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

Nitrate Intoxication in Cattle

by G.A. KENNEDY, F.W. OEHME, J.A. PICKERELL
Kansas State Veterinary Comparative Toxicology Laboratory and Diagnostic Laboratory

Cattle grazing a central Kansas stalk field in March are breathing rapidly and show some signs of weakness. Occasionally, they drop while walking to drink from their watering tank. They drop so rapidly, sometimes minutes after signs are noticed, that your client thinks it appears they're getting too much exercise. 10% of them are down and two have died. Both the dead animals and those remaining down and hyperventilating have darkened mucous membranes with a muddy-brownish cast to them. The blood sampled from those down is brownish-black; it does not brighten (redden) when gently mixed with an equal volume of air.

In a second situation in late summer, 4 of 150 cows are dead with dark mucous membranes, agonal petechiae and ecchymoses, while others were asymptomatic. The following morning, an additional cow is hyperventilating.

In another instance in November, 5 of 50 cows are acutely involved; 3 of 50 die rapidly with darkened mucous membranes and 2 of 150 abort. These cows were fed mixed grass forage with 7,100 ppm nitrate as submitted (28,400 ppm nitrate as dry weight).

Each of these cases is an example of nitrate toxicity. The pastures or fields should be examined closely by county agents and veterinarians looking for plants such as redroot pigweed, lambs quarter, fireweed (kochia weed), beets, jimson weed, thistle or smartweed. In addition, cereal grains such as sorghums (sudan, sudex or Johnson grass), corn, oats, millets, ryes, or sunflowers can concentrate nitrate under the right circumstances. These plants concentrate nitrate in their stems[1, 3]. Not all plants concentrate enough nitrate to become a threat to ruminants consuming them. For example, some grasses concentrate almost no nitrate and may have levels as low as 50 ppm nitrate. However, crop plants bred to produce high quantities of protein pick up nitrate-nitrogen with their roots. This nitrate may be in the soil as excess chemical fertilizer, manure or as oxidized ammonia from previously applied anhydrous ammonia fertilizer. Phenoxy herbicides, such as 2,4-D, can increase palatability in plants and, thus, the consumption of nitrates[1].

Plants that grow more slowly than intended may continue to accumulate nitrate at normal rates and store it in vesicles in their stems for later use. Plants growing more slowly because of stress during conditions of drought or reduced temperatures accumulate nitrate-nitrogen. After prolonged drought, soil nitrate may be washed deeper into the soil where it is readily available to the plant roots. Newly sprouted *sorghum* plants will continue to rapidly concentrate the nitrate-nitrogen, but coolness may delay the plant's use of it; thus, nitrate can become concentrated in the stems of new regrowth.

Ruminant ingestion of forage nitrate makes it possible for rumen microbes to reduce it to nitrite. Upon absorption into the blood, nitrite oxidizes ferrous (+2) iron of hemoglobin to ferric (+3) producing methemoglobin. Only animals with fully functioning rumens develop methemoglobinemia from dietary nitrates. Direct ingestion of nitrite produces methemoglobinemias in all animals. Methemoglobin will neither carry oxygen nor revert back to hemoglobin following exposure to oxygen. Methemoglobin is brownish-black, accounting for the same color in blood[2].

Different levels of methemoglobinemia will produce different clinical signs. Individuals holding their breaths usually have 5%

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BRD continued from page 1

methemoglobin; 15% methemoglobinemia causes a slight brownish cast to blood drawn into a syringe. However, cows and calves will not begin to hyperventilate noticeably until they have about 30% methemoglobinemia. At 40-60% methemoglobin, cattle will hyperventilate noticeably and may collapse if exercised. Even walking to drink from a stock tank or pond can be sufficient exercise to cause collapse. Because of hyperventilation, their mouth dryness and metabolism increase the animal's desire to drink. Cattle with 65-70% methemoglobinemias are more likely to die acutely[3].

To make certain that we are dealing with plant-induced methemoglobinemias, nitrate toxicity must be differentiated from sodium chlorate toxicity, caused by the granular herbicide applied to county "right-of-way" roads and causing methemoglobinemia and significant gastrointestinal pain. We also must differentiate acute death from nitrate from other causes of rapid death in the bovine; for example cyanide intoxication where the cow's blood is often bright red or *taxus* (yew) poisoning where rumens contain significant visible quantities of the evergreen-like plant. Additional causes to differentiate include urea with a high rumen pH, grain overload with a low rumen pH, hypomagnesemia from lush green pastures[1], and various infectious diseases whose organisms can be cultured.

