



FROM THE DIRECTOR
H. Leon Thacker, DVM, PhD

Good day from the Purdue ADDL. This is a great time of the year with harvest of crops, crisp, changing air temperatures, and football. In case any of you have not noticed lately, the Purdue team, under the tutelage of Joe Tiller is doing quite well this fall, perhaps foreboding of post season invitation to a high ranking bowl game.

The activity in the ADDL continues to be busy but, with the passing of a fairly hard frost, it is now permissible to state that the occurrence of West Nile Virus infection in our state this year has been markedly reduced from that of '02. We have had only one horse posted in the lab diagnosed with West Nile Virus infection as the cause of death. We're told by our entomologist friends that there was a lower mosquito population in the state this year; it is also likely, or a given, that immunity among horses vaccinated in the state and among our bird population from natural exposure, was higher than last year.

Among the personnel of the ADDL, we are sorry two valued staff members, Dr. Randy White, pathologist since 1988 who went to work for Bristol, Myer, Squibb in Evansville, and Ms. Janeice Samman who has worked in our histology lab for the past 38 years and retired in September. We wish them both success and happiness in the years ahead. We have had the opportunity to welcome two new faculty members in September, Dr. Peg Miller and Dr. Pepe Ramos, both accomplished anatomic pathologists who came to us from the University of Missouri. We are very happy and fortunate to have Peg and Pepe on the ADDL faculty and look forward to many years of their contributions to the ADDL and School of Veterinary Medicine strategic plans, accomplishments, and services.

A change of shipping regulations which includes transport of cultures of known content has effected change of the means whereby we can ship cultures back to practicing veterinarians. In order to receive known/identified bacterial cultures, a permit and permit number from the USDA is required. As long as the shipped material is either suspicioned to have bacterial presence or is sent as diagnostic specimen, i.e. being sent to the laboratory for culture, the requirements for shipping remain unchanged.

We hope that you are enjoying the changing of the fall season and that you will keep us apprised of your diagnostic needs. It is our desire to provide you with animal disease diagnostics in the most accurate and timely manner currently available to veterinary application.

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FINAL DIAGNOSIS

Poxvirus Infection in a Prairie Dog

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

History: A 12 week-old female prairie dog was euthanized after presentation to the referring veterinarian with respiratory distress. It was then

submitted to the Animal Disease Diagnostic Laboratory at Purdue University to determine the specific cause of the clinical signs since deaths of prairie dogs with similar clinical signs had occurred where this animal was bought.

Gross Findings: Gross lesions included numerous variably sized, round, discrete ulcers on the tongue and hard palate, dark red consolidation of both cranial and right middle lobes of the lungs, affecting approximately 40% of the pulmonary parenchyma, and small (<3 mm), white, firm, slightly raised, plaque-like lesions sparsely distributed throughout the wall of the gastrointestinal tract.

Histopathologic Findings: The main microscopic lesions included severe multifocal to coalescing necrotizing bronchopneumonia, with vasculitis and poorly defined eosinophilic inclusions either within the cytoplasm of cells presumed to be of histiocytic or fibroblastic origin or freely scattered throughout the necrotic debris. Multifocal necrotizing lesions, often accompanied by myxoid edema, were also present in sections of nasal turbinates, trachea, thymus and adjacent brown fat, tracheobronchial lymph nodes, lips, tongue, esophagus, stomach, jejunum, cecum, colon, liver, kidney, adrenal gland, vagina, vestibule, female accessory genital glands, conjunctiva, and cornea.

Additional Tests: Ultrastructural examination of lung and intestinal tissue revealed scattered aggregates of immature and mature, non-enveloped, to 320 x 200 nm

viral particles located within the cytoplasm of degenerating cells. The immature viral forms were semicircular to round and had a granular matrix, whereas the mature virions were oval or brick-shaped, with an electron-lucent core and two lateral bodies surrounded by an outer membrane. The morphology of the virions was consistent with poxvirus. Specimens of selected tissues were submitted to the Centers for Disease Control and Prevention (CDC) to confirm the presumptive diagnosis of monkeypox infection. Laboratory evaluation of these tissues is in progress.

