapted beef cattle is roughly 75°F. As temperatures rise above that level, appetites are progressively suppressed, additional energy is expended through panting and sweating, cattle spend most of their time seeking out water holes and shade, and animals may get into trouble. The inability of cattle to dissipate heat is compounded as humidity increases.

In anticipation of hot weather, cattle producers should be reminded of several

management activities that increase the heat-related risk to cattle, including working cattle during the heat of the day, tightly bunching groups of cattle in areas with poor air circulation (holding pens, barns, low spots, pens with solid fences, etc.), bunching for extended periods of time, and holding cattle away from water. Providing free access to water at all times is extremely critical. Providing access to shade during periods of extreme heat is also helpful. Many feedlots and some cow/calf producers use overhead shades and/or sprinkler systems to keep cattle cool, not only to benefit cattle safety, but also to maintain optimal performance during hot weather.

As in all species, when hyperthermia becomes a problem, rapidly reducing the core body temperature to normal is the primary goal. Sprinkling or hosing down the animal with cold water will help. In the case of ruminants, the ability to force feed cold water via a stomach tube into the rumen is an excellent option that usually provides faster results.

Hopefully, your clients will avoid letting their cattle get into heat-related trouble this summer. Common sense management teamed with a water hose and garden sprinkler is probably the cheapest preventative medicine available.

Patton joins diagnostic laboratory

Dr. Kristin M. Patton has joined the Kansas State Veterinary Diagnostic Laboratory as a veterinary pathologist. Patton earned a D.V.M. degree from Oregon State University in 1995. After graduation she entered a residency program in veterinary pathology at Cornell University, which she completed in 1998. She then began work on a Ph.D. at Washington State University. In 2000, Patton became a diplomate of the American College of Veterinary Pathology and in 2004 received a Ph.D. from Washington State University. After graduation from Washington State, Patton took a position as a veterinary pathologist at the New Mexico Department of Agriculture veterinary diagnostic laboratory in Albuquerque, N.M., where she worked until coming to K-State.

Patton's doctoral research focused on the immunopathogenesis of pulmonary diseases of the horse, specifically *Rhodococcus equi* infection. She has a good background in pathology of production and pet animals, and is especially interested in equine pathology with special interests in respiratory and reproductive diseases. Patton is also inter-

ested in dermatopathology and all diseases of horses and cattle. While at the New Mexico lab, Patton was responsible for setting up laboratories for immunohistochemical staining for chronic wasting disease and bovine virus diarrhea virus. In New Mexico, she actively participated in educational meetings for veterinarians and producers. Client education is very important to her and she looks forward to interacting with veterinarians and animal owners at K-State.

You can contact Dr. Patton by phone at 785-532-5650 or by e-mail: kmpatton@vet.k-state.



Dr. Kristin Patton

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Gulf Coast ticks make their presence known in Kansas

Alberto B. Broce, Ph.D., Entomology; Michael Dryden, DVM, Ph.D., Diagnostic Medicine/Parasitology; All photos by Dr. Michael Dryden

The Gulf Coast tick, *Amblyomma maculatum*, used to be considered incapable of overwintering in Kansas – the infestations seen in the state were thought to have been brought in by

cattle from states along the Gulf. But persistent and early spring populations in the state can only be explained by overwintering populations. This tick was originally thought to be restricted to Gulf Coast States and up to southeastern Kansas. It is now found on cattle in central, northcentral, and northeastern Kansas.

Outbreaks of this tick have been recorded in central and northeastern Kansas since 1998. This is an ornate three-host tick typically found in late March through June in Oklahoma and southern Kansas. In central and eastern Kansas, populations are at their highest from April through early June. Larvae feed on small rodents and ground-dwelling birds such as quail, cattle egrets and meadowlarks. Nymphs use hosts similar to

those of the larvae, plus dogs. Adults feed on larger hosts such as dogs, bobcats, coyotes, rabbits, rodents, deer and humans, preferring livestock such as cattle, horses, pigs, and goats. Immature forms feed on migratory birds such as cattle egrets and meadowlarks. These birds may be responsible for the spread of this tick.

