

# VETERINARY

## FOR THE PRACTICING VETERINARIAN

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### Milk Fever Gel is Licensed

By *JESSE P. GOFF*

*Agricultural Research Service-USDA*

*Agricultural Research Service (ARS)*

has granted an exclusive license to Kemin Industries, Inc., of Des Moines, Iowa, for an in dairy cows by about 50 percent. Each year, about 500,000 U.S. dairy cows develop severe milk fever—usually within a day after giving birth. The disorder costs producers \$150 million a year. According to ARS researchers, the gel may be given orally to cows when they give birth and for the first two days of lactation. Other oral formulations contain calcium chloride, which can irritate the cow's mucous membranes and the skin of the person administering the treatment. The ARS-formulated gel delivers calcium propionate, a less irritating form of calcium. Another advantage of calcium propionate is that cows can use it to make glucose for energy. All lactating dairy cows are energy deficient, because they use much glucose to make milk, and they can't eat enough immediately after calving to meet their energy needs.

In ARS field trials with an Iowa Jersey dairy herd, the gel reduced milk fever from 50 percent in untreated cows to 29 percent in treated cows.

## Effects of Growing and Finishing Steer Health on Performance, Carcass Traits, Meat Tenderness, and Net Return

By *B.A. GARDNER, H.G. DOLEZALI,*

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Steer Calves (n=204) were used to evaluate effects of respiratory disease during a 150-day finishing period on daily gain, carcass traits, longissimus tenderness, and net return. Steers were monitored daily for clinical signs of respiratory infection and medicated as needed. At harvest, lungs were inspected; lungs were evaluated for the presence of bronchopneumonia lesions in the anteroventral lung lobes and activity of the lung lymph glands. Net return per steer was calculated using average feeder steer prices from 1985 to 1995, average 1995 and 1996 carcass premiums and discounts, and a feed cost of \$165/907 kg DM (net return did not account for differences in feed efficiency). Lung lesions were present in 33% of all lungs but were equally distributed between medicated (37%) and non-medicated groups (29%). Steers that were medicated (n=102) had reduced (P<.05) final live weights, ADG, carcass weights (HCW), less external and internal fat, and more desirable YG. Temporal classification of lungs revealed that steers with lesions (n=87), regardless of lymph gland activity, and those with lesions and active lymph glands (n=9) had lower (P<.05) daily gains, lighter HCW, less in-

ternal fat, and lower marbling scores than steers without lesions or steers with lesions and non-active lymph glands, respectively. Morbid steers, regardless of classification system, yielded more U.S. Standard carcasses than Anon-sick@ steers. No differences (P>.10) in longissimus shear force (WBS) values were evident, except that 7-day aged steaks tended (P=.05) to have lower WBS values when obtained from steers without than with lung lesions. Net return per medicated steer was \$17.46 and \$73.50 less for those pulled once and more than once, respectively, than for non-medicated steers. A pronounced effect of health on net profit was evident for steers classified by the temporal lung scoring system: net return for steers without lung lesions averaged \$20.03 greater than for those with lung lesions but non-active lymph glands and \$73.78 greater than for those with lesions plus active lymph glands. Overall, morbidity suppressed daily gains, decreased carcass quality, and reduced profit. Classification of lung lesions by lesion age was more predictive of production, carcass trait, and meat tenderness differences than antemortem health evaluations.

(Key words: *Morbidity, Performance, Carcass Traits, Profitability*)

Source: *1998 Plains Nutrition Conference - Abstract*

# Chronic Wasting Disease in Elk in Northeastern Colorado

By TERRY SPRAKER  
*reprinted from Spring 1998 Colorado State  
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Chronic Wasting Disease (CWD) is a specific transmissible spongiform encephalopathy affecting free-ranging and captive mule deer, white-tailed deer, and elk. This disease was first observed by biologists with the Colorado Division of Wildlife in captive mule deer in the late 1960s and was diagnosed as a spongiform encephalopathy in captive deer and elk in 1978. In 1981, free-ranging elk from Rocky Mountain National Park was found with CWD and the first free-ranging mule deer with CWD was found northwest of Fort Collins in 1984. Presently, CWD is found in free-ranging deer and elk in northeastern Colorado and southeastern Wyoming, and in captive deer and elk at wild animal facilities at Fort Collins, Colorado, and at Sybille, Wyoming. Recently, several elk were positively diagnosed with CWD in a game farm in South Dakota.