The clinical toxicologist's most useful test to confirm ruminant nitrate intoxication is measurement of forages high in nitrate (>3,000 ppm nitrate may interfere with production or growth, while >15,000 ppm nitrate is sufficiently high to produce acute nitrate intoxication). If submitting forage from a stalk field, submit the lower portion of the plant or stalk and not the leaves. Testing mostly leaves or upper portions of the plant could give a false negative result. Our test at The Comparative Toxicology Laboratories, Kansas State University Diagnostic Laboratory, costs $15 and is completed the day samples are received. The ensiling process for silage reduces nitrate content by as much as 50%. Nitrate is soluble in water and will settle to the bottom of an upright silo, so that the nitrate content of the bottom portion of silage may be as much as 5-fold higher than the nitrate content in the silage at the top.

Rumen nitrate has been suggested as an indication that animals consumed high levels of forage nitrate. Although specific in the sense that it provides evidence of animal exposure, it may be insensitive diagnostically because rumen bacteria rapidly reduce forage nitrate so it is no longer measurable as nitrate. High levels of nitrate in the eye aqueous humor of cattle (>30 ppm) provides evidence of animal exposure and accumulates in animals consuming high forage nitrates[2]. Our test for aqueous humor nitrate costs $10 and is run the day the fresh eye fluid is received.

Veterinarians treat ruminant methemoglobinemia with 4-30 mg of methylene blue/kg body weight intravenously as a 1 % solution. This treatment may be repeated after 2-3 hours if signs persist. Methylene blue reduces the +3 iron of methemoglobin to the +2 iron of hemoglobin. It brings blood methemoglobin to about 10%, a level consistent with good health. Cattle should be rechecked the following day to determine the need for further treatment[1-3].

The prognosis is good if cattle not showing overt signs are gently moved to new pasture, or if those having signs of toxicity are treated with methylene blue and not stressed. If affected cattle are not noticed and treated, or have signs and are forced to move, the likelihood of a favorable outcome is significantly reduced.

Veterinarians should educate their clients that cattle consuming forage with 500-3,000 ppm nitrate will likely have no clinical signs. At higher forage levels, 20,000 ppm nitrate for example, blending such high nitrate forage with forage known to have safe concentrations of nitrates (<3,000 ppm nitrate), and supplementing the animals' diet with vitamin A and iodine, will reduce the likelihood of developing clinical methemoglobinemias.

Nitrate comes from the soil. Plant parts nearer the soil are likely to have higher nitrate concentrations[2]. Conditions that stress or reduce the rate of plant growth will cause high concentrations of nitrate to accumulate. Rain after a drought may cause regrowth of sorghum plants that have potential to accumulate nitrate[1-3]. We are available at the Comparative Toxicology Laboratories, Kansas State University Diagnostic Laboratory, to provide consultation with such cases. VQ

Further Readings

Aiello S, Mays A: 1998. Nitrate and nitrite poisoning. IN *The Merck Veterinary Manual 8th Edition*, National Publishing, Philadelphia, PA, p 2091-2094.

Haliburton JC; 1999. Nitrate poisoning associated with the consumption of forages or hay. IN Howard JL, Smith RA eds, *Current Veterinary Therapy 4 Food Animal Practice.* WB Saunders Co, Philadelphia PA, p 278-279.

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Report from the Food Animal Health and Management Center

In January 2000, the Food Animal Health and Management Center will begin its sixth year of operation. At its founding, the Center was envisioned to generate new knowledge in food animal health and management. The mission of the Center has been to solve problems of the food animal industry by building interdisciplinary research teams. The Center's faculty has secured funding from federal, state and private resources to provide an environment for academic excellence and an opportunity to conduct leading-edge research in food safety, advanced diagnostic methodologies and production management. USDA-CREES and USDA-FSIS funding has provided researchers the opportunity to study the ecology of E. coli O157:H7 in cow/calf operations and wildlife populations, determine the existence and extent of drug resistant microbial populations in feedlots and to define the prevalence of Salmonella in culled dairy cattle. From these projects, the Center's researchers have developed the methodology and diagnostic capabilities to efficiently collect, propagate and characterize pathogens that can seriously jeopardize food safety in products of microbiologists, pathologists, animal

