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Summer 2003

FROM THE DIRECTOR
H. Leon Thacker, DVM, PhD

In the good ole summertime, the "lazy daze of summer" aren't likely to happen this year. It doesn't look like we are going to run out of things to do. The Exotic Newcastle Disease in the Southwest has put us on alert for detecting its occurrence as early as possible if it gets to Indiana; the finding of Bovine Spongiform Encephalopathy in Canada has brought increased surveillance efforts to the U.S.; the import of some giant rats from Africa has ostensibly brought monkey pox to our shores and into Indiana and the West Nile virus season has started a bit earlier than it did last year. We've had one case of monkey pox through the ADDL in a prairie dog; the case is awaiting confirmation by CDC, but gross and histologic lesions and finding pox virus particles with electron microscopy in the lung are very suggestive of the diagnosis. At the time the animal came through the lab, we were not suspicious of monkey pox as news of its occurrence in this country had not yet been released. Fortunately, no one in the lab contracted pox from it.

As charges for sending samples via public transportation that contain known infectious agents have gone to such high levels, it has become necessary to recharge shipping charges if you request return of infectious cultures. The high cost of shipping is incurred if we are shipping known infectious material; if you are sending us material or we are sending material that is "diagnostic" and perhaps suspicioned, but not known to have, infectious agents present, the cost is much lower.

We are getting up to speed on running immunohistochemistry for transmissible encephalopathies. It is an involved procedure and, up to now, we have been running tests on samples received and contracted with the federal lab in Ames, Iowa. If all goes well we should be set up to do the surveillance testing for CWD in Indiana white-tail deer this fall using this procedure.

We hope you have an enjoyable summer and we hope that you are unhesitating to contact us with your diagnostic needs.

FINAL DIAGNOSIS

Disseminated Blastomycosis in a Dog

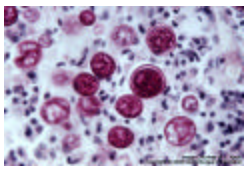
In each issue, we will feature a case submitted to ADDL that we hope will be of interest to you.

History: A 3-year-old, male German Shepherd dog was submitted to the Animal Disease Diagnostic Laboratory for necropsy. Reportedly, the dog had a history of anorexia and lethargy for three weeks. The dog had been treated with a course of antibiotics for 10 days with some improvement. Six days prior to death, the dog reportedly had an Addison's crisis and was treated with steroids. The dog was referred to Purdue University Teaching Hospital. On presentation, the dog was tachypneic, pale and painful in the abdomen. Ophthalmology exam revealed retinal granulomas in both eyes. An aspirate of the popliteal lymph node showed pyogranulomatous inflammation and *Blastomyces dermatitidis* organisms. The dog was humanely euthanized.

Gross Findings: Fibrous tags adhered the right caudal lung lobe, right middle lung lobe, and pericardial sac to the parietal pleura of the thoracic cavity. The lungs were diffusely tan and dark red, mottled and firm on palpation. Multifocal to confluent, circumscribed, firm, gray, variably-sized granulomas ranging from 1-10 mm in diameter were disseminated throughout the lungs. The bronchial lymph nodes were enlarged and firm and measured approximately 2 x 1.5 cm. The gastric contents were dark brown to black, liquid, and opaque. The pyloric antrum of the stomach contained numerous multifocal mucosal ulcers which ranged from 4-7 mm in diameter. Some of the ulcers had blood clots adhered to them. Similar mucosal ulcers were found in the small and large intestine.

Histopathologic Findings: The primary lesion in the lungs was a multifocal to confluent pyogranulomatous pneumonia. Numerous intralesional, 10-20µm in diameter, spherical yeasts with a thick, double refractile cell wall, basophilic granular central zone, and occasional broad-based budding consistent with *Blastomyces dermatitidis* were present. Bronchial lymph node lesions included pyogranulomatous lymphadenitis with intralesional *Blastomyces dermatitidis.* All sections of brain examined had multifocal pyogranulomatous encephalitis with intralesional *Blastomyces dermatitidis* organisms. The primary morphologic alterations in the right and left eyes were multifocal sub-retinal accumulations of *Blastomyces dermatitidis* organisms with very little inflammatory reaction, retinal detachment, and small multifocal granulomas in the choroid. Sections of stomach, duodenum, ileum, and jejunum contained acute multifocal mucosal ulcers.

Discussion: Blastomycosis is a systemic mycotic disease caused by the dimorphic fungus *Blastomyces dermatitidis.* The disease primarily affects dogs and humans, but has been reported in cats, horses, sea lions, lions, wolves, ferrets and polar bears. Young, male, large breed dogs (especially sporting breeds and hounds) living near water are at an increased risk. It is generally restricted to the Mississippi River and Ohio River basins and the central Atlantic states.



