VIRGINIA COOPERATIVE EXTENSION SERVICE

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THE FOOD ANIMAL VETERINARIAN

VIRGINIA-MARYLAND REGIONAL COLLEGE OF VETERINARY MEDICINE

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Dear Food Animal Practitioner,

For the first time, the Virginia Academy of Food Animal Practice and the Virginia Veterinary Medical Association met jointly in February in Richmond. The meeting was very successful with over 50 in attendance in the Food animal program.

The Academy elected to hold its 1999 meeting also in connection with the VVMA. That meeting will be held in February 1999 at the Hotel Roanoke as long as the fee structure and other accommodations are favorable to food animal practitioners, as they were in Richmond. Please make plans to attend this meeting with us. Meeting with the VVMA gives us the chance to participate in several of their events such as the trade show, their fun night, banquet, etc. For mixed practitioners there is the opportunity to participate in CE for more than one practice area. The year 2000 VVMA meeting will be held at the Homestead in Hot Springs, VA. No decision about our participation in that program has yet been made.

At the Academy business meeting the decision to continue the two Academy scholarships was made. The Academy of Food Animal Practice will award the following scholarships:

- Outstanding graduating food animal student - Heather Brazzell

- Summer internship - Rob Ratcliffe.
  This program is designed to encourage the development of an interest in food animal practice by providing support for a student to spend the summer in Academy member's food animal practices. Rob will be spending parts of the summer in four practices.

We are preparing for graduation of another class of veterinary students. We continue to have a select but outstanding group of students who elect the food animal track in the fourth year of the curriculum. The numbers of food animal track students for the following graduation years are: 1997- 7; 1998-6, 1999-5. Andrew Holloway is a food animal track student who will graduate May 8, 1998. He was named the outstanding senior student at the Virginia Tech Founders Day in April. Two other food animal track students, Dr. Andy Meadows and Dr. Tom Massie have received the same award in 1995 and 1996. It speaks very well for our area of practice that we attract such outstanding students.

I sincerely hope that things are going well for your practices.

W Dee Whittier, DVM

Dr. Dee Whittier, Extension Veterinarian

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MINIMIZING MYCOTOXIN PROBLEMS

Mycotoxin contamination of grain is a serious problem. A large portion of the world's grain supply is contaminated with mycotoxins. Mycotoxin-contaminated feeds depress livestock and poultry performance causing U.S. producers millions of dollars in losses. Often these economic losses are due to subtle, non-specific effects on animal performance.

Mycotcxins cause a wide variety of adverse clinical signs depending on the nature and concentration of mycotoxin present, duration of exposure, and the animal species, age, and nutritional and health status at the time of exposure to contaminated feed.

Molds and mycotoxins are widely distributed in the environment and have complex metabolisms. The mold species that are of concern to grain handlers and feed manufacturers are routinely found in soil. Spores from some types of mold, such as *Fusarium,* infect the plant seed as it germinates. Other types of mold spores become airborne and infect the immature grain during the growing or silking stages. Under normal conditions, the sugar and moisture content of the grain prevents the spores from developing into molds and producing mycotoxins. However, unfavorable cropping conditions, poor management, or damage to the grain seed coat can cause the spores to grow and mycotoxins to form.

Before grain facility operators and feed manufacturers can control mycotoxin contamination, they must first understand its etiology. Mycotoxins are usually not evenly distributed in a given field, railcar, or bin. For example, only a few kernels of corn in an entire load could be contaminated with mycotoxin, the level of contamination on those few kernels of corn in an entire load could be contaminated with mycotoxin, and the level of contamination on those few kernels could be very high--500,000 ppm. Because of the uneven distribution of mycotoxins, grain sampling becomes critical when one seeks a meaningful measure of the level of contamination. Proper sampling requires drawing several small samples from various locations in the storage vessel.

Researchers have identified more than 350 different mycotoxins. Like their distribution, the chemical characteristics of the different types of mycotoxins also vary widely. In the U. S. the most commonly reported mycotoxins are aflatoxins, zearalenone, deoxynivalenol, T-2 toxin, and ochratoxin A. Several years ago, researchers added fumonisin to the list.

