

VETERINARY

FOR THE KANSAS PRACTICING VETERINARIAN

Quarterly

January-April 1998
Volume 1, Number 1

Welcome to the first issue of the Kansas Veterinary Quarterly published by the Kansas State Diagnostic Laboratory and K-State Research and Extension.

To better serve our readers, we have changed our format and added some members to the team. Contributors from the Kansas State Diagnostic Laboratory will now report in each issue on topics relevant to practitioners in Kansas and the surrounding region. In addition, we want KVQ to be an avenue for veterinarians in private practice to publish their articles as well. Our goal is to provide information to Kansas veterinarians that they might not otherwise receive, so we will continue to provide research summaries from the K-State College of Agriculture, diagnostic notes from the Diagnostic Laboratory and abstracts from publications normally not available to private practitioners.

This newsletter is for you—we encourage your input on the subjects you would like to have addressed in future issues.

Contact us with your thoughts and suggestions:

K-State Research and Extension

Phone (785) 532-5694

Web: http://www.oznet.ksu.edu/dp_ansi

Kansas State Veterinary Diagnostic Laboratory

Phone (785) 532-5650

Web: <http://www.vet.ksu.edu/pathology>

A Toxic Dietary Problem in Cattle

By JOHN A. PICKRELL
AND FREDERICK W. OEHME
Diagnostic Medicine/Pathobiology

The following is not an unusual field case for cattle practitioners: It is a Monday in early spring, and you are called out because 80 of some 2,000 feedlot cattle have died over the past 24 hours. The calves are mixed breed, mostly steers, 550 to 700 pounds. All vaccinations are current. The cattle have been in the lot for 30 to 45 days and are being fed mixed grains, mixed tall grass hay, and a protein and mineral concentrate that has 25 percent of the protein equivalent as urea. As of last Friday, average daily gain and feed conversion were in the top third of your feedlot clients. As you head out to the lot, here are some things to think about . . .

Urea—Ammonia Toxicosis: This is most commonly observed in ruminants with microorganisms whose urease metabolize urea to ammonia. Ammonia is rapidly absorbed from the rumen leading to hyperammonemia, bloat, dribbling of urine, ataxia, nervous signs, convulsions and rapid deaths. Cattle on full feed who then “miss” two to three feedings can lose their adaptation to urea. For example, cattle without feed for 24 or more hours can tolerate less than one-third the urea fed when fully adapted. Urea toxicoses can also result from miscalculations of the urea placed in feed, inadequate mixing or consumption of a urea-protein-mineral block instead of grain. The diagno-

sis of urea intoxication is greatly strengthened by detecting a rumen pH above 8.

Ionophore Overdoses: Monensin (Rumensin), Lasalocid (Bovatec, Avatec), Salinomycin (Biocox), Narasin. Toxicities occur from cattle consuming higher than recommended amounts from unmixed or improperly mixed concentrates, producing anorexia, depression, reluctance to move, dyspnea, leg weakness, ataxia and finally death. At necropsy, pale areas of skeletal and cardiac muscles and pulmonary and peritoneal edema are seen. Monensin may interact with zearalenone to heighten estrogenic reactions and reduce bull fertility.

Lactic Acidosis: Grain overload, feeding “hot carbohydrates” (flaked corn/maltose, sucrose, lactose or glucose), or soluble proteins or amino acids induces this condition. Rumen production of propionic acid shifts to lactic acid, and as the rumen pH lowers (frequently into the 5.0 to 6.0 range) a lactic acidemia is produced. Signs occur 6 to 24 hours after engorgement and include weakness, rumenitis, bloating and diarrhea. It is not unusual for losses to continue for 3 to 4 weeks. Associated liver abscesses, laminitis or rumen parakeratosis may be present.

Gossypol Toxicosis: Cottonseed meal or cottonseed residues (cotton burrs) are cheap sources of protein and major sources of gossypol. Adult cattle tolerate 800 to 2,000+ ppm gossypol, but calves

Continued on Page 2

BIV Affects Cattle Industry

By HARISH MINOCHA

Diagnostic Medicine/Pathology

Bovine immunodeficiency virus (BIV), a lentivirus, was first isolated from a dairy cow with lymphocytosis, lymphadenopathy, neuropathy and progressive emaciation. Structurally, it reassembles human immunodeficiency virus (HIV) and has more than 40 percent genetic homology with HIV. The infection is prevalent worldwide. A limited serological survey data showed that 15 to 50 percent cattle may have been exposed to BIV. At Kansas State University's College of Veterinary Medicine, we have used recombinant BIV gag protein to test sera of naturally and experimentally infected animals by Western blot assay and polymerase chain reaction (PCR) of blood sample to detect the virus. Approximately 18 percent of cattle in Kansas were positive for BIV.