Most cases of blastomycosis are acquired by inhalation of aerosolized conidia into the lungs. After inhalation, the conidia are phagocytized by alveolar macrophages and transform the mycelial phase to the yeast phase. The yeast stimulates local cell-mediated immunity which results in a marked suppurative or pyogranulomatous inflammation. The incubation period varies from 5-12 weeks.

The acute pulmonary phase of the illness may be asymptomatic or self-limiting or may result in acute fulminate infection. The preferred sites of dissemination in the dog are the skin, eyes, bones, lymph nodes,

subcutaneous tissues, external nares, brain, and testes. Less commonly affected sites are mouth, nasal passages, prostate, liver, mammary gland, vulva, and heart. The size of the yeast when it grows at body temperature prevents it from entering the terminal airway in an aerosol. Therefore, aerosol transmission of the yeast phase from infected animals is not possible. However, penetrating wounds can transmit the disease. Primary cutaneous blastomycosis has been reported in veterinarians after accidental laceration while performing a necropsy on a dog with blastomycosis, following a dog bite from a dog with blastomycosis, and accidental inoculation with a needle used on a dog with blastomycosis. Cutaneous blastomycosis is rare in the dog and should be considered a manifestation of disseminated disease.

The dog appears to be more susceptible to infection than humans, and serves as a sentinel for the disease. Dogs have a shorter prepatent period and develop the disease before people do when exposed at the same time. Most dogs with disseminated disease are probably immunosuppressed, since the majority of dogs experimentally infected by exposure of contaminated soil recover from blastomycosis without treatment.

Clinical signs in dogs with blastomycosis include anorexia, weight loss, cough, dyspnea, ocular disease, lameness, lymphadenopathy, and proliferative or draining skin lesions. Signs of disease usually have been present for a few days to a week but may have been apparent for several months. In many cases there has been a history of antibiotic therapy with minimal or temporary improvement. A majority (85%) of dogs with blastomycosis have lung lesions, and up to 40% have ocular lesions, the most common of which is uveitis. Skin lesions are found in 20-40% of dogs with blastomycosis. Bone involvement occurs in up to 30% of infected dogs. Diffuse lymphadenopathy is seen in 40% of dogs with blastomycosis.

The yeast organisms range from 5-20 µm in diameter and are characterized by broad based budding with a thick, refractile, double-contoured cell wall. Diagnosis is usually based on finding the characteristic yeasts in cytologic or histologic preparations. Hematology is usually not helpful in the diagnosis since complete blood count (CBC) results are often normal. Radiographic assessment can be helpful in the diagnosis. Thoracic radiographs often reveal a nodular interstitial pattern or diffuse interstitial pattern. Serology should be used diagnostically only when a high degree of suspicion for *Blastomyces* exists and repeated attempts have failed to demonstrate the organisms.

Spontaneous recovery from symptomatic blastomycosis rarely occurs in dogs but has been reported in people. Therefore, it is suggested that all cases of symptomatic blastomycosis in dogs should be treated. Itraconazole is considered the treatment of choice, except in cases of moderate to severe hypoxemia when amphotericin B should be considered. Approximately 70-75% of treated cases recover from blastomycosis. Treatment failure most likely occurs in dogs that are hypoxemic or have three or more systems involved.

-by Dr. Phaedra Stiles, ADDL Graduate Student

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Rabies in Raccoons

Raccoons act as a reservoir for rabies in the United States. In fact, the majority of rabies cases in the U.S. are in wildlife, with raccoon cases predominating. In the U.S., 90% of all rabies cases occur in wildlife. During 2000, raccoon rabies made up 41% of wildlife cases diagnosed with skunks, bats and foxes making up the balance. To limit the exposure of humans and domestic animals to rabies, it is important for veterinarians to know the current geographic distribution of rabies, the procedure if a human or animal is bitten by a wild animal, and be familiar with rabies diagnostic testing.

Geographic distribution: Raccoon rabies was much less prevalent in the U.S. prior to 1950. From 1950-1970, the incidence of raccoon rabies began to rise, especially in Florida and Georgia. In 1977, a variant of raccoon rabies distinct from the southern variant was detected in Virginia and West Virginia. This variant has since spread north along the eastern seaboard to Ontario, Canada, and was reported in northwest Ohio in 1996. Eventually this rabies variant converged with the southern variant in North Carolina.

In most states with raccoon rabies, there has been an increased number of cases; however, Ohio has reported fewer cases. In 2000, 10 of 19 states which have reported raccoon variant rabies reported an increase in the number of cases. However, Ohio reported no cases in 2000. This was a decrease from the 6 cases reported in 1999. This may be because Ohio has used extensive wildlife rabies control programs, including rabies vaccine baiting.

Human or Domestic Animal Exposure:

Veterinarians should discourage ownership of raccoons or any wildlife. No parenteral vaccines are approved for use in raccoons and prior vaccination does not contraindicate euthanasia and testing. Captive raccoons in exhibits may have been incubating rabies when caught and, therefore, should be quarantined for a minimum of 180 days before exhibition.

