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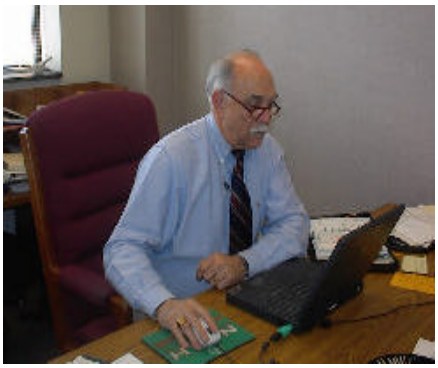
Spring 2004



FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Welcome to another beautiful spring. It is one of the perks of living in our latitude and longitude to see the change of seasons and the newness that each brings. This time of the year brings a new set of conditions to the diagnostic lab. The national animal scene is also bringing changes to our operation with the threat of intentional introduction of disease agents to our animal population. We are reminded to be especially alert to the possible occurrence of a foreign or new disease entity being brought to us for diagnosis. Recognition of some foreign animal diseases must, of necessity, be relegated to study of the conditions via distance learning mode as many or most of these have not occurred in this country. Many individuals in the ADDL have had foreign animal disease recognition training on Plum Island put on by USDA-APHIS. This training is available only on this single site of our country where the most severe of many foreign animal diseases are confined for study and teaching of animal disease diagnosticians. If a foreign or newly developed disease entity occurs in our country, the economic impact will be directly proportional to the time it takes to discover the existence of the disease. If the condition is recognized early, the economic and physical loss will be much less than if the condition is widespread before it is diagnosed and elimination procedures are initiated. The threats of avian influenza, exotic Newcastle disease, classical swine fever (hog cholera), foot and mouth disease, anthrax, and others are continually with us.

The histopathology services of the ADDL have been hampered over the years by delay in getting the samples delivered to ADDL by the postal service. This will be somewhat alleviated by the availability of next day delivery of histo samples to the lab via FedEx. Special mailers for sending samples for next day delivery are now available. We will send out mailers in boxes of 12 for $80; this will cover the cost of pickup anywhere in Indiana and return of the formalin container to the lab via next day delivery. We expect that tissues received will be processed for reading the next day, results will be available on the internet or by fax transmission immediately after the pathologist writes the report. This will markedly shorten the turnaround time of histopathology in the lab.

The ADDL is also now receiving boar semen for PRRS testing with same day turnaround. This available test will be a major factor in controlling this most economically important of swine infectious diseases of this day in time.

The faculty and staff of the ADDL continue to strive to serve the veterinarians, animal owners and researchers of Indiana with the most accurate, expedient and appropriate animal diagnostics available. If you have suggestions for improvement of our services, please do not hesitate to inform us. Have an enjoyable spring!

FINAL DIAGNOSIS:

Polycystic bile duct disease

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

History: A female-spayed domestic short-haired cat, reportedly 14.5 years of age, was submitted for necropsy to the Purdue Animal Disease Diagnostic Laboratory. The cat had been euthanized by the referring veterinarian following a chronic decline in health and a one year history of blood in the urine. Radiographs revealed multiple cysts in the liver. Additional history of the cat included diagnosis of hyperthyroidism four years previously which had been treated with I-131.

Gross findings: The subcutis of the cat was jaundiced and the liver was markedly enlarged and misshapen, and weighed 12.6% of the cat's total body weight. The capsular surface of the liver was irregular, mottled red to white, and had multiple white umbilicated depressions. The left lateral lobe of the liver had a cavernous cyst that contained 20-30 ml of serosanguinous fluid. On cut section, the lobe had a dark red lining with multifocal embedded white strands of fibrous tissue. The right medial liver lobe was expanded by a cyst that contained 10-15 ml of clear, viscous fluid. The lining of the cyst was dark tan to pale white. The remainder of the liver was mottled dark red to tan with multifocal white and green foci that extended into the parenchyma.

