Winter 2002

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Welcome 2002. What changes we have seen in the past 12 months! Perhaps the coming twelve will bear better. We continue to serve as a monitoring center for several potential diseases of animals for local, state, and national queries. The continued threat of purposefully introduced diseases of animals remains among our thoughts in our day to day activities in the Laboratory. It is most unfortunate that the potential and possibility for someone to introduce a disease agent into our animal populations exists but, at this point in time, we remain vigilant to detect such an action as soon as possible in the event that it should occur. It is well accepted that the eventual loss to be incurred in the event of the occurrence of a noxious known or heretofore unknown disease condition will be proportional to the time elapsed before discovery of the existence of the disease. The sooner the diagnosis is made and control procedures initiated, the lower the eventual cost to taxpayers, owners, and consumers. In many past instances, the initial recognition of an unusually destructive, foreign animal or new disease entity has been made by a private veterinary practitioner. We hope to be of assistance to the sharp diagnostic eyes of Indiana veterinarians and animal owners.

Christina Wilson, assistant chemist and graduate student in our toxicology lab, won the certificate and cash award for the best graduate student scientific presentation at the annual meeting of the American Association of Veterinary Laboratory Diagnosticians in November. A major recognition for a job well done. Cheryl Parker, Barb McDonald, Brenda Turner and Tammy Crowell, technicians in our serology area, also recently completed check tests for EIA, bluetongue, bovine leucosis, brucellosis and PRV (by three different methods) with 100% accuracy. Congratulations to each of you.

If there is something that we are doing that we shouldn't be doing or something that we are not doing that we should, let us know. Have a good day and Happy New Year.

FINAL DIAGNOSIS

Chlorate toxicosis

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

History: Two 700-800 pound castrated-male Angus calves, reportedly about 1 year old, were submitted to the Heeke ADDL in late summer for a legal necropsy. The owner reported waking up that morning to find 10 dead animals (out of a herd of 16). Affected animals included weaned grass-fed calves and mature cows. Five of the dead were lying next to the water trough; the remainder were scattered over a 10-acre pasture. The owner had seen no previous signs of illness or unusual behavior in the herd. The animals were housed in an open pasture composed of fescue and other grasses, and had access to woods. Water came from three separate ponds, two of which were spring-fed. One pond drained a piece of pasture and roadside that had been sprayed by the county on the previous day with a product allegedly similar to Roundup herbicide. All animals had been given a multivalent vaccine for blackleg at three months of age. No other vaccines or treatments had been given to the herd.

Gross Findings: The only gross lesion in either animal was a muddy-brown discoloration of the blood and body tissues (methemoglobinemia).

Preliminary laboratory findings: A drop of clear ocular fluid from both calves was placed on a white ceramic spot dish and mixed with one drop of diphenylamine reagent. A dark blue color immediately developed, suggesting the presence of nitrate in the ocular fluid. A presumptive diagnosis of nitrate toxicosis was made.

Preliminary follow-up: The owner was contacted that afternoon and told to search for a source of nitrate. Possible sources would include nitrate accumulating plants (sorghum or sudan grass, green corn silage), spilled concentrated fertilizer, or possibly runoff from a manure pit or fertilizer storage bin into drinking water. The owner reported that the pasture had no known sources of nitrate, but he did find that the cattle had broken into an old storage shed at the back of the pasture and an old rusted 1- pound can of white granular material was present on the floor of that shed. The can had been broken open and the white granular material had been scattered over the floor. It appeared that the cattle had licked the can and the white material. The owner did not know what the material was, but did not think it looked like fertilizer. A sample of this white material was submitted to the lab for analysis. The cattle were locked out of the shed.

Toxicology Findings: The white granular material was composed of irregular white crystals measuring 1-2 mm in diameter, and had no detectable odor. It was easily dissolved in water, producing a clear solution. In qualitative colorimetric analysis, the material reacted identically to sodium chlorate.

Discussion: Chlorate toxicosis has been reported in cows, sheep, horses, chickens, dogs and humans. It occurs rarely in southern Indiana, and is usually caused by accidental consumption of sodium chlorate or potassium chlorate. It apparently has a "salty" taste that cattle crave. (I did not taste the material myself.) Before the advent of modern herbicides these compounds were frequently used in southern Indiana to kill Johnson grass, and old forgotten bags or canisters of the material can still be found in many old farm buildings. Chlorate toxicosis also occurs in pet animals and young children by accidental consumption of match heads (potassium chlorate).