This tick commonly clusters in the inside of the ears of large mammals, causing intense soreness. Under heavy infestations, ear muscles become weakened, which results in a flop-eared, permanent deformity called “gotch ear.” Research on growth performance of cattle infested with Gulf Coast ticks has shown it to be reduced by as much as 20 percent. Before the screwworm fly was eradicated from the United States, the presence of Gulf Coast ticks producing bloody ears through which the screwworm could penetrate into cattle heads, resulted in cattle mortality of up to 7 percent some years.



Male Gulf Coast tick



Female Gulf Coast tick



Ear infested with Gulf Coast ticks

There are 41 products registered in Kansas for the control of Gulf Coast ticks. Rapid control of these ticks can be achieved with direct animal applications of pesticides, such



Cow with “gotch ear”

as amitraz (Tactic), coumaphos (Co-Ral) or permethrin (Ectiban, Atroban, Permethrin, Expar etc.). Most of these chemicals should provide control and prevent reinfestation for 3 or 4 weeks. To control Gulf Coast ticks in the ears, spray for good neck, shoulder and ear coverage. Long-term control can be best achieved by using ear tags impregnated with any of several insecticides.

Tags containing organophosphate (OP) tags include: etion (Commando); coumaphos + diazinon (Co-Ral Plus); diazinon (Patriot, Optimizer, Z Diazinon, X-Terminator); and chlopyrifos + diazinon (Warrior, Diaphos).

Tags containing pyrethroid insecticides include permethrin (Atroban Extra, Gard-Star Plus); beta-cyfluthrin (Cylence Ultra); fenvalerate (Super Deckem); and zeta-cypermethrin (Zeta-Gard).

Even an organophosphate-pyrethroid combo is available as cypermethrin + chlorpyrifos (Max-Con).

Lead poisoning: A cause of sudden death in young calves

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Veterinary Diagnostic Laboratory

Lead poisoning is one of the most important causes of toxicity in cattle. Although lead poisoning occurs throughout the year, numbers of cases increase in the spring and summer. Two primary reasons for this are that the acute toxic dose of lead is much lower for calves than for adults, and calves are curious and small and can explore places that adults cannot. If their environment contains an old battery or something else that contains lead, calves, will find it and eat it. Approximately 20 grams (0.7 ounces) of lead is an acute lethal dose for a neonatal calf.

Text books list the clinical signs of acute lead poisoning in cattle as being primarily referable to the central nervous system (CNS).^{1,2} Signs that appear within 24 to 48 hours of exposure include: ataxia, blindness, hyperesthesia, muscle tremors, seizures, head pressing, aggression, and excessive salivation. Diarrhea, bloat, tenesmus, and constipation can also occur, but are often overshadowed by the CNS symptoms. Differential diagnoses are rabies, thromboembolic meningoencephalitis, polioencephalomalacia, listeriosis, nervous coccidiosis, ammoniated forage toxicosis, and hepatic encephalopathy.¹ Blindness is an important finding and can occur with few other noticeable signs of lead toxicity. Easily the two most common causes of acute blindness in cattle are lead toxicity and polioencephalomalacia. In any outbreak of CNS disease in calves where the clinical signs resemble polioencephalomalacia, if affected calves do not respond to treatment for polio, test them for lead.

Something that is rarely, if ever, mentioned in textbooks as a clinical sign of lead poisoning is sudden death. Each year we receive several cases where someone has several calves die unexpectedly. Often the calves are simply found dead in the pasture, but sometimes they are found recumbent and minimally responsive. Usually, there is nothing in the history to suggest CNS disease. If there are signs to suggest CNS disease, they are very subtle, like the case where an owner pulled up to the veterinarian's clinic with seven dead calves and one live calf in a trailer. The live calf was in lateral

recumbency, hypothermic, nonresponsive, and died within minutes of beginning the examination. The veterinarian said that he thought that the calf had nystagmus, but noticed nothing else to assist in determining the cause of death. The calves were getting through a small hole in the fence and chewing on a cracked car battery. Typically, the calves that die suddenly due to lead poisoning are less than two to three months old.

