

# VETERINARY

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## Polioencephalomalacia in Cattle

by *KARAMEET PANDHER, B.V.Sc., Ph.D.*  
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Polioencephalomalacia (PEM) literally means the softening of the gray matter of the brain. The lesion of PEM can occur due to many etiologies (discussed below), however, the term is also used to describe a syndrome in ruminants characterized by laminar necrosis of the deep cortical neurons. This is a disease of young ruminants (sheep, weaning to 18 months, and cattle, three months to a year) and occurs in both feedlot and pastured animals, although the severity and incidence of the disease is less in pastured animals.

Clinical signs include ataxia, head pressing, opisthotonus, nystagmus, and blindness. Recumbency, muscular tremors and convulsions are seen in more severely affected animals. Gross examination of the brain may reveal cerebral swelling and flattening of the gyri. The dorsal surface of the cortices is soft on palpation. In more severe cases of cerebral swelling, herniation of the cerebellum and medulla through the foramen magnum can occur. On cut surface, 0.5 to 1 mm wide band of the gray matter adjacent to the underlying white matter of the cerebral cortex may be pale, but lesions may be difficult to appreciate grossly. In more advanced cases, these areas may be necrotic and look yellow to pink and friable. Necrotic areas exhibit autofluorescence if exposed to UV light. This change is more conspicuous in the gyri than in the sulci. The lesion usually involves the dorsal middle and caudal part of the cerebral hemispheres.

Microscopically, minimal change is necrosis of the deep cortical neurons. In more advanced cases, spongiosis of the neuropil, presence of macrophages (gitter cells) in the cortex and in the meninges, and hypertrophy and hyperplasia of capillary endothelial cells are observed.

The malacic lesion is mostly found in the field of distribution of the middle cerebral artery. In more severe cases, areas supplied by the posterior cerebral artery may be involved.

### Etiology

#### Thiamine Deficiency

Thiamine deficiency can occur in ruminants if they ingest thiamine antagonists, such as amprolium. Certain microbes produce thiaminases, which cause breakdown of thiamine in the rumen. Sulfur-containing compounds, especially sulfites, are capable of cleaving thiamine. (The role of sulfur in PEM is discussed in detail in the following paragraphs.) Production of thiamine analogs by thiaminase I in the rumen may interfere with biologic effects of thiamine, even if the concentrations in blood are normal. Indeed, thiamine administration to calves showing onset signs of PEM leads to rapid recovery. Delayed administration following onset of neuronal necrosis may be of more modest benefit, since the neuronal deficit created is never recovered, and such calves, if they survive, may continue to show CNS deficit.

However, association of thiamine deficiency with PEM is not always consistent. Low levels of thiamine do not always correlate with development PEM. Thiamine levels may be normal in animals showing signs of PEM. Also, low levels could be the result of anorexia and indigestion in animals showing signs of PEM.

It is now thought that thiamine might have nonspecific therapeutic effects in a range of cerebral diseases. Thiamine has been reported to be beneficial in cases of lead toxicosis. (Benefit of thiamine treatment in calves showing sulfur-induced PEM is discussed later.)

*continued on page 2*

**Polioencephalomalacia***(continued from page 1)***Lead poisoning**

Clinical signs of lead poisoning are similar to PEM, but the characteristic laminar necrosis is not a salient feature in the brain. Actually, the CNS lesions of lead poisoning can be quite nonspecific and subtle. Laminar cortical necrosis can occur in subacute cases when the course of the disease is at least a few days. Diagnosis of lead poisoning is mainly through demonstration of elevated levels of lead in the blood, liver and kidneys. Brain is not a good tissue to submit for lead analysis.

**Water deprivation, salt intoxication**

Salt intoxication is a management problem and is usually the result of water deprivation followed by ingestion of large quantities of salty water. The disease is also precipitated by free access to salt in animals that have gone through a long period of salt restriction. This phenomenon is usually seen in cattle that are grazed on mountain pastures. Along with nervous signs of blindness and paresis, clinical signs involving the digestive tract — such as vomiting, diarrhea, and abdominal pain — are also seen. The disease is seldom seen in animals that have access to water and low-salt diet.

