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A Quarterly Newsletter from the Animal Disease Diagnostic Laboratory
Purdue University, West Lafayette, Indiana 47907 (765-494-7440)

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Good day from ADDL. The weather has started to cool and fall is in the air. Perhaps the major weather related issue for our state this late spring, summer, and fall has been the lack of rain. this has brought about shortage of pasture and hay over much of Indiana. We have encountered some cattle problems from feeding of alternate feed sources. The shortage of roughage normally present this time of year and earlier have brought about changes in feeding practices of feeding corn stalks, small grain straw, soybean pummies, raw soybeans and other materials. caution is to be exercised that these feedstuffs are, first of all, not toxic.

Two other animal diseases that have been newsworthy in our state this fall have been epizootic hemorrhagic disease of deer and occasionally cattle and Eastern equine encephalitis of horses. Epizootic hemorrhagic disease (EHD) is caused by a virus that is related to, but distinctly different from, Bluetongue virus. It is transmitted by biting midges; when we get a killing frost or two, the vector of the disease should be markedly reduced and the occurrence of the disease in deer reduced as well. We have received reports of deaths of large numbers of white tail deer, both captive and wild, in southern Indiana and adjoining states. At the time of this writing, no vaccine is available to protect susceptible animals.

Eastern equine encephalitis (EEE) is also caused by a virus, spread is via mosquito vectors. Most of the reports of EEE affected horses have come from northern Indiana; we have had positive results of horses as far south as Clay County. Again, as the vector is an insect, killing frost(s) should reduce the means of the disease distribution. In the instance of EEE however, effective vaccines to protect horses from the disease are available and in routine use among many or most Indiana horse owners.

The Indiana Department of Natural Resources will again send tissue samples from hunter killed white tail deer to us for chronic wasting disease testing. In accordance with requirements of the Indiana Board of Animal Health, any captive deer or elk that die of any cause in Indiana are to be tested for CWD. Over the past six years, we have tested several thousand Indiana deer and elk for CWD. To date, we have found no positive cases of CWD in Indiana deer or captive elk. We expect to test somewhere around twelve or thirteen hundred deer again this year.

The ADDL has a new fee schedule that took effect October 1, 2007. We've put a great deal of thought and comparison with other labs into setting the fees. It is our hope and intention to provide the best diagnostic services for the most reasonable prices we can offer. Hope you are enjoying the fall weather; it is beautiful in Indiana.

Final Diagnosis
Canine Mammary Gland Tumor

Introduction: Mammary gland tumors are the most frequently diagnosed neoplasms in female dogs. Roughly 80% of canine mammary tumors are diagnosed in bitches older than 7 years. About 30% of the cases are malignant mammary carcinoma. These tumors can be solitary or multicentric (Figure 1).

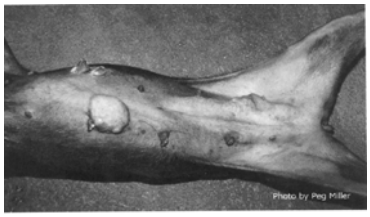

Figure 1. Doberman Pinscher with mammary tumors in multiple glands

Tumor types: There has been a long-lasting debate about classification of canine mammary tumors. According to the World Health Organization (WHO), canine mammary tumors are histologically classified into four categories: malignant tumors, benign tumors, unclassified tumors, and mammary hyperplasias/ dysplasias. This classification reflects cellular atypia, pattern of neoplastic growth (e.g. tubulopapillary vs. solid), the origin of neoplastic cells (e.g. epithelial vs. mesenchymal), and descriptive morphology of each cellular component. Subclassification depends on clarification of neoplastic cell profile. The most common types are benign mixed tumor, adenoma, and adenocarcinoma. Benign mixed tumors consist of epithelial tumors and mesenchymal tumors, which can be manifested as bone, cartilage, or fibrous tumor.
Adenoma and adenocarcinoma have many different subtypes. In order to incorporate prognostic factors, reclassification of canine mammary carcinoma has been attempted with statistical backups. This reclassification, in the order of increasing malignancy, includes non-infiltrating carcinoma, complex carcinoma (Figure 2), simple carcinoma and simple anaplastic carcinoma.