Older calves typically display clinical signs of CNS disease before dying.

The younger calves may very well display signs of CNS disease, but the clinical course is probably very short and easy to miss if the calves are not being observed closely. Infectious diarrhea is by far the most common cause of illness and death in neonatal calves and affected calves can also be found dead. Lead should be suspected in calves that are found dead or dying, do not have clinical evidence of diarrhea or dehydration, and do not have significant necropsy findings. Basically, lead poisoning should be considered in cases where several calves have died peracutely for no apparent reason.

Diagnosis

For live animals, whole blood is the specimen of choice. More than 90 percent of circulating lead is absorbed by erythrocytes, so serum is not suitable. Most laboratories (including the K-State Veterinary Diagnostic Lab) can determine lead levels on EDTA, citrated, or heparinized blood. If all that is available is clotted blood, we can analyze that, it just takes a little longer. Do not send serum without the clot. EDTA can interfere with some procedures, so if you are unsure of the sample needed by the lab you plan to use, call them to determine the appropriate sample. In most species, blood lead levels greater than 0.3 ppm indicate significant exposure to lead and are usually considered diagnostic if accompanied by the appropriate clinical signs. Levels greater than 0.6 ppm are diagnostic of toxicity. Kidney and liver are the tissues of choice for diagnosis of toxicity in dead animals. Levels of greater

than 10 ppm lead in either organ are usually considered diagnostic of toxicosis. In cases of acute toxicosis, the kidney is more likely to contain elevated lead, while in cases of chronic toxicity, lead is more likely to be elevated in the liver. Virtually all cases we see are due to acute exposure, and the lead levels are higher in the kidney than the liver. In fact, occasionally the liver levels are normal while the kidney levels are considerably above 10 ppm.

Sources of lead

Textbooks give long lists of possible sources of lead exposure,^{1,2} which makes one realize that lead is indeed widespread in our environment. However, by far the most common source of lead

in livestock poisoning cases is discarded or forgotten automotive-type batteries. Lead-based paint accounts for a small percentage of cases, and improperly discarded oil and other lubricants that contain lead account for a few cases. If you suspect or have had lead toxicity diagnosed and do not find one of these sources, pull out a toxicology, medicine, or pathology text and keep looking for one of the lead-containing compounds listed.

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1. Gwaltney-Brant S. Lead. In: Clinical Veterinary Toxicology. Plumlee KH, editor, 2004, Mosby, St. Louis, MO. pp 204-210.
2. Lead Poisoning. In: The Merck Veterinary Manual, 9th ed., Kahn CM, editor, 2005, Merck & Co., Inc., Whitehouse Station, NJ. pp. 2404-2405.

Lead poisoning should be suspected in calves that are found dead or dying, do not have clinical evidence of diarrhea or dehydration, and do not have significant necropsy findings.

Feline tularemia cases may pose risk to humans

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Editor's note: The following is very important as it seems that the incidence of tularemia has increased dramatically over the past five to 10 years. Growing up, I was always warned to be careful not to catch rabbit fever (tularemia), but I never knew of anyone contracting the disease. In the past five years I know of at least seven people who have developed tularemia. None of the cases were because of cat bites, but it is still important that people be very careful when handling potentially infected cats. Within the past year, *Francisella tularensis* was isolated from seven cats at the KSU Diagnostic Lab. In addition, we had several cases where only formalin-fixed tissues were received, but the cats had gross and microscopic lesions that we only see in cases of tularemia.