In addition, within the mid-jejunum, there was a dilation of the small intestine, approximately 5 cm in length, which contained a 1.4 cm vertical, transmural tear that was surrounded by a focal, dark purple to red discoloration of the adjacent serosa. Within the intestinal lumen in this area, there was a 1.2 cm in diameter foreign body that was black, jagged, fetid, and had an orange-brown core. Both kidneys were mildly shrunken and palpably firm with a pitted appearance to the capsule. The renal medullas were prominently bright white.

Histologic findings: The liver was characterized by multiple, variably sized, cystic compartments that were lined by flat, cuboidal epithelium that was suggestive of bile duct epithelium. These polycystic bile ducts were occasionally encapsulated by moderate amounts of fibrous stroma and contained proteinaceous material. Adjacent hepatic parenchyma was congested, compressed, and disorganized. Portal areas contained numerous proliferating bile ducts and increased fibrous connective tissue. Bile pigment was observed in sinusoids and numerous hepatocytes (bile stasis).

The capsular surface of both kidneys was irregular and thickened by fibrous connective tissue. Numerous tubules contained proteinaceous casts and the renal medulla was characterized by tubular mineralization.

The small intestinal tear was characterized by necrotic edges, fibrinous connective tissue, and large numbers of mononuclear inflammatory cells.

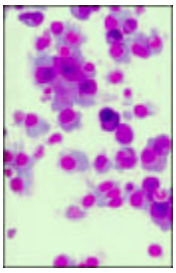
Discussion: Polycystic bile duct disease was the prominent lesion in this case and appeared to be chronic and responsible for generalized icterus and loss of condition in the patient. Polycystic bile duct disease, although not common, can be a primary or incidental finding, especially in cats. In the last 6 months, there have been two cases of this nature at the ADDL. Some intrahepatic bile duct cysts can be congenital and may go unrecognized until they are discovered incidentally at necropsy; however, in this case, it was causing pathological lesions.

The renal lesions were generalized, the likely cause of hematuria, and were suggestive of chronic renal failure. The intestinal tear, a surprising and incidental finding in this case, proved to

be an acute lesion that was evidenced to be ante-mortem in nature. The core of the foreign body resembled cat or human food.

-by Angela Smith, ADDL Graduate Student

Q-fever (*Coxiella burnetti)* Zoonoses



An historical and persistent rickettsial disease

First identified in Queensland, Australia in 1935, infection with *Coxiella burnetti* became known as QUERY (Q) fever due to its dubious etiology and pathogenesis. It was recognized at that time in Australian slaughterhouse workers and, in the United States, it was found to be the source of human infection from a laboratory. The pathogenic rickettsial agent was identified by Harold Cox and MacFarlane Burnet in 1935, which lends explanation to the proper name for Q-fever becoming *Coxiella burnetti*. *Coxiella burnetti* is a gram negative coccobacillus that lives and replicates within host monocytes and macrophages. Since the identification of *C. burnetti* as the causative agent for Q-fever, exposure and disease has, as of the year 2002, been reported in every country except New Zealand.

Q-fever is an important zoonotic disease for several reasons. There are multiple sources of exposure, human infection with *C. burnetti* may result in clinical signs ranging from headache to death, and animals with *C. burnetti* are seemingly normal, with the exception of occasional abortion storms occurring in the herd. Finally, culturing the organism from tissues of suspected animals is difficult, making serology necessary for diagnosis. Considering all of these factors, it is clear that measures for monitoring prevalence, risk assessment and active prevention protocols should be in place for all individuals at risk for exposure to *C. burnetti*.

Sources of exposure to *C. burnetti* include, but are not exclusive, to slaughterhouses, farms, research institutions and veterinary facilities. People have also acquired Q-fever following contact with urine, milk and feces from infected animals. In Idaho, people with no animal contact but who lived within a mile of an abattoir which frequently processed sheep from a research institution, were diagnosed with Q-fever. Awareness of zoonotic risk is especially important for abattoir workers, farmers, veterinarians, research employees and veterinary students due to their increased contact with animals, animal tissues and fluids and animal waste. However, all individuals exposed to *C. burnetti* are susceptible to infection.