Like nitrate, chlorate is a potent oxidizing agent that converts hemoglobin to methemoglobin. Clinical signs of chlorate toxicosis reportedly include methemoglobinemia, ataxia, prostration, purgation, hematuria and hemoglobinuria. Death occurs very rapidly and is the usual outcome as treatment is very difficult. Unlike nitrate toxicosis, methylene blue treatment of chlorate toxicosis is often disappointing because chlorate also denatures glucose-6-phosphate dehydrogenase. (The antidotal effect of methylene blue depends on NADPH produced by glucose-6-phosphate dehydrogenase.) In addition to methylene blue treatment (4 mg/kg in dogs, 10-15 mg/kg in cattle), treatment should also include gastric lavage and vitamin C (20 mg/kg).

Some veterinarians and county agents keep diphenylamine reagent on hand to monitor nitrate levels in silage corn. The diphenylamine reaction is a useful qualitative benchtop test that can make a rapid and timely diagnosis of nitrate poisoning. Because chlorate reacts similarly to nitrate in a number of chemical reactions including the diphenylamine reaction, it can be confused with nitrate toxicosis. Chlorate toxicosis should be considered as a differential diagnosis in cases of suspected nitrate toxicosis where no nitrate source can be found.

- by Duane Murphy, Pathologist,
Heeke ADDL

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Pedunculated lipomas in horses

Pedunculated lipomas are benign tumors that most frequently arise from the mesentery of the small intestine and remain attached by a pedicle. Obstruction of intestine by a pedunculated lipoma is one of the more commonly encountered causes of colic requiring surgical management. Geldings and ponies are know to be at a greater risk of colic resulting from pedunculated lipomas than are other horses. Upon review of the records of 17 horses that were evaluated and treated because of colic caused by pedunculated lipomas between 1983 and 1990, the mean age of the horses was determined to be 16.6 $\pm$ 3.9 years. Most obstructions are closed loop strangulating obstructions resulting from the pedicle becoming wrapped around a length of intestine and the lipoma tucking itself underneath this band. The resulting tight compression occludes the lumen of the intestine and corresponding mesenteric vessels. At laparotomy, the strangulated loop of bowel frequently shows advanced infarction appearing reddish-black with very thin walls, suggesting that arterial occlusion may occur very soon after initiation of the obstruction. In contrast, in horses with non-strangulating obstruction, the lipomas are frequently large and the pedicle is drawn firmly across the intestine by the weight of the lipoma resulting in obstruction without vascular impairment. Alternatively, having encircled the gut, the lipoma may become enveloped in the mesentery.

Pedunculated lipomas start as localized plaques of fat between the two serosal layers of mesentery. As these aggregations enlarge, the overlying serosa stretches forming a pedicle, which lengthens as the weight of the lipoma increases. While many old horses and ponies have one or more lipomas attached to mesentery, only a small proportion of these animals develop strangulating obstruction. How the lipoma becomes intricately wrapped around a loop of intestine, which may be several meters long, is unknown. The weight of the lipoma, the length of the pedicle and sufficient momentum resulting from movement of the horse or intestinal motility would seem likely contributory factors in this bizarre accidental obstruction of small intestine and, much less commonly, small colon. Only one report of strangulation of the small colon has appeared in the literature. The paper describes the extensive resection and anastomosis of the descending colon of a horse after strangulation by the pedicle of a mesenteric lipoma originating from the mesocolon.

Gastric reflux is a common clinical finding in cases of small intestinal obstruction associated with ileus and buildup of fluid anterior to the obstruction. Deboom (1975) suggests that this reflux may result from compression of the

proximal duodenum by the gradual distention of the large intestine through the proximity of the attachment of these two structures, or perhaps from the effects of severe pain leading to a generalized ileus. Some reports suggest that horses with descending colon obstruction often present with mild changes and, as time progresses, there is a slow deterioration of clinical signs, and hematological and peritoneal fluid values associated with buildup of fluid and gas anterior to the obstruction.

Treatment of a strangulating lipoma involves resection of the lipoma at the base of its pedicle and resection of any compromised intestine. The decision on whether to resect, and how much to resect, is a judgement made for each individual case. Evaluation of the color of the serosa and mesentery, reflex motility, a pulse in the mesenteric vessels and evaluation of the mucosa via enterotomy are the most frequently used criteria. The incidence of complications following resection and anastomosis of the affected intestine appears to be high. Contamination is difficult to control because of high intraluminal bacterial count and particulate matter within the lumen.