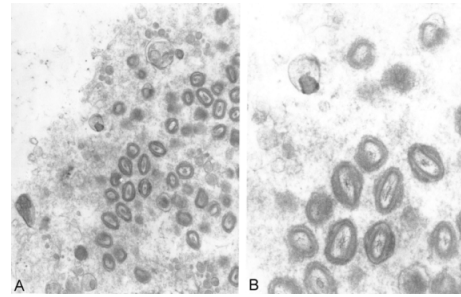
Discussion: Monkeypox virus, a member of the orthopoxvirus genus, appears to be enzootic among wild mammals in the west and central African rainforest, where the principal reservoirs are thought to be squirrels and other rodents. Despite the name of the virus, primates are infected only accidentally through direct or close contact with infected reservoir hosts. The disease is usually transmitted to humans from rodents and primates through a bite or contact with the animal's blood. The infection with this virus causes a vesicular and pustular rash similar to, but usually milder, than smallpox. The incubation period is approximately 12 days, and the death rate among infected humans in Africa has ranged from 1-10%. In primates, monkeypox should be considered in any outbreak of a systemic febrile illness that involves a skin rash. Rashes can be severe and generalized in some species. Dyspnea caused by pneumonia may develop in severe cases. Concurrent bacterial septicemia might be present.

The diagnosis is confirmed by histological and electron microscopic examination of tissues, by serological tests (ELISA and hemagglutination inhibition test), and by virus isolation; however, the characteristic lesions on inoculated chicken chorioallantoic membrane and the large eosinophilic intracytoplasmic inclusions typical of poxvirus infections may not be seen with monkeypox virus infection.

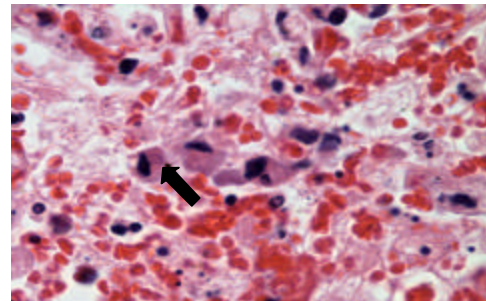
Immunohistochemistry, Western blot, and polymerase chain reaction (PCR) tests for monkeypox viral antigen detection are now available.

In the United States, the disease was reported in early June 2003 among several residents of the Midwest who became ill after having contact with sick pet prairie dogs and, in one case, a rabbit. As of 8 July, 2003, a total of 71 cases of monkeypox had been reported to the CDC from Wisconsin, Indiana, Illinois, Missouri, Kansas, and Ohio; these included 35 (49%) cases laboratory-confirmed at CDC and 36 (51%) suspect and probable cases under investigation by state and local health departments. Trace-back investigations have determined that all 35 confirmed human cases of monkeypox were associated with prairie dogs, which appeared to have been infected through contact with Gambian giant rats and dormice that originated in Ghana. Laboratory tests have demonstrated the presence of monkeypox virus in several rodents that died unexpectedly without exhibiting characteristic signs of monkeypox that originated from a shipment from Ghana on 9 April 2003. This outbreak underscores the potential threat to animal and public health by introduction of exotic species.

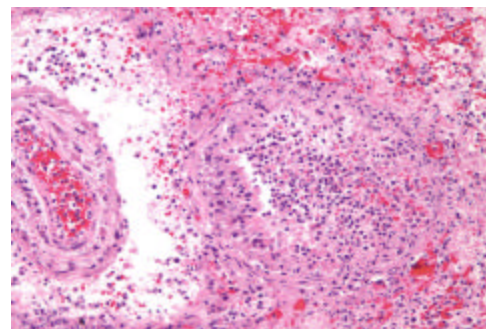
-by Dr. Ingeborg Langohr, ADDL Graduate Student



Lung, prairie dog, poxvirus infection, transmission electron microscopy. 50,000X (A) and 112,500X (B). Aggregates of immature and mature, non-enveloped virions were located within the cytoplasm of scattered degenerating cells. Mature virions had the typical structure of poxviruses, with an outer membrane enclosing a biconcave core and two lateral bodies.

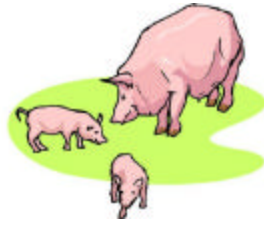


Lung, prairie dog, poxvirus infection, 1000X. An eosinophilic inclusion body (arrow) is within the cytoplasm of a degenerating cell presumed to be an alveolar macrophage.



Lung, prairie dog, poxvirus infection, 200x. Bronchioles (right), blood vessels (left) and the surrounding pulmonary parenchyma were extensively necrotic.