If a human is bitten by a raccoon (or any wildlife) the animal should be regarded as rabid. The raccoon should be humanely euthanized and the brain tested for rabies. If human exposure occurs, it should always be reported to the local or state health department. Bites or scratches should be thoroughly washed immediately. A bite to a human requires the initiation of post-exposure prophylaxis.

Animals potentially exposed to rabies by a raccoon or other wild animal not available for testing should be handled as an exposure. Unvaccinated domestic animals which are exposed should be euthanized immediately. If the owner is unwilling to do this, the animal should be placed in strict isolation for six months. The animal should be vaccinated one month before release. Animals that are current on rabies vaccinations should be revaccinated immediately and observed for 45 days.

It is important to remember that the guidelines for bites from wildlife are different from those for bites to humans from healthy domestic animals. If a dog, cat, or ferret bites a human, the animal should be quarantined and observed for ten days. The animal should not be given a rabies vaccination during this time. At the first sign of illness, the animal should be evaluated by a veterinarian and reported to the local health department. If signs of rabies develop, the animal should be euthanized, appropriate brain tissue removed

and shipped refrigerated to the State Board of Health. Any unwanted animal that bites a human may be euthanized immediately and submitted for testing.

Diagnostics: No antemortem tests are sensitive enough to be considered reliable for diagnosis. Handling live suspect animals should be done cautiously using safety equipment such as rabies poles, cages, and heavy gloves. The animal should be euthanized humanely in a way that does not damage the animal's brain. The head can be removed and the whole animal or head should be chilled until examined. The head should not be frozen as freezing causes tissue damage and may delay diagnosis.

The standard diagnostic test for rabies is the direct fluorescent antibody test (FAT). This test can be performed quickly and has a sensitivity and specificity approaching 100%. In this test, microscope slides of brain from the affected animal are fixed in acetone. The slides are then exposed to rabies specific nucleocapsid fluorescent antibody. They are evaluated on a microscope with an energy source which allows visualization of fluorescent marker fixed to the antibodies. It is not necessary that the animal be showing neurologic signs at the time of examination. If there is virus in the saliva, there will be detectable virus in the CNS by FAT.

Rabies can also be diagnosed histopathologically through the identification of intracytoplasmic inclusions in large neurons called Negri bodies. Negri bodies can be found in the thalamus, hypothalamus, pons, cerebral cortex, and dorsal horn of the spinal cord. In carnivores, they are most common in the neurons of the hippocampus. In herbivores, they are often found in the Purkinje cells. The bodies are best seen with Seller's or van Gieson's stain; they stain magenta. Negri bodies are not seen during all stages of infection and this test is no longer used for routine diagnosis.

In Indiana, animals or brain tissue is sent directly to the Board of Health as soon as possible for official examination at:

Rabies Laboratory
Indiana State Board of Health
P.O. Box 7203
635 N. Barnhill Dr.
Indianapolis, IN 46207
(317) 233-8036

Conclusion: There have been no confirmed human deaths associated with rabid raccoons; however, there has been an increased number of rabies cases in domestic animals in the northeast due to raccoon rabies. Therefore, rabid raccoons could potentially lead to human exposure through rabid domestic animals.

-by Paul Rennekamp, Class of 2003
-edited by Dr. Christine Hanika, ADDL Pathologist

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Sharon Young, Mary Woodruff, Tom Hooper and **Dr. Ching Ching Wu** attended the Association of Veterinary Microbiologists Heartland Chapter annual meeting in Madison, Wisconsin, April, 2003.

Dr. **Ching Ching Wu** spoke to swine and poultry groups on antimicrobial usage in London, England and Paris, France, May 2003.

Drs. Leon Thacker, Bill VanAlstine, Alok Sharma, Ingeborg Langohr, and **Phaedra Stiles** attended the North Central Conference for Veterinary Laboratory Diagnosticians in Minneapolis, MN, June, 2003.

Dr. Leon Thacker attended the American Veterinary Medical Association annual meeting in Denver, Colorado, July, 2003.

Dr. **Tsang Long Lin** attended the Ixth International Symposium on Nidoviruses in Egmondaar Zee, Netherlands. May, 2003.

Dr. **Ching Ching Wu** attended the 2003 Conference on Modern Vaccines, Adjuvants and Delivery Systems in Dublin, Ireland, June, 2003.

Idiopathic hypertrophic cardiomyopathy in the cat

One of the most common primary feline cardiac diseases is idiopathic hypertrophic cardiomyopathy (HCM). HCM is characterized by a massive left ventricular hypertrophy without dilation and is present without any other cardiac or systemic disease. The secondary form of HCM is usually associated with disease processes such as acromegaly, systemic hypertension, or hyperthyroidism.

