Spring 2002

FROM THE DIRECTOR
H. Leon Thacker, DVM, PhD

Greetings. I hope this finds you enjoying spring weather and looking forward to the freshness that this season brings to us. I understand that the arctic and gulf streams had unusual interaction this winter so that we received mild weather through most of the season in the Midwest. As a consequence of the mild winter, perhaps the anticipation of spring has been somewhat diluted this year. It seems that even with the mild winter, this spring has brought a high incidence of "weak calf syndrome". We received reports that this is occurring in high numbers in other states as well; there are a number of theories as to causes and contributing factors including copper deficiency, vitamin A deficiency, protein/energy deficiency of the dam, BVD and others. Following the mild nature of the winter we just experienced, perhaps the influence of a harsh winter can be placed lower on the list of causes than has been heretofore thought. WCS likely has multifactoral etiologic contributions and may be influenced by different factors in various areas of the country and in different fall-winter-spring weather patterns.

I regret to report that Dr. Evan Janovitz will be leaving ADDL at the end of April to take a position at Bristol, Myer, Squibb. Evan has been a vital part of the ADDL faculty for the past 14 years, his excellent contributions to the training of our anatomic pathology graduate students, his expertise in the diagnosis of neural disorders and neoplasia, as well as his general diagnostic acumen, will be sorely missed. ADDL is fortunate to have had Evan's input for the years he has been here. We wish him highest success in the coming years in his new position. I am sure that he will soon prove to be as vital to BMS as he has been to ADDL. We are still attempting to fill the diagnostic virologist position that was vacated by Dr. Chuck Kanitz last year. It remains to be seen whether or not the state budget status will allow efforts to fill this position to proceed. We will continue to make every attempt to provide you with up-to-date diagnostic assistant with as prompt as possible turn-around time.

As always, I hope that we are providing you with the diagnostic needs that you have. If not, I hope that you will let us know. Wishing you a most pleasant spring season.

FINAL DIAGNOSIS
   Hepatic lipidosis
   Pancreatitis

History: A 12-year-old female domestic shorthaired cat was submitted for complete necropsy. The cat had been diagnosed with diabetes mellitus one month previously based on clinical pathology findings, but was not being treated with insulin. The cat also had a reported history of recurrent urinary tract infections and vomiting for the previous two months. Reported clinical pathology findings from shortly before the cat's death revealed glucosuria and bilirubinuria as well as hypoalbuminemia, bilirubinemia, and elevated liver enzymes. Physical examination as well as radiographs revealed ascites.

Gross Findings: The carcass had abundant body fat stores and icterus was noted in multiple body tissues including the body fat, sclera, intima of great vessels, and skin. The liver was diffusely yellow-tan with depressed, dark red foci, and floated in formalin. The pancreas was pale, firm, and small. The fat on either side of the pancreas had multiple white, chalky foci. The abdomen was grossly distended with approximately 300 ml of serosanguinous fluid.

Histopathology: Alterations included severe, diffuse hepatic vacuolar degeneration characterized by large clear vacuoles that displaced the nuclei to the periphery of cells. The pancreatic acini were reduced in size and populated with cells devoid of zymogen granules. There was fibrosis between lobules, sometimes extending into acini. Several endocrine islets were expanded and replaced by a homogenous eosinophilic material that stained red with Congo red stain and had an apple-green birefringence under polarized light. There were also locally extensive regions of peripancreatic fat necrosis.

Discussion: The histopathologic lesions were consistent with pancreatitis and hepatic lipidosis. The pancreatitis probably represented a chronic condition in which repeated episodes of mild to moderate pancreatic inflammation resulted in progressive loss or atrophy of pancreatic parenchyma and replacement by fibrous tissue. The amyloid deposition in the pancreatic islets (detected with Congo red stain) is a common lesion in cats with diabetes mellitus. Pancreatitis in cats has been associated with ileus and increased capillary permeability due to vascular injury with resultant abdominal effusion.

Chronic relapsing pancreatitis with progressive loss of both exocrine and endocrine cells, followed by replacement with fibrous connective tissue, is a frequent cause of diabetes mellitus, especially in dogs. Selective destruction of islet cells by infiltration of islets with amyloid is a common cause of diabetes mellitus in cats. Elements of both processes were present in this case. Diabetes mellitus is often, in turn, involved in the pathogenesis of hepatic lipidosis. Hepatic lipidosis is a manifestation of abnormal metabolism in which the accumulation of lipids in the liver of a diabetic animal can result from increased fat mobilization and decreased utilization of lipids by injured hepatocytes.