Evaluation of immune function of cattle experimentally infected with BIV demonstrated transient decrease of CD4/CD8 ratio during weeks 2 to 7 after infection suggesting a possible immune dysfunction. The infected cattle had decreased antibody titers when vaccinated with bovine viral diarrhea virus vaccine. This suggested BIV-exposed animals may not fully respond to the vaccination process. In infected animals, the virus was predominately detected in the brain and lymphoid tissues by PCR in situ hybridization techniques.

In summary, cattle herds may have a number of animals exposed to BIV. These animals may be low responders to vaccines because of some immune dysfunction due to BIV infection. This may lead to vaccine breaks in a herd. Further survey of cattle herds showing vaccine breaks needs to be conducted to determine implications of BIV infection in cattle.

Fat Necrosis and Calcification Causes Carcass Discounts

Reports of carcass discounts have surfaced due to a condition called by inspectors as calcification of fat. Calcification of fat occurs when fat undergoes necrosis. The fat breakdown products combine chemically with calcium, potassium or sodium to form a soap. The soap is in the fat cell and is not dissolved with fat solvents that are used in the staining and sectioning technique. Grossly, the material becomes opaque, whitish, and solid and may calcify.¹

Cases of fat necrosis in cattle have not been described in relationship to hypothermia. However, there are reports in humans of this condition.² It is likely this condition occurred as a result of cold

injury due to the cold weather and snowstorm that occurred at the end of October in the High Plains area. Cattle most susceptible to this condition would be those with thinner hides, typically the dairy crosses and *bos indicus* crosses. This condition should not be considered as detrimental to the carcass, it may be noted but not discounted.

¹*Veterinary Pathology, Smith, H.A., Jones, T.C., Hunt, R.D., 4th ed., pp. 22.*

²*Duhn, R., Schoen, E.J., Siu, M., Subcutaneous Fat Necrosis with Extensive Calcification after Hypothermia in Two Newborn Infants, Pediatrics 1968 Mar; 41(3):661-664.*

A Toxic Dietary Problem in Cattle *from Page 1*

may be sensitive to 400 ppm or less gossypol. The condition usually requires 2 to 3 weeks to develop, but much of it is silent with cattle "suddenly dying." Animals will have sporadic labored breathing, occasional hemoglobinuria, and death at or near market weight if stressed. Because of its polyphenolic nature, gossypol will bind free essential amino acids such as lysine. Diluting out gossypol with other protein sources may prevent this toxicosis. The condition affects feed efficiency, the liver, gastrointestinal tract and heart.

Some diagnostic thoughts: In the field case presented, urea intoxication would be likely if the rumen pH were 8 or greater and lactic acidosis if the rumen pH were very acidic. Ionophore toxicity can be assayed for in feeds and should also be differentiated in the feedlot from lead toxicity, sudden death syndrome with myocardial necrosis, and vitamin E deficiency (differentiated by dietary assay, or vitamin E responsiveness). Gossypol toxicosis can be confirmed by the source of protein and quantitation of gossypol in the ration. Any gastrointestinal

signs should be differentiated from entero- and endo-toxemia by fecal culture and from plasma endotoxin, especially in young calves by endotoxemia's persisting diarrhea. The digestion toxemias and ionophore overdose are important concerns in calves 30 to 45 days into feeding.

In this case, the feed had appropriate amounts of urea as non-protein-nitrogen. There had been no interruption in feeding. The feed protein was not from cottonseed and no gossypol was in the feed. Necropsy of three dead calves revealed large collections of thoracic and abdominal fluids, a pale streaked heart, pale sections of the rear leg extensor muscles and excess fluid oozing from the cut surface of lungs. Rumensin was present in the grain mix at recommended levels, but the mix was contaminated with quantities of a macrolide antibiotic. The macrolide antibiotic interfered with rumensin metabolism and produced a classic ionophore toxicity. No new cases developed after the contaminated grain mixture was replaced with freshly prepared concentrate.