**Sulfur**

Sulfur is essential for ruminants, and the microbial population in the rumen. The ruminal microflora that utilize sulfur to produce amino acids and other essential organic molecules are called assimilatory sulfate reducers (*Bacteriodes*, *Butyrivibrio*, and *Lachnospira*), whereas the ones that utilize sulfur for their own energy needs — and in the process produce excess of sulfide ions — are called dissimilatory reducers (*Desulfovibrio* and *Desulfotomaculum*).

Recently, the role of sulfur in PEM has been quite convincingly elucidated through many experimental and field studies. High levels of sulfide in ruminal fluid and ruminal gas cap have been consistently correlated with the development of PEM. Indeed, direct experimental administration of sodium sulfide to cattle induced PEM. It was also noted in some of these studies that thiamine levels were within normal limits. Researchers in Colorado found that consumption of water with high sulfate content, along with a copper-deficient diet, led to PEM. Again in this study, thiamine deficiency was not noted.

Sulfur compounds — sulfates and sulfur-containing amino acids — are nontoxic to ruminants, but these are converted to more toxic ions, such as sulfides, by rumen microflora.

In ruminants, sulfate and sulfide form a

recycling system. Sulfate is reduced to sulfide in the rumen and is absorbed or is utilized by the ruminal microbes to produce proteins. The absorbed sulfide is converted into sulfate in the liver and is recirculated into the rumen through saliva and blood. Sulfide ions inhibit cytochrome oxidase in the electron transport system and therefore decrease the production of ATP. Neurons are extremely sensitive to depletion of ATP and rapidly undergo necrosis. Sulfite is generated in ruminants during the process of elimination of sulfide. Sulfite is a potent nucleophile capable of causing considerable oxidative damage, and the brain tissue, with its large lipid content, is very susceptible to oxidative damage.

Recommended dietary sulfur is 0.3 percent of total dry weight consumed; maximum tolerated levels of sulfur in diet are about 0.4 percent. Sulfur is available in diet from many sources, including sulfated water, molasses (because of treatment with sulfuric acid), dietary supplementation of concentrate and cruciferous forages (e.g., corn gluten feed). Water levels of sulfur appear to be quite significant. A feedlot had PEM episodes every summer after water supply was switched over to a new well that had twice the amount of sulfur as water from the old well. Since water consumption by cattle in this feedlot was 2.4 times higher in summer than the winter months, these cattle consumed considerably higher amounts of sulfur during summer. PEM developed 15 to 30 days after initiation of high sulfur intake.

It takes about 10 days for the ruminal microflora to reach maximal generation of sulfide following initiation of a diet high in sulfur. This coincides with increased incidence of PEM and high levels of sulfide in ruminal fluid. It has been shown in vitro that ruminal microflora from animals fed sulfate-rich diet had a better capacity to convert sulfate to sulfide than microflora from animals that were fed low-sulfate diet. Animals have a hydrogen sulfide smell in their eructations, ruminal content might be dark due to formation of sulfides with iron and copper.

Measurement of the ruminal gas cap hydrogen sulfide content has been suggested as a measure of ruminal sulfide production. Ruminal gas cap can be sampled percutaneously, through the paralumbar fossa, using an 18-gauge cerebrospinal fluid needle and connecting this needle to a commercially available hydrogen sulfide detector tube. In one study, the hydrogen sulfide content in the gas cap of animals fed high sulfate diet was 40 to 60 times higher than the animals fed similar diet without sulfate. In this case, the increased ruminal hydrogen sulfide coincided with onset of clinical signs of PEM. Sulfide levels in the rumi-

nal fluid may decrease sharply as anorexia sets in after the onset of clinical signs of PEM. This should be taken into account while determining sulfur levels in animals suffering from PEM. Sulfur content in diet should be assessed in cases of PEM, even if the ruminal sulfur levels are normal.

Diet influences the pH of the rumen. During acidosis (or simply decreased ruminal pH), hydrogen sulfide is released into the gas cap because it becomes insoluble in water. Ruminants inhale a significant portion of the eructed gas. Thus hydrogen sulfide can be absorbed through the respiratory tract. Interestingly PEM can be one of the sequelae to acidosis in ruminants.

Although the lesions of PEM are now being more closely associated with sulfur excess rather than thiamine deficiency, higher availability of thiamine can be protective against sulfide and sulfite-induced nervous damage. Thiamine may offer protection by scavenging the free radicals produced during oxidation of lipid. Deficiency of thiamine may thus potentiate the lesion induced by sulfur-containing nucleophiles. Also, since sulfides and sulfites cleave thiamine, a secondary deficiency of thiamine might follow sulfur toxicosis.