-by Dr. Theresa Boulineau, ADDL Graduate Student
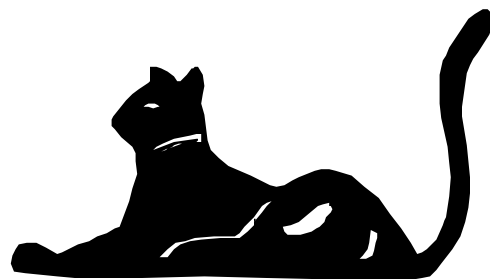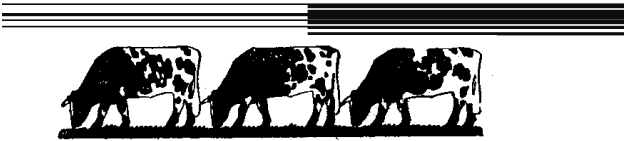
Bovine Granulosa Cell Tumors

Granulosa cell tumors are the most common neoplasm involving the ovaries of cattle. Even though they are the most common ovarian neoplasm, the incidence has been reported to be approximately 0.5 per cent. Granulosa cell tumors affect all breeds of cattle but appear to occur more often in dairy cattle rather than beef cattle. Of the dairy breeds, Guernseys and Holsteins are most frequently affected; however, these breeds also represent the majority of the dairy breeds in absolute numbers.

The higher incidence of these neoplasms occurring in dairy cows may also be partially due to more intense reproductive management practiced on most dairy operations compared to beef cattle operations. The reproductive status of a cow on a dairy farm is evaluated more frequently through repeated rectal examinations than beef cattle on average. It has been proposed that this intense management results in a higher percentage of these tumors being diagnosed rather than dairy cattle being predisposed to developing them. These tumors have been diagnosed in every age of cow, ranging from virgin heifers to very old cows. Granulosa cell tumors have also been reported in pregnant cows, but the occurrence is rare.

These tumors vary in size and structure and are rarely malignant. They have been reported to range from 11.9 grams – 12.3 kilograms. Granulosa cell tumors are most often unilateral and may suppress the function of the contralateral ovary. When this occurs, the contralateral ovary becomes atretic while the ovary with the tumor may continue to grow. These tumors vary in size from relatively small, solid, yellow to white structures to large structures composed of multiple cysts (some being as large as 7.5 cm in diameter), a single large cystic structure, or a combination of solid and cystic structures. The surface of these tumors may be smooth or lobulated. They may be so highly vascularized that one can palpate fremitus in the middle uterine artery. The occurrence of malignant granulosa cell tumors is rare and metastasis is even more rare. It has been reported, however, that an aged cow experienced metastases to the liver.

Cows with granulosa cell tumors may present with a variety of clinical signs ranging from anestrus to nymphomania to male-like behavior. Other clinical signs that may be present include abnormal estrous cycles, deepening of the voice, virilism, and mammary gland development. Mammary gland development and colostrum-like secretions have been reported in virgin heifers. The presence of vaginal discharge and enlargement of the vulva and clitoris may also be present. Cows have also been presented for chronic weight loss due to persistent heat or male-like behavior. These cows may be noticed constantly riding other cows within the herd expressing bull-like behavior. The presence of each of the above clinical signs depends on how much functional luteal tissue is present within the tumor. The amount of functional tissue relates to the dominant hormone being produced within the tumor. In the majority of cases, the tumor continues to grow and may cause affected cows to progress through a series of various stages beginning with nymphomania and ending with virilism.

Diagnosis of granulosa cell tumors is most frequently made through rectal palpation. Most often one will palpate one ovary that is abnormally enlarged while the contralateral ovary will be small. As a rule, granulosa cell tumors should be suspected when the affected ovary is greater than 10 cm in diameter. In some cases, due to the size and/or weight of these tumors, it may be impossible to palpate if located deep in the abdominal cavity. Using a board to pry the abdomen from ventral to dorsal may be one way to make rectal palpation possible. The use of cervical forceps may also be an

option to retract the uterus and ovaries to make rectal palpation effective. Ultrasound examination is another means of diagnosing granulosa cell tumors. Upon ultrasound examination the affected ovary may appear to have a honeycomb appearance. If the majority of the tumor tissue is solid the tumor will appear solid and may be misdiagnosed as a large corpus luteum. A granulosa cell tumor should also be suspected when a chronic cystic structure does not respond to conventional treatment regimens. Cystic ovaries may produce the same clinical signs as a granulosa cell tumor. Monitoring a cow's response to exogenous hormones or prostaglandins may aid in differentiating between the two conditions. Another more practical way to differentiate between a granulosa cell tumor and cystic ovaries is serial rectal palpations. Most often a granulosa cell tumor will continue to enlarge, whereas cystic ovaries tend to be more static. However, the only way to have a definitive diagnosis for a granulosa cell tumor is through histopathological examination of the tumor sections.

