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Dr. George Kennedy retires

Larry C. Hollis, DVM
Extension beef veterinarian

After serving Kansas State University for more than three decades, Dr. George Kennedy has decided to seek other employment in the form of the proverbial "honey-do" list that goes along with retirement. His last day as director of the Kansas State University Diagnostic Laboratory will be July 16.

Kennedy has a long and distinguished record at K-State. He is best known for his work at the Veterinary Diagnostic Laboratory where he has served for the past 26 years. A gifted pathologist, Kennedy has seen numerous new diseases emerge, old diseases defined and differentiated, and changes in the way veterinary medicine is practiced in the field and clinic. He has been influential in ensuring that K-State's diagnostic capabilities evolved with the needs of the future. He has shaped the careers of many graduate students and veterinary pathologists during his tenure. As co-editor and regular contributor to the Veterinary Quarterly, he prompted his fellow diagnosticians to contribute as well.

Kennedy is the friendly face that first comes to mind when we think of the Diagnostic Lab. We will miss his personal touch along with his professional ability and seasoned wisdom. Everyone who had the privilege of working with him and talking with him on the phone will surely miss the characteristic "goooodbye" at the end of our conversations. Well done, George!

Equine becoming less susceptible to WNV

Molly McCue
Resident, equine internal medicine
K-State College of Veterinary Medicine

West Nile virus (WNV) was first recognized in the United States in New York in 1999. Since its introduction, the virus has spread across the country to more than 40 states. WNV is an endemic disease of the United States, and will maintain that status for an extended time. West Nile virus became important to veterinary practioners in this area in 2002. In 2002, more than 700 cases were confirmed in Kansas and more than 1,200 in Nebraska. Additionally, the IgM seroconversion (exposure) rate in nonclinically affected horses was approximately 27 percent. In 2003, West Nile virus was more sporadic because of factors including natural immunity, vaccination protocols and environmental influences on mosquito populations. Natural immunity and vaccination protocols should decrease the susceptibility of the equine population and help avoid outbreaks seen in 2002. Because WNV is endemic to the Midwest, continued equine vaccinations are necessary.

> In 2002, more than 700 cases of West Nile virus were confirmed in Kansas.

Two West Nile Virus vaccines are available for horses. Innovator, a killed whole virus vaccine made by Fort Dodge, was the first fully licensed vaccine. Merial has recently produced Recombitek, a modified-live canary

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Be on alert for vesicular stomatitis

Vesicular stomatitis (VS), a foot-and-mouth disease (FMD) look-alike, has been found in nine locations in Texas and four in New Mexico this summer. VS occurs primarily in horses, cattle, pigs, and sheep. As the name implies, VS causes vesicle formation, primarily around the oral cavity. Vesicles are seen only early in the course of disease, while erosions and ulcers are more frequently observed. These oral lesions usually result in profuse salivation and anorexia. Lesions also may be found around the coronary bands and on teats, sometimes causing profound lameness.

Because VS is indistinguishable from FMD, it is a federally reportable disease. Please contact Dr. Kevin Varner, USDA Area Veterinarian-in-Charge, at 785-235-2365 (*kevin.p.varner@aphis.usda.gov*) or George Teagarden, Kansas Animal Health Department, at 785-296-2326 (*gteagard@ink.org*) if you see vesicular disease in livestock.

Also in this issue

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

Applied Reproductive Strategies in Beef Cattle Conference

A conference titled, "Applied Reproductive Strategies in Beef Cattle" is set for Sept. 1-2 at the Sandhills Convention Center in North Platte, Neb. The meeting is hosted by the North Central Region Bovine Reproductive Task Force and features outstanding speakers. Veterinarians can obtain up to 14 hours of CE credit for attending this meeting.

