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Pathogen Blamed for Ovine Abortions in Kansas

Submitted by Tawfik Abollelail, Kansas State University Veterinary Diagnostic Laboratory

The rickettsial pathogen Coxiella burnetii is the causative agent of Q-fever in humans and is also capable of infecting a wide variety of other hosts, including arthropods, fish and various domestic and sylvatic mammals. Goats, sheep, cats, and cattle are the species most frequently infected. Coxiella burnetii is a ubiquitous organism in North America, but to our knowledge, it has not been previously identified as a cause of abortion in Kansas.

In the spring of 2000, a flock of 500 sheep had at least 15 late-term abortions. Coxiella burnetii was confirmed as the causative agent, and all other tests for the causes of abortion in sheep were negative. Gross lesions were confined to the placentas. The cotyledons and intercotyledonary areas were diffusely and markedly thickened by edema and inflammation, and the intercotyledonary areas were often thickened and leather-like with surface exudate.

Histologically, the gross lesions were the result of multi-focal areas of necrosis and marked exudation of fibrin and inflammatory cells, particularly neutrophils. The syncytial trophoblasts and macrophages contained numerous large numbers of organisms measuring approximately 1.0µ (micron).

The organism is difficult to grow in the laboratory and in this case was definitively identified in fixed placental tissue via immunohistochemical staining using monoclonal antibody against the organism.

Coxiella burnetii, in its spore stage, can withstand harsh environmental conditions and especially desiccation. Domestic animals are probably most frequently infected by ticks of which more than 40 species can be infected by the organism. Transmission to other animals and humans can be via ticks, airborne in contaminated dust, or accidentally ingested via infected fetal fluids or milk.

Other, more common causes of abortion in sheep include campylobacteriosis, chlamydiosis, and toxoplasmosis. All of these diseases can have various degrees of placentitis. Brucella ovis, more commonly a cause of epididymitis in rams, can also occasionally cause abortions in ewes with a grossly obvious placentitis. Diagnostically, submission of placental tissue along with the aborted fetus is critical for diagnosis of each of these diseases.

As with chlamydiosis and toxoplasmosis, coxiellosis can be a zoonotic disease. In humans, it causes a flu-like condition with an interstitial pneumonia and can be quite severe. As such, care should be taken in handling all aborted lambs, placentae, and fetal fluids.

KSU Diagnostic Lab ReportsTrends in Salmonella Serotypes 1995-1999

The frequency of isolation of most Salmonella serotypes has remained fairly constant. However, a few serotypes have had notable changes. Salmonella serotype Typhimurium isolates in bovines have decreased in recent years, but Salmonella serotype Typhimurium (Copenhagen) isolates have been fairly constant. During the same time period Salmonella serotype Newport has increased drastically. See Table 1.

Further analysis is needed to determine if the increase in isolation of Salmonella serotype Newport and the decrease in isolation of Salmonella serotype Typhimurium are real, and if so, what factors have lead to this change in serotype.

During the first three weeks of January, 2000, another five Newport isolates were isolated. However, none has been recovered from late January to the middle of February. It may be that Salmonella serotype Newport is emerging in Kansas, but further monitoring is essential for confirmation. It has been previously published that Salmonella enterica subspecies enterica serotype Dublin is an emerging pathogen in the northeastern United States.(1) We

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Also in this issue

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

Bulk Tank Milk Culturing May Help Reduce Mastitis

Dan N. Waldner, Dairy Specialist
Oklahoma State University

Reprinted from Dairy Lines, August 2000.

Although based on limited scientific data, the practice of bulk tank milk (BTM) culturing may provide clues to help reduce or prevent mastitis problems. It may be used to identify the presence or absence of a bacterial group and predominant bacterial groups.

Sampling

The more often BTM is sampled, the more useful the information. Samples taken over consecutive days or weeks are most useful. Use caution when interpreting results from a single BTM sample.

To get the most out of bulk tank sampling, follow these suggestions:

1. Take samples for four or five days in a row.
2. Agitate the milk in the bulk tank for 10 to 15 minutes before sampling.
3. Take samples from the top of the tank with a sterile syringe and needle or vial to avoid contamination from the outlet valve. If sampling from the top is not possible, allow a gallon or two of milk to flow through the outlet valve before taking a sample in this manner.
4. Freeze the sample immediately and pack for shipment to ensure the sample will stay frozen until it reaches the laboratory. It is a good idea to not ship samples to the laboratory after Wednesday in order to avoid long storage times.
5. If results are inconclusive or inconsistent with current management practices, retake samples or identify and sample individual cows with high somatic cell counts to provide further information.