Immune Function Tests

By MELINDA J. WILKERSON

Diagnostic Medicine/Pathology

The College of Veterinary Medicine at Kansas State University has an Immunology/Flow Cytometry Laboratory that serves as a reference laboratory for a variety of immunology tests. These tests are performed for the practitioner who needs diagnostic results that supplement and confirm clinical immune dysfunction. Many of the tests that are performed in this laboratory point to immune mediated or autoimmune diseases as a basis for immune dysfunction.

The following tests are offered through this laboratory.

(1) A direct Coombs test identifies immune mediated hemolytic anemia in anemic dogs, cats, and horses induced by warm and cold antibodies. One mililiter of whole blood in EDTA is required, sent on ice overnight delivery. The results are available the day of arrival. (\$20/sample)

(2) Anti-nuclear antibody test identifies anti-nuclear antibodies in the serum of dogs, cats, and horses supportive of a diagnosis of autoimmune disease. A serum sample is required sent either on ice or frozen. (\$15/sample). This test should be supplemented with clinical history, signs, and other more specific tests. For example, skin biopsies will help assess the presence of an autoimmune skin disease such as pemphigus, rheumatoid factor in the dog if arthritis is a clinical sign, direct Coombs test for immune mediate hemolytic anemia, and tests to evaluate the presence of infectious diseases (i.e. Equine infectious anemia, Ehrlichia titers, Rocky Mountain Spotted Fever titers).

(3) Rheumatoid factor test is specific for canine rheumatoid factor identified in serum samples. (\$15/sample)

(4) We can test canine platelets for the presence of platelet surface associated IgG (PSAIGG). Elevated numbers of platelets coated with IgG have been identified in dogs with immune mediated

thrombocytopenia. This test does not distinguish between primary ITP (autoimmune) or secondary ITP induced by drugs, neoplasia, or infectious diseases (Ehrlichia). Platelet counts, mean platelet volumes, and reticulated platelets are also determined in conjunction with PSAIGG. These additional procedures improve the interpretation of the PSAIGG test.

The reticulated platelet test is a new test that we are currently evaluating. It identifies young platelets or reticulated platelets in the circulation, indicative of platelet regeneration by the bone marrow. This test supplements, but does not replace a bone marrow assessment of thrombopoiesis. All of the tests associated with the PSAIGG package require 13 mililiter of whole blood in EDTA sent on ice overnight delivery. The results are available on the day that the sample is received. (\$50/sample)

(5) Direct immuno-fluorescence assays are available for demonstration of IgG bound to bone marrow elements in dogs, cats and horses. Identification of bound IgG on marrow stem cells would support the immune mediated destruction of bone marrow elements. Multiple (at least 2) unfixed, unstained bone marrow smears are needed for this test. (\$15/sample)

(6) Radioimmunoassay (RIA) assays are available to determine the quantity of IgG, IgM, IgA, IgT in equine serum, IgG in llama and feline serum, IgG, IgM and IgA in canine serum, IgG in bovine and porcine serum. Ig RIA assays are helpful in determining failure of passive transfer and primary (genetic deficiencies, i.e. Combined immunodeficiency) or secondary immunodeficiencies due to infectious processes, agents, or neoplasia. Serum samples are required, sent on ice or frozen. (\$30/sample)

If you have any questions contact Wilma Shuman or Dr. Melinda Wilkerson at 785-532-4617 or 785-532-4818.

The Kansas State Veterinary Diagnostic Laboratory was established in 1961. Today, the laboratory is fully accredited by the American Association of Veterinary Laboratory Diagnosticians and has a staff of more than 60 dedicated employees.

Our goal is to provide high-quality diagnostic and consultation services to the veterinary profession and animal industries of Kansas and surrounding states. We are continually striving to develop and maintain state-of-the-art techniques that are responsive to the changing needs of veterinarians and the animal industry.

If you have questions regarding laboratory testing, available tests, fees, results, etc., please feel free to call the central diagnostic lab number: 785-532-5650.



Dr. Sanjay Kapil, from The Virology Laboratory, Kansas State Diagnostic Laboratory has recently expanded the services provided to Kansas Practitioners to include the following diagnostic tests. For more information contact Dr. Kapil at 785-532-4457 (e-mail kapil@vet.ksu.edu).