Deoxynivalenon (DON): DON--also known as vomitoxin--is produced by *Fusarium* fungi DON, at concentrations of 300 to 500 ppb in feed, and may indicate the presence of other mycotoxins. DON may cause reduced feed intake and milk yield in ruminants. Pigs are more sensitive to vomitoxin than poultry and other animals. In pigs, DON has depressed feed intake and weight gain, caused unthriftiness, and reduced performance. One key symptom is vomiting.

Prevention and Control Methods

Feed mill and grain handling facility operators can utilize specific strategies and technologies to minimize mycotoxin problems. Poor storage practices will enhance mold growth and mycotoxin production because mold spores are already present in feed grains. Feed manufacturers must store grain at less than 14% moisture. Feed ingredients must be either dry, oxygen-free, fermented, or treated with a mold growth-inhibiting chemical. Grain bins and feed handling equipment should be routinely cleaned. In silage crops, the application of good management practices during harvesting and feeding is important.

This involves:

- Harvesting the crop at the correct moisture content to assure rapid fermentation;
- Filling the silo at a rapid rate and packing the material tightly to exclude oxygen;
- Sealing the silo completely.

Silage preservatives may enhance fermentation. Proper removal and feeding of the silage from the silo will minimize secondary fermentation that may occur when loosened silage is exposed to the air for more than 6 to 10 hours, especially in summer. Good feed bunk management will prevent moldy feed from accumulating in the trough. Researchers have investigated the use of active clays as tools against the effects of mycotoxin contamination. The clays, such as sodium calcium aluminosilicates and bentonites, bind or adsorb the mycotoxins and prevent them from being absorbed from the animals' intestines.

In addition to the clay absorbents, mannanoligosaccharides, which are derived from yeast cells, may be effective against mycotoxin contamination. Mannose sugars in the mannanoligosaccharides (MOS) influence the immune system by stimulating the secretion of mannone-binding protein from the liver, which binds to the capsule of invading bacteria and triggers the complement fixation system. Trenholm et al. (1994) conducted an in vitro experiment in which MOS bound at least 80% of the zearalenone in a feed. Mahesh and Devegowda (1996) compared the aflatoxin binding capacity of a commercial aluminosilicate and a commercial MOS. At the highest level of inclusion, both products bound approximately 80% of the aflatoxin added to the feed. Subsequent studies found that MOS also binds DON. MOS may provide feed manufacturers new ways to counteract mycotoxins as well as pathogens and other toxins present in feed. However, in the U. S. neither clays nor MOS are approved as anti-mycotoxin feed additives. **--Barney Harris, Jr., excerpted from Feed Management, Oct '97, as reported in Veterinary News, Feb. 1998, Penn State University, University Park, PA.**

DIAGNOSIS OF BVD FOR VIROLOGY AND SEROLOGY

The following recommendation for BVD sample submission is presented to clarify selection of appropriate diagnostic samples. Keep in mind that laboratory evidence for BVD should be considered within the context of accompanying clinical signs and/or pathological examination.

1. *Diagnosing BVD in the live animal with clinical signs of acute infection:* Virus isolation is the preferred test. Submit a whole, unclotted blood sample (EDTA) from the affected animal. The sample must be obtained early in the course of the disease, preferably during the febrile period. Send the sample on ice or dry ice. DO NOT FREEZE the sample since virus isolation will be done on buffy coat cells.

   Submission of paired serum samples (sera collected early in the course of the disease and approximately 3 weeks later) from 5-10 exposed herdmates is also suggested. Serology results are diagnostic provided that animals have not been vaccinated between the two sampling points.

2. *Diagnosing BVD when from a dead animal where pathology is consistent with BVD:* It is best to submit the whole animal for a complete necropsy. If that is not possible, submit fresh tissues for virus isolation and formalin-fixed tissues for histopathology and immunochemistry. Samples of intestine (including Peyers patches), spleen, lymph nodes, lung and kidney are recommended for virus isolation. Send samples on ice or dry ice (DO NOT FREEZE) by overnight courier.

3. *Diagnosing BVD when abortions are the only clinical sign:* Submit the aborted fetus (or fetal tissues), a sample of placenta, and serum from the dam for virus isolation and pathologic examination. It submitting fetal tissues. collect and submit fresh tissues on ice for virus isolation and also formalin-fixed tissues for histopathology and immunohistochemistry.