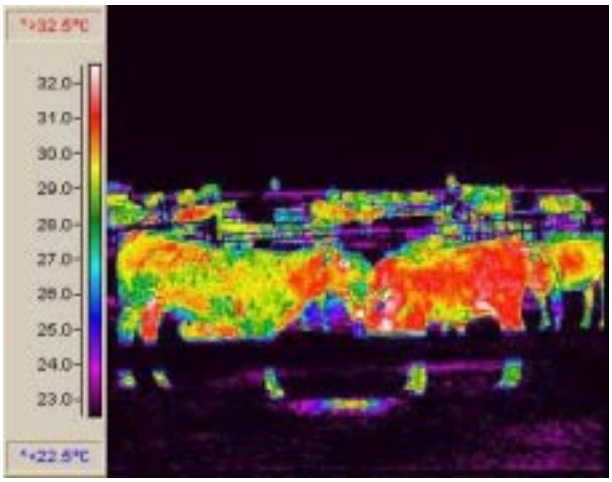

Figure 1.

scientists, nutritionists, agricultural engineers, agricultural economists, practicing veterinarians, and producers to develop and evaluate management tools that significantly impact production efficiency and animal welfare in the swine and beef industries.

An example of the Center's research activity is the use of infrared thermal imaging to predict the well being of feedlot cattle. This research initiative is being lead by Dr. Mark Spire who has been in the Center since its inception. For the past 4 years his research has focused on the diagnostic uses of infrared thermography and its applications in livestock management. As illustrated on page 2, infrared images can be captured chute side or cattle can be remotely monitored for radiant energy exchange rates in their natural settings (Figure 1). High and low energy expenditures are associated with the metabolic well being of an animal and are influenced by its health, nutritional and environmental status.

As the Center moves into the new millennium it will remain committed to enriching the environment in the College of Veterinary Medicine at Kansas State University for collaborative research for the food animal industry. VQ

New Diagnostic Test to Detect Erythrocyte Associated Antibody in Horses and Dogs With Immune Mediated Hemolytic Anemia

MELINDA J. WILKERSON

The Clinical Immunology/Flow Cytometry Laboratory at the College of Veterinary Medicine, Kansas State University, has available a sensitive and specific method to determine the classes of antibody bound to erythrocytes in horses and dogs with immune mediated hemolytic anemia (IHMA). This test is a direct immunofluorescence (DIF) assay that uses flow cytometry and fluorescein isothiocyanate (FITC)-conjugated antibodies against immunoglobulin (Ig) classes (IgM, IgG, IgA) and C3 to detect erythrocytes coated with Ig or C3. We have established background levels of antibody binding in samples from 12 normal horses and dogs and compared the DIF test to the direct anti-agglutination Coombs' test in patient's with IMHA (Tables 1 & 2—page 4). The former assay is more sensitive in dogs with IMHA than the Coombs' test (100% vs. 66%). A negative Coombs' test does not rule-out immune-mediated hemolytic anemia. Several factors may reduce sensitivity of the assay and cause false negative results including: insufficient quantity of antibody or complement on erythrocytes, improper antiglobulin-to-antibody ratio, not incorporating the offending drug into the test, corticosteroid treatment greater than one week duration, and improper or warm temperatures which disperse cold agglutinins. The DIF assay detected low levels of bound antibody (<11%) in two canine patients determined to be negative by the Coombs' test (Table 1). For both species, performance of the DIF test is independent of the prozone effect, a common cause for false negative results in agglutination and precipitation based tests. The Coombs' test is based on detection of agglutination or clumping of erythrocytes that have surface bound immunoglobulin following addition of an anti-species polyvalent mixture of antibody to IgM, IgG, and complement protein C_3. Serial dilutions of the polyvalent Coombs' reagent are prepared and tested against patient erythrocytes to provide the proper concentration equivalence between anti-globulin reagent and the antibody-coated erythrocytes at which agglutination occurs. To sort out warm (typically IgG) versus cold (IgM or IgA) reacting agglutinins, a duplicate set of the reactions are prepared and read following incubation at 4° C and 37° C. The prozone effect or lack of agglutination occurs at low dilutions of Coombs' reagent, the point at which the antibody concentration is excessive and does not agglutinate or precipitate antigen. The DIF test readily identifies erythrocytes coated with antibody without the necessity of serial dilutions of FITC-conjugated antibody. This is best illustrated in Table 1 (equine case #1) and Table 2 (canine cases #1, #2, and #4).