Treatment of granulosa cell tumors is limited. Unilateral ovariectomy is the only treatment option available. Most often, this is only used in valuable breeding stock. It is not recommended to perform this on animals that exhibit changes in secondary sexual characteristics such as enlargement of the vulva and clitoris. Animals showing these changes have a reduced fertility post surgical removal of the tumor. It has also been reported that removing the ovary in cows exhibiting these changes resulted in cystic follicles on the contralateral ovary. As a rule, cattle have reduced fertility after a unilateral ovariectomy when compared to the mare. Economically it is often not feasible to perform surgery to remove the tumor on animals with average genetics. The surgical approach for removing the ovary depends on the size and location of the ovary and the demeanor of the cow.

Most dairy cows will stand for the procedure, whereas beef cattle often need to be cast into lateral or dorsal recumbency. If the ovary fits in the palm of one's hand, most often the ovariectomy via colpotomy approach is successful. In order to safely remove large tumors the surgical approach recommended is through a laparotomy incision in the paralumbar fossa or on ventral midline.

Whichever technique is used, special care should be taken to avoid damaging the vasculature of the tumor to prevent hemorrhage.
 - by Brandon Brackenbury, Class of 2002
  -edited by Marlon Rebelatto, ADDL
     Graduate Student

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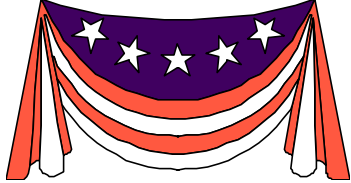
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Purdue ADDL and Heeke ADDL will be closed on the following University holidays.

May 27, 2002   Memorial Day
July 4, 2002   Independence Day

ADDL
STAFF
NEWS

ADDL welcomes its newest graduate student, **Dr. Ingeborg Langohr**.

The bacteriology laboratory has successfully completed Johne's

organism-based check tests, including both Polymerase chain reaction and culture, and was certified according to the criteria established at the last National Johne's Working Group meeting.

A reminder that ADDL test results are available on the Internet. If you'd like to have an account established for your practice, please contact our systems manager, Steve Vollmer, at

www.addl.purdue.edu/onlineaccess/contact.asp
or phone 765-494-7440.

3

Bluetongue

Virus

Bluetongue is a non-contagious, athropod-borne viral disease of both domestic and wild ruminants. Bluetongue virus (BTV) is endemic in some areas with cattle and wild ruminants serving as reservoirs for the virus. Epizootics of Bluetongue virus killing approximately 179,000 sheep within 4 months have threatened the livestock industry in recent years. For this reason, regulatory veterinarians have heightened their interest in this devastating disease.

The threat of decreased trade associated with Bluetongue outbreaks has become an even bigger threat to the livestock industry than the actual disease itself. According to Kahrs, "bluetongue is a major obstacle to exportation of U.S. ruminants and ruminant products and probably affects the United States more than most countries." This is because of the prevalence of BTV in conjunction with competent vectors within the U.S., vague surveillance and reporting policies, and extensive BTV research emanating from U.S. laboratories.

Bluetongue is an orbivirus which cross-reacts with many antigenically related viruses including Palyam virus and the viruses that cause epizootic hemorrhagic disease of deer and African Horse sickness. Bluetongue virus replicates in both arthropod and mammalian host cells. The virulence of BTV varies quite markedly; even strains with matching serotypes have variable virulence. A total of 25 serotypes have been identified worldwide with only 5 recognized within the United States.