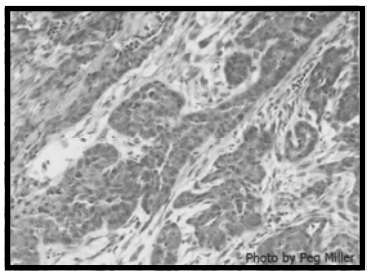

Figure 2. Photomicrograph of a complex mammary carcinoma. Tubular structures and solid nests of neoplastic epithelial cells are surrounded by proliferative myoepithelial cells in myxomatous stroma

It may also be of note that inflammatory carcinoma is not a distinct entity of mammary carcinoma, but rather, a particularly invasive mammary carcinoma with prominent inflammation. Inflammatory carcinomas account for less than 5% of all mammary tumors.

Causes and risk factors: The cause of canine mammary tumors is unknown; however, there are several factors that may influence the development of mammary gland tumors. That hormones such as estrogen and progesterone can be a risk factor is shown by the fact that ovariohysterectomy decreases the incidence of mammary tumors by avoiding the influence of these hormones. However, risk of developing a mammary tumor in later life is closely associated with the time when a bitch is spayed. If the dog is spayed before the first estrus, such risk is less than 1%. If the spay is done before the 2nd or 3rd estrus, the risk is 8% and 26%, respectively. Little benefit has been reported with spaying after the 3rd estrus. Obesity has been correlated with higher prevalence of mammary tumors in humans and dogs. Inherited predisposition for the development of mammary neoplasms has been described in human medicine, but studies are still underway in veterinary medicine.

Clinical signs: What will the pet owners notice? Canine mammary tumor usually appears as variably-sized, single or multiple, soft to firm, discrete to poorly-defined masses or lumps associated with mammary gland(s). Dog owners may notice color change (red to purple) and/or ulceration on these masses. Clinical signs relevant to tumor-associated disease may be noticed on rare occasions. In particular, inflammatory carcinoma has been described to cause significant pain in the mammary region, axilla, groin, or medial aspects of the limbs due to extensive inflammation and/or edema secondary to tumor invasion in regional lymph vessels and nodes. With metastatic disease, clinical manifestations would reflect the affected organ's location and function; for instance, a dog with pulmonary metastasis might show dyspnea or intolerance for exercise.

Diagnosis: Clinical evaluation of the patient is important as part of the diagnostic procedures. Thorough palpation of both mammary chains is indispensable. Tumors are more often found in the caudal pair of mammary glands. Multicentric involvement is common. Care should be taken to distinguish neoplastic from non-neoplastic masses such as cystic hyperplasia or duct ectasia if a patient is in estrus or has recently experienced pregnancy or pseudopregnancy. These masses sometimes simulate neoplasia; however, they usually regress as the influence of estrogen declines.

As a part of the clinical evaluation, complete blood count and serum chemistry profiles are performed. Thoracic (3 views-left and right laterals and ventro-dorsal) and abdominal (2 views—lateral and ventro-dorsal) radiographs are useful to detect metastases. Ultrasound examination is indicated if metastasis to the abdominal organs is suspected.

The definitive diagnosis is based on histopathology on excisional biopsy specimens. In addition to aforementioned histological classification, WHO suggests tumor-node-metastasis (TNM) staging on canine mammary tumors to provide more practical prognostic information.

This staging is based on the size of the primary tumor, presence/absence of tumor metastasis to regional lymph nodes, and presence/absence of distant metastasis (Table 1). The greater the stage, the poorer the prognosis.

Stage	Primary Tumor	Regional LN Status	Distant Metastasis
I	T1	NO	MO
II	T2	NO	MO
III	T3	NO	MO
IV	Any T	N1	MO
V	Any T	Any N	M1

Table 1. Modified TNM staging of canine mammary gland tumor.