We have identified three IMHA dogs with surface IgG and IgM, two dogs with IgG, and one dog with IgM (Table 1). Two equine cases with IMHA had surface bound IgG, a horse with suspected penicillin-induced IMHA and a foal with neonatal isoerythrolysis (Table 2). Because of the quantitative nature of the DIF assay, we can use it to monitor the response to therapy. For example, a reduction in surface IgG was detected over the course of blood

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Production data analysis course

Enroll in the "Statistical Analysis in Production Medicine" self-study course through a combination of at-home study and informal workshops. Drs. Steve Dritz and Gerald Stokka will be hosting an informal discussion group workshop every other month at a Kansas location. The workshop will be in conjunction with the Statistical Analysis in Production medicine certificate series. Several experts in the areas of Epidemiology and Statistics will provide the at-home study videos. Enclosed is a brochure and registration form with further details. All you need to do is sign up and we will contact all Kansas participants with the location and time of the Kansas discussion group. We look forward to the active participation of practitioners willing to improve their quantitative analytic skills. For further information contact Dr. Dritz (785-532-4202 or dritz@vet.ksu.edu) or Dr. Stokka (785-532-5694 or jstokka@oz.oznet.ksu.edu)

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www.vet.ksu.edu/depts/itc/cvmce.htm.
For more information or to request a brochure, contact:
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e-mail johnson@vet.ksu.edu,
Veterinary Medical Continuing Education, College of Veterinary Medicine, Kansas State University
or Veterinary Extension
www.oznet.ksu.edu/pr_vetext

Test continued from page 3

transfusions and supportive therapy in the foal with neonatal isoerythrolysis. Neonatal isoerythrolysis develops after high titer maternal antibody against the neonatal foal's red blood cell antigens is passed through the colostrum, initiating an acute episode of hemolysis. The foal was given 5 liters of washed packed red cells from the dam over a period of 8 days following admission. There was a greater than 2 fold decrease in the proportion of erythrocytes coated with surface IgG by Day 8 of treatment (34% red blood cells had surface bound IgG compared to 100% at the time of presentation), and a further 2 fold decline 7 days later (17%). The reduction in IgG on the surface of foal erythrocytes corresponded with a steady increase in hematocrit from 18% to 24% by discharge (Day 15). One week after discharge the foal had no detectable IgG on the surface of its erythrocytes and normalization of hematocrit.

This test is performed in conjunction with the direct Coombs' test for a total charge of $25. Send fresh whole blood collected in EDTA anti-coagulant on ice by overnight mail. Please direct any questions to Dr. Melinda J. Wilkerson, DVM, PhD at 785-532-4818 or 785-532-4617, Dept. Diagnostic Medicine/Pathobiology, 1800 Denison Ave. Manhattan, KS. 66506. VQ

Table 1: Canine Patient Cases; Clinical Summary and Comparative Test Results

Breed	age sex	Hct%	Clinical parameters Dx = Diagnosis	Coombs	Dilution* 4° C	DIF# 37° C
Dog 1. Dachsund	5 yr	11	Chronic regenerative anemia, responsive to prednisone, transfusion, azathioprine, Dx: Primary IMHA	Neg	1:32	IgG-35%
Dog 2. Irish Wolfhound	4 yr MN	14	Lethargy, exercise intolerance, dyspnea, ANA neg, regenerative anemia, expired the day after testing Dx: Primary IMHA	Neg	1:16	IgG-88% IgM-4%
Dog 3. Miniature Schnauzer	9 yr FS	11	Nonregenerative anemia, spherocytosis, negative tick titers died from Rt. Sided heart failure, splenic hemosiderosis Dx: IMHA	Neg	Neg	IgG-7%
Dog 4. Maltese	8 yr MN	7	Chronic anemia, spherocytosis, responsive to prednisone, transfusion, azathioprine Negative tick titers Dx: Primary IMHA	1:64-1:128	1:64-1:128	IgG-28% IgM-38%
Dog 5. Dachsund	8 yr FS	9	Chronic regenerative anemia, spherocytosis, ANA+ responsive to prednisone Dx: Primary IMHA	Neg	Neg	IgM-10%
Dog 6. Cocker Sp.	7 yr FS	12	Regenerative anemia, responsive to prednisone and transfusion, negative tick titers Dx: Primary IMHA	1:2-1:64	1:2-1:64	IgM-11% IgG-5%

*The dilution or range of dilutions of Coombs' reagent at which agglutination occurred.
#Mean percentage of erythrocytes labeled with isotype specific antibody.