HCM has been more frequently observed in males than females. It is most commonly described in middle-aged cats with a range distribution from 5 months to 17 years. In a study done by Atkins et al, the median survival rate of cats with HCM was about two years. Cats without clinical signs survived longer than those with heart failure or signs of embolism. In the same study, the survival rates of cats having heart rates $\leq$ 200 beats/minute survived longer than those with heart rates $\geq$ 200 beats/minute.

Etiology: The etiology of HCM is usually not known. Multiple etiologies have been proposed including hereditary predisposition, elevation in circulating catecholamines, abnormal myocardial calcium metabolism, abnormal compensatory myocardial hypertrophy due to ischemia and fibrosis, or abnormal primary collagen resulting in secondary ventricular hypertrophy. Researchers have found evidence that some cases of HCM are inherited in the Maine coon cat and the American shorthair as an autosomal dominant pattern. Kraus et al identified a litter of five 18-month-old mixed breed cats that all had HCM.

Pathophysiology: Clinical signs are generally associated with myocardial

hypertrophy due to decreased left ventricular diastolic filling and myocardial ischemia. In many cases there are also systolic abnormalities such as intraventricular pressure changes leading to asynchrony of the contraction and relaxation of the heart muscle.

Depending on the type of myocardial alterations found, there are several complications that can occur. Pulmonary edema can be present when there is an increase in left atrial and pulmonary venous pressure. Arrhythmias can also be present where then is myocardial ischemia. Also, due to localized endocardial injury, circulatory stasis and altered blood coagulability, there is a potential risk of developing a thrombus within the left atrium that could potentially lead to a thromboembolic episode.

Clinical signs: Cats with HCM can be asymptomatic and often experience sudden and unexpected death. Others can show symptoms of acute dyspnea after stressful episodes. On a routine physical exam, HCM can be suspected if there is a murmur or gallop rhythm. When clinical signs are present, they are associated with left sided heart failure or arterial embolization.

Clinical signs observed with HCM are variable and may include dyspnea, tachypnea, pulmonary crackles, lethargy, reluctance to move, syncope, gagging, anorexia, and sometimes abdominal distention or vomiting. When pericardial or pleural effusion is present, the heart and lung sounds can be muffled.

Thromboembolism has been recognized in approximately 50% of cats with HCM. When aortic thromboembolism is present, the clinical signs will vary according to its location. The most common sign of thromboembolism is unilateral or bilateral pelvic limb paresis or paralysis. When the embolization occurs at the brachial artery, pain and paresis of forelimbs has been observed. Blockage of the renal artery will produce acute renal failure. If the thromboembolism occurs at the cranial mesenteric artery, it is possible to observe clinical signs of colic. Central nervous system abnormalities can be seen if the embolus affects the cerebral artery. Respiratory distress can be observed after embolization of the pulmonary vasculature.

Diagnosis: It is possible to have a tentative diagnosis based on the history, physical examination, electrocardiographic findings, and thoracic radiographs.

Arrhythmias and conduction disturbances have been detected in 60-70% of cats with HCM. Several types of arrhythmias have been observed, including premature ventricular contraction, atrial fibrillation, atrial tachycardia, atrial premature contraction, paroxysmal ventricular tachycardia, and ventricular bigeminy. Varying degrees of atrioventricular block, right and left bundle branch blocks, and Wolff-Parkinson-White syndrome are some of the conduction disturbances that have been observed. Prolongation of P waves greater than 0.04 seconds and prolongation of the QRS complex greater than 0.04 seconds with R wave amplitude greater than 0.9 mV in lead II are parameters that have been used as electrocardiographic indications of an enlarged left ventricle.

Depending on the degree of compromise, the thoracic radiographs may show different degrees of cardiomegaly with pulmonary edema. On the ventrodorsal view, the classic radiographic sign of HCM is a “valentine” shaped cardiac silhouette with biatrial enlargement and normal looking apex. It is common to observe a banana-shaped cardiac silhouette on the lateral view. When pericardial effusion is present, it is possible to observe generalized enlargement and rounding of the cardiac silhouette on the ventrodorsal view. Radiographic evidence of cats with pulmonary edema usually has a patchy and focal distribution along the pulmonary vessels.

Definite diagnosis can be done using echocardiography. With this method, it is possible to rule out secondary causes of HCM such as congenital aortic stenosis, chronic systemic hypertension, chronic anemia and hyperthyroidism. Typical echocardiographic abnormalities found in HCM are symmetrical hypertrophy of the

left ventricular caudal wall and interventricular septum, reduced dimensions of the left ventricular chamber, and ventricular hyperkinesis.