   The serum sample can be used to determine if the darn was exposed to BVD virus prior to the abortion. A single serum sample for each individual animal is usually sufficient. Serological evidence for a BVD abortion is based upon a titer that is significantly higher than can be explained by the vaccination history. If more than one cow has recently aborted, collect and submit sera from each of these cows.

4. *Diagnosing BVD when repeat breeding (early embryonic death) is the only clinical sign:* Submit single serum samples from as many recent repeat breeders as possible for serology. Evidence of exposure to BVD virus is based on whether or not the titers are significantly greater than those expected from the vaccination history.

5. *Identifying persistently infected (PI) animals:* Submit a single serum sample from each animal to be tested for virus isolation. Samples should be taken either as precolostral samples or after the animal is at least 3 months of age to avoid interference with maternal antibody in virus isolation testing. Virus isolation from serum is preferable to serology since PI animals sometimes develop SN titer following vaccination, particularly with an MLV vaccine.

--These guidelines are based, in part, on recommendations of the Cornell University Veterinary Diagnostic Laboratory. *Dr. Gary Osweiler in Iowa State University VET·MED, SEP 97*, as reported in Veterinary Newsletter, Utah State University.

REASONS FOR TREATMENT FAILURES

Mastitis results when bacteria penetrate teat duct keratin, overcome the defenses in milk and multiply within the gland. The establishment of an infection is greatly influenced by the interaction of bacteria with milk leukocytes. These leukocytes function by phagocytosing bacteria and killing the organisms intracellularly. However, phagocytosis is inefficient in the udder due to the lack of an energy source, low opsonic activity, and interference caused by casein and butterfat. Therefore, antibiotics continue to be relied upon in attempts to treat clinical quarters during lactation. However, antibiotic treatment often fails.

Reasons for treatment failure include:

- √ lack of contact between bacteria and antibiotics
- √ due to scar tissue formation
- √ protection within leukocytes
- √ poor drug diffusion
- √ inactivation by milk and tissue proteins
- √ microbial resistance to antibiotics
- √ development of bacterial L-forms
- √ metabolically inactive organisms
- √ improper treatment procedures

For effective intramammary therapy, antibiotics must reach infection sites at concentrations exceeding the drug's minimum inhibitory concentration (MIC) and remain at adequate concentrations for sufficient time to kill or inhibit growth of the infective agent. Unfortunately, therapeutic concentrations may not be achieved in mammary tissue for a sufficient period of time when presently used doses of antibiotics are administered intrammarily. Infections are frequently refractory to intramammary therapy because of the inaccessibility to bacteria -- this inaccessibility is due to deep tissue lesions, swelling and reduced patency of milk ducts.

A hallmark of acute inflammation is the formation of inflammatory exudates composed of necrotic tissue debris, leukocytes, bacteria, fibrin and other blood components that occlude milk ducts draining areas of secretory tissue. The frequent therapy failures during acute mastitis are due, in part, to poor or uneven distribution of the drug throughout the intensely swollen udder parenchyma in which the duct system is either compressed or blocked by inflammatory products.

If bacteria and their toxins are eliminated (by host defenses combined with antibiotic therapy), inflammation subsides, occluded ducts open and milk composition returns to normal in several days or weeks. However, if occluded areas are not flushed at every milking and continued bacterial growth and toxin production occur, the bacteria may become inaccessible to drug action. **--Source: 1996 Western Canadian Dairy Seminar Proceedings, Vol. 8, Red Deer, Alberta, "Clinical Mastitis: To Treat or Not to Treat," p. 355; Udder Topics - Vol. 20, No. 5, Oct - Nov 1997 -- Nebraska Veterinary and Biomedical Sciences Newsletter, Volume 27, Number 1, January 1998, as reported in Animal Health Spectrum, Mississippi State University, Volume 9, No. 1, Spring 1998.**

FDA APPROVES SELENIUM AS ANIMAL FEED ADDITIVE

On August 25, FDA adopted without change provisions of an interim rule approving use of selenium as an additive in animal feeds. The interim rule was published in 1995 in response to Congressional action overturning the agency's attempt to stay earlier amendments to selenium food additive regulations.