Table 2. Equine Patient cases; Clinical Summary and Comparative Results.

Breed	Age Sex	Hct%	Clinical parameters response to treatment Dx = Final Diagnosis	Coombs 4° C	Dilution* 37° C	DIF#
Eq 1. Quarter	12 yr MC	12	acute hemolytic anemia, responsive to discontinued administration of penicillin and dexamethasone Dx: drug induced IMHA	1:128	1:64-1:128	IgG-96%
Eq 2. Warm blood	2 days F	18	acute hemolytic anemia thrombocytopenia, post suckling–responsive to transfusion with maternal packed cells Dx: Neonatal isoerythrolysis	1:2-1:128	1:2-1:128	IgG-100%

*The dilution or range of dilutions of Coombs' reagent at which agglutination occurred.
#Mean percentage of erythrocytes labeled with isotype specific antibody.

Rabies: Prevention and Control

DR. DEBORAH J. BRIGGS
DIRECTOR RABIES LABORATORY CVM

Although rabies is prevalent in Kansas year round, a seasonal increase in the number of confirmed positive cases occurs in the spring. Since the 'rabies season' is rapidly coming upon us, it is a good time to review the current protocols for preventing the spread of this dread disease. There are several definitions to keep in mind as you read this article: An 'exposure' occurs when the saliva from a rabid mammal enters into an open cut, wound or mucous membrane of a human or other animal. Simply petting a rabid mammal or being in the same area as a rabid mammal is not considered to be an exposure. The term 'wild mammal' refers to any mammal that is not a dog, cat, ferret or considered to be livestock. Additionally, a wild mammal is any mammal that has descendents within the wild mammal population (this includes wolves and wolf-hybrids).

The basic points of rabies prevention can be separated into three parts: (1) What to do with a mammal that exposes a human, (2) What to do with a mammal that was bitten by a rabid mammal and (3) How veterinarians and their assistants can protect themselves against contracting rabies.

1. What to do with a mammal that exposes a human: If the exposure occurred from a wild mammal, that mammal should be humanely destroyed and tested as quickly as possible for the presence of rabies virus. If the mammal that exposed the human was a healthy dog, cat or ferret, the vaccination history should be reviewed. A healthy dog, cat or ferret that is current on its rabies vaccination may be closely observed for a ten day period. If the dog, cat, or ferret is healthy at the end of the ten-day period, the person exposed is not in any danger of contracting the disease from the exposure. The ten-day period is recognized as a safe observation window because any dog, cat, or ferret that was rabid at the time of an exposure will sicken or die prior to the end of ten days. It is important to remember that the ten-day observation period is only applicable for dogs, cats, and ferrets. This is because the shedding period for rabies virus in wild mammals is unknown and, therefore, rabies virus may be present in the saliva of these animals for longer than ten days prior to presentation of clinical signs of rabies. In the case of exposure to livestock, ie horses, cattle or sheep, the circumstances of the exposure should be taken into consideration. Each incident should be investigated on a case by case basis, taking into consideration vaccination history, type of exposure (provoked or unprovoked), and the current health of the animal.

2. What to do with a mammal that was bitten by a rabid mammal: If the bitten animal is current on its rabies vaccination, it should be immediately re-vaccinated and carefully observed for 45 days. The local health officer will decide the circumstances under which the animal should be observed. Generally if the owner is responsible, the observation can take place at the owner's residence. An animal is considered currently vaccinated if it received a USDA rabies vaccine licensed for use in that species. For example, a wolf or wolf-hybrid vaccinated with a canine vaccine is not considered currently vaccinated because there are no canine rabies vaccines licensed for use in wolves or wolf-hybrids. If the animal received only one rabies vaccination, the vaccine should have been administered at least 30 days and not more than 12 months prior to the exposure. If the bitten animal was never vaccinated, it should be humanely destroyed. It is important to remember that in Kansas, each community has its own regulations regarding the number of years a rabies vaccine is considered valid. On the other hand, the USDA recognizes a three-year rabies vaccine as being valid for three years in animals that have received at least one rabies vaccine previously. This causes some confusion as to when a rabies vaccination is considered out of date. Therefore, if there are any questions concerning whether an animal is current on its rabies vaccination, contact Dr. Gail Hansen, the State Public Health Veterinarian in Topeka Kansas.