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# Warm winter results in flies, flies and other kinds of flies!

by DONALD E. MOCK, Extension Specialist  
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In the popular press, robins are the “harbingers of spring” but flies may get more springtime attention from entomologists and ranchers. With “hardly any winter this past winter,” we’ve had flies in Kansas every month since March 1999.

## Face Flies

In many homes surrounded by pasture land (and in a few that are not so close to the source) people contend with overwintering face flies every year. In the typical scenario, the outside of the home is plastered with flies for a few days in early October each year. Then it turns cold, and flies seek shelter within the home’s attic and walls. Gradually, they find their way into the interior of the home and become especially pesky during occasional warm spells in December and January. By March, they’re trying to get out and are very active around windows. Those that do get outside are not noticed any more. But this past fall and winter they were abundant and active both outside and inside of homes from late September continuously through March. There must have been an unusual number of them on cattle last September! Despite the fact that a high percentage of them burned up their fat reserves and died because the temperature kept them active, jillions of them were still alive and well this spring. For example, reports of those flies being a nuisance **inside** homes came in as late as March 6 in Wabaunsee and Reno counties and March 7 in Pottawatomie County. On February 20 they “covered the **outside** of a home in Cloud County. On March 7, they were numerous on a home in Cherokee County, and “tens of thousands” of them covered a home near Auburn in Shawnee County. On March 27, Bill Wood reported “masses of face flies” on a home in Douglas County. Watch out, cattle producers; pinkeye season may start early this year!

## House Flies

A large poultry operation spread hundreds of tons of manure on fields in Rice County between mid-October and mid-November, 1999. In an ordinary fall, fly development in open fields would not be expected that time of year but, as one fellow put it, “with three

months of September” the countryside was inundated by flies coming out of those fields all through November. Most of our Kansas experience with house flies emanates from cattle feedlot, dairy, and swine units, and we usually don’t expect house flies to be numerous except from mid-June through mid-September. On March 21, construction workers on a site a couple miles east of Manhattan, Kansas, complained of being bothered by hordes of flies. I expected these to be part of the unusual face fly population emerging from overwintering sites, but they were house flies.

## Black Flies

“Gray gnats” might be a more descriptive term for the black fly species that has been bothering livestock lately. On March 27, Dr. Ridley at the K-State College of Veterinary Medicine reported black flies in horses’ ears north of Manhattan; on April 3, Matt Pfeiffer sent specimens from the ears of a horse in Wabaunsee County; and on April 5 a woman in eastern Pottawatomie County complained on behalf of her horse. The species involved is *Simulium vittatum*. Although more numerous in some years than others, this species is bothersome from mid-March to mid-April throughout Kansas. There are also an early-summer and a latesummer generation, but they seem to be less numerous. *S. vittatum* bites mostly in the ears of horses, sheep, and cattle, but may also be numerous on the necks of these hosts. Although it does not happen frequently, he black fly species occasionally does bite humans.

Control efforts at the source are not feasible, as these gnats develop in running streams. Cattle and sheep can be protected with sprays of permethrin, malathion, methoxychlor, dichlorvos, or Ravap (Rabon + dichlorvos) applied to necks and ears. Horses can be protected with wipes or rubs of insecticides containing permethrin, cypermethrin, pyrethrin or methoxychlor including products such as Techtrol, Flysect, Repel-X, and Wipe.

## Horn Flies

This time of year I habitually watch for first signs of horn flies. When they emerge so early that cattle still have their winter hair (and especially on cool, windy days such as we’ve had

recently) they may be down in the haircoat and difficult to observe. I haven’t seen any. Lyle Lomas reported on April 10 that they’ve been on cattle at the Southeast Agricultural Research Center “for the past two weeks.” This is a bit on the early side, but not surprising given that this past winter was so warm that development was probably taking place within the overwintering horn fly pupae during months in which they are ordinarily dormant. We expect horn flies to be numerous about mid-May.