Predominate Bacterial Isolates

Bacterial isolates from BTM are typically a mixture of various groups. Theoretically, any bacterial isolate from BTM could arise from an intramammary infection. The probability of an isolate originating from a mammary infection depends on the bacteria. For example, coliforms and environmental streptococci may originate from intramammary infections, but more common sources of elevated counts caused by these bacteria are milking wet udders, organic soil in milk lines, cracked inflations, inadequately heated wash water and inadequate milk cooling.

Interpreting Results

The first question to ask when interpreting BTM cultures is whether or not the samples are positive for *Streptococcus agalactiae, Staphylococcus aureus* or *Mycoplasma* spp. Presence of these pathogens in BTM almost always indicates the presence of infected quarters in the herd. But negative culture results do not necessarily mean the herd is negative for infection caused by the pathogens. It is important to remember that BTM cultures are not useful as indicators of mastitis prevalence in the herd, nor should they be used as a substitute for determining infection incidence and prevalence based on quarter milk samples.

Use the table below to help determine the response to BTM culture results.

Interpreting results of bulk tank milk cultures

Bacteria Type	Source	Suggested Control Procedures	
Streptococcus agalactiae	Infected udders	•Use separate towels to wash and dry udders •Use postmilking teat dip	•Dry treat all cows at dry-off
Staphylococcus aureus	Infected udders	•Use separate towels to wash and dry udders •Use postmilking teat dip •Dry treat all cows at dry-off	•Cull chronically infected cows •Milk infected cows last
Mycoplasma species	Infected udders Respiratory tract Urogenital tract	•Follow proper milking procedures •Use premilking teat disinfectio •Use postmilking teat dip •Milk infected cows last	•Culture all replacement animals •Culture all cows and heifers at calving •Cull infected cattle when possible •Maintain a closed herd
Non-agalactiae Streptococci	Environment	•Milk only clean, dry udders •Improve cleanliness of housing environment •Use premilking teat disinfection	•Use postmilking teat dip •Dry treat all cows at dry-off
Coliforms	Environment	•Milk only clean dry udders •Improve cleanliness of housing environment •Use premilking teat disinfection	
Coagulase-negative staphylococci	Environment Skin	•Milk only clean dry udders •Improve cleanliness of housing environment	•Use postmilking teat dip •Dry treat all cows at dry-off

Source: National Mastitis Council, 1999. Laboratory Handbook on Bovine Mastitis

Watch for 10 Spring Plants Poisonous to Kansas Cattle

John Pickrell and Fred Oehme, Kansas State University, College of Veterinary Medicine, Department of Diagnostic Medicine Pathobiology

Spring brings lush green plants to Kansas pastures. It is important to watch carefully for 10 common poisonous plants. The seven most toxic of these are described with references for additional detail. A CD-ROM with color pictures of the plants and information about their effects and collection for identification is available for $10.69 from the College of Veterinary Medicine Integrated Technology Center.

Ten Kansas plants risky to cattle in the spring and their poison potential:

Japanese yew	(Taxus)	nervous signs and sudden death occurs
Milkweeds	(Asclepias)	nervous and gastrointestinal effects, sudden death
Locoweed	(Astragalus)	nervous signs, erratic behavior
Poison hemlock	(Conium)	nervous, neuromuscular effects, nicotinic signs
Cockleburr	(Xanthium)	hepatic failure
Fireweed	(Kochia)	hepatic failure
Mustards	(Brassica)	skin changes, hepatogenous photosensitivity
Tansy mustards	(Descurania)	skin changes, hepatogenous photosensitivity
Oak	(Quercus)	renal and gastrointestinal signs
Death camus	(Zygadenus)	cardiac effects, hypotensive shock

Japanese yew is an ornamental grown throughout North America and Kansas. Toxic alkaloids are present in the bark, evergreen leaves and seeds, but not in the fruit. As little as 6 to 8 ounces of leaves will kill a cow. Cattle seem to find Japanese yew quite palatable and may seek out the yew plants. Cattle eating toxic quantities are often found dead, without other signs. Those affected show gaseous distress, diarrhea, tremors, dyspnea, dilated pupils and respiratory difficulty, weakness, collapse, coma, convulsions or seizures, bradycardia and circulatory failure. Diagnosis depends on history and finding remnants of the plant in rumen content.

Locoweeds are present mostly in the western half of Kansas. Swainsonine (the toxic principle) causes depression, emaciation, incoordination, dry lusterless hair and erratic behavior. Cattle who have eaten locoweed will seek it out in the future. There are no gross lesions, but there are suggestive histologic lesions in the brain.