- T1 < 3 cm maximum diameter
- T2 3-5 cm maximum diameter
- T3 > 5 cm maximum diameter
- NO Histologically no metastasis
- N1 Histologic metastasis
- MO No distant metastasis
- M1 Distant metastasis detected.

(Reference: Philbert JC et al: 2003.J Vet Intern Med 17:102-6)

Though fine needle aspirate (FNA) and cytological evaluation of the sample has been reported as part of the diagnostic workup, they rarely provide information as to the malignancy of a mammary tumor. Mast cell tumor in a mammary location can be readily ruled in/ out by FNA.

Treatment: There are several options to treat canine mammary tumors including surgery, chemotherapy, radiation, immunotherapy, hormonal therapy, and diet. Nonetheless, surgical excision is still the most effective modality. The choice of surgical methods has been vigorously discussed; however, it is important to tailor the remedy to clients' needs and to treat patients individually. If surgery is successful, with clear histologic margins, and the patient has no evidence of lymph node involvement or metastasis, chemotherapy is not recommended. The most commonly used chemotherapeutic agents are doxorubicin and cisplatin. One potential side effect of doxorubicin is cardiotoxicity. Potential renal toxicity may occur with the use of cisplatin. Paclitaxel has been experimentally used to treat canine malignant mammary tumors with high incidence of side effects. Hormonal therapy, such as tamoxifen citrate (an antiestrogen drug used in human estrogen receptor-positive breast cancer), has been used in a limited number of dogs. An advantage of this medication is that it can be given orally. The disadvantage is that 25% of the animals have side effects such as vulvar swelling and pyometra. Radiation therapy and immunotherapy have been studied, but efficacy is not proven. A theory proposing that a high protein/ low fat diet may prolong survival has been advanced.

Prognosis: The most important prognostic information is derived from the WHO staging system and histologic diagnosis. Size of the tumor is thought to be a good prognostic indicator. Dogs with smaller (less than 5 cm in diameter) malignant tumors have a better prognosis for long-term survival. Metastasis is observed in about half of malignant tumor cases. Histologic evaluation is critical not only to determine the origin of the neoplastic cells, but also to find microscopic evidence of possible metastases. For example, invasiveness of tumor cells into adjacent normal tissue can be demonstrated by immunohistochemistry with antibody for calponin (marker for myoepithelial cells), which will distinguish *in situ* from invasive carcinoma. Early surgical intervention and the method of surgery play important roles in prolonging survival. Ovariohysterectomy at the time of tumor excision has been recognized to increase survival time.

-by Dr. Ikki Mitsui, ADDL Graduate Student

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

2007

November 22-23……..Thanksgiving
December 24-25…….…….Christmas
December 31……..…..New Year's Eve

2008

January 1……New Year's Day
January 21……..……..Martin Luther King Day
May 26……..……..Memorial Day

Canine Lymphocytic-Plasmacytic Gastroenteritis

Canine lymphocytic-plasmacytic gastroenteritis (LP) is one disease in a group of idiopathic, chronic intestinal diseases collectively termed inflammatory bowel disease (IBD), and is considered to be the most common cause of chronic vomiting and diarrhea in dogs. LP gastroenteritis is characterized by a diffuse infiltration of lymnphocytes and plasma cells into the lamina propria of the stomach and/or the small intestine resulting in diffuse mucosal inflammation. Lymphocytic-plasmacytic is the most prevalent form of IBD. Other forms of IBD may be due to other cellular infiltrates (i.e. eosinophilic gastroenteritis, granulomatous enteritis, chronic histiocytic ulcerative colitis in Boxers).

The definitive cause and pathogenesis of this disease remains unknown. It is thought that an abnormal mucosal immune response to various environmental antigens, potentially dietary or parasitic in origin, results in a host hypersensitivity reaction that is responsible for the recruitment of inflammatory cells. Continued exposure to the antigen perpetuates the gastrointestinal inflammation. Subsequent damage results from the elaboration of cytokines, release of proteolytic and lysosomal enzymes, complement activation secondary to immune complex deposition, and the generation of oxygen free radicals. Delayed gastric emptying can then occur secondary to the inflammation. Hereditary factors may also influence the development of IBD. Basenjis and Ludenhunds are known to have familial forms of IBD; German shepherd dogs and Shar-peis have been reported to be predisposed to LP gastroenteritis specifically. Typically, middle aged to older dogs are affected and no sex predilections have been described.