Treatment: Medical therapy of HCM is aimed to control the clinical signs and has been adapted from that used in human medicine. To relieve signs of pulmonary edema, diuretics such as furosemide have been used. If refractory right-sided heart failure develops, a second diuretic such as hydrochlorothiazide can be used. For treatment of congestive heart failure, an angiotensin-converting enzyme inhibitor such as captopril can be used. Beta adrenergic-blocking agents (propanlol) or calcium channel blockers (dialtiazem hydrochloride) have been used for the treatment of the diastolic dysfuntion.

Medical management of thromboembolism has been mostly empirical. Heparin therapy has been used to prevent additional formation of thrombi. The use of thrombolytic agents such as streptokinase, urokinase, and tissue plasminogen activator are expensive and have not shown to be consistently effective. A potential complication of their use is uncontrolled bleeding due to their fibrinolytic effect.

Aspirin has been used to prevent platelet aggregation and thrombus formation. Follow-up studies have not shown that aspirin prevents clot formation in all cases. Some cats with long term aspirin therapy, although presenting thromboembolic episodes, have shown shorter recovery periods.

Cats with HCM have very little cardiovascular reserve and are very sensitive to stress. Caution should be observed when administering rapidly intravenous fluids since there is the potential to cause rapid decompensation and the initiation of congestive heart failure.

Pathology: HCM is characterized by hypertrophy of the left ventricle free wall ($\geq$ 0.6 cm), papillary muscles and interventricular septum. The size of the left ventricular lumen is decreased. The muscle hypertrophy can be symmetrical or asymmetrical. It is sometimes possible to find thickening of the mitral valve with enlargement of the left atria.

In a study performed by Liu, a total of 51 cats with HCM were studied. In this study, 70% of the cats presented symmetric, concentric ventricular hypertrophy with normally arranged cardiac muscle cells in the septum. In the remaining 30%, the cardiac muscle cells in the septum were disorganized. This lesions seems to be very specific to primary HCM because it was rarely found in cats with secondary HCM.

In a previous study (Van Vleet), 10 cats with HCM had gross lesions including cardiomegaly, diffuse symmetric left myocardial hypertrophy, small left ventricular cavities, and dilated left atria. The histopathology lesions found were hypertrophy and disorganization of cardiac muscle cells, interstitial fibrosis, and fibromuscular hyperplasia of small intramural coronary arteries. Other lesions observed include hypertrophy and disorganizaiton of myocytes of the left ventricular wall and septum. The endocardium, conduction system, or myocardium may present focal or diffuse degeneration, interstitial fibrosis, and chondroid metaplasia. In 50% of cats with HCM, the intramural coronary artery walls were thickened and had narrow lumens.

-by Luz Borrero-Yu, ECFVG Student
-edited by Dr. Theresa Boulineau, ADDL Graduate student

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

September 1, 2003.....Labor Day
November 27-28,2003.....Thanksgiving
December 25-26,2003.....Christmas
January 1-2,2004.....New Year's

An Update On Bovine Pinkeye

Infectious bovine keratoconjunctivitis (IBK), commonly known as pinkeye, is an economically important and often frustrating disease of cattle. The disease is quite common and highly contagious. A 1993 survey of Kansas cattle producers found IBK to be the second most common disease. A Missouri study found 45.4% of cattle herds to be affected by the disease. Cattle affected by the disease are painful and often become temporarily blind and, therefore, do not consume as much feed. This leads to economic losses due to decreased daily gain and lower weaning weights. The cost of treatment adds to economic losses. A 1984 study estimated that $200 million were lost due to IBK. This is a significant disease to the cattle industry and prevention and control should be of utmost importance. However, current treatment and prevention measures can be unrewarding and often do not circumvent the economic losses.

IBK is a multifactorial disease. The primary etiologic agent is the bacterium *Moraxella bovis.* However, there are many factors that add to the virulence of this microorganism. In addition, other bacteria have been isolated from eyes with clinical IBK in the absence of *M. bovis*, indicating that other bacteria may be a primary pathogen in some cases of IBK.

M. bovis possesses virulence factors that allow it to colonize the eye and result in pathology. Cell membrane pili are responsible for colonization of the cornea. Two functionally distinct pili have been identified. The Q pilus is responsible for attachment and the I pilus allows for local persistence and maintenance of an established infection. It is thought that bacteria possessing the Q pilus can convert to the I pilus during the course of disease. Non-piliated strains do not cause clinical IBK. *M. bovis* also possesses hemolysins. The hemolysins damage neutrophils that are recruited to the area of infection. It is the destruction of neutrophils and release of collagenolytic enzymes that ultimately lead to corneal liquefaction and ulceration. Non-hemolytic strains of *M. bovis* are non-pathogenic. Seven serologically distinct groups of *M. bovis* have been identified based on antigenic differences in membrane pili. Of these serogroups, groups 3 and 4 (strains Epp63 and Fla64, respectively) are most common in the United States.