Poison hemlock is found in roadside ditches and damp or disturbed areas throughout Kansas. Piperidine alkaloids (coniine and others) are present in vegetative plant parts. Animals become weak, ataxic, stagger, experience convulsions, and develop a weak thready pulse. Ascending paralysis leads to slow breathing and death from respiratory failure. The toxin is excreted by the lungs and kidneys. Both have a typically diagnostic mousy odor as may the rumen contents.

Cockleburr is in fields, disturbed areas and along the shores of ponds, streams and rivers. Carboxyatractyloside is present in sprouted seedlings called dicotyledons. Although more common in pigs, cattle also show weakness, ataxia and appear to be blind by stumbling over objects. Calves have convulsions. Lesions include acute hepatitis, nephritis, gastroenteritis, and possibly edema fluid in the peritoneal cavity, thoracic cavity, pericardial sac and gall bladder wall.

Fireweed grows throughout grazing areas. This plant's toxin is likely to cause a liver disease in the Kansas spring. Disease is intensified by dietary or water sulfates. Photosensitivity and polioencephalomalacia have been associated with this plant.

Oak is found in most deciduous woods and is toxic to the gastrointestinal tract of all grazing animals. Gallotannin is in the young leaves and shoots in the spring. Cattle experience gastrointestinal tract irritation, anorexia, rumen stasis and constipation followed by the appearance of dark, tarry stools. They often have a dry muzzle, urinate frequently and develop a rapid weak pulse several days after ingestion as the intoxication progresses. Necropsy lesions may include perirenal edema, nephrosis and gastroenteritis.

Death camus is present in foothill grazing lands, boggy grasslands and low open woods. The plant's bulb is especially toxic in early spring and will cause salivation, vomiting, muscle weakness, ataxia and collapse. A fast, weak pulse, coma and death also occur. It is not uncommon to find dead cattle with no preceding signs. There are no consistent necropsy lesions because death occurs so rapidly.

Aiello S ed: 1998. *The Merck Veterinary Manual-Toxicology;* Merck and Co, White Stackhouse, NJ, pp 2108 – 2139.

Osweiler GD: 1996. *Toxicology, The National Veterinary Medical Series.* Williams and Wilkins, Philadelphia, PA, pp 361 – 408.

Murphy M: 1995. *A Field Guide to Common Animal Poisons.* Iowa State University Press, Ames, IA, pp 58, 62, 110, 115, 144, 147.

Toxicology: What Does the Diagnostic Laboratory Need?

John Pickrell and Fred Oehme, Kansas State University, College of Veterinary Medicine, Department of Diagnostic Medicine Pathobiology

"You mean you can't do the test with the samples I sent?"

Problem-solving efforts can be delayed periodically if incorrect samples or information are sent to the diagnostic laboratory. Here is a review of the materials and information the KSU Diagnostic Laboratory needs to give optimum service to you, your clients, and your patients.

Careful sample collection, preparation, and submission are the first steps. When combined with complete written case information, rapid and credible results will follow.

Tissue samples are key. In cases of suspected infectious problems or poisonings, too much is always better than too little. Extra material sent to the laboratory can always be ignored, but good samples for histopathology, organism cultures, or chemical analysis cannot be performed on tissues from decomposed carcasses or inadequately preserved samples. From live large animals, at least one large EDTA or heparinized tube of whole blood should be collected. At least one large red-top tube of serum should be submitted. Urine is an excellent sample, as is feces or vomitous from animals demonstrating digestive tract problems. Samples of feed, hay, water and plants that are accessible to the animals, provide environmental specimens for etiologic investigations.

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Bacterial Name Changes of Veterinary Importance

Jerome Nietfeld, DVM, Veterinary Diagnostic Laboratory, Kansas State University

Linda Cox of the Kansas State University veterinary diagnostic laboratory compiled a list of bacterial name changes that are of veterinary importance. Some of the changes are quite recent, while a few have been around for a few years. A partial listing of those changes is in the table at right.

In 1980, two species in the genus Chlamydia (trachomatis and psittaci) were recognized. Chlamydia isolates that contained glycogen within their inclusions and were susceptible to sulfa drugs were classified as C. trachomatis and everything else as C. psittaci.

In the past 10-15 years there have been many advances in the ability to detect and characterize Chlamydia isolates. This led to the recognition of several biotypes or groups within the genus and to the creation of two new species: Chlamydia pecorum and Chlamydia pneumoniae.