Sorting Out Sow Mortality



Introduction:

Over the past decade, many farms and production systems have experienced problems with high sow death loss. Thorough investigation of sow mortality is an important area where veterinary expertise can add value to client herds. Unfortunately, veterinary intervention may not be sought until mortality rates exceed 10%. Partial budget analysis of the economic benefits of lowering sow mortality may provide substantial evidence to support the cost of veterinary services. The expense associated with sow mortality can be categorized as replacement costs, opportunity costs when a female dies during gestation, and the negative impact that mortality can have on worker morale. Decreased morale is difficult to value, but replacement and opportunity costs can account for \$400-\$500/sow death. One financial analysis predicted that lowering sow mortality by 4% would save \$18 per inventoried female per year. Another study showed a 4:1 return on investment for an intervention strategy aimed at decreasing sow mortality.

Sow mortality rate has been defined as the number of breeding females that died or were euthanized on a farm throughout the year divided by the average female population. Sow death rate has been calculated similarly to mortality rate but does not account for females that were euthanized on the farm. Essential components of a sow mortality investigation should include a thorough herd record evaluation, multiple necropsy examinations, and an epidemiologic evaluation of risk factors present on the farm. A systematic approach to evaluating sow mortality has been described previously in detail.

The most common causes of sow mortality are discussed below.

Heart failure: Several criteria have to be taken into account to make a diagnosis of heart failure. First, other causes of death must be excluded and evidence of heart failure such as transudate in body cavities, pericardial effusion, pulmonary edema, passive congestion of lungs, liver, kidneys, and spleen, must be observed or altered cardiac chamber measurements must be observed. In a prospective study, heart failure accounted for 31.4% of all sow deaths. Approximately 2/3 of these females died during the peripartum period, defined as the period from 3 days prior to 3 days after farrowing. Ambient temperatures greater than 32.0°C (89.6°F) have also been associated with an increased risk of heart failure in sows. One recent conflicting study suggested, however, that heart failure was not a common cause of sow mortality because microscopic lesions in the ventricular wall or interventricular septum were rare.

Torsions and other accidents of abdominal viscera: The diagnosis of torsions and other accidents of abdominal viscera is based on characteristic gross findings, including gastric dilation and torsion, splenic torsion, hepatic torsion, intestinal volvulus, intussusception, herniation, and intestinal rupture. No correlation was established between death from torsions or other abdominal accidents and housing type or feeding management. Deaths due to gastric dilation occurred most frequently within a few hours after feeding. One study concluded that feeding three times per day resulted in a lower risk of sow mortality than feeding twice per day. A genetic predisposition for gastric torsion was suspected when all sows in a herd that died from this condition were traced back to a single Landrace boar. Once the boar was removed, no further cases of death from gastric torsion were reported.

Cystitis-pyelonephritis: Urinary tract infections in sows are usually the result of ascending bacterial infections. Isolates obtained from cases of cystitis-pyelonephritis included *Escherichia coli*, *Actinobaculum (Eubacterium) suis*, *Proteus*

spp, *Klebsiella* spp. and *Enterococcus faecalis*. One study showed that the mean aqueous humor area concentration from sows that died from cystitis-pyelonephritis was 52.3 mmol/L compared to 9.9 mmol/L observed in sows that died of other causes. In this study, the types of waterer did not correlate with the sow deaths attributed to cystitis-pyelonephritis, although adequate water supply is thought to decrease incidence of urinary tract infections.

***Clostridium novyi* infection:** *C. novyi* infection may cause sudden death in pigs. Gross lesions reported in these cases include a large, bronze-colored, emphysematous liver, generalized edema, subcutaneous emphysema, foul-smelling sanguinous effusion in body cavities and pericardium, and congested submandibular and superficial inguinal lymph nodes. The diagnosis can be difficult in sows that have been dead for an extended period of time prior to necropsy because clostridial bacteria proliferate rapidly after death. Fluorescent antibody (FA) tests on liver smears were reported to have sensitivity superior to that of culture; however, the frequency of false positive FA test results increased as duration between death and necropsy increased. *C. novyi* types A and B have been isolated from sows with the lesions described above. Death occurs as the result of toxemia.