Chronic small bowel diarrhea is the most common clinical sign of lymphocytic-plasmacytic gastroenteritis. If only the stomach is involved, vomiting might be the most common sign in dogs. Initially, the chronic diarrhea and vomiting is intermittent, but eventually increases in frequency over time. Additionally, weight loss with a waxing and waning appetite is usually reported. On physical exam, poor BCS, dehydration, ascites, thickened bowel loops, fluid filled intestines, or abdominal pain may be noted. Initial diagnostic findings may include a neutrophilic leukocytosis with a left shift, microcytic anemia, hypoproteinemia, hypoalbuminemia, and hypocholesterolemia. Additionally, extra-intestinal manifestations of IBD to be aware of include growth retardation, thrombo-embolic episodes, polyarthritis, nephrolithiasis, dermatologic disease, and immune-mediated anemia.

The exclusion of diseases that mimic IBD along with histopathology is the primary way to diagnosis LP gastroenteritis. A differential diagnosis list should include infectious diseases such as histoplasmosis, Intestinal bacterial overgrowth, other infiltrative inflammatory bowel conditions (e.g. eosinophilic enteritis, granulomatous IBD), neoplastic conditions (e.g. lymphoma), and other diseases such as lymphagiectasia, gastrointestinal motility disorders, and exocrine pancreatic insufficiency. Additional diagnostic tests that should be performed to rule out these diseases may include urinalysis to rule out a renal cause for protein loss, zinc sulfate fecal flotation (*Giardia* sp.), fecal culture (*Salmonella* sp., *Campylobacter* sp, *Clostridium* sp.), folate/cobalamin assays (bacterial overgrowth), TLI or trypsin-like immunoreactivity test (exocrine pancreatic insufficiency), abdominocentesis to classify any abdominal effusions (IBD typically results in a pure transudate), and diagnostic imaging including abdominal radiographs and ultrasound and/or contrast studies to help define the disease distribution.

Following a diagnostic exclusion protocol, upper gastrointestinal endoscopy and biopsy is the diagnostic test of choice. Studies indicate that increased granularity, increased friability, and erosions are the predominant mucosal lesions seen endoscopically. Grossly, the stomach and intestine may also appear edematous, thickened, or completely normal. Focal gastric ulceration alone is not frequently seen in dogs with LP gastroenteritis. Some reports indicate that visible endoscopic lesions on the gut mucosa are present in only 50% of canine IBD cases. Histologic findings reveal an infiltrate of lymphocytes and plasma cells in the lamina propria. Less commonly, the cellular infiltrates may extend into the submucosa and muscularis tissue layers. Villous clubbing, atrophy and fusion can be seen on biopsy in some severe cases. The distribution may be irregular, so several biopsy specimens need to be collected.

Even though endoscopic biopsies of the gastrointestinal tract are less invasive with minimal complications, limitations of this procedure include a requirement for expensive equipment and highly skilled personnel, the entire bowel is not accessible, and the small sample size can occasionally cause problematic diagnostic interpretations. Since the diagnosis of IBD is subjectively based on the evaluation of the cellularity of the lamina propria, there can be considerable variability in biopsy interpretation. This may be due to technical constraints of specimen size along with procurement and processing artifacts involved in collecting endoscopic samples. Studies have shown that significant interobserver variation exists when attempting to describe histopathologic lesions of intestinal biopsy specimens from dogs with and without inflammatory intestinal diseases. The alternative to endoscopic biopsies is full thickness surgical biopsies of the gastrointestinal tract acquired through a laparotomy. Large, full thickness biopsies obtained are reported to provide definitive histopathologic diagnosis more readily than endoscopic biopsies, especially to rule out gastrointestinal lymphoma. Disadvantages of surgery in an IBD patient include increased morbidity and a necessary delay before initiating steroid therapy.