## Heel Flies

If you see baby calves running and jumping it’s usually because they’re feeling frisky. This time of year, and for the next few weeks, if you see yearlings and mature cattle suddenly tear out across the pasture with tails held straight up in the air they aren’t just cavorting for the glory of springtime — they’re “gadding.” Gadding is the term applied to cattle running to escape the buzz and tickle of female heel flies hovering as they oviposit on hairs on the pasterns of the hind feet. These are the adult warbles or cattle grubs. They don’t bite, but they cause panic.

By the way, when most producers (and some vets and county agents) speak of heel flies they are talking about stable flies — a biting species that draws blood from the lower legs of cattle and horses — that we’ll be dealing with mostly in May and June.

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# What's New in Feline Heartworm Diagnostics

DR. PATRICIA PAYNE and DR. PROBERT RIDLEY  
Department of Diagnostic Medicine/Pathobiology

In every survey published for all geographical areas studied, the prevalence of feline dirofilariasis is approximately 10 percent of the prevalence of canine heartworm. Therefore, if you are seeing 10 heartworm-positive dogs in your practice, there may also be one or two cats with the disease. You will only find feline heartworm disease by seeking it out, since feline dirofilariasis is often overlooked — even on necropsy.

Clinically, most infected cats will not show obvious signs on presentation but will have various non-specific, vague signs, including tachypnea, intermittent coughing and anorexia. Vomiting, not related to eating, is one of the more common complaints. Other positive cats may be critically ill and may die without showing any clinical signs.

Heartworm disease in cats is different than in dogs because of the adapted host-parasite relationship. The feline immune system is more effective in preventing the larvae from maturing. The disease and diagnostic plan are different. The following plan is recommended by the American Heartworm Society.

## Step one: Antibody testing

A positive antibody test is only indicative of exposure within the last 60 days.

Antibody tests will not distinguish among the presence of maturing or dead larvae, living or recently deceased adults. A negative antibody test indicates no exposure to heartworms or an early infection.

The tests: ASSURE®/FH or Solo Step™FH Heska

## Step two: Antigen testing

A positive antigen test indicates that the cat is harboring at least one adult female heartworm. A negative antigen test indicates that there are no heartworms present in the animal or that a few adults or only males are present.

The tests: DiroCHEK® is highly recommended but Snap™ tests will sometimes be sensitive enough.

## Step three: Microfilaria concentration

These tests are rarely positive in cats because most cats do not develop a patent infection. However, the presence of *D. immitis* microfilaria is a definitive diagnosis.

The tests: Knott's or Difil

## Step four: Echocardiography

The body wall of the adult heartworm is strongly echogenic. The signature of the live adult heartworm is short, segmented, parallel lines. An experienced sonographer can make a definitive diagnosis of the presence of live adult *D. immitis* parasites. You will be surprised how many clients will gladly accept a referral to KSU or your local referral center.

## Step five: Radiography

Good quality chest films will indicate the severity of disease and are valuable for monitoring the course of the disease.

An extremely conservative treatment plan that includes decreasing doses of prednisolone, strict confinement and low stress is recommended for cats. Adulticide treatment is not recommended, and in the minds of most is contraindicated. Periodic reevaluation at six-month intervals with serology and radiography is suggested. In an emergency situation, immediate response with appropriate shock therapy to an acutely ill cat may be life-saving.

Chemoprophylaxis is a safe and effective

option for your feline patients. Using feline heartworm antibody testing for screening will provide you with prevalence data for your area. All antibody positive cats have been bitten by an infected mosquito; most develop a transient infection only to the 4<sup>th</sup> larval stage. However, exposure confirms that the cat is at potential risk for the life threatening disease. Heartworm preventatives can be safely administered to cats 6 weeks of age (5 lbs) or older, including those that are seropositive. The choices for chemoprophylaxis include selamectin (Revolution™) and ivermectin (Heartgard™ for Cats). Milbemycin (Interceptor™) is effective but not yet approved for the use in cats.

These recommendations are obviously a drastic departure from tradition. We highly recommend consulting the Web site for the American Heartworm Society at ([www.heartwormsociety.org/](http://www.heartwormsociety.org/)) or calling their office at 630-844-9676 for further information. Other good sources of both technical and educational information can be found at [www.synbiotics.com](http://www.synbiotics.com), [www.idexx.com](http://www.idexx.com), and [www.heska.com](http://www.heska.com).