Testing diagnostically for BTV can be difficult. Two types of viral antigen are used for BTV testing. All Bluetongue serotypes share a common antigenic determinant called antigen protein P7, while the antigen protein Ps is variable and is used to determine the specific serotype of a virus (1-25). The serum is often tested by complement fixation, AGID, or one of several Elisa techniques. The AGID test can detect antibodies that have persisted for years in BTV-exposed animals and can cross-react with related orbiviruses, thereby producing high numbers of false negatives (low sensitivity and specificity). While difficult to perform, complement fixation is still used to determine BTV exposure status
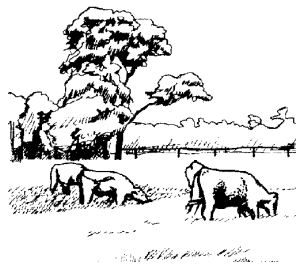
for export since it can detect shorter-lived antibodies. However, because of BTV's wide pathogenic variability and the fact that cross-reaction may occur between other orbiviruses (especially EHD), a positive result on Bluetongue group test does not mean clinical signs seen were caused by BTV itself. The competitive ELISA (C-ELISA) has proven to be the best serologic test for BTV antibody detection. Monoclonal antibody detection is used with ELISA to decrease the chance of cross-reaction. In any case, the detection of BTV antibodies is poorly correlated with BTV viremia.

Virus isolation from blood of a viremic animal is the most definitive means of BTV diagnosis. Virus isolation can be labor intensive, time-consuming, and expensive; however, spleen and brain tissues (often from aborted fetuses) are used to isolate the BTV. According to James Mechan, "Isolation of BTV has traditionally relied on inoculation of cell cultures, embryonated chicken eggs, or sheep with blood from infected animals or with homogenates of insects collected in endemic areas." Currently, PCR is taking over previous methods of virus isolation. The PCR tests have proven to be very sensitive and specific for BTV RNA. A positive PCR is not always indicative of infection, however, since viral RNA can be detected in some tissues after viremia has passed.

BTV can replicate in a variety of mammalian cells. According to Smith, "Clinically, the BTV seems to present as underlying endothelial cell damage resulting in a vasculitis causing edema and eventually necrosis of epithelial and mucosal surfaces." Teratogenesis occurs in the developing fetus due to virally induced disruptions of organogenesis.

It has recently been hypothesized that development of clinical disease in cattle may be mediated through Type 1 hypersensitivity (aranylaris). Likewise, clinical disease in sheep appears to be most severe when previous exposure has occurred.

BTV infection occurs in both wild and domestic ruminants/camelids from the bite of the vector midge of the genus *Culicoides*. The *Culicoides* vector infects most species during mid-summer to early fall when it is most active. The virus can also be transmitted sexually in infected semen and transplacentally from dam to offspring. Transmission via embryo transfer may also be a concern if the embryo is not washed at least ten times. *Culicoides* transmission is by far the most important method of transmission in endemic areas. BTV is mostly seen in the southern United States where *Culicoides* are widespread. In the absence of competent vector populations, animal to animal transmission is not capable of maintaining an endemic state. The overall seroprevalence of cattle in the United States is >18%.

Bluetongue is clinically manifested as two syndromes: 1) vascular insult of several organ systems and 2) a reproductive syndrome. Sheep are commonly seen with clinical disease, but other domestic ruminants such as cattle and goats only rarely show clinical signs. Differential diagnoses of Bluetongue in sheep include Orf (contagious ecthyma), foot and mouth disease, any vesicular disease, and sheep pox.

After a prepatent period of 3-8 days, sheep may begin to show clinical signs such as transient fever (up to 106F), edema of the face, lips, muzzle and ears, excessive salivation, and hyperemic oral mucosa. The disease name stems from the fact that affected sheep begin to develop a mucopurulent nasal discharge after the first few days and the tongue may become cyanotic. This is actually an infrequently reported sign; however, the oral lesions may progress to petechial hemorrhages, erosions, and ulcers. A marked pulmonary edema is often seen. Late in the disease (7-12 days), lameness characterized by petechial hemorrhages at the coronary band may occur and the hooves may eventually slough. Fragile wool and diarrhea are commonly seen.

Many affected animals become depressed and die while others make a full recovery.

The reproductive portion of the disease varies greatly. Signs include abortions, stillbirths, and weak "dummy lamb" live births. BTV can be both abortigenic and teratogenic in cattle experimentally, but neither is commonly seen in field conditions. Early embryonic loss and decreased reproductive efficiency is a more frequently seen manifestation of the disease in cattle and can be devastating to their calf/milk production. Clinical signs in cattle also include hyperemia and necrosis of the muzzle ("burnt muzzle") and patchy dermatitis. Differentials for BTV in cattle include Bovine Viral Diarrhea virus, Malignant Catarrhal Fever, vesicular diseases, Rinderpest, photosensitization, Bovine Papular Stomatitis and Infectious Bovine Rhinotracheitis. Regulatory officials should be notified if an outbreak in cattle occurs or is suspected.

