VIRGINIA COOPERATIVE EXTENSION SERVICE

EXTENSION DIVISION - VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY - BLACKSBURG, VIRGINIA 24061

VIRGINIA-MARYLAND
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OF
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VIRGINIA VETERINARY NOTES

November-December 1981 No. 11

WHAT'S INSIDE - - - - -

Virginia Veterinary Medical Association Annual Meeting
Hotel Roanoke - February 18-21, 1982
Roanoke, Virginia

G. A. MacInnis
Extension Specialist

OUR APOLOGIES

The editors of Virginia Veterinary Notes apologize for the long delay in mailing the September and October newsletters. The problem was not with us but has been traced to an apparent computer foulup in printing the necessary mailing labels. While we thought our eagerly awaited publication was being enjoyed all over the country, it was in reality sitting totally neglected in a distribution center. For this problem we are sincerely sorry and promise to take steps to prevent a repetition of this regretable snafu.

CHLORAMPHENICOL RESIDUES NOW DETECTABLE BY USDA: FDA TO TAKE REGULATORY ACTION AGAINST VIOLATORS.

Chemists with the USDA's Food Safety and Inspection Service (FSIS) have recently developed a method to detect residues of the antibiotic drug Chloramphenicol in tissues of food-producing animals.

In recent weeks USDA has reported finding several samples of veal muscle tissue which contained significant levels of Chloramphenicol, an antibiotic drug long suspected of being widely used illegally in food-producing animals. This drug, when used in humans, has sometimes been associated with such serious adverse effects as aplastic anemia and other blood-related disorders.

USDA and FDA are undertaking collaborative inter-laboratory studies of the new Chloramphenicol residue detection procedure in their facilities. Because the procedure contains a confirmatory step based on mass spectroscopy, it will now allow FDA to take regulatory action against those who use Chloramphenicol in food-producing animals. This regulatory action will be based upon USDA's report of a residue which is supported by inspectional evidence.

Although it is FDA policy to permit veterinarians to use any legally obtainable drug in the treatment of a food-producing animal, it is also policy that the veterinarian is responsible for any illegal drug residue found in any edible tissue of a food-producing animal intended for human consumption.

Because Chloramphenicol is not the subject of an FDA New Animal Drug Application (NADA) for use in food-producing animals, nor has any permissable tolerance been established, any confirmed residues found in edible tissue of food-producing animals will cause that carcass to be considered adulterated as defined in the Federal Food, Drug, and Cosmetic Act.

BVM Update
Bureau of Veterinary Medicine
September 29, 1981

SUCCESS IS NO ACCIDENT

One real test of your self confidence and self control is dealing with the difficult client. Unfortunately every practitioner faces an

occasional confrontation with an animal owner who seems determined to cause trouble because of dissatisfaction with service or fees.

The most effective approach in dealing with these clients is to remain calm and composed while attempting to patiently answer their accusations. Much easier said than done, but resist the temptation to argue and have the conversation degenerate into a bitter shouting match. Don't be afraid to admit a mistake. Patience and understanding will accomplish much more than loss of self control and abrasiveness.

In the long run the "customer" is almost always right.

CONTROL OF LEG PROBLEMS IN SWINE

Condition	Cause(s)	Prevention	Treatment
Rickets and osteomalacia	Deficiency of Ca, P and/or vitamin D. Congenital and other causes	Adequate fortification of the diet with Ca, P and vitamin D.	Supplement the diet with Ca, P and vitamin D. Injections of vitamin D.
Osteomyelitis	Infection	Hygiene. Prevent tail-biting	Chemotherapy (may not be successful)
Osteoarthritis	Not known. Poor conformation. Earlier rickets? Zinc poisoning.	Genetic selection. Good early nutrition? Exercise?	None suggested
Arthritis	Infections. Injury	Hygiene. Ensure early suckling. Enysipelas vaccination	Chemotherapy
Leg weakness	Not known. Poor conformation?	Avoid individual confinement. Provide exercise. Good early nutrition. Genetic selection?	Move to pasture or dirt pens? Provide exercise.
Foot lesions	Rough concrete	Avoid rough concrete. Supplementation of the diet with biotin. Genetic selection?	Supplementation of the diet with high levels of biotin?
Spraddle leg	Not yet fully established. Congenital. Choline deficiency.	Genetic selection. Supplementation of the diet with choline. Avoid slippery floors.	None. Bind hind legs for first few days.

Robert Blair - Feedstuffs, 9/7/81
as reported in Penn State University
"Veterinary News"
November 1981

SELENIUM DEFICIENCY IN CATTLE IN KENTUCKY

Awareness of the importance of selenium deficiency in livestock has increased in recent years. This deficiency is thought to be a growing problem in central Kentucky. Selenium deficiency in cattle has been associated with white muscle disease in calves; retained placentas and reproductive problems in cows; ill thrift in yearling cattle; weak, premature, and stillborn calves; and downer cows. Due to problems with weak calves and undefined abortions, a survey was conducted to determine the incidence of selenium deficiency in cattle presented to the University of Kentucky Livestock Disease Diagnostic Center.