Wednesday, September 1

7:30 a.m. Registration
8:00 Welcome – Rick Funston
8:10 Follicular growth and the estrous cycle – *Freddie Kojima*
8:50 PGF2a in estrous synchronization: History, efficacy and utilization *Jim Lauderdale*
9:20 Estrous synchronization systems: GnRH – *Darrel Kesler*
10:00 Estrous synchronization systems: MGA – *Dave Patterson*
10:40 Break
11:00 Estrous synchronization systems: CIDR – *Cliff Lamb*
11:40 Questions for morning speakers
Noon Lunch
1:00 p.m. Fertility of natural vs synchronized estrus – *George Perry*
1:40 Timing of vaccinations in estrous synchronization programs – *Richard Randle*
2:10 Cost and comparisons of estrous synchronization systems – *Sandy Johnson*
2:45 Break
3:05 Puberty and anestrus: dealing with non-cycling females – *Jeff Stevenson*
3:45 Nutrition and reproduction interactions – *Rick Funston*
4:20 Supplementation and weaning strategies to optimize reproductive performance – *Don Adams*
5:00 Questions for afternoon speakers
5:15 Producer and industry panel: Utilizing advanced reproductive technologies

Thursday, September 2

7:30 a.m. Registration
8:00 Welcome – *Darrell Nelson, dean and director, UNL Ag Research Division*
8:15 Breeding soundness exams and beyond – *Peter Chenowith*
9:00 Insemination-related factors affecting fertilization in estrous-synchronized cattle – *Richard Saacke*
9:45 Ubiquitin as an objective marker of semen quality and fertility in bulls – *Peter Sutovsky*
10:30 Break
10:55 Industry application of technology in male reproduction – *Mel DeJarnette*
11:40 Questions for morning speakers
Noon Lunch
1:00 p.m. Ultrasound – early pregnancy diagnosis and fetal sexing – *Cliff Lamb*
1:20 Reproductive tract scoring – *Dave Patterson and Richard Randle*
1:40 Semen quality assessment – *Peter Chenowith*
2:00 Break
2:30 Veterinarian panel: Utilizing advanced reproductive technologies
4:00 Adjourn

Accommodations

Sandhills Hotel & Convention Center
I-80 & Hwy 83, North Platte, NE
(800) 760-3333
www.sandhillcc.com

Mention the name of the conference when reserving rooms. Reserve rooms by Aug. 3 for special rates. Deluxe sleeping rooms are $59.95 and two-room suites are $89.95.

Registration Form

Applied Reproductive Strategies In Beef Cattle
September 1-2, 2004

Name _____

Company Affiliation _____

_____

Address _____

_____

City, State, ZIP _____

_____

Daytime Phone Number _____

E-mail Address _____

_____

Registration for

Wed., Sept. 1 on or before Aug. 16	$100	_____
Thurs., Sept 2 on or before Aug. 16	$100	_____
Wed. and Thurs., Sept. 1-2 on or before Aug. 16	$150	_____
Registration after Aug. 16 add late fee	$25	_____
TOTAL ENCLOSED		_____

Registration fee includes lectures, proceedings, lunch and refreshment breaks.

Refunds, minus a $25 handling charge, will be given if notification is received by Aug. 27, 2004.

Make check payable to: University of Nebraska
Return form and payment to:
Rick Funston
University of Nebraska
West Central Research & Extension Center
461 W University Dr.
North Platte, NE 69101

Beware of summer plant hazards to livestock

Frederick W. Oheme and John A. Pickrell
Veterinary Diagnostic Toxicology Laboratory,
Diagnostic Medicine/Pathobiology
Kansas State University

Animals, particularly those outdoors or on pasture, are constantly at risk of ingesting toxic forage or being injured from contact with plant spines or dermal irritants. Summertime presents unique environmental challenges. High temperatures, too much or too little rainfall, and the application of fertilizers, herbicides, or other agricultural chemicals can change the toxic capacity of pasture plants.

The specific hazards to animals vary depending on the plant and the amount consumed, as well as the animal species. Environmental conditions and stage of plant growth, as well as the specific genus and species of the hazardous plants also affect the level of danger. Common Midwestern plants are hazardous to livestock if consumed or touched.