Endometritis: The diagnosis of endometritis is based on characteristic gross lesions in the uterus, for instance edematous mucosa and presence of exudate, and on the isolation of the etiologic agent. The most common isolates from cases of endometritis have included *E. coli*, group C streptococci, and *Arcanobacterium (Actinomyces) pyogenes*. Severe PRRS outbreaks have been reported to cause death losses ranging from 5-10% over the course of the outbreak. This increased mortality may partly result from increased bacterial endometritis and septicemia associated with retained fetuses.

Pneumonia: The diagnosis of pneumonia is based on distinctive changes in the lungs, including abnormal color and texture of the pulmonary parenchyma and frequently presence of exudate, as well as on the

isolation of the specific etiologic agent. Bacterial isolates from sows that died of pneumonia have included *Actinobacillus pleuropneumoniae*, *Pasteurella multocida*, *Arcanobacterium (Actinomyces) pyogenes*, *Streptococcus* spp., and *Klebsiella* spp.

Gastric ulcers: The diagnosis of gastric ulcers is based on the characteristic gross lesions consisting of mucosal defects in the stomach. The etiology of the gastric ulcers has been an area of recent controversy. Small particle size of cereal grains, pelleting, and grain type used in the food, stressful conditions, and heritability have been implicated as predisposing factors for the gastric ulcer development. Speculation has been made regarding *Helicobacter* spp. infection as a cause of gastric ulcers in pigs, but this has not yet been clearly established.

Downer sows: The pathologic changes observed in downer sows include vertebral abscesses, vertebral fractures, arthritis, osteochondrosis, osteomalacia/osteoporosis, and degenerative joint disease. One study evaluating sow mortality reported that musculoskeletal problems were the cause of death or reason for euthanasia in 38.2% of all sows. Histologically, necrosis of muscle fibers and an associated inflammatory response have raised questions of whether myodegeneration is a primary process, i.e., a direct result of injury, or if it occurs secondary to a debilitation process. Regardless of the cause, the end result is an animal that is unable to rise, ultimately leading to natural death or euthanasia.

Enteropathy:: Causes of sow death attributable to pathologic conditions of the intestine include swine dysentery, idiopathic hemorrhagic enteropathy, proliferative hemorrhagic enteropathy, salmonellosis, and cecal perforation. One study reported that all cases of proliferative hemorrhagic enteropathy were observed in parity zero females.

Uterine prolapse: The diagnosis of uterine prolapse is based on characteristic gross findings. This condition is likely underreported since it is often already diagnosed on the farm with no need for further examination by a veterinarian.

Septicemia: The diagnosis of septicemia is based on bacterial culture of spleen, liver, kidney, or other affected organs. Isolates from cases of septicemia have included *Erysipelothrox rhusiopathiae*, *Klebsiella* spp., *Streptococcus* spp., and *Salmonella* spp.

Effects of parity: One study reported that gilts had the lowest risk of mortality, with the risk of death increasing with each subsequent increase in parity. Authors of another study, however, reported a substantial variation in parity effects on risk of sow mortality. The initial parity had high mortality risk, but older sows were also at high risk. These authors concluded that sow mortality rates in older parities may be dependent on sow culling practices and size of the available gilt pool. Reportedly, younger sows tend to be at high risk of death from endometritis, pneumonia, and downer cow syndrome, whereas older sows tend to be at high risk of death from torsion or other accidents of abdominal organs, cystitis-pyelonephritis, uterine prolapse, and *C. novyi* infection.

Effects of season: In one study, 1/3 of all sow deaths occurred during July and August. Another study also concluded that the sow mortality in the United States (US) was significantly higher during the summer months of July through September. A Canadian study retrospectively evaluated sow deaths during the seven consecutive days in 1994 when the average temperature was the highest. Three of these seven days made up only 0.8% of the year, but accounted for 11% of the annual death loss.

Effects of management: A study evaluating the production data obtained in 1997 from 604 US farms concluded that a higher risk of sow mortality was associated with a larger herd size. A case-control study reported that multiplier herds were more likely to have higher sow mortality than commercial herds. Lactation length of 28 days or more resulted in a higher risk of mortality, but shorter lactation lengths have also been shown to result in higher mortality. High herd prevalence of metritis, urinary tract

infections, or lameness were also associated with high levels of sow mortality rate.

Effects of stage in reproductive cycle: It has been reported that 42% of all sow deaths occurred in the peripartum period, 35% occurred during gestation, 16.5% during lactation, and 6% post-weaning. In another study, nearly half of all mortalities occurred within the first three weeks after farrowing, and a sow had 24% more chance of dying if she had one or more stillbirths.