A broad spectrum of clinical signs in humans with Q-fever has been reported since the earliest identified case. Clinical signs range from mild self-limiting fever, headache, nausea, eye pain, shaking, chills, anorexia, nausea, arthralgia, myalgia and non-productive cough, to pulmonary disease, meningitis, pericarditis and valvular endocarditis. Meningoencephalitis and myocarditis have occurred with *C. burnetti* infection. Individuals with valvular heart conditions or susceptible immune status are at increased risk for development of more severe clinical signs if infected with *C. burnetti*. Up to ½ of exposed individuals will seroconvert without having exhibited any clinical illness. Others will experience a variable incubation period anywhere from one to three weeks before onset of clinical signs. While mild forms of infection resolve spontaneously, treatment with doxycylcine will shorten the course of disease. If cardiac involvement occurs, doxycycline and hydroxychloroquine are administered long term. Valvular

replacement may be required if severe valvular disease occurs.

Species of interest in the transmission of *C. burnetti* are primarily the bovine, caprine and ovine. However, felids and canids have been associated with human infection. In the host animal, whatever the species, the *C. burnetti* organisms concentrate in fetal/placental fluids and tissues. The concentration may exceed one billion times the ID50 (the dose of enough quantity to infect 50% of exposed people). Animals and people alike acquire the infection by contacting these infected tissues, or contacting contaminated items. *Coxiella burnetti* organisms have two life cycle stages: the large-cell variant (LCV) and the small cell variant (SCV). The LCV is the vegatative form seen in infected monocytes and macrophages. The SCV is seen extracellularly and is presumed to be the infectious form of the organism. When the environment is contaminated with these organisms, via contact with infected tissues and fluids, the SCV variant survives exposure to physical and chemical disruption. Inhalation of the aerosolized organism is the most common route of infection.

There are two primary ways to diagnose Q-fever in humans and animals: isolation of the organism and serologic testing. Organism isolation requires laboratories with established biosafety level 3 conditions and the use of tissue culture, lab animals or embryonated eggs. Serologic testing can be performed using indirect fluorescent antibody (IFA), enzyme immunoassay, and complement fixation. Antigenic detection assays, immunohistochemical staining (IHC), nucleic acid detection assays and polymerase chain reaction (PCR) are also available testing.

Because of the ease of sampling and assaying, serologic testing is the preferred method to diagnose Q-fever in humans and animals. Two antigenic forms of *C. burnetti* are important for serologic diagnosis: avirulent smooth lipopolysaccharide microorganism (S-LPS) phase I antigen and avirulent rough lipopolysaccharide microorganism (R-LPS) phase II antigen. Currently, the Centers for Disease Control are responsible for maintaining epidemiologic surveys of disease in the United States. Case reporting is the limiting factor to valid epidemiologic distribution of a disease. With the serologic assays available and the relative ease of accessing laboratories for performing these assays, monitoring the prevalence of Q-fever would not be an overwhelming task. In addition, the potential life-threatening outcome for some individuals when infected with Q-fever substantiates the importance of monitoring prevalence and establishing risk.

The total number of cases of a specific disease in a given population at a certain time is the prevalence of that disease. Prevalence of Q-fever can be established through serologic monitoring. The number of new cases of a specific disease during a fixed time is the incidence of that disease. Incidence of Q-fever can be established through serologic monitoring. Risk is defined as the chance of an unfavorable event occurring. In this instance, that event would be exposure to or contraction of Q-fever. Risk can be assessed by examining prevalence and incidence.