Various postoperative treatment regimes have been recommended to minimize complications at surgery. A combination of antimicrobials to provide broad spectrum activity is recommended. Ideally, food should be withheld for a minimum of 24 hours, then re-introduced slowly to minimize the bulk of feed passing through the surgical site and combined with fecal softeners to reduce tension on the anastomosis. Aggressive peritoneal lavage, with heparin added to the lavage solution, frequent walking, anti-inflammatory medications, and motility stimulants may be used to minimize formation of abdominal adhesions.

The clinical signs associated with pedunculated lipomas depend on the segment and extent of intestine involved and the degree of strangulation. Horses often present with mild to moderate abdominal pain and may initially respond well to analgesic medication. The majority of horses with obstructing lipomas have palpable small intestinal distention on rectal examination. Fluid obtained by abdominocentesis may be normal early in the course of the obstruction; however, most horses with strangulating lipomas have increases in peritoneal fluid total protein and WBC counts. Lipomas are also usually discovered as incidental findings at surgery or necropsy.

The short-term survival rate for strangulating pedunculated lipoma obstruction cases alone is 43%. This may be due to a reluctance to refer older animals with colic, leading to a greater delay between the onset of obstruction and surgical correction. Endotoxemia and post-operative ileus are frequent causes of mortality in cases of strangulating obstruction. Long term survival rate is known to be 38%, indicating an appreciable mortality rate due to colic in the first year after surgery. Adhesions, mesenteric stump abscesses or stenosis of the anastomosis are all possible strangulations.

-by Saeed Bashir, ECFVG Student
-edited by Dr. Kaori Sakamoto, ADDL Graduate Student

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ADDL REMINDERS

- ✓ If specimens of the same organ are to be submitted to two or more labs in ADDL, please package and label separately.
- ✓ Labels provided on the histopath mailing jars may not be transferred to other containers or boxes.
- ✓ ADDL is closed on Saturday and Sunday. No mail or packages can be accepted on those days.
- ✓ As we have no control over the testing procedures, ADDL cannot give interpretation of results secured from testing in other laboratories.

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

Please plan accordingly.

January 21, 2002	Martin Luther King holiday
May 27, 2001	Memorial Day

Dr. Zheko Kounev joined the Purdue ADDL faculty in October, 2001 as Avian Diagnostician and Food Safety Specialist. Dr. Kounev received his DVM from the University of Sofia, Bulgaria and his PhD in Food Safety Microbiology and Meat Technology from the National Agricultural Academy, Sofia, Bulgaria. Since 1991, Dr. Kounev has served as Corporate Manager of the Biotechnology Center at Maple Leaf Farms, Milford, Indiana. Prior to that he worked for the World Health Organization at the Collaborating Center on Food Virology at the Food Research Institute, University of Wisconsin, Madison WI.

Please join us in welcoming Dr. Kounev to the Purdue ADDL staff.

ON THE ROAD

Drs. Leon Thacker, Randy White, Bob Everson, Duane Murphy, Steve Hooser, Ching Ching Wu , Charles Kanitz and Christina Wilson, Steve Vollmer and Linda Hendrickson attended the annual meeting of the American Association of Veterinary Laboratory Diagnosticians in Hershey, Pennsylvania, November, 2001.

Drs. Christine Hanika, Tom Bryan and Tsang Long Lin attended the North Central Avian Disease Conference in Grand Rapids, Michigan, October, 2001

Dr. Christine Hanika attended the Foreign Animal Disease pathologists training course at Plum Island, New York, October, 2001

Dr. Ching Ching Wu attended the National Poultry Improvement Plan meeting in Athens, Georgia, November, 2001 and the Antimicrobial Colloquium in Albuquerque, New Mexico, November, 2001

Drs. Evan Janovitz and Marlon Rebelatto attended the American College of Veterinary Pathologists annual meeting in Salt Lake City, Utah, December 2001.

Nasopharyngeal Polyps

Nasopharyngeal polyps are relatively uncommon lesions in cats which, when large enough, can be an important cause of chronic upper respiratory signs. Several reports have described the clinical presentation and the management of this disease in cats, which may reflect the increased incidence or may simply be the result of increased awareness of this lesion. Only one case report on nasopharyngeal polyps in dogs was found. The age at presentation, presenting signs, histological appearance of the polyp, treatment and postoperative course in this dog were similar to those reported in cats.