The published rule provides for currently acceptable levels of selenium supplementation of animal feed, i.e., levels not to exceed 0.3 parts per million in completer feeds of chickens, swine, turkeys, sheep, cattle, and ducks; in feed supplements for sheep not to exceed 3 mg per head per day; in free-choice salt-mineral mixes for sheep up to 90 ppm but not to exceed 0.7 mg per head per day; and, for beef cattle, up to 120 ppm in a mixture for free-choice feeding not to exceed an intake of 3 mg per head per day. **--Food Chemical News, Vol. 39, No. 28, Sept. 1, 1997; AABP Newsletter, Sept. 1997, as reported in Veterinary Newsletter, Utah State University.**

NUTRITIONAL RISK FACTORS IN THE ETIOLOGY OF LEFT DISPLACED ABOMASUM IN DAIRY COWS: A REVIEW

The transition period occurring 2 wk prepartum through 2 to 4 wk postpartum is the major risk period in depression of intake and the slow postpartum increase in intake are risk factors causing lower ruminal fill, reduced forage to concentrate ratio, and increased incidence of other postpartum disorders. Uncomplicated ketosis, retained placenta, metritis, and hypocalcemia at parturition are risk factors for left displaced abomasum. Excessive amounts of concentrate during the prepartum period increase the risk of left displaced abomasum, which may occur from the lower ruminal fill caused by greater prepartum intake depression and reduced forage to concentrate ratio, decreased ruminal motility from lower ruminal fill and higher volatile fatty acid concentration, and decreased abomasal motility and emptying from higher concentrations of volatile fatty acids. Effects of volatile fatty acids on motility may be exacerbated by low ruminal absorption of volatile fatty acids during the transition period. Minimal intake of concentrate during the prepartum period may increase the risk of left displaced abomasum through failure to increase the absorptive capacity of the ruminal papillae and failure of the microbial population of the rumen to adapt prior to the intake of high energy postpartum diets. Increased risk of left displaced abomasum in cows that are hypocalcemic at parturition may be due to decreased ruminal and abomasal motility.

Because of low feed consumption, the transition period is the major risk period in the etiology of LDA. Feeding and management practices that prevent other postpartum disorders reduce the risk of LDA. Ketosis and LDA are closely related postpartum disorders, and cows that have excess BCS at parturition are at increased risk of ketosis and LDA.

Both excessive and minimal amounts of dietary concentrates during the prepartum period may increase the risk of LDA. More research is needed on lead feeding strategies. Prepartum concentrate lead feeding of 0.5% of BW with an upper limit of 0.75% of BW is recommended. For herds that are not fed TMR, postpartum concentrate DM can be increased at the rate of 0.20 to 0.25 kg/d until peak concentrate intakes are reached; concentrates should be fed at least three to four times daily. A TMR that has been formulated to control F:C and to consider nutritional needs of early postpartum cows is recommended.

There is increased risk of LDA in hypocalcemic cows at parturition, suggesting a role for the formulation of prepartum diets for dietary cation-anion difference in the prevention of LDA. Although a pelleted TMR increased the incidence of LDA, research is needed to determine the critical forage and TMR physical form for preventing LDA. Rations composed entirely of corn silage should not be fed to dry cows. Feed bunk management is an important risk factor for LDA that should be monitored closely on commercial dairies. **--R. Shaver, Journal of Dairy Science, Vol. 80, No. 10, pp. 2449-2453, Oct. 1997; AABP Newsletter, Dec. 1997. --Nebraska Veterinary and Biomedical Sciences Newsletter, Vol. 27, No. 2, February 1998, as reported in Animal Health Spectrum, Mississippi State University, Volume 9, No. 1, Spring 1998.**

E. *COLI* IN WATER TROUGHS AND MOIST FEEDS

Washington State University began research into the on-farm ecology of *E. Coli* 0157:H7 in 1990. The research has come a long way in the past seven years from knowing very little about this agent to a point where we can begin talking of farm-level strategies, states Dale Hancock, DVM, veterinarian at Washington State University. The research conducted by Hancock found that *E. coli* 0157:H7 exists on a majority of cattle farms in the U.S. However, less than five percent of the cattle had *E. coli* 0157:H7 detected in their feces.