IBK is most often seen in summer and early fall. This is most likely because ultraviolet light and face flies are the two most important contributing factors. Ultraviolet light causes nuclear fragmentation and corneal epithelial loosening, which enhances the ability of *M. bovis* to colonize the cornea. Flies are the primary vector for *M. bovis. Musca autumnalis* is the important specie of fly in transmission. Direct transmission of *M. bovis* between cattle is rare. Therefore, face flies are likely the most important factor in outbreaks of IBK. Wind, dust, tall grasses, hay, and high ammonia levels in the air contribute to eye irritation and initiation of IBK. A relationship between vaccination with a modified live IBR vaccine and the development of IBK has been found,

suggesting the ability of IBR virus to enhance *M. bovis* infection.

Several other bacteria have been implicated as a predisposing factor and primary cause of IBK. *Mycoplasma bovoculi* and *Branhamella ovis* have been cultured from normal eyes and eyes displaying clinical signs of IBK. *M. bovoculi* is known to enhance IBK caused by *M. bovis* by extending its colonization. *M. bovoculi* alone can cause a mild catarrhal conjunctivitis. *B. ovis*, alone, has been shown to cause a keratoconjunctivitis similar to *M. bovis.* However, like *M. bovis*, factors contributing to ocular irritation are required for clinical disease. *B. ovis* has also been cultured concurrently from eyes with *M. bovis* infection. This suggests that *B. ovis* plays a role as a primary pathogen and a contributing factor in IBK.

Diagnosis of IBK is by clinical signs which include blepharospasm, epiphora, photophobia, chemosis, corneal edema, corneal ulceration and blindness. Culture and sensitivity is recommended in outbreaks to determine the bacteria involved and the best treatment options. Conjunctival swabs and lacrimal secretions are the best samples to submit for culture. *M. bovis* is extremely susceptible to dessication; therefore, the sample should be transferred to appropriate growth media prior to shipment to a diagnostic laboratory. For best results, the sample should be transferred to 5% blood agar within 2 hours of collection. It may be necessary to request *Mycoplasma* culture in refractory cases or in outbreaks where predisposing factors are unclear. *M. bovis* cultures may be serotyped for pili through immunofluorescence or immunogold electron microscopy. A fluorescent antibody test is available to demonstrate *M. bovis* in lacrimal secretions. An ELISA for identifying serum or lacrimal IgG is useful in determining the *M. bovis* serogroup responsible for the outbreak; however, these special tests for serotyping are not widely available.

Treatment of IBK relies on the use of antibiotics and the prevention of further ocular irritation. Oxytetracycline is the only antibiotic labeled for the treatment of IBK. Two doses of long-acting oxytetracycline given 72 hours apart is usually effective in accelerating healing time. Parenteral florfenicol can also shorten healing time. Daily subconjunctival injections of penicillin have also been used in cases of IBK. This treatment is economical but requires more handling and labor. Two doses of benzathine cloxacillin at 375 mg given topically 72 hours apart has been shown to be effective. However, commercial mastitis preparations currently available are not concentrated enough for use in IBK. Topical gentamicin and betamethasone spray preparations have also been used topically. Corticosteroids, such as dexamethasones, are often used to reduce the inflammation and pain associated with IBK although classically contraindicated in cases of corneal ulceration. The use of 3rd eyelid flaps, tarsorrhaphy and eye patches can aid in reducing irritation due to environmental factors. Although treatment reduces healing time, research has not proven the ability of treatment to improve weight gain in affected animals.

Several commercial vaccines are available, including whole cell monovalent and multivalent *M. bovis* bacterins. Vaccination for *M. bovis* has met with mixed results. It has been shown that lacrimal secretory IgA is required for resistance to reinfection. The available bacterins produce adequate serum IgG titers which have little correlation with resistance to infection; therefore, currently available bacterins may offer little protection. A mucosal vaccine would be much more effective, but none are currently available. In addition, there is little cross-protection among serotypes. Monovalent vaccines do not protect against heterologous serotypes. Studies using multivalent vaccines have reduced the incidence of IBK, but not the severity of disease. Currently, multivalent bacterins, including the Fla64 and Epp63 strains of *M. bovis* are available. While providing cross-protection for other strains within their serogroups, this bacterin cannot protect against strains in the other five serogroups. These serogroups are less

commonly encountered in the United States, but can still cause outbreaks. Recombinant multivalent vaccines will allow for more serotype inclusion but are not currently available. Autogenous vaccines may be considered where uncommon serotypes are isolated from a herd.