Standardization of histopathologic descriptions of intestinal tissues that correlate with clinical signs would be ideal for clinicians attempting to diagnose lymphocytic-plasmacytic gastroenteritis. However, unlike in human medicine, uniform and objective morphological criteria for IBD lesions have not been established. Attempts have been made at creating a system of clinical indices to assess and classify mild, moderate, and severe cases of IBD in dogs. Factors such as attitude/activity, appetite, vomiting, stool consistency, stool frequency, and weight loss have been used to correlate clinical signs with histologic lesions. Through the development of a standardized scoring index, not only can an accurate diagnosis be made histologically, but, by knowing the extent of the disease, the appropriate therapy can be correctly instituted.

Finally, if finances are a concern to the client, a hypoallergenic diet trial may be started first in order to rule in or out dietary allergy or intolerance. Dietary management is the most important aspect of the treatment of LP gastroenteritis and it is sometimes possible to control the disease with diet alone. Therefore, if clinical signs resolve, no further work-up is necessary and dietary manipulations should not be overlooked as a diagnostic tool in some patients. Controlled diets are the most common dietary manipulation used for the treatment of IBD cases. Prescription or homemade controlled diets include a highly digestible carbohydrate source, are gluten-free, low in lactose and fat, and contain a novel protein source. Prescription low residue diets with a hydrolyzed protein source may also be effective. Additional aspects in the treatment of LP gastroenteritis may include providing immunosuppression using corticosteroids, azathioprine, budesonide, cyclophosphamide, or cyclosporine, treatment with immunomodulators and antibiotics such as metronidazole, tylosin, and salicylates, and empirical treatment with fenbendazole and cobalamin supplementation.

There are several possible routes to reach a diagnosis of lymphocytic-plasmacytic gastroenteritis, making it a challenge to quickly and accurately diagnose. Once a diagnosis is established, the short term for prognosis is good for control, but poor for a cure. Chronic therapy is usually indicated making owner compliance important.

-by Scott Trapp, Class of 2007

-edited by Dr. Robert Johnson, ADDL Graduate Student

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ADDL NEWS

Dr. Steve Hooser, ADDL Head of Toxicology and Analytical Chemistry, has been named Chair of the AAVLD Veterinary Analytical Toxicology and Mycotoxin Committee and Chair of the Toxicology Working Group of the National Animal Health Laboratory Network.

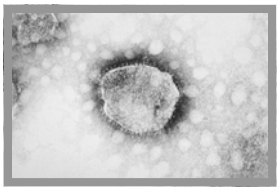
Congratulations to our **Serology technicians** who successfully completed proficiency tests for Bluetongue, Equine infectious anemia, Bovine leukosis virus and Brucellosis.

Dr. Christina Wilson was recently named Head Analytical Chemist in the ADDL Toxicology section.

Laboratory Records Clerk **Mary Fran Nelson** retired on June 30, 2007 after 17 years of service to ADDL. Please join us in wishing her well.

Dr. Joshua Webster, ADDL graduate student, was awarded a travel grant to the American Association of Veterinary Laboratory Diagnosticians meeting in October, 2007.

Lymphocytic Choriomeningitis



Introduction: Lymphocytic choriomeningitis is a viral disease of mice that poses a zoonotic risk to humans. The lymphocytic choriomeningitis virus (LCMV) is an arenavirus (single-stranded RNA virus). Arenaviruses derive their name from their characteristic morphology which includes the unique feature of host ribosomes within the virion (*arena*=sand). There are several strains of the virus; three that exhibit differences in mouse pathogenicity are WE, Armstrong, and Traub.

Wild mice are the resident host for LCMV, and laboratory mice and Syrian hamsters can be naturally infected.

Transmission: LCMV can be shed in saliva, urine, and milk; chronically infected mice can shed the virus for a very long time. Most commonly, the virus is transmitted transplacentally, although bite wound and aerosol spread can occur.