According to Hancock, *E. coli* 0157:H7 occurs naturally and sporadically in the gastrointestinal flora of several animals and humans. *E. coli* 0157:H7 can be found in deer, sheep, dogs, horses, flies and birds. However, there is evidence that indicates that most *E. coli* 0157:H7 infections in humans are foodborne and that the ultimate source of the agent in food is a non-human reservoir. Since *E. coli* 0157:H7 can be sporadic, it can be hard to detect in one research test. In early studies, *E. coli* 0157:H7 appeared to be present on a minority of farms. Repeated studies demonstrated that *E. coli* 0157:H7 is present on most farms. In one study 60 dairy farms were sampled for *E. coli* 0157:H7 on a single occasion. The first sample found only five farms with *E. coli* 0157:H7 present. Later sampling was conducted each month on eight of the farms that tested negative on the first sample. After repeated sampling *E. coli* 0157:H7 was found present in four of these eight farms. According to Hancock this research proves how sporadic *E. coli* 0157:H7 can be. Also, the agent is most often undetectable, or detectable at very low percentages.

Hancock has observed that fecal shedding of *E. coli* 0157:H7 is more common in warm weather. Research was conducted in nine herds where samples were taken monthly for approximately one year. The data revealed that *E. coli* 0157:H7 was more prevalent in June through October than in December through March. This same pattern is consistent with other research conducted in an English dairy herd.

Currently, Hancock's research focuses on water troughs and on feed in which E. *coli* can survive. The 0157:H7 strain can survive in water troughs for up to four months and can multiply during this time. In feed, E. *coli* 0157:H7 can replicate to infectious doses during summer months. According to Hancock, most dairy farms clean water troughs in intervals of six months or greater. During this time the sediments can become several centimeters in depth. He says that *E. coli* can multiply increasing by I ,000 to 10,000-fold in moist feeds. "Producers should clean feed bunks out after a rain and make sure there are no slime layers on the water troughs," Hancock says.

Research on commercial feeds found that half of the feeds contained detectable E *coli,* indicating widespread fecal contamination. Due to limited sampling, naturally occurring *E. coli* has not been detected in commercial feeds. Feeds play a key role in new *E. coli* strains due to the high daily intake and the ability of *E. coli* to multiply in feeds when moisture is added.

According to Hancock, the likelihood that cattle will become colonized by *E. coli* is a function of exposure dose and susceptibility. The exposure dose is influenced greatly by replication in feeds and/or water troughs. Susceptibility to colonization by a transient *E. coli* strain is a function of age and factors, such as temporary feed withholding, which disturbs the gastrointestinal flora, states Hancock. Traditional means of controlling infectious agents such as eradication or test and removal of carrier animals do not appear to be feasible for this agent, states Hancock. "Our main goal is to minimize *E. coli,"* Hancock says. "Farm management practices can provide a practical means to reduce the prevalence of *E. coli* 0157:H7 in cattle on farms and in slaughter plants," **--LCI Food Safety Digest JAN/FEB 98 as reported in Herd Health Memo No 10 April 1998, University of Kentucky, Lexington, KY.**

DETERMINING CAUSES OF ABORTION

Practitioners, as well as pathologists, are aware of the difficulties involved in diagnosing the cause of an abortion. There are many potential causes in addition to infectious agents; toxic reactions, hormonal changes, concurrent illness in the dam, genetics, and dietary changes to mention a few. Infectious agents may be bacterial, viral, fungal, or protozoal, and are the types which pathologists are most likely to make a diagnosis..

Practitioners can improve the chance for a pathologist's diagnosis of a fetal abortion by following certain important guidelines in submitting tissue to the laboratory. Tissues should be as fresh as possible and should include the following in order of importance:

- placenta
- lung
- brain
- skin
- eyelid
- intestine (tied off - not opened up)
- skeletal muscles
- heart, kidney, liver, spleen, thymus

Remember to identify each specimen and to include samples of any identifiable gross lesions. Samples should be preserved in 10% formalin as soon as possible. Tissue degeneration is the worst enemy of the pathologist. Tissue to formalin ratio should approximate 1:10. A complete and accurate history of circumstances surrounding the abortion is critical to the investigation and diagnosis.