In 1999, a new classification system for chlamydiae based on their genetics, patterns of virulence, antigens, and animal species affected was adopted. In this system the genus Chlamydia was split into two genera, Chlamydia and Chlamydiophila (Chla.my.do'phil.a.), that include nine species. The new classifications are listed at right.

Online version has appendix of table in text for copy and paste.)

Changes in bacterial nomenclature

New Name	Old Name	Year of change
Arcanobacterium pyogenes	Actinomyces pyogenes	1997
Actinobaculum suis	Eubacterium suis Actinomyces suis	1997
Branchyspira hyodysenteriae	Serpulina hyodysenteriae Treponema hyodysenteriae	1998
Branchyspira innocens	Serpulina innocens Treponema innocens	1998
Branchyspira pilosicoli	Serpulina pilosicoli	1998
Burkholderia mallei	Pseudomonas mallei	1993
Burkholderia pseudomallei	Pseudomonas pseudomallei	1993
Clostridium piliforme	Bacillus piliforme	1993
Haemophilus felis	New species	1999
Lawsonia intracellularis	New species	1995
Mannheimia granulomatis	Pasteurella granulomatis	1999
Mannheimia haemolytica	Pasteurella haemolytica	1999
Mannheimia ruminalis	New species	1999
Mycovacterium avium subsp. paratuberculosis	Mycobacterium paratuberculosis	1990

Changes in the genus *Chlamydia*

New Name	Old Name	Species affected & primary diseases
Chlamydia trachomatis	Chlamydia trachomatis	Human-venereal disease, conjunctivitis
Chlamydia suis	Chlamydia trachomatis	Swine-very common in intestines conjunctiva, respiratory tract.
Chlamydia muridarum	Chlamydia trachomatis	Hamsters and mice
Chlamydophila psittaci	Chlamydia psittaci	Birds; psittacosis in birds; transmissible to humans; 8 serovars
Chlamydophila abortus	Chlamydia psittaci	Sheep & goats; abortion; transmissible to humans, esp. pregnant women
Chlamydophila felis	Chlamydia psittaci	Cats; conjunctivitis, rhinitis
Chlamydopila pecorum	Chlamydia pecorum Chlamydia psittaci	Ruminants, swine, koalas; very common in ruminants & swine and associated with many diseases, but most infections are subclinical
Chlamydophila pneumoniae	Chlamydia pneumoniae Chlamydia psittaci	3 biotypes: human-respiratory disease; koalas-isolated from respiratory, ocular, and urogenital tracts; equine-1 isolate from respiratory tract

Comparison of In Vitro Activity of Danofloxanin, Florfenicol, Oxytetracycline, Spectinomycin and Tilmicosin Against Recent Field Isolates of Mycoplasma Bovis

The Veterinary Record, Vol. 146, No. 26, pp. 745-746
Reprinted from the AABP Newsletter, July 2000

The minimum inhibitory concentrations (MICS) and minimum mycoplasmacidal concentrations (MMCS) of danofloxacin, florfenicol, oxytetracycline, spectinomycin and tilmicosin against 62 recent British field isolates of *Mycoplasma bovis* were determined in vitro by a broth microdilution method. The isolates were most susceptible to danofloxacin with MIC_{90} and MMC_{90} values of 0.5 g/ml, respectively. They were less susceptible to florfenicol with a MIC_{90} of 16 g/ml and MMC_{90} of 32 g/ml. Oxytetracycline and spectinomycin had only a limited effect against the majority of isolates tested with MIC_{50}s of 32 g/ml and 4 g/ml, respectively and MIC_{90}s of 64 g/ml and more than 128 g/ml, respectively. Nearly 20 percent of the isolates were highly resistant to spectinomycin, and tilmicosin was ineffective, with 92 percent of the isolates having MIC values of 128 g/ml or greater. There was no evidence of resistance by M *bovis* to danofloxacin.

Toxicology, continued from page 3

A detailed written clinical history is as important as your interview with the client and following physical examination.

From dead animals, a full set of formalin-fixed tissues should include a sagittal half of the brain, lung, spleen, liver, kidney, appropriate digestive tract wall (rumen, abomasum, jejunum, ileum, colon), skeletal and cardiac muscles, and any lesions found in organs such as lymph nodes, adrenals or urinary bladder. For the identification of possible causative agent, a variety of fresh tissues are necessary: fresh liver and kidney, the non-formalized other sagittal half of the brain, any urine available, at least one intact eye globe, at least one OB sleeve full of rumen content, fresh reticulum and abomasal content (each bagged separately), any organs showing gross lesions and for which there is enough tissue to send fresh as well as fixed, and feed, hay, water, and plant samples as appropriate and available. Finally the clinical history and detailed necropsy findings should be clearly and completely documented.