CWD is a neurological disease characterized by a spongiform degeneration of the brain, primarily affecting the thalamus and

brain stem. Clinical signs of CWD are excessive salivation, emaciation or wasting, behavior changes, and weakness. At necropsy, few changes besides emaciation, ulcers, and secondary pneumonia are seen. CWD is believed to be caused by an altered prion protein. This prion protein is antigenically similar to the prion protein thought to be the cause of scrapie in domestic sheep and goats.

Over the last several years, the Colorado Division of Wildlife has collected deer and elk heads from hunted and road-killed animals throughout Colorado to delineate the regional distribution of CWD in cervids. We remove the brains from these heads and examine them both histologically and immunohistochemically to determine if they are affected by CWD. To date, we have examined approximately 3,500 cervids. The prevalence of CWD in deer is highest (approximately 5%) in a relatively small area bounded by the Wyoming border, Fort Collins, Rocky Mountain National Park, and Estes Park. The prevalence of CWD in elk in the same area is less than 1%. CWD has

been diagnosed in deer east of Fort Collins, but the number of animals found positive in this area is extremely low.

CWD has been known to occur around the Fort Collins area for over 30 years and during this time there have been no cases of a spongiform encephalopathy in cattle here, or anywhere else in the United States. The few cases of scrapie in domestic sheep from the Fort Collins area were from sheep recently brought into the state or were from scrapie-infected flocks and were not associated with deer or elk. Scrapie has not been linked to any disease in humans, however, bovine spongiform encephalopathy (mad cow disease) has been linked to new variant Creutzfeldt-Jacob disease in humans in Great Britain.

The only method to diagnose CWD is to examine several specific areas of the brain histologically and immunohistochemically. The immunohistochemical stain was developed for diagnosis of scrapie but also stains the prion protein in deer and elk brains and is used for confirmation of diagnosis for all cases of CWD.

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# Induced Heat Stress Resistance in E. Coli

By DORIS STANLEY  
*Agricultural Research Service*

*Escherichia coli* O157:H7 bacteria that get only a sublethal dose of heat can become more heat resistant than bacteria that are not so exposed, report Agricultural Research Service scientists in Wyndmoor, Pennsylvania.

The microbiologists at the ARS Eastern Regional Research Center (ERRC) say the finding reiterates the continuing need to adequately cook food to kill *E. coli* O157:H7 and other food-poisoning microorganisms, or pathogens. Cooking remains the primary means to kill these organisms in foods.

"Our increasing understanding of the wide range of factors that can affect pathogens' thermal resistance indicates the need for a standard way to measure that resistance," says Vijay K. Juneja, who conducted the study in ERRC's Food Safety Research Unit.

Juneja and colleagues subjected beef gravy samples containing *E. coli* O157:H7 to 114.8°F for 15 to 30 minutes, heat-shocking the bacteria at a temperature not quite sufficient to kill them. Then they cooked the gravy to a final internal temperature of 140°F.

The results: The preheated *E. coli* survived longer (a 1.5-fold increase in heat resistance) than other *E. coli* not subjected to the sublethal heat. The increased thermotolerance lasted for at least 48 hours.

Therefore, says Juneja, food processors should realize that bacteria will not be killed in foods that are heated slowly to the final cooking temperatures normally used. Heat-shocking conditions may occur in minimally processed, refrigerated, cook-in-bag foods such as filled pasta products (ravioli, tortellini, cannelloni, etc.), mousaka, lasagna, and chili

con carne. The slow heating rate and low heating temperatures used in preparing these foods expose potential pathogens to conditions similar to heat shock—which would make them more heat-resistant.

This induced heat-resistance could also be a concern in meat products kept on warming trays before final heating or reheating, or when equipment failure interrupts the cooking cycle during processing.

Juneja says that traditional research methods to determine if heat kills pathogens are cumbersome because of lengthy sample preparation times and nonuniform heating. He and colleagues used a submerged stainless-steel coil-heating apparatus that allows quick temperature control by a thermostat, eliminating the customary problems.

# Cytauxzoonosis in Cats: An Emerging Disease for Kansas Practitioners

By GORDON A. ANDREWS

Diagnostic Medicine/Pathobiology

Cytauxzoonosis is a rapid and highly fatal febrile disease of cats caused by the blood protozoan parasite *Cytauxzoon felis*.

## Etiology & Epidemiology

Cytauxzoon is classified in the order Piroplasmida and family Theileriidae. Like other members of this family, *Cytauxzoon* has an erythrocytic and tissue phase. The tissue phase in cats consists of large schizonts that develop within macrophages or monocytes in the vascular system. The erythrocytic form is referred to as a piroplasm.