Prevention of IBK centers on reducing the predisposing factors of the disease. Since face flies are the major vector of IBK, prevention should focus on fly control. The use of insecticide ear tags, back rubs and feed-through insect growth regulators can significantly reduce the incidence of IBK. In the case of ear tags, both ears must be tagged to effectively reduce the number of face flies. Affected cattle should be isolated from other cattle if possible. New additions should be isolated for 30-60 days prior to introduction to the herd. Mowing tall grasses and noxious weeds from pasture and ensuring adequate head space at the bale feeder can aid in reducing ocular irritation. In case of an outbreak, mass treatment with parenteral oxytetracycline may be beneficial. The use of oxytetracycline in the feed may also be considered. Vaccination with a modified live IBR vaccine should be avoided during an outbreak. Vaccination with an *M. bovis* bacterin should be considered in herds with recurring problems with IBK. A multivalent pili vaccine is recommended. In addition, culture, serotyping and sensitivity should be performed to determine the bacterial pathogens and serotypes involved in the outbreak. This information can aid in tailoring treatment and vaccination programs.

-by Jill Franks, Class of 2003
-edited by Dr. Duane Murphy, ADDL Pathologist

Visit our website at www.addl.purdue.edu

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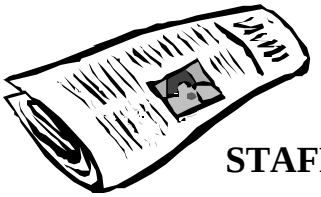
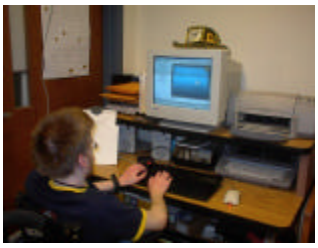


STAFF NEWS

The Purdue Graduate Student Government recognized **Dr. Sandra Schoeniger**, ADDL Pathology Resident, as Outstanding Teaching Assistant in the Department of Veterinary Pathobiology for the 2002-2003 school year.

Nathan Stembel, a junior at Harrison High School in Lafayette, IN, has been working at ADDL as part of the Student Training Employment Program (STEP), a cooperative work program for high school students with special needs.

Nathan helps with a variety of tasks in the ADDL clerical office.



PLEASE NOTE

Beginning July 1, 2003

Due to changes in shipping regulations, ADDL will begin re-charging postage for bacterial cultures sent to veterinarians. The charge is generally $42-$50. We will continue to re-charge postage for isolates sent to outside labs at the request of the submitting veterinarian

Percent of Micro-organisms that are Resistence to Selected Antibiotics for July-Dec. 2002 and Jan.-June 2003

Antibiotic	Canine										Equine												Feline							
	E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus		Staph. intermedius		E. Coli		Salmonella sp.		Staph. aureus		Staph. epidermidis		Strep. equi		Strep. zooepidemicus		E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus	
	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June
Amikacin	1	1	38	18	2	0	4	0	0	0	0	0	0	0	0	0	0	0	100	83	100	88	0	0	71	100	0	0	0	0
Amoxycillin/Clauvulinic acid	18	12	19	27	95	100	4	29	2	16	27	6	20	0	50	33	0	18	0	0	0	0	14	12	43	0	100	100	0	0
Ampicillin	48	46	23	27	95	100	46	57	42	48	50	44	40	14	50	67	50	45	0	0	5	0	38	47	43	0	100	100	33	50
Cefazolin	18	11	62	82	98	100	4	0	0	0	27	13	20	0	50	17	0	0	0	0	5	0	10	6	100	100	100	100	0	0
Cefotaxime	7	1	33	27	10	25	4	0	0	0	0	0	0	0	50	17	0	0	0	0	5	0	5	0	57	0	20	0	0	0
Cefoxitin	17	10	81	73	95	100	4	0	0	0	19	6	20	0	50	17	0	0	0	0	5	0	0	12	86	0	100	100	0	0
Ceftiofur	16	8	82	91	98	100	4	0	0	0	12	6	20	0	50	17	0	0	0	0	5	0	5	0	100	100	80	75	0	0
Cephalothin	23	18	57	73	95	100	4	0	0	0	31	19	20	0	50	17	0	0	0	0	5	0	19	6	86	0	100	100	0	0
Chloramphenicol	16	24	0	0	80	92	0	0	0	0	38	13	40	14	0	0	0	0	0	0	0	0	5	0	0	0	60	75	0	0
Ciprofloxacin	16	14	10	18	17	0	8	29	2	0	4	0	0	0	0	0	0	0	0	0	0	0	10	0	43	0	0	0	67	0
Clindamycin	100	99	86	73	95	100	8	14	9	13	100	100	100	100	0	0	0	0	0	100	5	100	100	100	86	100	100	100	0	0
Enrofloxacin	18	13	45	27	55	42	17	43	2	0	4	0	0	0	0	0	0	9	0	100	0	60	14	0	57	0	40	25	67	0
Erythromycin	100	98	23	18	95	100	17	29	9	16	100	100	100	100	50	17	50	18	0	100	5	100	100	100	43	0	100	100	0	50
Gentamicin 500 microgm/ml	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	18	0	0	0	0	0	0	14	0	0	0	0	0
Gentamicin	11	14	32	9	10	25	1	14	0	10	31	38	0	14	50	67	0	0	100	100	55	88	19	0	57	100	0	0	0	0
Sulphadimethoxine/Ormetoprim	29	29	10	0	95	100	13	14	9	6	62	16	40	14	50	33	50	0	0	0	5	4	19	6	14	0	100	75	0	0
Oxacillin + 2% NaCl	100	100	76	91	95	100	4	0	0	0	100	100	100	100	50	17	0	0	0	0	0	0	100	100	86	0	100	100	0	0
Penicillin	100	100	27	27	98	100	42	57	38	35	100	100	100	100	50	67	50	27	0	0	5	12	100	100	43	0	100	100	33	50
Rifampin	97	97	33	27	95	100	0	0	0	0	100	100	100	100	0	0	0	9	0	0	0	0	100	100	29	0	100	100	0	0
Tetracycline	33	43	52	45	27	42	17	14	16	26	42	56	20	14	50	33	50	27	0	0	50	40	24	18	86	0	40	25	0	0
Ticarcillin	42	45	24	27	7	25	42	57	38	35	42	38	20	14	50	67	50	36	0	0	5	0	29	47	71	0	40	0	33	50
Tribrissen	29	29	10	0	29	33	42	14	47	23	62	56	40	14	50	50	50	18	0	0	9	0	14	0	14	0	40	50	0	0
Vancomycin	100	100	10	0	98	100	0	0	0	0	100	100	100	100	0	0	0	0	0	0	0	0	100	100	0	0	100	100	0	0
number of isolates	90	68	21	11	41	12	24	7	45	31	26	16	5	7	2	6	2	11	1	6	22	25	21	17	7	1	5	4	3	2