When mice are infected *in utero* they are born without virus-specific cytotoxic T lymphcytes and are considered immunotolerant,subclinical carriers. Perinatal exposure to the virus results in persistent infection. Experimental inoculation of immunocompetent adults results in fatal disease characterized by hepatitis and encephalitis; natural infection is generally cleared after an exaggerated immune response by adult mice.

Pathology: There are two forms of the infection in mice: persistent form and acute form. The persistent form of the disease is acquired perinatally, results in lifelong viremia and shedding, and animals are asymptomatic due to immune tolerance. These animals appear normal until approximately 7-10 months of age, at which time they develop immune complex-induced glomerulonephritis and die. The acute or non-tolerant form of the disease arises from infection of immunocompetent adults and results in viremia without viral shedding. In these animals, the disease is caused by T lymphocyte-mediated immune injury in response to the virus; immunosuppressive therapy halts the disease. A similar pathogenesis has been described for Syrian hamsters.

Many tissues can have non-suppurative inflammation in severe infections. Both cytolytic and proliferative lesions can be seen and the type is determined by the strain of the mouse affected. Lesions in the liver can include necrosis, accompanied by nodule infiltrates of lymphoid and Kupffer cells, and activated sinusoidal endothelium. In lymphoid organs, cytolysis, cell proliferation, and fibrinoid necrosis can be seen. In later stages of the disease, the lesions observed are inflammation associated with immune complex deposition. The most severely affected tissues are renal glomeruli and choroid plexus, but immune complex deposition can also occur in blood vessel walls, synovial membranes, and skin. It is interesting that the lymphoid tissues in these animals are so severely affected, yet the immune system is still sufficiently strong to mount a response vigorous enough to kill the animal.

Clinical signs: The clinical signs seen in LCMV infection vary with the strain of the virus involved. The most virulent strain in mice and humans is WE. In the cerebral form, convulsions, photophobia, and weakness characterize the disease.

In rats, viral inoculation causes retinitis, auditory and visual impairments, and cerebellar hypoplasia.

Diagnosis: Commercially available enzyme-linked immunosorbent assays (ELISA) and immunofluorescence assays (IFA) can be used to diagnose LCMV. Adult mice infected with LCMV will exhibit sero-conversion after exposure but carrier mice may develop poor humoral immune response or consume antibody through immune complexes, which can lead to a false-negative result. Therefore, using an adult contact sentinel becomes useful to detect the presence of LCMV by seroconversion.

In mice that develop neurologic signs, lymphocytic choriomeningitis must be differentiated from mouse hepatitis virus, mouse encephalomyelitis virus, bacterial meningoencephalomyelitis, and other causes of neurologic disease such as toxicities, neoplasia, or trauma. Presentations of early onset must be differentiated from other causes of early mortality such as ectromelia virus, mouse hepatitis virus, and Tyzzer's disease (*Clostridium piliforme*).

Control: Control of LCMV infection includes barrier maintenance in holding facilities, parasite control, and obtaining mice from clean sources. It is imperative to ensure that the animal feed has not been contaminated by wild rodents. In the case that LCMV infection is diagnosed, all animals exposed should be tested and those positive should be eliminated. The facilities should be decontaminated with appropriate disinfectant.

Human implications: LCMV poses a serious zoonotic concern and human infection is characterized by fever, myalgia, sore throat, photophobia, nausea, and vomiting. It can range from mild disease (fever and respiratory symptoms) to severe illness (meningitis and meningoencephalitis). In humans, the incubation period for LCMV is approximately 6 to 13 days. An influenza-like prodromal stage develops and, after a brief recovery, central nervous system (CNS) signs may appear. Additionally, CNS signs may appear without a prodromal stage. When meningitis or meningoencephalitis develops, the course of the disease can be lethal.

In utero hamster-induced human infections have led to the development of hydrocephalus, uveitis, and chorioretinitis. Also, these children were noted to have mental retardation and reduced visual acuity.