Conclusion: Sow mortality can be a large expense for the modern swine producer. Systematic diagnostic intervention, including proper record analysis, multiple necropsies, and evaluation of the risk factors, are necessary to determine the specific causes of this problem and to formulate intervention strategies. Fortunately, veterinarians are in a unique position to provide valuable diagnostic services to farms experiencing problems with sow mortality. Often a small decrease in mortality can lead to a big increase in farm profitability.

-by Jason Kelly, Class of 2003

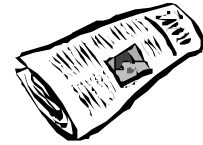
-edited by Ingeborg Langohr, ADDL
Graduate student

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ADDL NEWS



ADDL welcomes its newest graduate students **Drs. Kim Maratea and Angela Smith.** Dr. Maratea is a 2003 graduate of the School of Veterinary Medicine at Purdue. Dr. Smith graduated from veterinary school at Michigan State and has been in practice in northern Indiana for the past few years.



Dr. Maratea

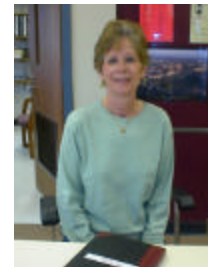


Dr. Smith

Dr. Ching Ching Wu, ADDL Chief of Microbiology/Avian Laboratory Services was elected Director of the Central Region of the American Association of Avian Pathologists at their annual meeting in Denver, 2003.

ADDL serology technicians recently completed the Bluetongue, Bovine Leucosis, and EIA check tests and were 100% accurate on all tests.

Janeice Samman, Supervisor of the Histology Laboratory, retired from ADDL at the end of September, 2003. Janeice has been in the histology lab at the ADDL for 38 years.



Visit our website at
<http://www.addl.purdue.edu>

ADDL Schedule

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

November 27-28, 2003Thanksgiving
 December 25-26, 2003.....Christmas
 January 1-2, 2004.....New Year's
 January 19, 2004.....Martin Luther King Day

Avian Chlamydiosis

Avian chlamydiosis is a disease of pet birds, poultry, and wild birds caused by *Chlamydophila psittaci* (formerly *Chlamydia psittaci*). In psittacine birds (parrots, parakeets, cockatoos, macaws, etc) and humans, avian chlamydiosis is also referred to as psittacosis. In other avian species, it is sometimes called ornithosis.

C. psittaci is an obligate intracellular organism that is endemic worldwide. The organism is well adapted to avian hosts and rarely causes clinical signs of pathologic lesions. Clinical disease is usually the result of exposure to new strains, poor husbandry, overcrowding, poor nutrition, or concurrent disease. Therefore, chlamydiosis is a common and important disease in pet bird medicine and in flock medicine.

Chlamydophila has a biphasic life cycle and exists as elementary and reticulate bodies. Elementary bodies are infectious, non-replicating particles that exist outside the body. Reticulate bodies, metabolically active particles that replicate by binary fission, form from elementary bodies that have entered epithelial cells. Rapid growth of the chlamydial organisms in the infected cells causes formation of multiple micro-colonies or intracytoplasmic inclusion bodies. Reticulate bodies then condense into elementary bodies, which may be released after cell lysis. Released elementary bodies may infect other host cells or may be released into the environment. Elementary bodies were found in feather dust, feces, urine, saliva and ocular, nasal and respiratory secretions. New hosts are commonly infected when elementary bodies are aerosolized and ingested or inhaled. Nestling birds may be infected during feeding, and vertical transmission has been documented. Manifestation of chlamydiosis in birds is variable, ranging from asymptomatic to sudden death. Asymptomatic infections are common and birds may shed the organism for several months without exhibiting clinical disease. Persistent infections in

carrier birds may be latent for years before a stressful episode leads to the emergence of clinical signs and shedding of the organism. Most acute outbreaks and deaths are in young birds exposed to high doses of a virulent strain.

When clinical signs occur in birds, they include yellow-to-greenish or watery gray droppings, weight loss, dehydration, lethargy, and ruffled feathers. Keratoconjunctivitis, rhinitis, sinusitis, dyspnea and, occasionally, CNS signs may also be seen. None of the aforementioned signs are pathognomonic, making a diagnosis of chlamydiosis somewhat difficult.