# Sampling and Interpretation of Copper, Selenium and Vitamin E

J.A. PICKRELL and F.W. OEHME  
Comparative Toxicology Laboratory

Copper, selenium and vitamin E are sampled as part of profiles evaluating the status of nutrition, metabolism and toxicology.

In live animals, both plasma and serum are equally representative samples. In the case of selenium, whole blood is an acceptable sample, but plasma is preferred if blood volume is sufficient. Serum or plasma samples to be tested should be unhemolyzed or only minimally hemolyzed.

If animals are dead, liver samples are the sample of choice to estimate the status of copper, selenium and vitamin E.

The liver's function is to store copper, selenium and vitamin E and make it readily available to keep the levels in serum or plasma constant within relatively narrow tolerances.

In nutritional excess, copper stores will continue to build until the liver's capacity to store the element has been exceeded. When excess occurs, cells will die, releasing large amounts of copper into the serum. This copper is also toxic to erythrocytes.

If only serum is available, the status of copper can still be evaluated and interpreted as having deficient, adequate or toxic status. However, the levels of copper, selenium and vitamin E in the liver change with greater magnitude, and thus are the best sample when animals have died, or when biopsy is available. Approximately 0.3 to 0.5 g (300 – 500 mg) of liver is needed for assay. Several punch biopsies are often necessary to obtain this amount of tissue.

# Effects of Supplementation on Performance of Lactating First-Calf Beef Heifers

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## Summary

Cattle consuming a protein block supplying rumen-undegradable protein with or without chelated copper, manganese, and zinc gained more weight during the breeding season and had increased conception rates compared to cows consuming agree-choice livestock mineral only. Intake of all products was greater than expected, even though pasture quantity was not limiting. Supplying copper, zinc and manganese as proteinates increased heifer gain but had no effect on conception or calf-weaning weight. Whether increases in conception rate are due to increased energy intake due to the amount of block consumed cannot be determined by this trial.

## Introduction

Reproductive efficiency is a major factor determining profitability for feeder calf producers. Compared to older cows, first-calf heifers take longer to return to estrus and have lower reproductive rates. Inadequate nutrition is often the cause of reduced reproductive efficiency, as first-calf heifers have increased nutrient requirements compared to older cows. Forage alone may not be adequate to meet the nutrient needs of spring-calving, first-calf heifers. Early spring fescue can be efficient in rumen-undegradable protein and the trace minerals copper and zinc. This study was conducted to determine the effect of supplementing rumen-undegradable protein and the trace minerals copper, manganese, and zinc in a proteinate form on the performance of first-calf heifers and their calves.

## Procedures

A total of 56 lactating first-calf heifers of Simmental breeding was used in this 96-day trial. Prior to trial initiation, male calves were castrated, and all calves were identified and matched with their dam. At trial initiation, all first-calf heifers were in a bloody condition score of four. Cow body weights were taken at trail initiation, midpoint, and trial termination. Calf weights were taken at weaning in the fall. Cows were rectally palpated for pregnancy determination at weaning.

Cow-calf pairs were assigned to one of three treatments by birth date of calf. Treatments were: 1) control, free-choice mineral only (FCM), 2) protein block (PB), and 3) PB with supplemental copper, manganese, and zinc in the proteinate form (PBPM). The free-choice mineral and protein blocks were formulated to supply macro and trace minerals in amounts adequate for the NRC requirements to be met. The protein blocks were also formulated to supply rumen undegradable protein from animal by-product sources. The protein blocks were identical except for the source of copper, zinc and manganese. A calculated composition of selected nutrients for all products is shown in Table 1. Product intake was determined by weight difference. Weight of fresh products was recorded when placed in the feeder. Product remaining in the feeder was weighed every three to five days, with the difference considered to be intake. Treatment groups were grazed in separate cool-season, grass-legume pastures. Pastures were rotated such that each treatment group received equal time in all pastures. Bulls used for breeding were also rotated between groups such that each treatment group received equal exposure to all bulls.

Growth data were analyzed by the GLM procedures of SAS. Procedures were used to account for birth date and calf sex differences between treatment groups. Differences in pregnancy percentages between treatments were analyzed using the Chi Square method.

## Results and Discussion

The effect of product supplementation on gain, weaning weight, and pregnancy is shown in Table 2. Heifers fed PBPM gained more weight during the breeding season and total trial than heifers fed FCM or PB. Heifers fed PB gained more slowly than other groups prior to breeding and were intermediate in gain to FCM- and PBPM-fed heifers during the breeding season. Total gain of PB- and FCM-fed heifers did not differ. All heifers gained in body condition throughout the grazing season.