Origin and Etiopathogenesis: The etiology and the pathogenesis of nasopharyngeal polyps are incompletely understood. Speculations concentrate on congenital or inflammatory origin.

The Eustachian tube and the tympanic cavity (middle ear) originate from the first pharyngeal pouch (tubotympanic recess), and it has been proposed that nasopharyngeal polyps develop as a result of a congenital defect in this tubotympanic recess. No other congenital defects associated with polyps have been described, so this hypothesis is weak. Nevertheless, the lesion does occur in young cats.

An inflammatory basis for nasopharyngeal polyps has also been proposed. This hypothesis is based, at least in part, on the usual histologic characteristic of polyps, in particular, the presence of inflammatory cells within well-vascularized connective tissue. This stromal core is typically covered by respiratory epithelium. The inflammatory component is a consistent finding, but whether it is a cause of the polyps is not known.

It is also uncertain whether the auditory tube or the middle ear is a site of origin for the polyps. One report of four cases provided evidence for the auditory tube as the site of origin. All cats in this series were under two years of age and had a short clinical history of respiratory disease with no clinical signs, or clinical or radiographic evidence of middle ear involvement. It was concluded that middle ear disease is not a primary factor in the development of the polyps.

In another report of four cases, affected cats ranged from two to five years of age. Three of these cats had clinical signs of otitis media either preceding or following the onset of the respiratory signs. In one cat, polyps were found in both the external ear canal and nasopharynx. Histologically, these lesions were similar to one another. This case provided evidence that these polyps had developed in association with chronic otitis media, probably involving the tympanic membrane.

Signalment and Presenting Clinical Signs: Nasopharyngeal polyps are diagnosed more frequently in young cats (with a mean age at the time of diagnosis of 1-1/2 years) than in older cats. Nevertheless, the lesions has been recognized in cats less than 6 months and up to 15 years. No sex or breed predisposition has been identified.

A wide variety of presenting signs has been reported in cats with nasopharyngeal polyps. The most common signs are partial upper respiratory obstruction including respiratory stridor, dyspnea, nasal discharge, sneezing, coughing and dysphagia. Less frequent signs include otitis, typically otorrhea, aural irritation, and vestibular signs such as head tilt.

Diagnosis: Diagnosis is based on finding a soft tissue mass above the soft palate, in the nasopharynx, or in the external ear canal. Examination of the oro- and nasopharynx of anesthetized cats is usually required to visualize the lesion. The caudal edge of the soft palate can be drawn forward with a non-traumatic hook to allow inspection of the mass, and a dental mirror placed within the caudal aspect of the pharynx to evaluate the area above the soft palate. Polyps appear as glistening, pedunculated, red, pink or grayish masses in the nasopharynx. Otoscopic examination to evaluate the ear canals and the tympanic membranes for signs of otitis media while the cat is under anesthesia is also recommended since most cats with nasopharyngeal polyps have otitis media.

Radiographs of the tympanic bulla are also recommended to assess for bony destruction. Computed tomography could be utilized in some cases when radiographs are inconclusive. Hematology and serum biochemistry values in cats with nasopharyngeal polyps are typically unremarkable. Calicivirus was isolated from two of three cats in one study.

Treatment: Surgical resection is the only reported successful treatment of nasopharyngeal polyps. Surgical removal of the polyp by traction is simple and usually uncomplicated. Retraction of the caudal edge of the soft palate may be sufficient in some cases to allow the removal of the polyp with Allis forceps, using traction at the base of the attached pedicle. However, in some cases, the free (caudal) border of the soft palate requires incising to improve access.

Performing bulla osteotomy in cats with nasopharyngeal polyps may be necessary when the middle ear is involved. Although the risk of complications is significant, some veterinary surgeons recommend an ipsilateral bulla osteotomy for every cat with a nasopharyngeal polyp. This recommendation is based on a reported recurrence rate of up to 35% without, but only 2% with, bulla osteotomy.

Temporary postoperative Horner's syndrome (ptosis, miosis, prolapsed third eyelid, enophthalmos) is the most common complication of bulla osteotomy. Other complications include temporary or permanent signs of vestibular nerve paralysis (head tilt, nystagmus, and ataxia) or, rarely, facial nerve paralysis (drooping of the lip, drooling of saliva, lack of palpebral reflex. Otherwise, the prognosis for complete recovery after polypectomy is good.