Bovine Rotavirus, an enzyme linked immunoassay (ELISA) to detect the virus in fecal specimens (1 to 2 grams [g]/animal; **\$8/sample**).

Bovine Coronavirus, an ELISA to detect the virus in fecal specimens (1 to 2 g/animal; **\$8/sample**).

Canine Parvovirus, an ELISA on fecal specimens (1 to 2 g/animal; **\$8/sample**).

PCR (polymerase chain reaction) for detecting porcine reproductive and respiratory syndrome (PRRS) virus in serum (**\$25/sample**).

Shrink Management in Cattle

By THOMAS R. FALKNER D.V.M.

K-State Research and Extension, Veterinary Medicine

Management and marketing are the two factors that have the greatest influence on beef enterprise profitability. Level and quality of management are the greatest single factors in ranch profitability, but as-tute marketing is the area most often overlooked by cattlemen. Marketing includes the buying and the selling of cattle; management is everything in between these stages. Utilizing “shrink” to the cattlemen’s advantage in both the purchase and sale of livestock will result in several dollars per head additional profit. The best thing about shrink management is that it is free. Many cattlemen are uncomfortable with other marketing management tools (i.e., futures and options), but shrink management can be easily mastered and implemented by anyone.

Definitions

Shrink: Shrink is the term used to describe the loss of weight in livestock between two consecutive weightings. The majority of the shrink seen in our cattle marketing and transportation systems is due to the withholding of feed and water—the loss of “gut fill” through the excretion of urine and feces. Most of this shrink is regained quickly once cattle are filled up. The remaining shrink is called “tissue shrink” and may take several days to recover.

Pencil Shrink: Pencil Shrink is a term used in cattle purchasing contracts to describe the process by which the cattle are weighed and then a certain percentage of that weight is subtracted before figuring the price. This calculated weight is often called the “pay weight”: $\text{Actual weight} - \text{Pencil Shrink} = \text{Pay Weight}$.

Most cattle are bought and sold on a price per pound basis. Shrink, whether “actual” or “pencil,” results in fewer pounds sold and, therefore, fewer dollars received. Research has shown that most of the shrink seen in cattle is due to the manure (60 percent) and urine (38 percent) excreted by the animal—commonly called “gut fill.” Time off feed and water is by far the biggest influence on the amount a particular animal shrinks. However, any activity that

raises an animal’s level of excitement increases shrink via increased urination and defecation. The math is pretty simple: manure or urine on the ground does not show up on the scales. Many cattlemen will fight tooth and nail for 25 cents per hundred pounds on selling price and then handle or contract cattle in a way that costs several dollars per hundred, maybe more. This is not good marketing.

Table 1 attempts to break down and combine the research data available on shrink so one can realistically compare different marketing scenarios. By smart trading on both ends, cattlemen can often increase profits by \$20 to 40 per head or more.

Using this table will give a fairly reliable estimate of shrink for a given set of cattle and allow the economic comparison of different buying or selling contracts. Of course, other factors such as initial “fill” of the cattle, time of year and disposition of cattle will also have an effect. If dealing with freshly weaned calves, figure them to shrink just as if feed and water were withheld the first 24 hours. Sharp buyers and sellers use actual shrink, pencil shrink, and other “tricks” as a marketing edge. For example, a buyer who sorts through the cattle several times before weighing them “steals” the shrink. Conversely, if all cattle are weighed before sorting and, after sorting, the weight of the “out” cattle is subtracted—full price will be paid for the cattle plus the cattlemen pays for the shrink of the “out” cattle. Also, buyers often convince cattlemen to contract cattle in a way that results in substantial actual shrink plus a pencil shrink.