Cows fed PB and PBPM weaned heavier calves numerically than cows fed FCM; however, differences did not reach statistical significance ( $P=.15$ ). The greater ( $P<.05$ ) gain during the breeding period of cows receiving either block indicates greater nutrient intake. Increased nutrient intake may have stimulated milk production, resulting in numerically increased weaning weights.

*continued on page 7*

**Table 1.** Calculated analysis of treatment products.

Nutrient Mineral	Free-Choice Proteinate	Block +	Block
TDN %	—	66.4	66.4
Crude protein %	—	20.3	20.3
Salt %	17.4	7.2	7.2
Calcium %	20.9	2.5	2.5
Phosphorus %	6.1	.7	.7
Copper, ppm	992	98.8	93.5
Zinc, ppm	2939	342	327
Manganese, ppm	2756	317	286
Selenium, ppm	52	1.79	1.79

**Table 2.** Effect of supplementation on heifer gain and pregnancy, and calf weaning weight.

Item	Treatments			SEM
	Control	Block +	Block	
Proteinate				
Prebreeding gain, lb	39 <sup>a</sup>	49 <sup>a</sup>	21 <sup>b</sup>	7
Breeding season gain, lb	39 <sup>c</sup>	91 <sup>d</sup>	61 <sup>e</sup>	8
Total gain, lb	78 <sup>f</sup>	140 <sup>g</sup>	82 <sup>f</sup>	10
Percent pregnant	61.1 <sup>c</sup>	89.4 <sup>d</sup>	83.3 <sup>d</sup>	—
Weaning wt, lb	457	490	488	13

Means in the same row with differing superscripts differ, <sup>ab</sup> $P<.1$ ; <sup>cde</sup> $P<.05$ ; <sup>fg</sup> $P<.01$ .

# Fair and Show Serology Testing

Summer, the season for fairs and shows, is rapidly approaching. This means a large increase in workload for our serology laboratory. Every year problems arise when samples are submitted too close to the date results are needed, and clients are disappointed. Our laboratory personnel strive to get results out as fast as possible, but each test has a minimum time-frame. When clients come to you just a day or two prior to leaving for

a fair or show, please warn them that the laboratory may not be able to accommodate their testing needs on such short notice.

The following is the minimum turn-around time for some of the tests commonly requested this time of year.

Test		Minimum Turn-Around Time	Cost/Sample
EIA	standard AGID	24 hrs.	\$5.00
	ELISA	4 hrs.	\$15.00
Pseudorabies	SN	48 hrs. (run MWF only)	\$3.50
Brucellosis	card test	4 hrs.	\$1.00
Leptospirosis	MAT	8 hrs. (run MWF, test run starts at 7:00 a.m. so sera must be in lab the day before)	\$7.50
BLV	AGID	24 hrs (run MWF only)	\$5.00
CAE	AGID	24 hrs (run MWF only)	\$5.00

### Hints for better service:

- Unless stated otherwise, sera needs to be in the lab by noon on the day the test procedure is started. If test results are to be faxed, PLEASE include fax number.
- Please be sure serum samples are properly identified on both the tubes and submission forms.
- Having serum already centrifuged and decanted off — or in separator tubes — saves lab time and allows us to be more efficient by getting results back to you and your clients faster.
- Please be aware that the Kansas State Veterinary Diagnostic Laboratory does not send test results directly to clients, nor does it give results to clients by phone. On Saturday mornings the lab is short-staffed, and we may not be able to get to the phone or to find tests results.
- If you have questions, please feel free to contact the Diagnostic Laboratory at 785-532-5650.

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.

# Requirements for Kansas County Fairs

*Courtesy of the Kansas Animal Health Department*

## Kansas Origin Livestock

All animals are subject to examination by the exhibition staff or their representatives, and shall be free of clinical signs of infectious or contagious disease. Health certificates and additional requirements shall be a county option.

## Cattle and Bison

The Kansas Animal Health Department (KAHD) requires no test on cattle and bison of Kansas origin. Cattle determined by exhibition staff to have lesions of ringworm, warts or infested with mange will not be permitted to exhibit.