Poison hemlock, European hemlock, spotted hemlock – *Conium maculatum*

Poison hemlock is toxic to humans, birds, wildlife, cattle, sheep, goats, pigs, and horses. People are usually poisoned when they mistake hemlock for plants such as yampa (*Perideridia gairdneri*), parsley (*Petroselinum crispum*), wild anise (*Pimpenella anisum*), wild carrot or Queen Anne's lace (*Daucus carota*). Livestock rarely eat hemlock because of its strong odor, but they will if no other forage is available or if it is in hay or silage. Cattle have been fatally poisoned by eating as little as 0.5 percent of their body weight of green hemlock. There is considerable variation in the toxic alkaloid content of the plant.

Signs of poisoning are similar in all species and develop within an hour of consumption. If a lethal dose has been consumed, death from respiratory failure occurs in two to three hours. First, salivation, abdominal pain, muscle tremors and uncoordination occur, followed by difficulty breathing, dilated pupils, weak pulse and frequent urination and defecation. Prolapse of the nictitating membrane across the cornea in cattle and pigs may cause temporary blindness. Cyanosis of the mucous membranes, respiratory paralysis and coma without convulsions precede death. Pregnant animals that survive the acute toxicity may abort.

Poison hemlock is teratogenic and causes abnormal fetal development if it is eaten by pregnant cows between the 40th and 70th days of gestation. Calves and piglets may be born with crooked legs, deformed necks and spines (torticollis or scoliosis) and cleft palates that are similar to deformities caused by lupines and some tobacco species. The congenital defects must be differentiated from viral infections such as BVD, blue tongue and Cache Valley virus. The teratogenic and toxic effects of poison hemlock are most severe in cattle, while sheep are the most tolerant. Mares are susceptible to poison hemlock poisoning, but foals born to mares fed conine between 45 and 75 days of gestation do not develop congenital deformities.

Because there is no treatment for hemlock poisoning, acutely poisoned animals should be given supportive treatment as necessary. If the hemlock has been recently consumed, saline cathartics and activated charcoal help remove the plant from the gastrointestinal tract. Destroying the plants by mowing or with herbicides before the seed stage reduces the risk of hemlock becoming an invasive weed and a problem to livestock.

Water hemlock, cowbane, poison parsnip, spotted water hemlock – *Cicuta maculate*

Cicutoxin (C17H22O2), a highly unsaturated diol, is one of the most toxic natural plant compounds. The toxin is concentrated in the tuberous roots, but the entire plant, including the fluid in the hollow stems, is toxic. The roots are highly poisonous at all times, and livestock that consume the root usually die. Mature plants often have a prominent root crown that protrudes above ground making it accessible to animals even in winter. The emerging plant in the spring is the most toxic, and the mature leaves in late summer have minimal toxicity to cattle. The dry stems are minimally toxic.

Humans and animals can be fatally poisoned by eating as little as 50 mg of green water hemlock per kilogram of body weight. Fatalities have occurred in children that sucked on the hollow stems of water hemlock.

Cicutoxin is a potent neurotoxin capable of causing rapid onset of muscle tremors and violent convulsions. Death often occurs in two to three hours after consuming a lethal dose of water hemlock. Excessive salivation, vigorous chewing movements, teeth grinding, frequent urination and defecation are common. Depending on the quantity of toxin absorbed, animals become ataxic and uncoordinated and develop grand mal seizures. During convulsions, animals may chew off their tongue or break bones due to the violent muscle contractions induced

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Poison hemlock in flower

Spotted stems of poison hemlock

Flowering water hemlock

continued from pg. 3

by the toxin. The signs, which may start within an hour of eating the plant or roots, progress rapidly to the convulsive seizures and lateral recumbency. Poisoned animals have dilated pupils and proceed to coma, before dying from respiratory paralysis and asphyxia. Death may occur in about 90 minutes after ingestion of a lethal dose. If animals consume a sublethal dose, they will recover if not stressed.