Tularemia is an acute bacterial infection of many avian and mammalian species, including dogs, cats and humans. It occurs throughout the northern hemisphere. *Francisella tularensis* is a small, gram-negative rod. Its two main biovars are A and B. Type A is associated with a tick-rabbit cycle, while type B is more complex, involving rodents, ticks, mud and water. Both strains have been isolated from cats. Various tick species can serve as both reservoir and vectors for tularemia. There appear to be four main species in the United States that are the primary vectors: *Dermacentor andersoni* (wood tick), *D. variabilis* (American dog tick), *D. accidentalis* (Pacific Coast tick) and *Amblylomma americanum* (Lone Star tick). It can be passed transovarially, and infection persists for the life of the tick. All three life stages of ticks can transmit tularemia, but the adults are of most concern. The deerfly, *Chrysops discalis* also can act as vector. Rabbits and rodents are important mammalian sources for infection. Transmission occurs from hunting or eating an infected host. Infection also can be transmitted to humans by contact of wounds when dressing a carcass from an infected rabbit.

Cats appear to be susceptible to developing tularemia, while dogs appear to be relatively resistant. The incubation period is one to 10 days. Important clinical signs in cats include fever, marked depression, dehydration, icterus, oral and lingual ulcers, pharyngeal and/or mesenteric lymphadenomegaly, and palpable splenomegaly and/or hepatomegaly. The cats often have a history of recent ingestion of a rabbit or rodent. The WBC may show an elevated neutrophil count with a left shift or it could show a panleukopenia. The neutrophils often show toxic changes. At KSU-VMTH we have noted that most of these cats have a decrease in their ALK. Gross necropsy often reveals hepatomegaly and/or splenomegaly. Regional or generalized lymph nodes are typically enlarged and contain multiple foci of necrosis. Small, military, whitish foci of necrosis are often found on the spleen, liver, and lungs. The heart may sometimes also be found with these foci.

Treatment

Treatment involves supportive care and appropriate antibiotics. It is important to remember that tularemia is NOT susceptible to penicillins. Enrofloxacin (Baytril) has

done well at the labeled cat dose of 5 mg/kg given once every 24 hours. Some veterinarians prefer to dose it at a higher amount; however, ocular toxicity is a potential sequelae. Baytril also has the advantage of coming in both injectable and oral forms. (This can be important later on when trying to decide which form of the medication

the owners can get into the cat with the least amount of risk to themselves.) If you suspect tularemia, it is best to start treatment with an antibiotic to which it is susceptible because it can be several days to weeks before you can get a definitive diagnosis. Definitive diagnosis can be done by paired serum titers, PCR on serum or ticks removed from the cat, or by cultures of liver/spleen. Serum samples for titers can be sent to Oklahoma State University to Dr. Becky Morton or to the CDC in Fort Collins, Colo. The CDC

does PCR on ticks and serum. Cultures need to be done on special media and by laboratories equipped with adequate biosafety equipment. Cultures can be submitted to Kansas State University Diagnostic Lab or to Dr. Morton. One should know that not all infected cats will develop a titer. I had a case of two cats that were seen feeding on the same rabbit. They both exhibited the same symptoms/bloodwork. One was clinically more ill than the other. Both were treated with the same therapy. The sicker of the two had a rising titer in two weeks. The other never did develop a titer. Another suspect cat had PCR positive ticks removed from it. The cat was negative on paired titers. The last case was not a proven tularemia case, but it was highly suspicious.

It is important to remember that tularemia is a zoonotic disease. Humans can be infected with as few as 100 organisms. Cat-associated cases are usually associated with bites or scratches. Accidental needle sticks from contaminated needles are a real occupational hazard with this disease as is inhalation by laboratory workers. When treating a known or suspect cat, it is best to limit the number of people who will have contact with it. This is equally important for the hospital personnel as well as the people in the cat's home environment. It is not known exactly how long a cat is infectious after treatment has started. It would be wise to treat it as being contagious until the entire treatment course is finished. This is approximately two weeks. It is important to warn the owners of the zoonotic potential so they may let their physicians be aware that tularemia should be on their differential list should that owner become ill. The incubation period for humans is two to 10 days. Humans have two main syndromes: ulceroglandular and typhoidal. Infection starts with an acute onset of high fever, chills and other "flu-like" symptoms. A skin ulcer at the site of entry and regional lymphadenomegaly are associated with the ulceroglandular form. Pneumonia is more characteristic of the typhoidal form. Mortality can exceed 30 percent if left untreated.