Prevention is defined as “measures designed to prevent the introduction of a disease in the areas where it does not already exist and improve the resistance of the population and decrease the chances of the infection from spreading when it already exists in the population”. The introduction of Q-fever is prevented from entering the population, the herd, by purchasing animals from serologically negative herds. Improving the resistance in the population, humans, is possible by vaccination. Vaccination is currently reserved for

individuals considered at greatest risk of exposure, such as biosafety-3 level laboratory workers. Isolating or depopulating affected animals and taking personal protective measures when working with suspected or confirmed infected animals or contaminated materials will decrease the chance of spread of Q-fever. Personal protective measures include masking, gowning, gloving, and wearing protective eye gear during high-risk exposure. High-risk exposure can include examining aborted materials, aiding in dystocia, cesarean section or necropsy of pregnant cows, goats, sheep, etc, and working in abattoirs, laboratories housing Q-fever infected animals or tissues. The objective of the personal protective measure is to decrease exposure to infective organisms by providing a physical barrier and minimizing environmental contamination and eventual aerosolization by discarding all contaminated items immediately following use. As mentioned previously, the organism is persistent in the environment. This should be considered when re-populating or isolating a herd.

Q-fever is an historical and persistent zoonotic disease. It is required in the state of Indiana to report cases of Q-fever in animals. For additional information about reportable diseases, visit www.cdc.gov or contact the Indiana State Veterinarians Office at http://www.in.gov/boah/.

-by Sherry Walters, Class of 2004
-edited by Dr. Leon Thacker,
ADDL Director

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ADDL NEWS

Dr. Dan Harrington (30 years), **Karen Crane (Supervisor of the Serology laboratory** (25 years), and **Dr. Ching Ching Wu** (10 years) were recently honored for their years of service to Purdue University and the ADDL.

Serology lab technicians **Brenda Turner** and **Barb Million** completed the Pseudorabies proficiency test with 100% accuracy. Procedures in this proficiency panel included Latex Agglutination, ELISA, Particle Concentration Fluorescence Immuno-assay, Serum Neutralization and G1 deletion.

The bacteriology laboratory has achieved certification for its Johne's testing.

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

May 31, 2004……Memorial Day
July 5, 2004……Independence Day
September 6, 2004……...Labor Day

Generalized Tremors: Identifying a White Shaker Dog

Tremors are involuntary, repetitive, rhythmic, oscillating contractions of antagonistic muscle groups. They are generally characterized by rate, rhythm and movement type and may be localized to one area or involve the entire body (generalized). They are often difficult to characterize because tremors are a relatively common neurologic abnormality in many diseases of the central nervous system (CNS) and peripheral nervous system (PNS).

Accurate characterization and diagnosis of generalized tremors is difficult. Discovering the cause requires a thorough history as well as a complete physical, neurologic and orthopedic exam. Other diagnostics that may be helpful include a CBC and chemistry panel to evaluate body systems and look for metabolic dysfunctions. An EMG and/or muscle and nerve biopsies may be warranted if a primary muscle disorder is suspected. Collection and analysis of cerebrospinal fluid is useful when looking for inflammation or an infectious cause. Tremors have been associated with congenital disease, chemical/plant intoxication, secondary to drug therapies, and bacterial or viral diseases.

Congenital diseases that cause generalized tremors in dogs, such as insufficient myelination of the CNS, often manifest themselves early in life. The tremors involved with these abnormalities are primarily noticed during goal-oriented activity (intention tremors), seem to lessen with rest and resolve with sleep. The pathogenesis of these tremors is unclear, but spontaneous discharge of the unmyelinated axons, and loss of coordinated muscle control may be the cause.

Ingestion of toxins such as organophosphates, hexachlorophene and bromethalin may result in generalized tremors. Suspected pathogenesis centers around altered nerve impulse conduction brought on by intramyelin edema. Mycotoxins have commonly been associated with tremors in dogs. Tremors resulting from drug therapies have also been documented. The pathogenesis is not always known, but discontinuing drug therapy will also stop the tremors.