Sheep that die acutely from water hemlock poisoning show no gross postmortem lesions. Because of the rapid lethal effects of the cicutoxin, pieces of water hemlock root may be found in the esophageal groove and not in the rumen during postmortem examination. Multifocal diffuse myocardial degeneration is characteristic in acute poisoning. In less acute poisoning, severe seizures cause skeletal muscle degeneration. The serum enzymes lactic dehydrogenase, aspartate transaminase and creatine kinase increase in animals that have long and frequent seizures.

There is no antidote for cicutoxin. Studies in sheep have shown that intravenous administration of sodium pentobarbital at the onset of seizures prevented lethal cardiac and skeletal muscle degeneration and resulted in complete recovery. When possible, early treatment should consist of heavy sedation with sodium pentobarbital to reduce the severity of the convulsions. Laxatives may help remove the plant from the digestive system. In cattle observed eating the water hemlock roots, it may be life-saving to immediately perform a rumenotomy to remove the plant parts from the digestive system before the toxin is absorbed.

Pigweed, amaranth – *Amaranthus retroflexus*

A variety of pigweeds grow throughout North America. The most common and typical of the genus is red-rooted pigweed (*A. retroflexus*). It is a common weed found in cultivated and disturbed soils along roadsides and waste areas, and is frequent in and around corrals and animal enclosures. Plants are stout, erect, quick-growing annuals that reach 3 to 4 feet (1 to 1.2 meters) tall depending on the growing conditions. The stems are usually branched, hairy and red to purple in color. The taproot is usually bright red. Numerous shiny, black seeds are produced that ensure successful proliferation of the pigweed.

Amaranthus spp. may accumulate significant quantities of an oxalate associated with a syndrome in pigs and cattle that causes perirenal edema. Renal tubular nephrosis probably causes death of the animal. However, renal nephrosis is often present without oxalate crystals, which suggests that Amaranthus spp. may contain other undefined toxic substances. Pigweeds also accumulate nitrates and can be a threatening source of explosive nitrate poisoning.

Hairy vetch – *Vicia villosa*

Introduced from Europe as a legume for pasture improvement and as a cover crop, hairy or woolly vetch is an established weed in many areas of North America, especially along roadsides, waste areas and in croplands.

The specific toxin(s) in hairy vetch responsible for the symptoms of hairy vetch poisoning has not been determined. The generalized granulomatous disease that is characteristic of hairy vetch poisoning in cattle and horses is suggestive of a hypersensitivity (type IV) reaction induced by a foreign substance that activates the immune system response. Vetch lectins might cause the immune-mediated response that causes the granulomatous inflammatory response seen in many different tissues of the affected animal. Not all animals are susceptible to vetch hypersensitivity. The disease is more prevalent in cattle that are more than 3 years old. Although reported in many breeds of cattle, hairy vetch poisoning is more common in Angus and Holsteins. Hairy vetch poisoning occurs most often when the plant is nearing maturity and forms a major part of the diet of cattle and horses. The plant is less likely to cause a problem in hay or when ensiled. However, the mechanism by which hairy vetch induces poisoning is unclear because the plant is frequently consumed by cattle without a problem.

Hairy vetch poisoning sporadically affects adult cattle and occasionally horses and is characterized by pruritic dermatitis, weight loss, conjunctivitis and diarrhea. The dermatitis is not confined to white-skinned areas. Initially, the hair coat is rough and stands erect where lesions develop. Papules then develop that exude serum and result in superficial crusts. Continual rubbing and scratching of the lesions causes hair loss and thickening of the skin. Abortions and red urine have also been associated with hairy vetch poisoning. Lymphocytosis and hyperproteinemia are a feature of hairy vetch poisoning. Acute hairy vetch poisoning is characterized by subcutaneous swellings, ulcers of the oral mucous membranes, purulent nasal discharge and coughing, with significant mortality. Ingestion of large amounts of hairy vetch seed may induce neurologic signs and death in cattle and horses.