Careful preparation of the specimens includes triple-bagging the formalin-fixed samples so that formalin does not contact the fresh tissue/sample submissions. If this happens, it negates opportunities for organism isolation and prevents meaningful chemical analyses. Careful bagging of fresh tissues to prevent leaking will also avoid contamination of the packing materials. Even the paperwork and written history should be bagged separately. Ice packs should be added to fresh tissues and the material shipped overnight to assure prompt arrival and the best laboratory results.

Everything starts with the on-farm observations and investigations when clinical histories are received, animals are observed showing clinical signs, and field necropsy may be called for. The fresh dead animal should be necropsied and thoroughly sampled. Blood, serum and urine from a live animal with characteristic clinical signs should be collected. All clinical signs and necropsy findings must be carefully documented for clinical and Diagnostic Laboratory application. As much history as possible must be gathered from the owner, including information on feeding programs, grain and hay used, pasture conditions, previous losses, and management activities.

If affected animals were not observed prior to this initial examination, the clinician should verify whether all live animals are showing the same clinical signs. If more than one set of clinical signs is present, blood, serum, and urine should be collected from the individual live animals clearly presenting each set of signs. At least two terminally-ill animals should be necropsied and samples collected representing each unique clinical syndrome. Carefully mark all specimens with correct animal identifications.

If there are no live animals, at least two of the most recent dead animals should undergo detailed post mortem examination. If the necropsy findings on these freshly dead animals are essentially the same, or there are no specific recognized lesions, a full set of specimens must be collected from the most freshly dead animal and gross lesions documented from the other. If necropsy of the first two animals provide significant lesions, but no pattern exists between the two, a third animal should be necropsied. If there is still not a pattern, samples from all three animals should be collected and carefully labeled.

As much history as possible should be collected from the owner of the necropsied animals, including information on feeding programs, hay and pasture available, any previous losses, and potential changes in management.

When in doubt about sample submission or what tests the Veterinary Diagnostic Laboratory is able to perform, call the laboratory to assure that the necessary specific samples and information will be provided.

Salmonella, continued from page 1

have also recently isolated Salmonella serotype Dublin from two calves in the same herd in Kansas. The only other bovine isolate of Salmonella serotype Dublin was one in 1998.

A recent study at Kansas State University found Salmonella serotype Newport to be the most commonly recovered serotype from raccoons. However, this study involved a small number of animals and any possible exposure of cattle to these raccoons is unknown. Further study would be necessary to determine if raccoons are an important factor in bovine infections of Salmonella serotype Newport.

Table 2 shows the frequency of isolation of Salmonella serotype Typhimurium in dogs and horses. They have remained fairly constant over time, except for a small cluster in horses in 1998. This cluster may not be as large as it appears even though the 10 horses listed had eight different owners. We are unable to tell from our records, but it is possible that several horses were housed at the same racetrack which would skew the results.

The numbers in canines is also skewed because of some greyhound research projects. The numbers in parentheses in Table 2 are dogs other than greyhounds involved in the research studies.

1. McDonough, PL; Fogelman, D; Shin, SJ; Brunner, MA; and Lein, DH. *Salmonella enterica serotype Dublin Infection: an Emerging Infectious Disease for the Northeastern United States.* J Clin Micro 37(8):2418-2427.

Table 1. Bovine Salmonella isolates

Year	Number of Newport	Number of typhimurium	Number of typhimurium (Copenhagen)
1995	5	12	20
1996	2	12	10
1997	1	10	17
1998	5	5	21
1999	14	3	10

Table 2. Frequency of Isolation of Salmonella serotype Typhimurium in Dogs and Horses

Year	Number in Canines	Number in Equines
1995	8 (3)	3
1996	1 (1)	0
1997	6 (0)	1
1998	2 (1)	10
1999	1 (0)	4

Online version has appendix of table in text for copy and paste.)