Unfortunately, no single gross or histologic lesion points with certainty towards BTV. Some animals appear normal at necropsy, while most show hemorrhage in some organ, most frequently the heart. Petechial and ecchymotic hemorrhages are also seen under the tongue, on the hard palate, esophagus, forestomach, lymph nodes, bladder, and spleen. Erosions and ulcers can be seen anywhere in the oral cavity. Gelatinous subcutaneous edema of the head, neck, forelimbs, and trunk is commonly seen.

Supportive treatment is used since no antibiotic for BTV exists. Since animals with severe oral lesions are reluctant to eat, they should be fed via stomach tube or encouraged to eat soft feedstuffs. Muscle and coronary band pain limits mobility and therefore shade and water should be made readily available. Sulfas may be administered to treat secondary bacterial pneumonia and NSAIDs are commonly used to control pain.

Environmental elimination is usually not possible so sheep should be kept indoors during peak midge activity, e.g. dusk. Environmental control using Ivermectin can be attempted, but transmission of BTV can occur before the insect's demise. Some modified live vaccines are available and should be based upon the local strains and serotypes.

-by Lisa McDill, Class of 2002
-edited by Dr. Theresa Boulineau, ADDL Graduate Student

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✔ TO EXPEDITE PROCESSING WHEN SUBMITTING SAMPLES TO TWO OR MORE LABS WITHIN ADDL, PLEASE REMEMBER TO SPLIT AND PACKAGE TISSUES SEPARATELY.

Biliary Cystadenoma in the Cat

Biliary cystadenomas are uncommon, benign hepatic tumors. Most feline cases have been reported in cats greater than ten years of age. Biliary cystadenomas may be unilocular or multilocular and consist of thin-walled cysts that contain clear, watery to slightly viscous fluid. The tumor is often raised above the capsular surface and may involve more than one liver lobe. The cysts vary in size from 1-15 mm and may be arranged in masses as large as 12.5 cm. The tumor is usually intrahepatic, but may rarely occur in extrahepatic ducts.

This neoplasm in man shows a predilection for females, but no sex predilection has been found in cats. Although at least one report claims it is more common in domestic shorthair cats, there is no confirmed breed predilection. In humans, biliary cystadenomas can undergo malignant transformation. It is unclear, however, whether this occurs in the feline species. Metastasis is not a reported feature of this neoplasm.

Abdominal pain is the most common sign in humans with this tumor, but does not appear to be common in cats. The most common signs in cats are anorexia, lethargy and weakness. Patients may present with a cranial abdominal mass on palpation. The tumor can also be demonstrated by radiography, ultrasonography, computed tomography, or may be an incidental finding on necropsy or exploratory laparotomy. In any case, signs associated with the tumor seem to be related to impingement on other organs rather than to the tumor itself. Blood abnormalities are often present due to the age of the patient and the aforementioned adjacent organ impingement, but there is no evidence of serum biochemical or hematologic abnormalities directly associated with biliary cystadenoma.

Radiographically, this lesion may be seen as a cranial abdominal mass. Association with the liver is often difficult to demonstrate. Ultrasound is the diagnostic tool of choice since the cystic nature and association with the liver are more apparent with this modality. The cyst walls are thin and smooth. The contents of these cysts are anechoic, but may occasionally contain internal echoes. The ultrasonographic features alone are not enough to definitively diagnose this tumor. Other differentials include hematoma, abscess, parasitic cyst, biliary cyst or tortuous biliary structures, cystadenocarcinoma, hemangiosarcoma, feline polycystic disease of the liver and kidney, or metastatic pancreatic and ovarian adenocarcinoma.

Final diagnosis is by histopathology. The lining of the cyst is cuboidal to attenuated epithelium with occasional papilla formation. The epithelium is histologically, immunologically and electron microscopically similar to typical biliary epithelium. The fibrovascular stroma surrounding the epithelium may contain frequent islands of entrapped hepatocytes and occasional muscle fibers and inflammatory cells. The cysts contain proteinaceous fluid and varying amounts of mucin. The cyst contents help to differentiate biliary cystadenoma from a biliary cyst, abscess, or hematoma. Those would contain bile, pus or blood, respectively. Aspiration and fluid cytology of the cyst contents are not adequate for final diagnosis.