At postmortem examination, granulomatous lesions can be found in multiple organs including the skin, liver, kidneys, heart, spleen, lymph nodes and digestive system. Histologically, the lesions consist of monocytes, eosinophils, multinucleated giant cells and lymphocytes.

Poisonous area of water hemlock stem

Pigweed (Amaranthus retroflexus)

Hairy vetch

Latest recommendations for controlling flies on cattle

Ludek Zurek, medical and veterinary entomologist, Kansas State University

The fly season has started and fly populations are growing in many animal production facilities, including cattle farms. There are three filth fly species that are major pests of cattle: house flies (mainly in feedlots), stable flies (feedlots and pastures), and horn flies (pastures). For control, management and prevention, it is important to know what fly species is causing the problem. This article includes basic information on biology, behavior and management of these three species, and an update on new insecticides on the market.

House flies

House flies do not bite. They are a nuisance pest and can transmit foodborne and animal pathogens. They generate brown or yellow spots on the walls and other surfaces where they rest, unlike stable flies that feed on blood and create dark red or black spots. They are common from spring until late fall. House fly larvae can develop in any decaying organic substrate, such as garbage, spilled feed or manure. The development from egg to larva to adults takes only eight days under ideal conditions.

Control

Sanitation. The key to managing house flies is a good sanitation program that includes cleaning around feedbunks (spilled grain, unused hay, straw, silage), under fences and gates, and around water systems, and maintaining good drainage.

Baits. There are many brands of baits. The newest bates include QuickBayt and QuickStrike. QuickBayt (from Bayer) contains imidacloprid, sugar and two other attractants. It's available in granular form (for scatter application or as a bait) or it can be dissolved in water for paint-on application (surfaces where house flies rest). QuickStrike (from Starbar) contains nithiazine and sugar attractant. This bait is marketed as an abatement strip. Both traps act quickly and kill flies in a few minutes or less. Remember that baits do not work against stable flies and horn flies.

Residual sprays. These insecticides are sprayed where flies rest – fences, walls, and outside walls of feedbunks. Residual sprays should be effective for several weeks. They are available in several forms: wettable powder (WP), microencapsulated and emulsifiable concentrate. Examples include Tempo WP (cyfluthrin), Atroban, Ectiban, Permectrin (all permethrin), or Ravap (tetrachlorvionphos+dichlorvos).

Stable flies

Male and female stable flies bite and feed on blood of animals and people. They look similar to house flies. The main difference is the proboscis – the piercing and sucking mouthpart. They leave black or dark red spots on surfaces where they rest. Populations are usually highest in the spring, and a second, smaller peak occurs in the fall. On cattle, bites most often occur on the legs. Cattle react to stable flies by bunching and standing in water, which reduces feeding and decreases weight gain. Larvae develop in manure mixed with soil, straw or hay, as well as decaying spilled grain and fermenting grass clippings. Development from egg to larva to adult takes three to four weeks. Stable flies are the most important insect pest of feedlot and pastured cattle.

Control

Control for stable flies includes good sanitation and residual sprays on feedlots. Baits and ear tags are not effective. Currently, there are no effective control methods of stable flies on pastured cattle. Research indicates that stable flies on pastures develop in the sites where round hay bales are fed to cattle during winter. A mixture of wasted hay, manure and soil is an excellent habitat for stable fly development in the spring. Steps should be taken to minimize hay waste and accumulation or spread the wasted hay into a thin layer.

Horn flies

Male and female horn flies bite and feed on blood of animals. They are about a half the size of the house fly. Horn flies usually gather on the shoulders, back, and sides of animals. On hot days they move on the underside of the belly. Unlike other flies, they stay on the animal and leave only when disturbed or when females lay eggs into fresh animal feces. In Kansas, they are active from April to October. They are pests on pastured cattle.