Bacterial and viral encephalitis are also rule-outs when presented with a tremoring dog. The agents most frequently associated with tremors are canine distemper virus, adenovirus, parvovirus, herpes virus and tick-borne diseases. An extensive evaluation of cerebrospinal fluid is necessary for diagnosis. This includes cytologic evaluation of, as well as laboratory titers for, the suspected agent in the fluid.

Finally, there are those dogs that are referred to as little white shaker dogs. The syndrome was given this name because it was historically recognized in small breed white dogs such as the Maltese, West Highland White Terrier and poodle, although dogs with all coat colors are susceptible. They are generally young adults, initially showing signs at less than two years of age, and are small to medium sized dogs (<15 kg/33lbs). The generalized head and body tremors can range in severity from mild to incapacitating and tend to worsen with exercise, stress or

excitement and lessen or resolve with sleep. Other neurologic signs (deficit in menace, nystagmus) may be present, but are not always noticed. Screening for infectious agents is generally negative. Cerebrospinal fluid analysis often reveals a mild lymphocytic pleocytosis, but may also be normal. Histologic exam of the CNS tissue of affected animals varies. A mild, non-suppurative meningoencephalitis with mild perivascular cuffing, most evident in the cerebellum, may be identified, but normal CNS tissue can also be found. The underlying disease process is still unknown. Some speculate that the tremors are due to an immune reaction targeted against the tyrosine producing cells. Tyrosine is important in the production of melanin as well as the neurotransmitters dopamine and norepinephrine. An imbalance of these neurotransmitters may lead to the clinical signs observed; however, variation in the pathologic changes identified in tissues suggests that inflammation is not the only mechanism involved in the pathogenesis. Diagnosing a white shaker dog is one of exclusion and response to treatment. These dogs respond to an immunosuppressive dose of cortico-steroids (prednisone). Tremors will generally resolve the first week or two after instituting therapy. The dose of steroids can then be tapered to the minimum effective dose, or discontinued completely. If the tremors return, reinstitution of the initial immunosuppressive dose may be necessary. Because of the response to steroids and the recognition of this syndrome in breeds other than those with a white hair coat, this disease is also referred to as steroid responsive tremor syndrome.

-by Kelly Smith, Class of 2004
-edited by Dr. Leon Thacker, ADDL Director

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Herpes B Virus Infection

Herpes B virus is relatively benign in the macaque monkey, its natural host. However, the alpha-herpesvirus can cause rapidly ascending encephalo-myelitis with a fatality rate of approxi-mately 80% if spread to humans. The transmission of the virus most likely occurs through bites, scratches, splashes, or needle-stick injuries. It is important that attempts are made to understand the etiology of the virus as well as ways to expedite diagnoses and prevent infection.

B-virus infection has been most commonly reported in the rhesus and cynomologus macaque. In the macaque host, B virus causes mild symptoms which may include oral or genital lesions. B virus is transmitted from a host when virus is shed from herpetic lesions or the affected mucosal sites. Most cases of human B-virus infection have been associated with apparently healthy macaques that have asymptomatic shedding of the virus.

How often or how long the host sheds is not yet fully understood.

These species of macaques are extensively used in biomedical research and, therefore, frequent contact is made between animal care technical staff and the non human primates. Detection of B virus is imperative to reduce the number of cases of the disease. It is the role of human and animal physicians to suspect infection early and diagnose it rapidly in order to control human infection. Taking samples from the exposed human as well as the source animal is important for virus culture and serologic testing.

Development of diagnostic methods that have the ability to differentiate between Herpes Simplex Virus (HSV) and the B-virus infection are required due to the extreme cross-reactivity of primate alpha-herpesviruses. Antibody testing by ELISA and western blot, virus isolation testing by cell culture, and virus DNA testing by PCR are the methods most commonly used today. Direct culture of the B virus itself has been the cornerstone for diagnosis of infection. This, however, comes with a risk for exposure to the laboratory personnel. Herpes B virus is a biosafety level 4 pathogen and, therefore, its culture requires a special containment facility. PCR methods have allowed direct demonstration of the B-virus infection without risk of exposure to a virus culture handling.