Two hundred eighty-seven bovine liver samples were analyzed for selenium between February, 1980 and February, 1981. Selenium analyses were completed on digested liver samples using atomic absorption spectroscopy with a graphite furnace. The liver samples analyzed were obtained from animals submitted for necropsy at the Diagnostic Center. Pathology data was available on each case to allow correlation of gross and microscopic findings, especially changes observed in cardiac or skeletal musculature, with liver selenium levels. The animals included in the survey were grouped according to age. The first group consisted of aborted fetuses and stillborn animals. The second group was composed of neonates or newborn animals up to one week of age. The third group was the largest group and represented calves older than one week but less than 6 months of age. The fourth group was composed of animals greater than 6 months of age. Animals with liver selenium levels between 0.02 ppm and 0.15 ppm were considered deficient; levels between 0.16 ppm and 0.25 ppm were considered marginal. Normal or adequate liver selenium levels were considered to be 0.25 ppm to 0.50 ppm. High liver selenium levels and toxic liver selenium levels were 0.75 ppm to 1.25 ppm and 1.25 ppm and above, respectively.

The results of the 187 bovine liver sample analyses for selenium were as follows: 147 samples (51.2%) were deficient, 69 samples (24%) were marginal, 65 samples (22.6%) were adequate, and 6 samples (2.09%) were in the high or toxic range. When animals were grouped according to age the selenium level was deficient in 16 (55.1%) and marginal in 5 (17.2%) of 29 fetuses or stillborn animals tested. The selenium level was deficient in 23 (57.5%) and marginal in 5 (12.5%) of 40 newborn calves less than one week of age. The selenium level was deficient in 70 (47.6%) and marginal in 45 (30.6%) of 147 calves between one week and 6 months of age. The selenium level was deficient in 10 (38.4%) and marginal in 5 (19.2%) of 26 animals greater than 6 months of age. The precise age was not available for 45 animals and they were not included in the specific age groups. Microscopic evidence of myodegeneration was observed in 3 of the selenium deficient fetuses or stillborn animals (18.8%)and in 11 of the selenium deficient and selenium marginal newborn calves less than one week of age (39.2%). Microscopic evidence of myodegeneration was observed in 75 of the selenium deficient and selenium marginal calves between one week and 6 months of age (65.2%) and in 7 of the selenium deficient and selenium marginal

animals greater than 6 months of age (46.6%).

This biased survey suggests that selenium deficiency may be a severe problem in cattle in Kentucky. Additional controlled studies or surveys are needed to document the true incidence of selenium deficiency in Kentucky. White muscle disease of calves appears to be an important cause or a contributing factor to calf mortality. Affected calves may die suddenly without exhibiting signs of illness, especially in the myocardial form of the disease; however, calves more commonly exhibit progressive weakness, poor growth, or secondary or associated pneumonia and/or diarrhea. These calves are often ineffectual nursers or have difficulty standing and nursing and there is often secondary aspiration pneumonia. Selenium deficiency may also be an important cause of abortions, stillbirths, and weak calves at birth.

Selenium may be supplemented in the diet by addition to salt, mineral mix, or grain ration. Dietary intake is most effective supplemented daily in amounts of selenium equal to 0.1 to 0.3 ppm of the total ration. It may also be administered by injection. Blood levels following injection rise rapidly, plateau for approximately 3 weeks and return to pre-injection levels by 4 weeks. Injections during the last trimester of pregnancy, particularly during the last three weeks, may be the best approach to insure an adequate selenium state in the newborn.

Selenium analysis is time consuming and expensive. The Livestock Disease Diagnostic Center performs selenium analyses; however, this is not available on an emergency basis and the reporting time will generally be 1 to 2 weeks. Discretion must be used in the selection of meaningful samples for analysis. Ten ml of non-hemolyzed refrigerated or frozen serum or 10 gm of fresh frozen liver are required for analysis. R. C. Giles, Jr., D.V.M., Lynne McMaine, B.S., Pamela Weaver, B. S. - Livestock Disease Diagnostic Center.

Notes for the Veterinary Practitioner
University of Kentucky
September 1981

ZEARALENONE AFFECTS LIBIDO IN BOARS

In a study at Purdue University, zearalenone, a toxin produced by Fusarium fungi, was fed to growing boars. Littermate pairs of crossbred boars were maintained in an environmentally controlled confinement facility. One member of each pair was fed a diet containing 40 p.p.m. purified zearalenone (97% pure) from 14 to 18 weeks of age, while the other member received the same diet without the toxin. Consumption of zearalenone did not affect body weight, feed efficiency or total feed intake during the feeding or after withdrawal. On the other hand, plasma levels of testosterone and luteinizing hormone were depressed during feeding of zearalenone.

A libido score was assigned all boars based upon their behavior during five minute exposure to a female in estrus. Boars fe

had significantly lower libido scores during the early part of the experiment, but no difference was observed by 33 weeks of age. Also by this time, circulating levels of testosterone and luteinizing hormone were similar in the two groups of boars. All of the boars were castrated at 36 weeks of age and no differences were observed between the two groups for testicular weight, epididymal weight, spermatid production or motility of cauda epididymal sperm. Histological examination of testicular tissue revealed no obvious differences between the boars in seminiferous tubule components or Leydig cells.

The data demonstrated that a level of 40 p.p.m. zearalenone fed for four weeks to prepuberal boars depressed testosterone and luteinizing hormone levels during feeding and delayed the development of sexual behavior. However, feed intake, growth and subsequent sperm production were not affected.--K. L. Esbenshade, Ph.D., Swine Extension Specialist, North Carolina State University.

"Veterinary Information"
N. C. State University
September-October, 1981

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