Control

Ear tags. There are many brands of ear tags and all of them based on organophosphate or pyrethroid active ingredients.

Recommendations for ear tag use:

1. One ear tag per animal is good enough. The difference in efficacy between two tags versus one tag per animal is small.
2. Not all animals need to be tagged for horn fly control. Tagging every third animal with one tag will provide good control because horn flies move between animals and eventually take blood from a tagged animal.
3. Rotate two years on organophosphate tags, one year on pyrethroid tags, two years on organophosphate tags, and so on. Read the label and find out if the active substance is an organophosphate or pyrethroid.
4. Control face flies if they appear in large numbers or if cattle have pinkeye infection caused by the bacterium Moraxella bovis that is transmitted by face flies.

Animal sprays. Co-Ral (coumaphos), Atroban, Ectiban, Permectrin (all permethrin), Rabon (tetrachlorvinphos), Ravap (tetrachlorvinphos +dichlorvos).

Pour-ons. CyLence (cyfluthrin), Ivomec Pour-on (ivermectin), Saber Pour-on (lamdacyhalothrin), many brands of permethrin. This year Elanco introduced a new insecticide, ELECTOR™, for control of horn flies and cattle lice. Elector is based on a new class of active substance, spinosad. Spinosad is a natural product produced by a soil actinomycete (bacterium) *Saccharopolyspora spinosa*. Spinosad is absorbed on contact and affects the insect's nervous system, which results in rapid paralysis and death. Elector is nonirritating to people and animals and requires no protective equipment. This product is also effective against horn flies resistant to other classes of insecticides. Elector is available in pour-on and dilutable spray formulations (on animal and premise) for dairy and beef cattle (lactating and non-lactating).

Dust bags. Co-Ral (coumaphos), Rabon (tetrachlorvinphos), many brands of permethrin.

Backrubbers. Co-Ral (coumaphos), Ravap (tetrachlorviphos+dichlorvos).

Oral larvicides. There is no convincing evidence that these products manage fly populations.

Follow product directions for all fly control products. Brand names appearing in this publication are for product identification purposes only. No endorsement is intended, nor is criticism implied of similar products not mentioned.

Additional information can be found in the publication, *Managing Insect Problems on Beef Cattle*, C-671, available at *www.oznet.ksu.edu/library/ENTML2/C671.PDF* or from your local K-State Research and Extension office.

Composting animal mortalities effective, but with limits

J. M. DeRouchey, livestock specialist, Kansas State University

Composting of animal mortalities has increased in popularity in recent years because of decreased availability and increased costs of the traditional animal rendering industry. With increasing concerns about foreign animal disease and transmission, composting has received more attention as a method for mass mortality disposal. Mass mortalities have traditionally been disposed of by burial or burning. However, burning raises concerns about airborne infectious agents. Burial is the preferred method of many state agencies. However, other disposal options must be available when carcass burial sites are limited because of shallow ground water or frozen soil.

Composting maintains a proper mixture of materials for effective carbon-to-nitrogen ratios, moisture, and temperature. When properly composting, the temperature and activity of microorganisms increase. Millner (2003) estimates that more than 99 percent of the pathogens and parasites are killed when heated to 55 °C for three consecutive days.

Senne et. al (1994) evaluated the survival of highly pathogenic avian influenza virus and egg drop syndrome-76 during composting. They used tissues from chickens infected with highly pathogenic avian influenza virus and tissues from chickens infected with egg drop syndrome-76 virus among the chicken carcasses. In the compost mixture, they included one part straw, one part carcasses and two parts manure. Senne et al. (1994) tested tissues at day 10 of composting, turned the pile and retested tissues 10 days later. The avian influenza virus was not detected after 10 days of composting. The egg drop syndrome-76 virus could be recovered at 10 days but not at 20 days of composting.