There is a real risk of fatal human infection following exposure to herpes B-virus due to the wide use of macaques in biomedical research. Personal protective equipment and continuing education on proper handling procedures have curbed the incidence of human exposure. Further efforts to better understand the biology of B-virus in its natural host is the next step to identify opportunities to prevent or limit zoonotic B-virus disease.

-by Adam Miller, Ross University
-edited by Dr. Leon Thacker, ADDL Director

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Proliferative Enteropathy in Rabbits

Proliferative enteropathy is a disease of domestic and laboratory animals; affected species include the pig, horse, dog, rat ,ferret, guinea pig and hamster. The causative agent of proliferative enteropathy is *Lawsonia intracellularis*, an obligate intracellular bacterium. The swine industry suffers the most significant impact from this disease. The prevalence of the bacterium and the incidence of associated clinical disease in lagamorphs are currently under investigation.

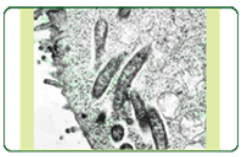
Lawsonia infection can be common in some rabbit colonies. Lesions are often mild and found as incidental findings in some rabbits at necropsy. Disease rarely results in death; however, severe fatal cases have been noted. Additional viral, bacterial and/or protozoal infections may be responsible for more severe clinical disease. Co-infection with enteropathogenic *Escherichia coli* has been documented in rabbits with clinical disease.

Epizootology and transmission

Proliferative enteropathy has a worldwide distribution and affects a wide variety of species. Since *Lawsonia intracellularis* isolates from different species have little genetic variation, intra- and/or interspecies transmission appears likely. Environmental contamination with feces of infected animals appears to be important for transmission of disease, but it is currently unknown how long *Lawsonia intracellularis* can remain infectious outside of the animal. Animals become infected by consuming fecal-contaminated material. Epizootics of the disease are usually confined to younger animals. Infection occurs often during the post-weaning period, when passive maternal immunity declines. After oral infection, the organisms infect intestinal proliferating crypt epithelial cells and multiply within the apical cytoplasm. There is no evidence of infection of tissue other than intestine. Most animals develop subclinical infection, but shed the bacterium within feces and contribute to environmental contamination. Stressors, such as overcrowding, transport, change in diet, and experimental manipulations have been identified as predisposing factors for clinical infection.

Etiology

Proliferative enteropathy is caused by infection with *Lawsonia intracellularis*, which is an obligate, intracellular, curved rod-shaped, argyrophilic bacterium located within the the apical cytoplasm of infected crypt epithelial cells. The means of cellular invasion is still under investigation at this time, but *in vitro* studies indicate that it involves receptor-ligand mechanisms. Bacteria associate with the cell membrane and are taken up by the enterocyte via an entry



vacuole. After lysis of the entry vacuole, bacteria multiply freely within the cytoplasm. Bacterial infection of enterocytes is associated with failure of enterocytes to maturate and increased crypt proliferation. The underlying mechanism is unknown. Animals with subclinical infection do not show clinical signs, but shed the organism within the feces for a limited period of time. Acutely infected animals are lethargic and anorectic, have matted hair coat and watery diarrhea. A common sequela to diarrhea is marked dehydration which may result in cardiovascular collapse. However, enteritis may also cause intestinal intussusception and sudden death.

Pathology: On gross inspection at necropsy, affected animals may be emaciated and/or dehydrated. They have proliferative jejunitis and/or ileitis characterized by thickening and corrugation of the small intestinal mucosa and the presence of semi-fluid mucinous contents within lumens of colon and cecum. Microscopically, intestinal crypts are elongated and often branched and lined by multilayered immature enterocytes. The number of goblet cells is decreased. Lumens of crypts may contain cellular debris. Villi are often shortened and blunted and the mucosa commonly has a mixed-cellular infiltration with histiocytes admixed with fewer macrophages, lymphocytes, plasma cells and heterophils and, occasionally, a few multinucleated giant cells. By use of special stains (Warthin Starry silver stain, PAS stain). Agyrophilic and PAS-positive curved bacteria consistent with *Lawsonia intracellularis* can be demonstrated in apical cytoplasm of enterocytes and within crypt lumens. Intralesional histiocytes may contain intracytoplasmic PAS-positive material consistent with bacterial fragments.