-by Suliman AlGhazlat, Class of 2002
-edited by Evan Janovitz, ADDL Pathologist

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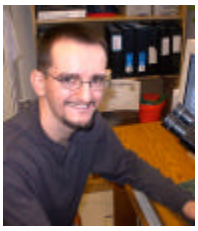
A reminder that ADDL test results are available on the Internet. If you'd like to have an account established for your practice, please contact our systems manager at

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

ADDL STAFF NEWS

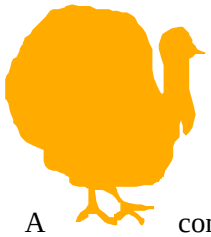
ADDL toxicology graduate student **Christina Wilson** accepts the award for best presentation by a graduate student at the annual American Association of Veterinary Laboratory Diagnosticians meeting in Hershey, Pennsylvania, November, 2001. Presenting the award is Purdue ADDL Director Leon Thacker. Ms. Wilson's work was titled "Species comparison of the warfarin-sensitive enzyme vitamin $K_1$2,3-epoxide reductase".



Dr. **Marlon Rebelatto** has completed his residency program at Purdue ADDL/ Veterinary Pathobiology and has accepted a position at MPI Research in Kalamazoo, Michigan. Please join us in wishing him well in his next endeavor.

Congratulations to Serology technicians **Cheryl Parker, Barb McDonald, Brenda Turner**, and **Tammy Crowell** who successfully completed check tests for Equine Infectious Anemia, Bluetongue, Bovine leucosis, Brucellosis, and Pseudorabies (by latex agglutination, serum neutralization, particle concentration fluorescent immunoassay, and G-1 FA. These Purdue technicians achieved 100% accuracy on all tests.

Dr. **Leon Thacker**, ADDL Director, participated in the State of Indiana Animal Health Emergencies Exercise in October, 2001. Several agencies that would be involved in responding to the occurrence of a foreign animal disease in Indiana livestock were involved in this program.



Hemorrhagic Enteritis In Turkeys

A commercial ELISA kit for Hemorrhagic Enteritis is now available to turkey clients of ADDL. Under the direction of Donna Schrader, Diagnostic Assistant, convalescent sera is measured for antibody levels. Hemorrhagic Enteritis in turkeys may manifest itself as an acute episode of frank hemorrhage throughout the intestine but, most of the time, it is subtle, weakening the immune system and allowing secondary *E. coli* to become prominent. Rarely is a flock not exposed to the virus but, when a flock reaches processing with ceca distended with "axle grease" contents, the degree of carcass contamination rises sharply. For processing and disease reasons, most producers want to alleviate problems with HE with a vaccination program. Most vaccinate at 4.5 to 5.5 weeks of age, using a mild vaccine. By taking 10-20 sera before vaccination and 10-20 sera two weeks following vaccine administration, the ELISA results give an excellent comparison to equip management with the tools to measure flock readiness.

-by Dr. Tom Bryan, Avian Diagnostician, Heeke ADDL

Johne's Disease

Johne's Disease is a disease of cattle resulting from a prolonged course of infection caused by a bacterium recently reclassified as *Mycobacterium paratuberculosis sbsp. avium.* It is an acid-fast organism that can also affect sheep, goats, other ruminants, swine and horses. However, lesions in swine and horses are minimal to none at all. In cattle, the disease has a long progression with no clinical signs for months to years after initial infection. It can eventually progress to a chronic wasting form with diarrhea, emaciation, and eventual death.

The infection is usually acquired in young animals by fecal-oral contamination. Some cattle can be infected *in utero* if born from infected cows and contaminated mother's milk can be a source of infection. A larger initial dose causes a shorter incubation period. Once infected, the organism replicates slowly in the intestinal mucosa. Over time, as macrophages are recruited to the area and the organisms continue to slowly multiply, the intestinal mucosa and submucosa become thickened and less able to absorb nutrients. The thickened wall eventually begins to leak proteins resulting in a hypoproteinemia. Weight loss and diarrhea result from the malabsorption and the albumin leakage.

In the first stage of infection, the adults that were affected as young still show no clinical signs, but antibodies may be detectable. Immunosuppression may also occur predisposing the animal to other diseases. Though the fecal culture is usually negative in this stage, shedding to the environment is possible. These animals pose an infection risk for other animals.

In the third stage of infection, clinical signs begin to appear. Gradual weight loss and diarrhea ensue. Emaciation can develop gradually and milk production decreases. Organism numbers are higher and more likely to be found in the feces. Antibody levels are also higher making them more readily detectable.