Internal Parasite Control Suggestions for Sheep and Goats

PARASITE	DRUG/TRADE NAME	DOSAGE FORM/DOSAGE[2]	COMMENTS[1]
Nematodes (roundworms of the stomach and intestines)	Thiabendazole E-Z Ex wormer, Omnizole Six TBZ (No longer manufactured but existing stocks may be used)	Suspension, crumble. medicated feed, pre-mix, bolus. Dose according to manufacturer s recommendations.	A microscopic fecal exam should be done prior to worming, as some species of worms do not respond to listed dosages. Consult your veterinarian. Protect from freezing. **Withdrawal:** 30 days before slaughter, eight milkings or 96 hours.
Small stomach worms, barber pole worm, whipworm, thread necked intestinal worm, roundworm and lungworms	<u>Benzimadazoles</u> Fenbendzole Oxfendazole Albendazole	Pellets, flaked meal, premix, blocks, paste, suspension Fenbendazole 7.5 mg/lb Albendazole 8 mg/lb	Approved for use only in cattle.
Small stomach, intestinal worms and lungworms	L. Levamisole (HCL) or phosphate Ripercole-L Tramisol Levasole	Bolus, soluble powder medicated feed, and pre-mix. Dose according to manu-facturer s recommendations.	Approved for sheep only. Careful weight estimates essential. Consult vet before using on severely debilitated animals. **Withdrawal:** 3 days before slaughter.
Gastrintestinal nematodes and for decreased egg production of same.	Phenothiazine (many different products), also combined with lead Arsenate.	Drench. Dry powder for compounding with feed or mineral. Follow manufacturer s directions. 2-50 lb. — 12.5 gm; 60 lb up — 25 gm; 1 gm/head/day in salt.	Reddish color in urine and milk, will stain wool. Don t use on ant females in last month of pregnancy. Do not use with organophosphates. When fed in salt or mineral, it will decrease parasite egg production. Does not destroy adult parasites at this dosage level. **Withdrawal:** None. Non-lactating.
Liver flukes	Curatrem Active ingredient: clorsulon	Oral drench 3.2 mg/lb	Approved for use only in cattle.
Nematodes and head grubs larval stages lungworms	Ivomec Dectomax Cydectin Moxidectin	Injectable. Pour-on. Ivomec also has oral drench. Follow label directions.	Approved for use only in cattle. Ivomec oral drench approved for sheep only. **Withdrawal:** 11 days.

Online version has appendix of table in text for copy and paste.)

Coccidia	Purina Sulfa-Nox Liquid: Active ingredient Sulfaquinoxaline Bovatec: Active ingredient Lasalocid Decox: Active ingredient Decoquinate Rumensin: Active ingredient Monensin Corid: Active ingredient Amploium	Purina Sulfa-Nox Liquid: 6 mg/lb of body weight daily in the drinking water for 3-5 days. Administer a 0.015% solution. Bovatec: 25 gm/ton Decox: 27 gm/ton Rumensin: 20 gm/ton Corid: 48 gm/100 gal water	**Withdrawal:** 10 days. Bovatec: Approved for sheep. Decox: Approved for non-lactating goats. Rumensin: Approved for non-lactating goats. *Corid: Treatment — 5 days; approved for prevention and treatment of coccidiosis in calves. *Do not use for more than 28 days.

[1]All withdrawal periods are subject to change. Consult with FDA Center for Veterinary Medicine *Communications* or the *Feed Additive Compendium* for updates. In many instances, specific wormers work better on specific parasites. It is always advisable to have fecal examinations conducted by your local veterinarian. Veterinarians may also have other products that are not available on the open market.

[2]Many times the same basic de-wormers will have different concentrations of active ingredients when produced or sold by different companies. To simplify and prevent errors *always read all the label instructions and package insert* if present before administering the drug.

To Diagnose Weak Calf Syndrome, Rule Out Unlikely Causes

GL Stokka DVM, MS
Extension Beef Veterinarian

The weak calf syndrome is a condition in which calves are born alive, but without the normal vigor. They are often unable to rise or nurse without assistance, if at all. Unfortunately, the cause is often not easily discovered. It is useful to construct a differential list to try and rule out some of the unlikely causes. According to veterinary medical references the list would include: Dystocia, cold stress, nutritional deficiencies, mineral imbalances that include copper and iron, Vitamin E / selenium deficiency, and Vitamin A deficiency.

Also possible are *infectious* causes such as IBR, BVD, Bluetongue, hemophilus somnus, leptospirosis, brucella, listeria, campylobacter, neospora, sarcosystis, aspergillosis. *Toxins* associated with locoweed, lupines, poison hemlock, nitrates, and *genetic defects.*

One text attributes weak calf/syndrome to fetal infection near the end of gestation, underdevelopment due to nutritional deficiencies, placental insufficiency, dietary deficiencies of selenium and vitamin E, hypothyroidism, traumatic injuries associated with dystocia, accidental injuries following birth, and fetal hypoxia from prolonged parturition.

For this type of diagnosis it is important to begin with a good case definition. A case definition for the weak calf syndrome would be a condition in which calves are born alive, but without the normal vigor, often unable to rise or nurse without assistance, if at all. If your case definition does not match this description, then the diagnosis may be incorrect, and the differential list will be different. For example, when calves are born healthy, attempt to nurse and show signs of weakness with death loss two or three days later, this would not be considered the weak calf syndrome. This example may seem simple, but the information required for a case definition may not be easy to derive.