Research by Granville et al. (2004) is evaluating the potential of composting to control other catastrophic viral disease outbreaks. Scientists are evaluating pathogen inactivation vaccine strains of poultry viruses inside the composting piles in retrievable containers (dialysis cassettes and cryogenic vials). A commercially licensed (B1Lasota) vaccine strain of Newcastle disease virus (NDV) was first used to evaluate the potential biosecurity risks from composting. NDV is a single-stranded RNA-enveloped virus that is highly representative of other viruses, such as influenza. Preliminary results indicate that no viruses were detected in either container at day eight of composting in mixtures of straw/manure, cornstalks or silage. They also studied whether viruses would escape the compost mixture and affect surrounding livestock. This was done by inoculating and propagating viruses inside chicken eggs and then distributing the eggs (and their contents) throughout the composting test piles before adding cover material. They used specific pathogen-free sentinel chickens in cages 10 feet from all sides of the composting test units. None of the 48 caged pathogen-free birds tested positive for NDV antibodies during the 10- to 12-week testing period. In addition, the authors are evaluating the effects of a commercially licensed vaccine strain of avian encephalomyelitis virus that was used to emulate foot-and-mouth virus. That data is not available.

> Composting maintains a proper mixture of materials for effective carbon-to-nitrogen ratios, moisture and temperature.

Recently, bovine spongiform encephalopathy has raised concerns about traditional rendering and its inability to kill prions associated with the transmission of the disease. However, Bagley et al. (1999) indicated that prions are not destroyed by composting. Animals infected with transmissible spongiform encephalopathies should not be rendered via composting because the infectious agents are still present after composting. Further research will determine if plants can absorb prions in soil when contaminated compost is spread on land.

Castastrophic composting can be an effective disposal method. This is especially true in destruction of viral disease agents, but further research is needed to verify safety and efficacy. While composting does show some effectiveness, burial is the major disposal method for mass mortality situations unless ground water or frozen ground limit its use.

Bagley, C. V., J. H. Kirk, and K Farrell-Poe. 1999. Cow Mortality Disposal. AG-507. Utah State University Extension.

Granville, T. D. 2004. Emergency livestock mortality composting in Iowa. *http://www.abe.iastate.edu/cattlecomposting/index.asp*. Accessed May 25, 2004.

Millner, P. 2003. Composting: Improving On a Time-Tested Technique. Available: *http://www.ars.usda.gov/is/AR/archive/aug03/time0803.htm.* Accessed May 25, 2004.

Senne, D.A., B. Panigrahy, R.L. Morgan. 1994. Effect of composting poultry carcasses on survival of exotic avian viruses: highly pathogenic avian influenza (HPAI) virus and adenovirus of egg drop syndrome-76. Avian Diseases 38:733-737.

West Nile virus, continued from pg. 1

pox vector vaccine. Efficacy between the two vaccines is comparable (94 and 86 percent preventable fraction, respectively). An initial vaccination is followed by a booster three to six weeks later. A single vaccination appears ineffective for prevention. Preliminary data indicates protection may begin 14 and 30 days after the booster vaccination for the recombinant and killed vaccines, respectively. Vaccination is recommended one, two or three times per year after the first vaccination, depending on geographic location. In the Midwest, we recommend vaccination in early spring with a second booster in July to boost immunity before peak viral exposure. Annual vaccination for WNV should mirror recommendations for EEE and WEE. Foals from vaccinated mares should begin their vaccination program at 3 months of age, and should be followed with two booster vaccines. Foals from unvaccinated mares should be vaccinated at 1, 2, 3 and 6 months of age. Vaccine recommendations may change as information becomes available regarding efficacy, duration of protection and immunity post-natural infection.

Continuing Education

November 6 -7
13th Annual Midwestern Exotic Animal Medicine Conference

For the most complete, up-to-date, conference information visit our Web site at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Linda M. Johnson, Ph.D., at 785-532-5696 or *johnson@vet.ksu.edu*

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