For more information or assistance, please contact:

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HIGH PREVALENCE OF BOVINE LEUKOSIS VIRUS IN U.S. DAIRY HERDS

A 1980 national study of BLV in Canada showed that 40% of its dairy herds and 11% of its beef herds were infected. Within-herd seroprevalence in dairy cattle was 18 times higher than it was in beef cattle. BLV control programs have been established in member countries of the European Economic Community (EEC) since the 1980s. According to a 1987 report, seroprevalence in the entire EEC cattle population rarely exceeds 0.5% to 1.5%. BLV infection has been reported by many other countries, but valid national estimates of seroprevalence are rare. In the U. S., previous studies of BLV have been restricted to a few sates, small regions within those states, or single herds. Seroprevalence varied among those herds from 0 to 95% of the cattle sampled. Data from herds participating in the leukosis certification program in New York suggest that when the seroprevalence is high, morbidity and mortality from malignant lymphoma may be economically significant. In herds with low seroprevalence, morbidity and mortality may not be economically significant. Other economic losses associated with BLV infection are due to restrictions on trade of infected animals and germplasm. *Standards for Certification of Cattle Herds as Bovine Leukosis Virus Free* was published by the Bovine Retrovirus Committee of the U. S. Animal Health Association. An assessment of BLV prevalence in U.S. dairy operations was part of the NAHMS Daily '96 study. Between February and May of 1996, randomly selected dairy operations with at least 30 milk cows in 20 states representing 79% of the U.S. dairy cow population were visited by federal or state animal health officials. Blood samples were collected from milk cows on 1,006 participating operations and sent to the USDA's National Veterinary Services Laboratories for BLV testing using the AGID test. Dairy '96 results showed that 89% **of U.S. dairy operations had cattle seropositive for BLV.** Herd prevalences in the west, midwest, and northeast regions were 87 to 89%, while the southeast region had a herd prevalence of 99%. Virtually all animals tested in some herds were seropositive. In the southeast region, the individual animal prevalence was higher than in other regions. Both herd and individual animal prevalences were slightly higher on operations with 200 or more cows than in smaller herds. **Individual animal seroprevalence was at least 25% in 75% of the positive herds.** These national estimates of BLV infection show a high prevalence and broad geographic distribution of the infection in the U.S. The high individual animal prevalence in seropositive herds indicates that culling alone will not be a cost-effective method for reducing BLV seroprevalence in those herds. Control strategies in which culling and risk factor management are combined may be the only cost-effective methods for reducing incidence of infection in high-prevalence herds. An accurate analysis of the risk factors for BLV infection will enhance the success of such control efforts where warranted. For more information, contact the Centers for Epidemiology & Animal Health, USDA:APHIS:VS, Attn, NAHMS; 555 South Howes, Fort Collins, CO 80521; (970) 490-8000. **--ISU Vet Med, Jan '98, as reported in Veterinary News, Feb. 1998, Penn State University, University Park, PA.**

FRESH DAIRY COW IMMUNE FUNCTION

BA Mallard and co-workers reported on immune alterations in the dairy cow around the time of calving in a recent Journal of Dairy Science article (J Dairy Sci 1998 Feb;81(2):585-595). The article was titled "Alteration in immune responsiveness during the peripartum period and its ramification on dairy cow and calf health." They stated the following: "Substantial evidence indicates that innate and acquired defense mechanisms are lowest from 3 wk precalving to 3 wk postcalving. This lowered responsiveness includes aspects of systemic and mammary gland immunity that may account, at least in part, for the increased incidence of peripartum disease. The physical and metabolic stresses of pregnancy, calving, and lactation may contribute to this decrease in host resistance and the subsequent increase in disease incidence. However, variation among cows in their host resistance mechanisms suggests that genotype and phenotype may possibly be used to identify cows that are able to mount beneficial immune responses over the periparturient period. Our own studies suggest that cows may be categorized as high or low responders based on the peripartum antibody responses to ovalbumin and Escherichia coli J5. Low responders were hyporesponsive to these test antigens and had a higher incidence of peripartum diseases, particularly mastitis. In many species, a functional link exists between the immune and endocrine systems, and, during periods of stress or physical injury, neuropeptides and neuroendocrine hormones function as immunomodulators. Initial investigations of peripartum cows reveal positive relationships between growth hormone kinetics and profiles of antibody response. Whether hormone fluctuations during the periparturient period are responsible for the alterations observed in immune responsiveness remains uncertain." **--Reported by W. Dee Whittier in Food Animal Veterinarian, Spring 1998.**

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