With rising tick populations, there also appears to be an increase in the number of feline tularemia cases. Be on the lookout for this disease so the cat can be treated more quickly and effectively, and owners can be cautioned of its zoonotic potential.

Important clinical signs of tularemia in cats include fever, marked depression, dehydration, icterus, oral and lingual ulcers, pharyngeal and/or mesenteric lymphadenomegaly, and palpable splenomegaly and/or hepatomegaly.

Trich on the rise in Kansas beef herds?

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Trichomoniasis (*Tritrichomonas foetus*) appears to be on the rise in Kansas. Increasing numbers of beef herds are coming up with reproductive problems that have proven to be caused by trich infection. Trich classically causes early embryonal death, which is manifest clinically as repeat breeding and poor pregnancy results with many late-bred or open cows. Likewise, calving will be spread out over a longer period of time resulting in wider than expected range in calf sizes. Trich is venereally-transmitted, with bulls being persistent carriers. Cows typically clear the infection within three months after infection and then go on to conceive. Mature bulls are typically more of a problem than younger bulls due to increased preputial wrinkling, which provides a better environment for growth of the organism.

One possible explanation for the increase in trich cases may be related to the drought in 2002 which forced many cattle to be sold out of drought-stricken areas such as eastern Colorado. Colorado has had trich problems for many years, to the extent that Colorado law requires trich testing to be performed on all non-virgin bulls sold within the state. Some carrier bulls may have been shipped to Kansas without being tested during the

drought.

Another possible explanation may be the increasing use of leased bulls or partnership bulls, where a beef producer can lease or share a higher quality bull (genetically-speaking) than he/she can normally afford to buy. If these bulls are not tested for trich before being moved from operation to operation, they can easily spread the disease.

Testing for the presence of the trich organism involves sampling the bulls. Preputial scrapings or washes are the sample of choice. The Diagnostic Lab at K-State¹ prefers samples to be submitted in a transport medium system, InPouch™ *Tritrichomonas foetus* Test². These pouches can be purchased directly from the company, or a very limited number can be purchased from the lab. (Please call for current pricing.) Please read and follow directions included with the pouches. These organisms prefer an anaerobic environment, so be sure to eliminate all of the air from the pouch. Label the pouches clearly, store upright (in a shirt pocket or plastic cup) and at room temperature. Please ship the pouches to the diagnostic lab overnight so they will arrive on a weekday. (Samples seem to disappear when they arrive on a weekend.)

Because it normally takes a week of incubation time in the lab before the sample can be called positive or negative, testing

requires planning and good communication. The lab will contact you with results if the test can be read before the normal incubation period is up, but owners should be warned not to plan to ship bulls the day after the samples are taken.

Young, unseasoned bulls may be infected with an intestinal trichomonad that is similar in appearance to the venereal one. We are fortunate to have a definitive PCR test available at K-State that will sort these out. Almost all positive tests in seasoned bulls turn out to be the venereal variety.

There is a very similar trichomonad that infects the intestinal tracts of a very small number of cats. Many scientists believe that this and the bovine venereal trich are exactly the same organism. Others think they are different. One thing you can tell the owner for certain – his bull did not get trich from the barn cats; he got it from infected cows.