To aid the clinician in diagnosis, various diagnostic tests are available, each with their own benefits and drawbacks. It is important to remember that samples for testing should be taken prior to initiating therapy with antichlamydial antibiotics (tetracyclines, macrolides, fluoroquinolones, and chloramphenicol). These drugs may interfere with diagnostic tests by reducing antibody production, antigen shedding, and *Chlamydophila* viability.

A definitive diagnosis of *Chlamydophila psittaci* in the avian patient is usually obtained by isolation and identification of the organism in culture or by demonstrating a four-fold rise in antibody titer to chlamydial group antigens. Because organisms are intermittently shed, the best opportunity for isolation and identification in a live patient is serial fecal samples or combined choanal/cloacal culture collected for 3 to 5 consecutive days and pooled in transport media supplied by the Avian Section of the ADDL. For postmortem diagnosis, a portion of the following tissues (approximately 1 gram each) should be collected aseptically for bacterial culture, packaged individually, and refrigerated: lung, liver, spleen, kidney and intestine. Alternatively, a dead bird may be submitted for necropsy by wetting the carcass with soapy water, wrapping it in wet paper, and placing it in a plastic bag. It is best to keep the sample refrigerated until submitted. Only freeze the dead bird if it cannot be

submitted to the ADDL within 48 hours after death.

Serologic testing for chlamydiosis can be used for diagnostic evaluation in ill birds or for screening purposes. It does not detect carriers. The two serologic assays currently being used are complement fixation (CF) and elementary body agglutination (EBA). CF detects anti-*Chlamydomphila* IgG, while EBA detects anti-*Chlamydomphila* IgM. Recently infected birds are expected to be EBA positive initially and then, approximately one week later, to be CF positive. Reportedly, CF titers remain detectable as long as an infection persists. After birds are treated, EBA and CF titers usually become negative, although reports exist of CF titers remaining elevated in appropriately treated birds. Although serologic testing for chlamydiosis is not performed at the ADDL, serum samples may be submitted and will be forwarded to an appropriate laboratory by ADDL personnel.

ELISA testing may also aid in the diagnosis of chlamydiosis. As with isolation and identification testing, however, ELISA may be unreliable due to intermittent shedding of the organism. For ELISA testing, a choanal and/or cloacal swab should be submitted to the ADDL.

Other diagnostics that are infrequently used include PCR and tests to detect ribosomal RNA (rRNA) and/or ompA gene from the family *Chlamydiaceae*. PCR can be performed on blood samples or from a choanal/cloacal swab. Although very sensitive, PCR is not very specific, and a positive result may only indicate environmental exposure and not true infection. Tests for rRNA or ompA will show the presence of replicating *C. psittaci* organisms and is not currently widely available.

When examining or collecting samples from a bird that potentially has chlamydiosis, every precaution should be taken to avoid self-infection. Psittacosis is a zoonotic and highly contagious disease. All persons in contact with infected birds, including staff and clients, should be

informed about the nature of the disease. Consider wearing protective clothing, gloves, and a respirator if indicated. When performing necropsies, wet the carcass with detergent and water and work under a biologic safety cabinet (or equivalent).

Confirmed diagnoses should be reported to appropriate local and state health officials.

Please call the ADDL with any questions regarding sample collection or diagnostic testing.

-by Sarah Janke, Class of 2004

-edited by Tom Bryan, Avian Diagnostician

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PLEASE NOTE

An incorrect address for the Indiana State Department of Health rabies lab was printed in the last issue of the Forum.

The correct address is

ISDH Microbiology/Rabies Labs
P.O. Box 7203
635 N. Barnhill Drive
Indianapolis, IN 46207



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

Drs. Zheko Kounev and Tom Bryan attended the American Association of Avian Pathologists meeting in Denver, Colorado, July, 2003.

Dr. Leon Thacker attended the Midwest Regional Carcass Disposal meeting in Kansas City, Missouri, August, 2002

Dr. Bill VanAlstine spoke at the Transcatheter Cardiovascular Therapeutics annual meeting in Washington, D.C., September, 2003.

Mary Woodruff, Margaret Gelhausen and Phyllis Lockard attended the Association of Veterinary Microbiologists annual meeting in Charleston, West Virginia, August, 2003.