3. How veterinarians and their assistants can protect themselves against contracting rabies: Veterinarians and veterinary assistants that handle animals should receive pre-exposure rabies vaccination. Every two years they should have their serum tested to determine the level of rabies virus neutralizing antibodies present. If the level of neutralizing antibodies falls below 1:5, one routine booster vaccination should be administered. There is no need to have an additional serologic test after the booster is given. If an exposure to a rabid animal occurs, all exposed persons that have received pre-exposure rabies vaccination should receive two intramuscular doses of rabies vaccine. One dose is given on day 0 and a second is given three days later. Again, there is no need to conduct serologic testing after the booster doses are administered unless the person has an immunosuppressive condition. If an exposed person has never received rabies vaccination, they should receive five intramuscular doses of rabies vaccine, one each on days 0, 3, 7, 14, and 28. Additionally, they should receive one dose of Human Rabies Immune Globulin (HRIG) given at the same time as the first dose of vaccine. The HRIG should be administered (as much as feasible) into the wound area away from the anatomical site that the rabies vaccine was administered.

Lastly, I would like to discuss a few changes that were published in the Rabies Compendium 2000. First, the issue of rabies vaccination in wolves and wolf-hybrids is currently being reviewed and a decision regarding USDA licensing will likely be made before the end of 2001. Secondly, although serologic testing is generally required for dogs and cats being imported to rabies free areas of the world, it should not be used as a substitute for rabies vaccination. This is because there is no level of rabies virus neutralizing antibodies considered 'protective' in animals. Third, there are new rabies vaccines licensed for use in 8-week-old puppies and kittens. These vaccines have met the requirements by the USDA for licensure and can add extra protection to young animals. If you have any questions regarding the efficacy of these vaccines, you should contact the manufacturer. VQ

The Relationship Between the Presence of Helicobacter pylori, Clostridium perfringens type A, Campylobacter spp, or Fungi and Fatal Abomasal Ulcers in Unweaned Beef Calves

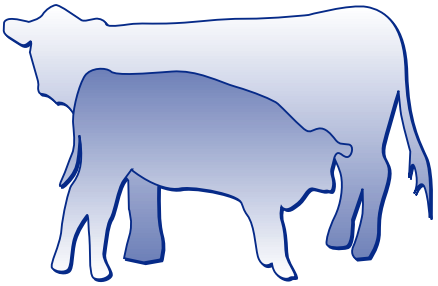
M.D. JELINSKE, C.S. RIBBLE, M. CHIRINO-TREJO, E.G. CLARK, E.D. JANZEN
THE CANADIAN VETERINARY JOURNAL
1995 JUNE;36(6):379-82

A case-control study involving 30 unweaned beef calves was conducted to determine whether specific species of bacteria or fungi were associated with fatal abomasal ulcer formation. Special microbiological and histological techniques were used to detect Clostridium perfringens type A, Helicobacter pylori, or Campylobacter spp. It has been speculated that these bacteria are potential ulcerogenic agents of unweaned beef calves.

Calves were recruited for the study at necropsy, with those dying of either a perforating or a hemorrhagic ulcer representing the cases, and calves of a similar age dying of a disease unrelated to the abomasum representing the controls.

Helicobacter pylori was not visualized in, or cultured from, any of the abomasal tissue samples. Clostridium perfringens type A was isolated from 78.6% of the cases and 75% of the controls. These isolates were further dichotomized into "heavy" and "light" growth; no significant association was found between ulcers and the amount of growth. A light growth of Campylobacter spp. was recovered from 3 cases and 3 controls.

There was no compelling evidence to suggest that Clostridium perfringens type A, Helicobacter pylori, or Campylobacter spp. were involved in ulcer formation. VQ



Kansas State University
Sensititre Epidemiological Survey
Date Range: 07/01/98 to 06/30/99

Bovine: E. coli; beta-hemolytic

Antibiotic	Interpretations (as %) Suscept.	Interm.	Resist.
NCCLS Group A - Approved Antibiotics			
Ampicillin	50	-	50
Ciftiofur	50	-	50
Erythromycin	-	-	100
Penicillin	-	-	100
Sulfachloropyridazine	50	-	50
Tetracycline	50	-	50
Tilmicosin	-	-	100
Tylosin	-	-	100
NCCLS Group B - Selective Group			
Enrofloxacin	100	-	-
NCCLS Group C - Extra Label			
Amikacin	100	-	-
Gentamicin	75	-	25
Tribissen	50	-	50