The origins of this tumor are obscure, but there is some evidence that this slow-growing tumor may sometimes be congenital. There is excessive production of the embryonic bile ducts that may not be continuous with the biliary tree. These areas would normally involute but may be retained as cysts or hamartomas and may be the source of this tumor. It has also been shown that these tumors can be acquired, as they have been experimentally induced in rats.

The treatment of choice for biliary cystadenoma is complete surgical excision with 1 cm margins. This may require complete lobectomy in the cat. Cholecystectomy may also be required. If complete removal is not possible, partial excision may be adequate for good prognosis due to the slow-growing nature of this tumor. Recurrence appears to be extremely rare, but has been reported in at least one case. Other treatments, such as aspiration, marsupialization, and partial excision have met with limited success and are not recommended since there may be a possibility of malignant transformation.

-by Mark Funk, Ross University Student
-edited by Theresa Boulineau, ADDL
  Graduate student

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Guidelines for Submitting Serology Samples for Fairs and Shows

The season for fairs and shows is fast approaching and plans should be made for performing inspections and tests.

The following information is a general review for submitting samples to ADDL/Serology.

1) Samples hand-carried to ADDL by owners must be sealed using the veterinarian's label or tape bearing the veterinarians' signature.

2) All regulatory charts must include **submitting veterinarian's signature** and complete animal identification.

3) ADDL Form 3 (Request for Serological Test) must be completed and attached to all regulatory test charts.
ADDL will run only those tests requested on this form.

4) A health certificate is the only necessary test record for 4-H exhibition. Do not submit duplicate test charts.

5) Use of BD-Vacutainer or Monoject tubes is preferred. Venoject, Jelco or EDTA-treated tubes will not be accepted.

6) Each tube must be identified with a tube number; additional identification is desirable.

7) Tube numbers and numbers on charts must match and be in consecutive order. Tubes should be packaged in consecutive order as well.

8) Clear serum is preferable to whole blood.

9) Tests for Pseudorabies will be performed daily (M-F). Turnaround time may be 10-14 days (may be slightly longer at peak testing periods).

10) Brucellosis tests are performed daily (M-F). Turnaround time is 2-4 days.

11) Swine samples for both Brucellosis and Pseudorabies will be tested first for Brucellosis, followed by Pseudorabies. Turnaround time may be 10-14 days.

12) ADDL will not release results to owners.

There will be no exceptions to the testing schedule. Please allow adequate time.

If you have questions, please contact ADDL/Serology at 765-494-7451 prior to submitting samples.
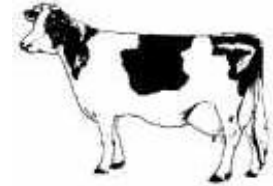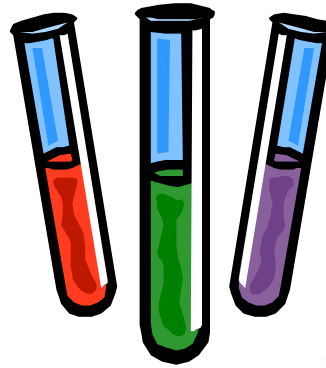
Identification, Control and Eradication of *Streptococcus agalactiae* Mastitis in Dairy Herds

Background: *Streptococcus agalactiae* is a gram positive obligate pathogen that affects pre-milking heifers, as well as older cows in dairy herds. It is considered one of the major causes of economic losses to dairy producers without a control program.

Although *Streptococcus agalactiae* can live outside the udder for short periods of time in the right conditions, it is considered to be an obligate pathogen of the udder. A high percentage of cows may be affected in herds where control procedures are not implemented. Fomites such as strip cups, towels, milkers' hands, cross suckling calves, milking machines and other milking equipment and unsanitary conditions are all potential sources of infection in cows. Even multi-use and hand-mixed antimicrobial mastitis preparations can be a potential source of infection for the udder.