Bovine Blue-Green Algae Toxicosis



Blue-green algae, also known as cyanobacteria, is capable of causing sudden death when high concentrations are ingested. This toxicity is known to occur worldwide and affects not only livestock, but wildlife, marine life, and humans as well. Blue-green algae tends to grow on the surfaces of farm ponds during the summer season. The algae's potency is derived from the toxins which it can produce under favorable environmental conditions. Good environmental growing conditions include warm, stagnant water with abundant nutrients and a breeze which blows the

algael organisms shoreward for easier livestock consumption.

There are several species of algae capable of producing harmful toxins. These include *Anabaena*, *Aphanizomenon*, *Microcystis*, and *Nodularia*. Some genera produce either hepatotoxins, neurotoxins, or both. The most common culprit is *Microcystis aeruginosa*. This organism produces microcystins, which are hepatopeptides that cause severe hepatotoxicosis. Normally this toxin is confined within the cell wall; however, with cell death (following an algael bloom) or cell damage (following contact with an acidic stomach environment) the toxin is released. Animal size and species help to determine an individual's toxic dose but, generally, monogastrics seem less sensitive than ruminants and birds. Also, the bloom density and toxin concentration determine if an animal shows signs following ingestions of a few ounces or a few gallons.

The pathologic lesions caused by microcystin toxins include hepatic enlargement and congestion. The cut surface is usually friable and hemorrhagic. Histologically, there is congestion, hemorrhage, diffuse centrilobular hepatocyte rounding, dissociation, and necrosis. This rapidly leads to massive liver insufficiency, shock and death. Less commonly, certain algael species (*Anabaena* and *Aphanizomenon*) produce neurotoxins. These are referred to as anatoxins and they behave as nicotonic agonists or cholinesterase inhibitors causing muscle paralysis and sudden death from respiratory arrest.

Clinical signs of cows affected by blue-green algae toxicosis may include muscle tremors, paddling, dyspnea, watery or bloody diarrhea, cyanosis, convulsions, becoming comatose, and death usually 4-24 hours following ingestion. Animals that survive, especially cattle and horses, may develop photosensitization. Liver enzymes will also be increased.

Diagnosis of blue-green algae toxicosis is based on history of exposure, clinical signs, gross and histopathologic lesions, and a

laboratory analysis of water samples and rumen content. The species of algae can be identified after fixing a fresh water sample and a rumen content sample in 1:10 dilutions of formalin. At least 2 liters of fresh bloom material should be kept refrigerated and submitted for high-performance liquid chromatography to identify potential toxins.

If one suspects blue-green algae toxicosis, cattle should be removed from the suspected water supply. Activated charcoal can be administered to decrease toxin absorption and atropine may serve to block acetylcholine receptors if neurotoxins are suspected. Copper sulfate (0.2-0.4 ppm) may be added to the water to control cyanobacterial growth. It is imperative that livestock not be watered from the toxic source for a minimum of five days following treatment because toxin release will be abundant with the onset of algae cell death.

-by Susie Lutz, Class of 2004

-edited by Dr. Leon Thacker, ADDL
Director

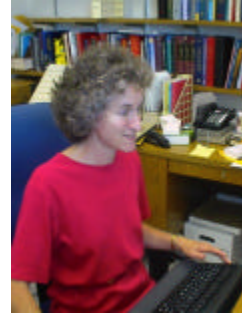
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ADDL is pleased to introduce **Drs. Peg Miller** and **Jose Ramos-Vara**, our newest pathologists. Drs. Miller and Ramos joined the ADDL faculty in September, 2003.

Dr. Miller most recently was pathologist

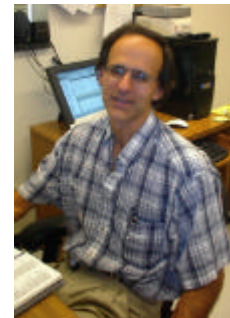


at the Veterinary Medical Diagnostic Laboratory in Columbia, Missouri. She received her DVM from the University of Missouri and her PhD in Pathology from Washington State

University, Pullman, Washington. She became a diplomate of the American College of Veterinary Pathologists in 1982.

Dr. Ramos-Vara received his DVM from

the Central University of Madrid (Spain) Veterinary School and his PhD in Animal Pathology from the Autonomous University of Barcelona (Spain). He is a diplomate of the European College of Veterinary Pathologists.



Dr. Ramos was most recently Clinical Assistant Professor in the Department of Veterinary Pathobiology at the University of Missouri, Columbia, Missouri.

Please join us in welcoming them and their daughter Maggie to Purdue.