Bovine: Haemophilus somnus

Antibiotic	Interpretations (as %) Suscept.	Interm.	Resist.
NCCLS Group A - Approved Antibiotics			
Ampicillin	77	15	8
Ciftiofur	96	-	4
Erythromycin	85	15	-
Penicillin	85	-	15
Sulfachloropyridazine	85	-	12
Tetracycline	-	92	8
Tilmicosin	96	-	4
Tylosin	77	4	15
NCCLS Group B - Selective Group			
Enrofloxacin	96	4	-
NCCLS Group C - Extra Label			
Amikacin	100	-	-
Gentamicin	96	-	-
Tribissen	88	-	8

Bovine: Pasteurella haemolytica

Antibiotic	Interpretations (as %) Suscept.	Interm.	Resist.
NCCLS Group A - Approved Antibiotics			
Ampicillin	79	-	18
Ciftiofur	99	-	-
Erythromycin	7	84	9
Penicillin	13	-	82
Sulfachloropyridazine	37	-	55
Tetracycline	63	7	28
Tilmicosin	90	3	6
Tylosin	6	-	93
NCCLS Group B - Selective Group			
Enrofloxacin	97	3	-
NCCLS Group C - Extra Label			
Amikacin	93	1	1
Gentamicin	93	4	1
Tribissen	94	-	4

Bovine: Pasteurella multocida

Antibiotic	Interpretations (as %) Suscept.	Interm.	Resist.
NCCLS Group A - Approved Antibiotics			
Ampicillin	93	-	7
Ciftiofur	98	1	-
Erythromycin	1	74	25
Penicillin	1	-	98
Sulfachloropyridazine	20	-	79
Tetracycline	61	13	26
Tilmicosin	78	8	13
Tylosin	6	11	83
NCCLS Group B - Selective Group			
Enrofloxacin	98	2	-
NCCLS Group C - Extra Label			
Amikacin	75	22	3
Gentamicin	82	8	8
Tribissen	82	-	18

Bovine: Salmonella sp.

Antibiotic	Interpretations (as %) Suscept.	Interm.	Resist.
NCCLS Group A - Approved Antibiotics			
Ampicillin	43	-	57
Ciftiofur	70	2	27
Erythromycin	-	-	100
Penicillin	-	-	100
Sulfachloropyridazine	20	-	77
Tetracycline	39	-	61
Tilmicosin	-	-	100
Tylosin	-	-	100
NCCLS Group B - Selective Group			
Enrofloxacin	100	-	-
NCCLS Group C - Extra Label			
Amikacin	95	-	5
Gentamicin	95	2	2
Tribissen	77	-	23

Heat-Stress Related Abnormalities Seen on Spermiograms

G. REED HOYOAK, DVM
OKLAHOMA STATE UNIVERSITY
DEPARTMENT OF VETERINARY CLINICAL SCIENCES

In recent weeks we have seen a higher than expected number of bulls receive a "deferred status" on their breeding soundness examination as well as a few stud dogs with similar spermiograms. The abnormalities have mainly been detached heads, proximal cytoplasmic droplets, acrosomol defects, diadem defects, other nuclear vacuoles, pyriform heads, and severely coiled tails. In some of the males, we have seen a greater number of abnormal forms of sperm than in other males, with some having one predominant abnormality and others having more. All of these defects can be associated with heat stress and/or other forms of stress in the male. This includes all domestic species, not just the bull. Additionally, young peripubertal males often have a high proportion of proximal cytoplasmic droplets. In many of these cases, the defects will no longer be detected after a few weeks to a couple of months due to the fact that spermatogenesis takes 60 days to occur in the bull, with an additional 8 to 14 days in the epididymis. Times for spermatogenesis range from 39 days in the boar to 62 days in the dog, with all of them having 8 to 14 days transit time in the epididymis. The spermiogram is a historical snapshot of what was happening at the time of spermatogenesis in the testis, and to a degree, during maturation, transit and storage within the epididymis. This is the case in all of the domestic species with which we are involved.