The annual cost of inefficient cattle catching, sorting and loading facilities is

Table 1: Factors affecting shrink (<24 hr period)

Factor	% Shrink	\$/100lb *	\$/head *
a Ease cattle to scales	0%	0	0
b → or 30 minute “round up”	1.0%	\$0.80	\$4.00
c Load, haul (<100miles), unload, weigh	+2.5%	\$2.00	\$10.00
d → + sort and/or wait extra hour before weighing	+1.0%	\$0.80	\$4.00
e 12 or more hours without feed or water	+2.5%	\$2.00	\$4.00
f → + Haul additional 500 or more miles	+2.0%	\$1.60	\$8.00
g Weigh on trailer (with pan) long haul	(-1.0%)	(-\$0.80)	(-\$4.00)
h Weigh on trailer (with pan) short haul	(-1.5%)	(-\$1.20)	(-\$6.00)

* Dollar values assume a 500-lb calf @ \$80/100 lb

To use the table, go down the list of factors and add together all those that pertain to the cattle in question. Using the above figures, cattle loaded at the farm and shipped 800 miles, unloaded and weighed would be expected to shrink 7% (c + e + f). The same cattle weighed on the truck would be expected to shrink 5.5% (c + e + f - g)

evident. These same inefficient facilities often cause other management problems. Interestingly, cattle bought through salebarns will often weigh more when shipped by an order-buyer three to four days later than they did when bought. This is due to poor cattle handling, resulting in large shrinkage prior to weighing. Veterinarians can use discussion of “shrink cost” with clients as an additional incentive to justify better cattle handling facilities and practices.

References:

Cole, NA, Camp TH, Rowe LD, et al. 1988. Effect of transport on feeder calves. *Am J Vet Res*, Vol 49, No. 2 178-183.

Asplund, JM, Mayes HF, Anderson ME, et al. 1982. Effect of transportation, handling, and environment on slaughter cattle. I. Weight loss and carcass yield. *Univ. of MO-Columbia Res Bull.* 1048.

Ribble CS, Meek AH, Shewen PE, Jim GK, Guichon PT. 1995. Effect of transportation on fatal fibrinous pneumonia and shrinkage in calves arriving at a large feedlot. *J Am Vet Med Assoc* 207 (5):612-615

Tarrant PV, Kenny FJ, Harrington D, Murphy M. 1992. Long distance transportation of steers to slaughter: effect of stocking density on physiology, behaviour and carcass quality. *Lvstk Prod Sci* 30 (1992) 223-238

Tarrant PV. 1990. Transportation of cattle by road. *App An Behav Sci*, 28 (1990) 153-170.

Banjaw K. 1979. Effect of transportation on body weight loss and muscle pH of cattle. *Ethiopian J Ag Sci* Vol 9, No. 2 115-125.

Bovine Leukosis Testing

The test kits that the diagnostic laboratory has been using for bovine leukosis, which uses an AGID format, will no longer be available and the laboratory has only a limited number of test components and reagents left.

Because of this, the Diagnostic Laboratory will be switching to an ELISA format test in the near future. This ELISA test kit is designed for running larger numbers of sera at a time and is not well suited for running small numbers of sera per run. Therefore, we will probably run bovine leukosis tests once or twice a week, which may delay your turnaround time depending on when the sample(s) arrive at the laboratory. We will no longer be able to routinely have results available the next day after arrival.

The cost per test will remain the same, at least temporarily, at \$5/sample. Volume discounts will be available for greater than 25 samples. Please call ahead regarding volume discounts and ask for either Dr. George Kennedy or Mrs. Sylvia Osborne.

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

Human Exposure to *Brucella abortus* Strain RB51—Kansas, 1997

On May 26–27, 1997, nine persons (a farmer, four veterinary clinicians and four veterinary students) in Manhattan, Kansas, participated in an attempted vaginal delivery, a cesarean delivery and a necropsy on a stillborn calf that died because of *Brucella abortus* infection. The infection was confirmed by isolation of *B. abortus* from placental and fetal lung tissue cultures. The National Animal Disease Center, United States Department of Agriculture (USDA), identified the *B. abortus* isolate from the calf as the RB51 vaccine strain. RB51 is a live, attenuated strain that was licensed conditionally by the Veterinary Services, Animal and Plant Health Inspection Service, USDA, on February 23, 1996, for vaccination of cattle in the United States.* Before 1996, vaccine was made by using the S19 strain. This report describes occupational exposure to animals infected with the RB51 strain and emphasizes the need for surveillance of unintentional exposure of humans to RB51 to assess outcomes of such exposures.

The vaccine had caused active *B. abortus* infection because the 14-month-old heifer delivering the calf was not known to be pregnant when she was vaccinated with RB51 at approximately 8 months of age, which was within the specified age range for vaccination. The heifer was administered the RB51 vaccine dosage recommended for adult or pregnant cattle.