In the last stage of Johne's disease, clinical signs are more evident and the animal's condition becomes more severe. Weakness, emaciation and "pipestream" diarrhea are usually present.

Diagnosing *Mycobacterium paratuberculosis* infection can be very difficult depending on the stage of infection. Obviously, since earlier (non-clinical) stages have lower organism counts, fewer organisms are present to be shed in feces or to trigger higher antibody production. This translates to decreased detection in the early stages. By the time the numbers are high enough for detection in the later stages, the likelihood of organism spread to the environment and to other animals is already high. For every cow that shows clinical signs, there are potentially several others that are infected due to environmental contamination. Moreover, since treatment is not efficient, infected animals are culled causing further economic losses.

Despite the problems associated with disease staging, there are several tests available to diagnose Johne's Disease in cattle. Fecal culture is one of these tests. It is considered the "gold standard" for diagnosis. The high specificity of the culture makes it a popular test. The culture test has been shown to detect organisms 1-4 years prior to the development of clinical signs, giving it a relatively high sensitivity. Yet, if an infected animal is not shedding at the time of sampling or is shedding low numbers, the culture may miss the true status of the animal. Another major disadvantage of the test is that completion takes from 12-16 weeks.

Other methods used for diagnosis involve serological testing (ELISA, AGID and complement fixation). These tests take a relatively short time to produce results and several samples can be processed each day. But the earlier the stage of infection, the lower the sensitivity will be. It is not until later stages that serum changes become adequate enough for these tests to gain respectable sensitivities.

Enzyme-linked immunosorbent assay (ELISA) measures serum antibodies directed against *Mycobacterium paratuberculosis.*

Because cattle are exposed to mycobacterial antigens from several different mycobacterial species, the specificity of this test is relatively low. Sensitivity, however, is considered relatively higher (50%) with ELISA than with the other serum tests and, if used with other diagnostic tests (e.g. fecal culture, DNA probe), is considered a good tool for herd screening.

Agar gel immunodiffusion (AGID) is considered highly specific (100%). Positive test results correlate well with clinical signs. But the sensitivity is low for this test. If the animal is not showing clinical signs, the likelihood of a positive result is low (less than 50%). When comparing AGID with ELISA, AGID is better for diagnosing infection in animals that are already showing clinical signs. On the other hand, ELISA is better for detecting infection in animals without clinical signs, with localized infections and low-level mycobacterial burden.

Complement fixation also has low sensitivity in lightly infected animals; however, positive results correlate well with active shedding of organisms. It must be noted that false positives have been experienced when using this test.

Cell-Mediated Immunity (CMI) is the first and strongest response to mycobacterial infections. Tests to detect CMI, which is mediated by T-lymphocytes, have low specificity but high sensitivity. Skin testing often misidentifies *M. tuberculosis* infections since the organism shares several antigens with other environmental mycobacterial species and the potential for cross-reactions with these is great. Tests that measure paratuberculosis specific gamma interferon, a sensitized T-lymphocyte mediator, have been shown to have higher sensitivities than other serological tests, but specificity is still low.

PCR testing represents a newer form of diagnosis and is used to detect *Mycobacterium parabuterculosis* in the feces of infected animals. At the Purdue ADDL, we have combined PCR with culturing to give better accuracy and shorter time for results. It is also highly specific (nearly 100%). However, it is dependent upon active shedding in the feces and subclinical infections may be missed.

In more than half of affected cattle, gastrointestinal lesions have been shown to extend to the rectal mucosa and colon. This occurrence offers the opportunity for diagnosis via rectal scrapings. Gently scraped mucosa is applied to and air dried on glass slides, then stained for acid-fast organisms (e.g. Ziehl-Neelsen stain). Microscopic exam should be able to detect the presence of clumps of acid-fast organisms still inside macrophages. Histopathology of the ileocolic lymph node and terminal ileum should also show the same acid-fast clumps inside the macrophages of an infected animal. These microscopic signs are enough to declare a positive diagnosis. These microscopic diagnostic methods are useful when testing an individual animal suspected of having Johne's disease. Also, herd culling can influence test performance. For example, if all of the positive and/or clinical animals have been removed from the herd, the remaining population will be all non-clinical and more difficult to diagnose. These and other factors (such as testing expense) must be considered when deciding on diagnostic testing to identify mycobacterial infection in a herd.

-by Lee Winnette, Class of 2002

-edited by Marlon Rebelatto, ADDL Graduate Student

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