Richard Saacke and others have documented that specific sperm defects will appear following mild thermal insult of the testis through evaluation of scrotal temperature in bulls. After only 48 hours of scrotal insulation, an increase in cytoplasmic droplets was observed within a week. Increasing numbers of decapitated heads followed these and then nuclear vacuolation defects, pyriform heads and finally acrosomol defects by 3 to 4 weeks post-insulation. Similar results were seen with insulation applied to the neck of the scrotum. This has application to excess fat in the inguinal area and scrotum and to inguinal hernias. Additionally, modifications of testicular function through either disturbance of heat regulation or stress-related endocrine disturbances would result in a change in the spermiogram. For example, in addition to obesity, extreme climatic heat, inflammation of the scrotum from frostbite, dermatitis, or trauma, fever from illness, vericoceles, and hematoceles can all interfere with temperature regulation within the scrotum.

In a separate study using similar techniques, significant seminal differences were found between semen samples for cryopreservation collected from bulls prior to and after heat stress. Even though sperm collected 3 to 9 days post-insulation did not appear significantly different from that collected prior to insulation, there was a significant post-thaw difference in both progressive motility and the proportion of intact acrosomes.

It has been noted that spermatozoa are very much like a biopsy of the seminiferous epithelium that affords us insight to the health of the testis. The spermiogram can give us information which, when combined with a complete history and the male's physical condition, may suggest specific causes of testicular malfunction, possible therapeutic plans, and the potential for recovery to normal spermatogenesis. The evaluation of an abnormal spermiogram often will raise several hard to answer questions. Having a thorough understanding of normal and abnormal spermatogenesis lends to your ability to provide the best evaluation and prognosis for potential fertility. Even though the "decision deferred" category is primarily intended for the peripubertal male with poor semen quality, it may also be appropriated for the mature male with a history of recent stress from which recovery is expected. VQ

Bovine Leukosis Virus

G.L. STOKKA DVM, MS

Recently, there have been numerous questions about Bovine Leukosis Virus and the accompanying disease, lymphosarcoma. In consultation with several experts in this area, including Dr. Ed Dubovi and Dr. Chris Rossiter from the College of Veterinary Medicine at Cornell University, I have put together a list of facts concerning this disease.

1. The virus is not highly contagious as compared to other viruses, (e.g. IBR).
2. The test (AGID vs ELISA) is not the important issue, but test positive means virus positive. The real threat is those animals that are test negative but virus positive; however, these are likely very few in number.
3. In large dairies in New York, the prevalence has increased from 30% to 60% because management procedures are being ignored which reduces transmission. The greatest risk occurs through the use of common needles, tattoo instruments, dehorning, castrating tools, ear tag pliers and tags, etc.
4. Economic impact is primarily through carcass condemnation, clinical disease and some infertility.
5. With a positive bull, semen can be used with basically no risk, the risk of transmission from natural service is real but, again, probably small.
6. Approximately 20% of calves born to positive cows will be positive and usually is associated with high lymphocyte counts in the positive animal.
7. There is no evidence that wildlife can be a reservoir or transmit the virus. Insects (biting and sucking) probably can transmit the virus, although the amount of transmission that occurs this way may not be very great.
8. The risk of transfer from adjacent herds (horizontal transfer) to negative cattle (recipients or otherwise) is low. In addition, the risk then to calves of the negative animals is likely even less.
9. Frequency of testing helps to identify positives and helps to identify when transmission occurs and, thus, perhaps the cause of transfer.
10. Vaccines at this stage are experimental only. VQ

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Coming Events

February 19, 2000
8th Annual Emergency Medicine
Conference on Neurology
Guest speaker: Dr. Curtis Dewey
Texas A&M University

February 20, 2000
Small Animal Internal Medicine
Continuing Education Series, Spring 2000
Endocrinology

March 5, 2000
17th Annual Frank W. Jordan Seminar
on Feline Infectious Diseases
Guest speaker: Dr. Michael Lappin
Colorado State University

March 11, 2000
Veterinary Technicians Conference
Behavioral Problems
Heartworm Giardia Lab
Equine Wound Treatment

March 12, 2000
Small Animal Internal Medicine
Continuing Education Series,
Spring 2000
Parasites

April 9, 2000
Small Animal Internal Medicine
Continuing Education Series,
Spring 2000
Geriatric and Neonatal Diseases

June 4-7, 2000
62nd Annual Conference
for Veterinarians

October 28-29, 2000
9th Annual Mid-Western Exotic Animal
Medicine Conference
Guest speaker: Dr. Kathy Quesenberry
Animal Medical Center, New York City

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