Streptococcus agalactiae may be transmitted from udder to udder in many ways. *Streptococcus agalactiae* breaks the natural barriers of the udder, enters the teat canal, and ascends in the milk through the quarter. The bacteria penetrates the acinar epithelium, causing edema and extravasation of neutrophils into the lumen, resulting in subclinical or clinical mastitis as well as possible systemic infection. In later stages, the acini become filled with scar tissue

which plugs the glandular-ductal system resulting in a chronic, smoldering infection which decreases milk production and increases the somatic cell count (SCC) of the quarter. Poor udder health due to *Streptococcus agalactiae* is slowly progressive over time, causing fibrosis and atrophy of the affected quarter. As a matter of fact, an individual cow with a high SCC typically has lower production that correlates with increased SCC of the herd.

Identification of *Streptococcus agalactiae*

As with other causes of mastitis, *Streptococcus agalactiae* may cause heat, pain and swelling of the udder as well as abnormal milk consisting of white to yellow clots and flakes. On closer examination, damage to the teat end may be apparent and can be the result of trauma, improper milking procedures and equipment or freezing. Poor udder health may be indicators for cows with an infection of the mammary tissue.

Subclinical mastitis is difficult to detect on visual examination of the milk. The California Mastitis Test (CMT) is a quick and easy way to identify chronic subclincal cases of mastitis, but is not specific for only this pathogen. As for clinical cases, the Hymast kit can give results of gram positive (most likely contagious; *Staphylococcus*, *Streptococcus*, etc) or gram negative (most likely environmental; *E. coli*, *Klebsiella* spp., etc.) organisms in 12 hours and is used frequently to aid in the diagnosis and treatment protocol of the mastitis. The most confirmative diagnosis for contagious mastitis is by the isolation and identification of the causative organism. Culture for *Streptococcus agalactiae* from the bulk tank with high CMT, Hymast and individual Dairy Herd Improvement Association (DHIA) SCC or linear scores, can identify those cows in the herd that may be infected and/or chronic shedders of *Streptococcus agalactiae*. The CAMP test, using a nurse streak of *Staphylococcus aureus* used to be the gold standard to identify *Streptococcus agalactiae;* however, it has now been replaced by the Latex agglutination test with high specificity and sensitivity. Most of

bacterial culturing and testing is still done in diagnostic laboratories; however, the practitioner in the field can easily use DMT, Hymast and DHIA records in conjunction with laboratory results during mastitis evaluation.

Control and eradication of *Streptococcus agalactiae*

Streptococcus agalactiae mastitis can be eradicated effectively from dairy herds. The loss of production due to poor udder health associated with *Streptococcus agalactiae* greatly exceeds the cost of implementation of some simple control measures.

All cows identified as infected with contagious organisms should be grouped together and milked last in the milking facility. These animals potentially are a source of infection to non-infected cattle. Also, newly acquired animals should be housed and milked separately to prevent spread of the organism. Animals with *Streptococcus agalactiae* mastitis usually have an excellent response to the use of intra-mammary antibiotics, penicillin in particular. The cost of the antibiotic and milk withheld from the bulk tank are far less than the long-term expenses associated with herd infection with *Streptococcus agalactiae*. However, if the cow is late in her lactation, it may be efficacious to wait until the dry period and use a commercial dry cow therapy (slow-release, wide spectrum antimicrobials). Often, dry cows are neglected and are rarely evaluated during the dry period predisposing them to udder infections. Intra-mammary infusion of antimicrobials following the last milking of the lactation (dry cow therapy) can greatly reduce the cases of *Streptococcus agalactiae* mastitis by eliminating existing infections and controlling new infections early in the dry period.

Pre-dipping and post-dipping the teat in conjunction with single towel use per animal is effective in the control of all contagious mastitis including *Streptococcus agalactiae* infections. Pre- and post-dippings will help kill any of the organisms that would normally be transmitted into the udder during milking. Also, regular and thorough

cleaning of the facilities and milking equipment with disinfectants will aid in eradication of the organism. Good hygiene always promotes milk production.

Heifers should also be considered in the eradication of *Streptococcus agalactiae*. Heifers that consume infected milk, whether from the teat or bucket feed unpasteurized milk, are a potential source of infection to their penmates or other cows through transmission of the organisms by mouth. This may explain why some heifers become infected prior to ever being milked. Housing these heifer calves separately will easily correct this potential problem.

Implementing these and other sanitation procedures described above can greatly minimize the economic impact due to *Streptococcus agalactiae* infections. These procedures are easily implemented and very cost effective. Launching this type of program will be a benefit to the producer economically, as well as to the consumer who will be receiving a more sanitary product.

-by Mark James, Class of 2002
-edited by Dr. Ching Ching Wu, Head of
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