References

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Chronic wasting disease identified in New York deer

The New York State Department of Environmental Conservation (DEC) recently announced that chronic wasting disease (CWD) was diagnosed for the first time in captive and wild deer in New York State. In 2005, chronic wasting disease was identified in white-tailed deer from two captive herds in Oneida County, New York. On April 8, 2005, the DEC announced that they had completed testing of the two herds, and that they had identified five CWD-infected deer. Because of the two infected captive deer herds, the New York DEC initiated intensive monitoring of wild deer in surrounding areas. As of May 24, 2005, the DEC has announced the identification of two wild white-tailed deer that were CWD positive. Both samples were collected in Oneida County, within a mile of each other. The first positive sample was from a yearling deer, and the second positive was from

a three-year-old doe. Both samples were identified as positive by the state's veterinary diagnostic laboratory at Cornell University. The positive result for the first sample was verified by the National Veterinary Service Laboratory (NVSL), Ames, IA. The results from NVSL for the second sample are still pending. More information concerning CWD in New York is available at: www.dec.state.ny.us/website/dfwmr/wildlife/deer/currentcwd.html

To date, CWD has been identified in captive deer in the following states and provinces (the number of infected herds is in parentheses): Colorado (12), Wyoming (1), Montana (1), South Dakota (8), Nebraska (4), Kansas (1), Oklahoma (1), Minnesota (2), Wisconsin (5), New York (2), Saskatchewan (40), and Alberta (3). Many of the herds in which CWD was found have been depopulated. CWD has been identified in

wild deer in the following states and provinces: Colorado, Wyoming, New Mexico, Utah, Nebraska, South Dakota, Wisconsin, Indiana, New York, and Saskatchewan. Additional information and links to other sources of information concerning CWD is available from the Chronic Wasting Disease Alliance, which is available online at: www.cwd-info.org/index.php

Ulcerative enteritis deadly for quail

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Ulcerative enteritis (quail disease) is one of the most common causes of high mortality in quail submitted to the KSU Veterinary Diagnostic Laboratory. Young quail raised in game bird facilities may experience mortality up to 100 percent within a few days. Quail are the most susceptible to ulcerative enteritis, but natural infections have been found in grouse, domestic and wild turkeys, chickens, partridge and pheasants. An outbreak has been reported in robins, indicating that birds other than those within the order *Galliformes* (poultry) can be infected.

Ulcerative enteritis of quail is caused by *Clostridium colinum*, a sporulating anaerobic bacterium that is resistant to harsh environmental conditions and chemicals. Quail at four to 12 weeks of age are most susceptible, although the disease occurs in adult quail. Outbreaks commonly occur after successive groups of quail or chickens have been raised on the same premises, and the premises are permanently contaminated after an outbreak.

Infected quail have very characteristic gross lesions, consisting of white spots in the small and large intestine that vary from pin-

point up to 5 mm diameter, visible through the serosal surface of the intestine (Figure 1). The spots are foci of mucosal necrosis that occasionally penetrate through the wall of the intestine to the serosa, resulting in peritoneal adhesions. Quail also may have multifocal necrosis in the liver, with variably-sized white foci randomly scattered through the liver lobes (Figure 2). Liver lesions probably occur from showering of bacteria from the intestinal lesions. The spleen often is enlarged and reddened. Coccidiosis is the most important differential diagnosis that needs to be considered, and birds may be infected with coccidia and *Clostridium colinum* simultaneously.

Diagnosis of ulcerative enteritis is made on gross and microscopic findings. Culture of *Clostridium colinum* from the lesions is confirmatory, but may not be necessary because the gross and microscopic lesions are distinctive. If liver lesions are present, culture of the liver is preferred over intestine because contamination by other intestinal flora is reduced.

Streptomycin, bacitracin, penicillin, lincomycin, and tetracyclines are listed as antibiotics that can be added to drinking water or feed for treatment of ulcerative enteritis, or as preventative if environmental contamination cannot be controlled.



Figure 1. Intestine from a quail with ulcerative enteritis. Note the distinct white to yellow foci of necrosis that are visible on the serosa.



Figure 2. Liver from a quail with ulcerative enteritis.

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