### Life Cycle

Little is definitively known about the life cycle of the organism. In cats, schizonts develop within monocytes or macrophages, develop into macroschizonts which develop buds or merozoites. The host cell ruptures, releasing merozoites which can infect other macrophages or erythrocytes. Bobcats (*Lynx rufus*) are believed to serve as biological reservoirs of the organism in the wild and are largely asymptomatic carriers. A survey of wild-trapped bobcats in Oklahoma revealed that 60% of the animals harbored an erythrocyte parasite indistinguishable from the intraerythrocytic form of feline cytauxzoonosis. Recently, the Florida panther (*Felis concolor coryi*) has also been implicated as another possible host for *Cytauxzoon felis*.

### Transmission

Ticks are the most likely vector of transmission from the bobcat to domestic cats, and appear to be necessary to induce the fatal form of the disease in cats. The fatal disease has been experimentally transmitted from wild-caught parasitemic bobcats to domestic cats via ticks (*Dermacentor variabilis*) (the American dog tick). However, blood from parasitemic bobcats inoculated into domestic cats induces a persistent erythroparasitemia rather than fatal disease. Parenteral injection of fresh or frozen blood or tissue homogenates from infected cats showing clinical signs to other cats induces fatal disease. The disease is not transmis-

sible between cats by direct contact or by ingestion of fresh or frozen blood or tissue homogenates.

## Clinical Findings

Clinical signs include the following. Those most frequently observed and most characteristic are in bold type.

Acute lethargy, anorexia, depression, icterus, pallor, high fever (103 to 107\_F), rapid course of disease (<7 days, more commonly 5 days or less from onset of clinical signs until death), dehydration, shock (capillary refill time >2 seconds, dyspnea, and occasionally neurological signs (seizures)).

## Clinical Pathology

### Hematology

Hematologic findings include the following. Those most common or characteristic are in bold type.

Mild to moderate normocytic normochromic anemia, thrombocytopenia, variable leukocyte counts (subnormal to normal to elevated with a left shift have been reported), may be slight increase in nucleated RBCs.

### Clinical Chemistry

Clinical chemistry findings include: Elevated total serum bilirubin, glucose, alanine transaminase, and BUN; decreased serum albumin, potassium; hemoglobinuria and bilirubinuria rarely and in the terminal stages of the disease.

## Diagnosis

Risk factors identified for cats include warm weather, access to a wooded environment, (i.e. outdoor cats that may share an environment with bobcats), and exposure to ticks. Therefore, cytauxzoonosis should be considered in the differential list for any cat with the above risk factors that presents with acute anemia, jaundice, and high fever.

Diagnosis is made by demonstrating the piroplasms (erythrocytic phase) in blood films, or by demonstration of macrophages containing merozoites or schizonts in tissue impression smears, or by histopathology. The piroplasms within erythrocytes appear as round "signet ring" forms 1 to 1.5  $\mu$  in

diameter, bipolar "safety pin" forms 1 by 2  $\mu$ , or round "dots" less than 1  $\mu$  diameter. All forms may appear in a single blood film. Some erythrocytes may contain more than one piroplasm.

## Prognosis

The prognosis in domestic cats is grave. There are rare cases reported in the literature of cats that have survived the disease. Cats that survive are apparently immune to reinfection.

## Pathologic Findings

### Gross Lesions

Gross necropsy lesions may include the following. Those most common or characteristic are in bold type.

Splenomegaly, icterus, pulmonary congestion with petechiation or mottling, hepatomegaly (hepatic congestion), dehydration, pallor, hydropericardium, enlarged edematous and hemorrhagic lymph nodes, intra-abdominal venous congestion, serosal hemorrhages.

### Histopathology

The pathognomonic histologic lesion is the accumulation of large numbers of parasitized macrophages containing schizonts and merozoites in various stages of development lining and filling the lumens of veins of multiple organs. Highly vascular organs such as liver, lung, spleen, and lymph nodes contain the largest numbers of organisms and are therefore the preferred organs for making impression smears for cytological examination. However, the organisms can be observed histologically in virtually any organ. Cats that present with neurological signs often contain large numbers of organisms within meningeal and parenchymal veins of the brain, and within choroid plexes.

## Treatment

Treatment in the vast majority of cases is unsuccessful and consists of aggressive supportive care. In any case, the chances of survival are low.

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