Percent of Micro-organisms that are Resistence to Selected Antibiotics for July-Dec. 2002 and Jan.-June 2003																								
Antibiotic	Beef								Dairy						Swine									
	E. coli		Past. Haemolitica		Past. Multocida		Salmonella sp.		E. coli		Staph. aureus		Salmonella sp.		APP		E. coli		Salmonella sp.		Strep. suis	
	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June		
Ampicillin	46	25	0	33	0	11	43	40	62	52	17	35	80	30	6	17	64	58	41	47	3	2		
Apramycin	12	6	22	17	67	56	29	20	35	28	na	na	26	9	13	7	20	20	9	8	41	46		
Ceftiofur	19	11	0	0	0	11	43	40	26	25	0	0	50	30	0	0	10	13	6	18	8	4		
Chlortetracycline	69	59	0	0	0	11	43	40	91	86	na	na	85	39	10	14	86	94	76	78	96	97		
Clindamycin	100	100	100	100	100	100	100	100	97	96	na	na	100	100	19	14	100	99	100	100	82	85		
Enrofloxacin	12	5	0	0	0	11	0	0	12	11	na	na	0	0	0	2	0	0	0	0	3	7		
Erythromycin	100	98	0	0	0	22	100	100	98	96	0	10	100	100	3	2	99	99	100	100	80	82		
Florphenicol	100	100	11	0	0	11	100	100	98	95	na	na	98	100	0	0	95	100	97	98	67	78		
Gentamicin	27	13	0	0	0	0	29	20	59	49	na	na	35	17	0	0	21	23	15	6	5	4		
Neomycin	42	24	44	83	67	22	43	20	71	75	na	na	67	22	3	2	43	45	15	25	63	61		
Oxytetracycline	69	67	33	67	50	44	43	40	92	86	na	na	85	43	35	43	88	95	76	78	97	95		
Penicillin	100	100	44	50	17	22	100	100	98	96	17	35	100	100	90	88	100	100	100	100	12	5		
Sulphadimethoxine	58	52	44	83	83	78	57	40	73	63	92	85	83	39	10	12	71	74	71	75	62	55		
Spectinomycin	38	32	78	100	50	67	100	100	76	76	na	na	93	91	0	9	55	55	100	92	29	22		
Sulphachloropyridazine	58	52	11	0	83	67	43	40	89	84	na	na	83	35	35	22	72	73	68	65	60	59		
Sulphathiazole	58	52	56	83	83	89	43	40	89	84	na	na	83	39	23	19	73	76	68	65	65	61		
Tiamulin	100	100	78	50	83	78	100	100	98	96	na	na	100	100	0	9	100	100	100	100	25	18		
Tilmicosin	100	100	0	0	0	22	100	100	96	96	na	na	100	100	0	2	98	100	100	100	77	82		
Triple Sulfa	42	16	0	0	0	11	43	20	61	68	na	na	37	13	0	0	18	17	12	16	6	1		
Tylosin	100	100	89	100	83	100	100	100	98	96	na	na	100	100	na	na	100	100	100	100	na	na		
number of isolates	26	63	9	6	6	9	7	5	85	80	12	20	46	23	31	58	96	128	34	51	117	109		