Diagnosis: Diagnosis should be made based on the combination of clinical signs and results of necropsy, histopathology, bacteriology and/or demonstration of intralesional organisms by histochemistry (Warthin Starry silver stain or PAS-stain, see above), electron microscopy, immunohistochemistry and/or polymerase chain reaction (PCR). Bacterial culture and isolation must be performed by use of cultured enterocytes since *Lawsonia intracellularis* does not proliferate in cell-free media. Electron microscopy can be used to detect the curved bacterial rods consistent with *Lawsonia intracellularis* within apical cytoplasm of enterocytes. Immunohistochemistry can be performed on formalin-fixed, paraffin embedded tissue of affected intestine to detect intralesional *Lawsonia intracellularis* organisms. In addition, a PCR is available to confirm presence of the *Lawsonia intracellularis* genome within tissue samples of altered intestine.

Differential Diagnoses: Differential diagnoses for diarrhea in rabbits include Rota and/or Coronavirus, Salmonellosis, antibiotic-associated enteritis, coliform enteritis and intestinal coccidiosis. Coinfections of *Lawsonia intracellularis* and other bacterial, viral or protozoal pathogens are common and may play an important role in the pathogenesis of the disease.

Treatment: Treatment of ill rabbits consists of symptomatic and supportive therapy. Antibiotics are commonly used to eliminate infection with *Lawsonia intracellularis.* Isolation of sick animals is advised. Supportive therapy includes administration of parenteral fluids and supplemental heat provided to those animals that become hypothermic.

-by Lou Turchyn, ECFVG Student
-edited by Dr. Sandra Schoeniger,
ADDL Graduate Student

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Selenium testing

Dr. Steve Hooser, Head of Toxicology

Analysis of serum and liver for selenium requires 3 days of processing and is labor intensive. With our existing equipment and personnel, we are unable to start and run selenium analyses on a daily basis. Starting, immediately, we will save frozen samples for selenium analysis and begin the procedure on Tuesdays with the goal of reporting the results of those analyses on Friday of each week. Samples which arrive after Tuesday will be held until the following Tuesday to begin analysis.

Serum for vitamin E and selenium should be collected in plain red-top tubes. Do not use clot separator tubes as the separator gel interferes with the analysis. The minimum amount of serum for running both tests is 3.5 ml. For accurate vitamin E analysis, it is important that the serum not be hemolyzed. If the serum appears at all pink to red, it is likely that significant degradation of vitamin E has occurred. A "golf ball" size piece of liver is enough for both tests.

Dr. Leon Thacker chaired a meeting of the American Association of Veterinary Laboratory Diagnosticians Accreditation Committee and attended a meeting of the AAVLD Executive Committee in Las Vegas, February, 2004.

Drs. Zheko Kounev and Tom Bryan attended the International Poultry Scientific Forum and International Poultry Trade Show in Atlanta, Georgia, January, 2004.

Alice Hardebeck, Serology Technician, attended an EIA training workshop at the National Veterinary Services Laboratory, Ames, Iowa, January, 2004.

Dr. Steve Hooser attended the Society of Toxicology annual meeting in Baltimore, Maryland, March, 2004.

Dr. Greg Stevenson was an invited speaker at the American Association of Swine Veterinarians Conference in Des Moines, Iowa, March, 2004.

Please join us in congratulating Dr. Steve Hooser, ADDL Assistant Director and Head of the Toxicology laboratory,

on being selected as the recipient of the *2004 Alumni Outstanding Teaching Award* for the Purdue School of Veterinary Medicine. Dr. Hooser has been on faculty at Purdue since 1994.