Producers tend to view similar events as related to the same cause. For example, during an episode of excessive calf death loss, a producer may lose 10 calves out of the first 40. During this time it may be logical to attribute all of the losses to a single cause such as infectious disease. It is important for the veterinarian to determine how many of the cases actually fit the case definition. Three or four different conditions may have contributed to the clincal conditions that are apparent.

To determine the cause, follow these steps:

1. Record any morbidity and mortality events.
2. Classify events to make a preliminary diagnosis.
3. Establish and apply a case definition in each case.
4. Determine if the majority of cases fit one case definition.
5. If so, follow the differential list and rule out causes that do not apply.

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For more information please contact: Linda M. Johnson, Ph. D.
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Newsletter Coordinators

Gerald Stokka
Extension Specialist, Beef Veterinarian
785-532-5694 • jstokka@oz.oznet.ksu.edu
G.A. Kennedy
785-532-4454 • kennedy@vet.ksu.edu

Contributors—K-State Research and Extension

Dale Blasi	Gerry Kuhl	Steve Paisley
Frank Brazle	Twig Marston	Lance Huck
Mike Brouk	John Smith	Sandy Johnson

Contributors—Veterinary Diagnostic Laboratory

G.A. Andrews	B.W. Fenwick	F.W. Oehme
S. Kapil	J.A. Pickrell	D.J. Briggs
K.S. Keeton	R.K. Ridley	M.M. Chengappa
W.E. Moore	P. Schoning	B. DeBey
D.A. Mosier	C.D. Seedle	M.W. Dryden
J.C. Nietfeld	Z. Fu	R. Ganta
T.G. Nagaraja	M.J. Wilkerson	

Cooperative Extension Service
K-State Research and Extension
131 Call Hall
Manhattan, KS 66506

KSU, County Extension Councils and U.S. Department of Agriculture Cooperating.

Appendix 1

Changes in Bacterial Nomenclature of Veterinary Importance

Jerome Nietfeld, DVM, Veterinary Diagnostic Laboratory, Kansas State University

Linda Cox of the KSU Veterinary Diagnostic Laboratory compiled a list of bacterial name changes that are of veterinary importance. Some of the changes are quite recent, while a few have been around for a few years. A partial listing of those changes are in the following table:

New Name	Old Name	Year of change
Arcanobacterium pyogenes	Actinomyces pyogenes	1997
Actinobaculum suis	Eubacterium suis Actinomyces suis	1997
Branchyspira hyodysenteriae	Serpulina hyodysenteriae Treponema hyodysenteriae	1998
Branchyspira innocens	Serpulina innocens Treponema innocens	1998
Branchyspira pilosicoli	Serpulina pilosicoli	1998
Burkholderia mallei	Pseudomonas mallei	1993
Burkholderia pseudomallei	Pseudomonas pseudomallei	1993
Clostridium piliforme	Bacillus piliforme	1993
Haemophilus felis	New species	1999
Lawsonia intracellularis	New species	1995
Mannheimia granulomatis	Pasteurella granulomatis	1999
Mannheimia haemolytica	Pasteurella haemolytica	1999
Mannheimia ruminalis	New species	1999
Mycovacterium avium subsp. paratuberculosis	Mycobacterium paratuberculosis	1990

Changes in the genus *Chlamydia*

In 1980, two species in the genus *Chlamydia* (*trachomatis* and *psittaci*) were recognized. *Chlamydia* isolates that contained glycogen within their inclusions and which were susceptible to sulfa drugs were classified as *C. trachomatis* and everything else as *C. psittaci*. In the past 10-15 years there have been many advances in the ability to detect and characterize *Chlamydia* isolates. This led to the recognition of several biotypes or groups within the genus and to the creation of two new species: *Chlamydia pecorum* ad *Chlamydia pneumoniae.* In 1999, a new classification system for chlamydiae based o their genetics, patterns of virulence, antigens, and animal species affected was adopted. In this system the genus *Chlamydia* was split into two genera, *Chlamydia* and *Chlamydiophila* (Chla.my.do'phil.a.), that includes nine species. The new classification is as follows:

New Name	Old Name	Species affected & primary diseases
Chlamydia trachomatis	Chlamydia trachomatis	Human-venereal disease, conjunctivitis
Chlamydia suis	Chlamydia trachomatis	Swine-very common in intestines conjunctiva, respiratory tract.
Chlamydia muridarum	Chlamydia trachomatis	Hamsters and mice
Chlamydophila psittaci	Chlamydia psittaci	Birds; psittacosis in birds; transmissible to humans; 8 serovars
Chlamydophila abortus	Chlamydia psittaci	Sheep & goats; abortion; transmissible to humans, esp. pregnant women
Chlamydophila felis	Chlamydia psittaci	Cats; conjunctivitis, rhinitis
Chlamydopila pecorum	Chlamydia pecorum Chlamydia psittaci	Ruminants, swine, koalas; very common in ruminants & swine and associated with many diseases, but most infections are subclinical
Chlamydophila pneumoniae	Chlamydia pneumoniae Chlamydia psittaci	3 biotypes: human-respiratory disease; koalas-isolated from respiratory, ocular, and urogenital tracts; equine-1 isolate from respiratory tract

Appendix 2

Internal Parasite Control Suggestions for Sheep and Goats

PARASITE	DRUG/TRADE NAME	DOSAGE FORM/DOSAGE[2]	COMMENTS[1]
Nematodes (roundworms of the stomach and intestines)	Thiabendazole E-Z Ex wormer, Omnizole Six TBZ (No longer manufactured but existing stocks may be used)	Suspension, crumble. medicated feed, pre-mix, bolus. Dose according to manufacturer's recommendations.	A microscopic fecal exam should be done prior to worming, as some species of worms do not respond to listed dosages. Consult your veterinarian. Protect from freezing. **Withdrawal:** 30 days before slaughter, eight milkings or 96 hours.
Small stomach worms, barber pole worm, whipworm, thread necked intestinal worm, roundworm and lungworms	Benzimadazoles Fenbendzole Oxfendazole Albendazole	Pellets, flaked meal, premix, blocks, paste, suspension Fenbendazole 7.5 mg/lb Albendazole 8 mg/lb	Approved for use only in cattle.
Small stomach, intestinal worms and lungworms	L. Levamisole (HCL) or phosphate Ripercole-L Tramisol Levasole	Bolus, soluble powder medicated feed, and pre-mix. Dose according to manu-facturer's recommendations.	Approved for sheep only. Careful weight estimates essential. Consult vet before using on severely debilitated animals. **Withdrawal:** 3 days before slaughter.
Gastrintestinal nematodes and for decreased egg production of same.	Phenothiazine (many different products), also combined with lead Arsenate.	Drench. Dry powder for compounding with feed or mineral. Follow manufacturer's directions. 2-50 lb. – 12.5 gm; 60 lb up – 25 gm; 1 gm/head/day in salt.	Reddish color in urine and milk, will stain wool. Don't use on ant females in last month of pregnancy. Do not use with organophosphates. When fed in salt or mineral, it will decrease parasite egg production. Does not destroy adult parasites at this dosage level. **Withdrawal:** None. Non-lactating.
Liver flukes	Curatrem Active ingredient: clorsulon	Oral drench 3.2 mg/lb	Approved for use only in cattle.
Nematodes and head grubs larval stages lungworms	Ivomec Dectomax Cydectin Moxidectin	Injectable. Pour-on. Ivomec also has oral drench. Follow label directions.	Approved for use only in cattle. Ivomec oral drench approved for sheep only. **Withdrawal:** 11 days.

Coccidia	Purina Sulfa-Nox Liquid: Active ingredient Sulfaquinoxaline Bovatec: Active ingredient Lasalocid Decox: Active ingredient Decoquinate Rumensin: Active ingredient Monensin Corid: Active ingredient Amploium	Purina Sulfa-Nox Liquid: 6 mg/lb of body weight daily in the drinking water for 3-5 days. Administer a 0.015% solution. Bovatec: 25 gm/ton Decox: 27 gm/ton Rumensin: 20 gm/ton Corid: 48 gm/100 gal water	**Withdrawal:** 10 days. Bovatec: Approved for sheep. Decox: Approved for non-lactating goats. Rumensin: Approved for non-lactating goats. *Corid: Treatment – 5 days; approved for prevention and treatment of coccidiosis in calves. *Do not use for more than 28 days.

[1]All withdrawal periods are subject to change. Consult with FDA Center for Veterinary Medicine *Communications* or the *Feed Additive Compendium* for updates. In many instances, specific wormers work better on specific parasites. It is always advisable to have fecal examinations conducted by your local veterinarian. Veterinarians may also have other products that are not available on the open market.

[2]Many times the same basic de-wormers will have different concentrations of active ingredients when produced or sold by different companies. To simplify and prevent errors *always read all the label instructions and package insert* if present before administering the drug.