Differential diagnoses for the disease in humans include herpes encephalitis, mononucleosis, meningitis, and influenza. Ninety percent of human outbreaks have been associated with pet trade. Of these, ninety percent are associated with hamsters.

Humans become infected with LCMV through direct contact or contact with infected food. In laboratory settings, human infections arise more commonly from handling materials such as cell lines, monoclonal antibodies, and transplantable tumors containing the virus rather than from direct handling of viral cultures.

Diagnosis of LCMV infection in humans is based on virus isolation from blood or cerebrospinal fluid in conjunction with IFA assay.

In humans, ribavirin has been used to treat infections with beneficial outcome when administered early in the disease. Still, because of the low incidence of the disease, there is no sufficient evidence of treatment success.

Impact on research: LCMV infection in mice has a variety of effects on research, especially immunologic research. LCMV infection induces development of severe cellular and humoral immune suppression, enhances natural killer (NK) cell activity, interferon production, macrophage function, and virus-specific cytotoxic T-cell proliferation. It also increases endothelial adhesion molecules in the serum, alters cytokine gene expression, and delays skin allograft and tumor rejection. Additionally, LCMV infection increases susceptibility to infection with various agents such as ectromelia virus, inhibits tumorigenic potential of polyoma virus, inactivates experimental hepatitis B infection, and alters behavior and cognitive functions in mice.

LCMV has been identified in transplantable tumors, cell cultures, strains of organisms that are passed on to laboratory mice, and monoclonal antibodies. As mentioned above, this poses a serious zoonotic risk.

-by Dr. Jose Vilches, Class of 2007
-edited by Dr. Pam Mouser, ADDL Graduate Student

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New Serology/Virology Tests Available

- PCV2 IFA (titration)—to determine level of antibodies to Porcine circovirus in swine serum sample……..$3.00
- PCV2 screening ELISA-to detect the presence of antibodies to PCV2 and PCV1 virus in porcine serum samples……$3.00
- PPV cELISA -to detect the presence of antibodies to porcine parvovirus……..$4.00
- Swine influenza virus (H1N1) ELISA -to detect the presence of antibodies to porcine influenza virus (H1N1) in porcine serum samples …….$4.00
- Swine influenza virus (H3N2) ELISA -to detect the presence of antibodies to swine influenza virus H3N2) ……...$4.00
- Sequencing and analysis of PRRS virus ORF5 region from positive samples tested by PCR or VI……$100.00

See additional information in the new 2007 ADDL fee schedule

Invoicing Changes

In the first quarter of 1007, Purdue University launched the OnePurdue program. This business operating system was designed to unify the various legacy computer systems into a single entity better able to serve Purdue's employees, students and customers. That's the upside.

The downside it that it also changed the look of ADDL invoices. We at ADDL do not have any control over this function and have been working on a solution to give you the information you were accustomed to seeing on an invoice. When a final report is issued, a "Billing Summary Report" will also be issued which will contain the line item details that were available to you in the previous invoice format. it will be sent to you in the same manner as the final report (fax, email, US mail).

NOTE: *The Billing Summary Report you receive from ADDL is not an invoice and is to be used for your information only. The actual invoice will still be sent to you from Purdue University.*

If you do not wish to receive the billing summaries, please contact us at 765-494-7440.

DIAGNOSTIC FORUM

DIAGNOSTIC FORUM is a quarterly newsletter of current services, regulations and research projects involving the ADDL which may be of interest to Indiana veterinarians and animal owners. It is our intention that the information provided will serve you. Please send your comments, suggestions, requests and questions to: Diagnostic Forum Editor, Animal Disease Diagnostic Laboratory, 406 S. University St., Purdue University, West Lafayette, In 47907.

ADDL SECTION HEADS

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Assistant to Director: Linda Hendrickson, BS, MA
Avian: Tsang Long Lin, DVM, PhD
Bacteriology: Ching Ching Wu, DVM, PhD
Business Manager: Tonya Byrd, BS
Computer: Steve Vollmer, BS
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Greg Stevenson, DVM, PhD
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Serology: Roman Pogranichniy, DVM, PhD
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