The heifer was euthanized after surgery because of the poor prognosis following a uterine rupture and the poor general condition of the animal. Necropsy findings included diffuse placentitis in the heifer and fetal pneumonitis. Evidence that intrauterine infection was caused by the RB51 vaccine strain, and not by field strains of *B. abortus* or by S19, included immunohistochemical staining specific for RB51 (negative for S19), RB51-specific titer of >1:10,000 on experimental dot-blot assay measuring antibody to RB51, and RB51-specific DNA sequences identified by polymerase chain reaction (PCR).

Persons at risk for infection with RB51 were those who contacted the calf, placenta, blood or amniotic fluid without wearing gloves, masks or eye protection. Six women and three men (age range: 23 to 45 years) were at risk for infection. None of the exposed persons reported having previously had brucellosis or being unintentionally inoculated with *Brucella* vaccine.

Within one week after exposure, eight of the nine persons started a prophylactic regimen of doxycycline (100 milligram twice daily for 21 to 24 days). Three of these persons also received rifampin (600 milligram once daily for 4 to 21 days). None of the exposed persons showed signs or symptoms consistent with brucellosis during the six-month follow-up period.

Since conditional licensure of the RB51 vaccine, 32 instances of unintentional inoculation or conjunctival exposure to the RB51 vaccine have been reported to the vaccine manufacturer or CDC. Three of the 32 persons, all of whom were unintentionally inoculated while vaccinating cattle, reported inflammation at the inoculation site; another person reported intermittent fever, chills, headache and myalgia and had elevated levels of serum transaminase and lactate dehydrogenase.

Reported by: B. Stauffer, Pottawatomie County Health Department; J. Reppert, MD, Lafene Health Center; D. Van Metre, DVM, R. Fingland, DVM, G. Kennedy, DVM, Kansas State University, Manhattan; G. Hansen, DVM, G. Pezzino, MD, State Epidemiologist, Kansas Department of Health and Environment; S. Olsen, DVM, National Animal Disease Center, Agricultural Research Service; D. Ewalt, PhD, Animal and Plant Health Inspection Service, United States Department of Agriculture; Meningitis and Special Pathogens Br, Division of Bacterial and Mycotic Diseases, National Center for Infectious Diseases, CDC.

*The vaccine was licensed conditionally to allow accumulation of additional data on field use under controlled conditions.

COOPERATIVE EXTENSION SERVICE
U.S. DEPARTMENT OF AGRICULTURE
KANSAS STATE UNIVERSITY
MANHATTAN, KANSAS 66506-3403

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, \$300

Coming Events

April 18, 1998

Veterinary Technicians Conference

June 7-10, 1998

60th Annual Conference for Veterinarians

The Kansas State University Diagnostic Laboratory and Department of Animal Sciences and Industry at Kansas State University greatly appreciates the sponsor(s) of the Kansas Veterinary Quarterly Newsletter. These sponsorships in no way imply the Departments' endorsement of the products and services offered by the sponsors. The Departments welcome inquiries from other individuals, associations and firms that may be interested in cosponsoring this publication.



Newsletter Coordinators

Gerald Stokka
Extension Specialist, Beef Veterinarian
785-532-5694 • jstokka@oz.oznet.ksu.edu

Thomas R. Falkner
785-532-1213 • rfalkner@oz.oznet.ksu.edu

G.A. Kennedy
785-532-4454 • kennedy@vet.ksu.edu

Contributors—K-State Research and Extension

Dale Blasi Gerry Kuhl
Frank Brazle Twig Marston
Dick Dunham John Smith

Contributors—Veterinary Diagnostic Laboratory

G.A. Andrews B.W. Fenwick F.W. Oehme
R.J. Basaraba S. Kapil J.A. Pickrell
D.J. Briggs K.S. Keeton R.K. Ridley
M.M. Chengappa W.E. Moore P. Schoning
M.C. DeBey D.A. Mosier C.D. Seedle
M.W. Dryden J.C. Nietfeld J.E. Smith
M.J. Wilkerson

**Cooperative Extension Service
K-State Research and Extension**
131 Call Hall
Manhattan, KS 66506

KSU, County Extension Councils and U.S. Department of Agriculture
Cooperating.

All educational programs and materials available without discrimination on the
basis of color, race, religion, national origin, sex, age, or disability.