## Swine

Swine originating in Kansas must meet the general requirements. Kansas has obtained Stage V status in the National Pseudorabies Program and the state is classified free of swine brucellosis. A negative pseudorabies and brucellosis test are not required for Kansas origin swine.

## Sheep

In addition to the general requirements sheep shall:  
Be free of any signs of "sore mouth."  
Be free of signs of active fungal (ringworm) infection, including club lamb fungus.

## Goats

Goats shall meet the general requirements. Goats determined by exhibition staff to have sore mouth, lesions of ringworm, warts or infested with mange will not be permitted to exhibit.

## Horses

Horses shall meet the general requirements. A negative coggins test within 12 months for equine infectious anemia should be considered, but it is not required.

# Gulf Coast Ticks Seen in Kansas

*DONALD E. MOCK, Extension Specialist  
Medical and Veterinary Entomology  
Kansas State University*

Gulf Coast ticks have been showing up fairly regularly in submitted samples from across Kansas. Adults of this species strongly prefer feeding inside large animals' ears. While doing so, they can cause both a 20 percent drop in livestock weight gains and permanent ear deformities.

The larval and nymph stages of the Gulf Coast tick's life cycle spend their time as parasites on wild birds. These ticks-in-progress particularly like quail and migratory birds such as meadowlarks, which spend time on the ground or in low thickets.

"Scientists have found up to 289 larvae on a single meadowlark," said Don Mock, Kansas State University entomologist.

As a result, the tick has plenty of chances to fly north each spring with the birds.

"We also know that the tick has been expanding its natural range. It spread through Oklahoma in the '60s and '70s and became part of that state's normal complement of pests," noted Mock. "Kansas may have been next on its list."

Despite the tick's seeming ability to adjust to a new type of living site, Kansas' weather still may be affecting the pest's activity level from year to year.

"Our data are limited. But we've got some indication these ticks don't survive well if we have a late summer that's extremely hot and dry—especially one followed by a bitterly cold winter with little snow cover," Mock said. "Concurrently, if mild summers and winters continue, the ticks may thrive."

Another possibility, however, is that the tick is settling in to stay. Oklahoma entomologists discovered the tick was able to adjust its normal seasonal cycles and adapt to a more northerly climate.

In Oklahoma, Gulf Coast larvae numbers peak in late June, but remain a factor in September. Nymphs show up from July to September, yet hit two population peaks: late July and early September.

In Oklahoma and Kansas, the species' cattle-harming adults appear in March, peak in late April, start to reduce numbers in June and gradually disappear in July.

"Ranchers legally can apply tick controls to livestock, but not to pastures," Mock said. "County Extension offices can provide recommendations ranging from quick sprays to long-term ear tags.

"I'd encourage ranchers to bring tick samples to their county office, as well. Submissions to K-State are how we've discovered the Gulf Coast tick may be far more prevalent than previously thought. Samples are how we discovered the problem can appear in the same place two years in a row."

Gulf Coast ticks look a lot like the American dog tick, except for two factors. The males of both species have a white, net-like pattern on their back. But Gulf Coast males' pattern has distinct edges, while the dog tick's pattern has blurry edges. Plus, both male and female Gulf Coast ticks have long, narrow mouthparts much like those of its near relative, the lone star tick.

### *Effects of Supplementation on Performance (continued from page 5)*

Cows receiving PB or PBPM had a greater ( $P < .05$ ) percent pregnant at weaning compared to cows receiving FCM only. In thin cows, conception rate can be expected to increase as rate of gain increases during the postpartum period.

Group average intake of the three products is shown in Table 3. Cows consuming FCM had greater intake than expected but

were consistent during the entire period. Intakes of both block products were similar, especially during the first half of the test period.

Intake increased greatly during the second half of the period, even though pasture mass was not limiting. Cattle apparently liked the product and consumed more than was expected.

**Table 3.** Product intake, group average, pounds per hd per day.

Item	Control	Treatments	
		Block +	Block
Proteinate			
Initial 48 days	.347	2.08	2.04

## Coming Events

**August 25, 2000**

### **Bovine Conference on Present and Future Feedlot Issues**

Speakers: Nathan Reese Ph.D., retired feedyard manager/nutritionist  
Bob Smith, D.V.M., Oklahoma State University  
Jim Sears, D.V.M., Bayer Animal Health

**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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