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Winter 2005

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Good day from ADDL. A few days ago the weather outside was quite appropriate for writing the "winter" edition of the ADDL Diagnostic Forum. Actually, in our part of the state, we had only a fraction of the snow and associated inconvenience and property damage that found its way to areas of Indiana more to the south. Our condolences go out to those of you who had losses due to that weather. As of now, however, we have somewhat overcompensated with 60 degree weather in January, not all that common.

In the fourth week of November of '04, a site visit team of the Accreditation Committee of the American Association of Veterinary Laboratory Diagnosticians came to the ADDL to check the capability, operating procedures, test suitability, quality assurance, personnel, administration and standards of our laboratory for accreditation by the AAVLD. The parting comments of the site visit team were encouraging of their findings and observations but we will not know of the accreditation status until the full committee meets to deliberate on the site visit team report in February. The ADDL offers thanks and gratitude to the users of the Laboratory who gave of their time to come to meet with the site visit team to offer their thoughts and opinions and answer questions from the team during the visit.

We are in process of testing the retropharyngeal lymph nodes by immunohistochemistry of about 1100 deer taken by hunters in Indiana in the '04 season for chronic wasting disease. Up to now, no deer from Indiana have been found to be positive for this disease though it has been found in a deer harvested in northern Illinois, not far from the state line.

In testing for unauthorized drugs administration to animals shown at the Indiana State Fair in '04 some animals of different species were found to be positive. Hearings have been held to determine the penalties of offenders in these instances. The outcome of the hearings are for other regulatory officials; our duty ends with running the tests.

I hope this finds each of you enjoying the winter and looking forward to a good year in'05. Our thoughts, prayers and assistance go out to those who have lost so much to the destructive tsunami in the Indiana Ocean.

FINAL DIAGNOSIS:

Zinc Intoxication in a dog

History: A female Pekingese dog, six months of age, was found dead by its owner. The dog had been lethargic for approximately one week, but was still eating. There was no history of diarrhea or vomiting. The puppy was current on all of its vaccines. A physical exam and fecal exam, performed five weeks prior to death, were both normal.

Gross necropsy lesions: The proximal portion of the duodenum at the junction with the pyloric sphincter contained five and ½ coins. The coins consisted of three pennies, one each from the years 1986, 1992, and 1978, and one half of a penny with a roughened surface, teeth marks, and an irregularly serrated edge with a year that was not visible. The other two coins were dimes. Significant gastrointestinal lesions were not found at the location of the coins. The serosal and mucosal surfaces of the bladder were discolored dark purple to black. The bladder contained approximately 20 ml of a thin, dark red to purple fluid. The liver was diffusely discolored dark red. The visceral surface of the kidneys was diffusely and bilaterally discolored dark red. No other lesions were found in the musculoskeletal, respiratory, cardiovascular, digestive, genitourinary, lymphatic, endocrine, or nervous systems or in the organs of special senses.

Toxicology testing: The penny with the serrated edge was cut longitudinally. The core of the penny was a silver metallic substance indicative of zinc. An ICP-MS zinc analysis of the liver revealed a zinc concentration of 236.087 ppm (normal range for liver zinc at the laboratory used by the ADDL is 30-90 ppm).

Microscopic lesions: The peripheral acini of the pancreas were necrotic and occasionally infiltrated with neutrophils, macrophages, and lymphocytes with individualization and destruction of acinar cells. Interlobular septa within the pancreas contained occasional neutrophils, macrophages, and lymphocytes. Hepatocytes within the liver were vacuolated and sinusoids were distended with edema fluid. There was intrahepatic cholestasis. The lungs were edematous. Lymphoid follicles within the small and large intestines were diffusely depleted of cells. The red pulp of the spleen contained multiple foci of extramedullary hematopoiesis.

Diagnosis: The gross findings (pigmented urine, icterus), microscopic findings (pancreatic necrosis), and liver zinc level support a diagnosis of canine zinc toxicity.

Discussion: Following the ingestion of zinc containing objects, zinc forms soluble salts in gastric acid and is absorbed mainly in the proximal small intestine. Approximately 67% of the zinc is bound to plasma proteins and carried to the liver. Protein-bound zinc is transported to the liver where approximately 30-40% of the zinc is extracted and returned to the bloodstream where it accumulates in high levels in the liver, kidney, pancreas, and bone. The remainder of the zinc is absorbed by hepatocytes and storied in the liver. Zinc is normally excreted in the feces via pancreatic secretions, intestinal mucosal secretions, and bile.

Zinc objects are directly irritating to the gastrointestinal tract. However, clinical signs at presentation are usually associated with severe intravascular hemolysis and organ damage (liver, kidney and pancreas). The pathogenic mechanism in dogs is not well-understood but oxidative damage to red blood cells plays a major role. Affected animals may appear asymptomatic prior to the hemolytic crisis. Clinical signs of zinc toxicity can be vague and non-specific or these animals may present with signs that are suggestive of hemolytic anemia and multiple organ damage.

Antemortem diagnosis of zinc intoxication is confirmed with elevated serum/plasma zinc levels. Either whole blood or serum can be submitted for zinc analysis. Whole blood can be collected into EDTA or

heparinized collection tubes. It should be noted that the rubber in syringes and red-top tubes can contribute zinc to the sample and falsely elevate the serum zinc level. Trace element-free tubes (Vacutainer, Venoject, royal blue stopper tubes) are preferred for sample collection. If these are not available, then the serum/plasma should be separated as soon as possible and placed in plastic-stoppered containers. Samples need to be refrigerated. The laboratory performing the zinc testing should be contacted for information regarding sample collection, storage, and shipping. Normal serum zinc level varies by laboratory but is usually less than 2mg/ml or approximately 0.7-2.0 ppm.

Typical gross necropsy lesions include icterus, splenomegaly, nodular appearance to the pancreas, and large, diffusely discolored red/brown kidneys. Postmortem diagnosis of zinc toxicity is confirmed by elevated tissue zinc levels. Sections of pancreas, kidney, and liver can be tested for zinc. Tissue samples should be stored frozen until analyzed. The testing laboratory should be contacted for information regarding tissue collection and submission. Normal liver zinc level varies by laboratory but is usually within the range of 30-90 ppm.

The main source of zinc toxication in dogs is pennies that were minted after 1982. These coins are composed mainly of zinc wafers (98% zinc) with a thin copper coating. Pennies minted before 1982 are composed mainly of copper. Other sources of zinc include galvanized coating on iron and steel (dog crates, carriers, and fencing), zinc nuts used in collapsible transport cages, automotive parts, batteries, fungicides, zinc-based paints (can be 50-55% zinc), shampoos, and zinc-containing topical medications (zinc-oxide lotions).

-by Dr. Chris Hoppe, ADDL Graduate Student

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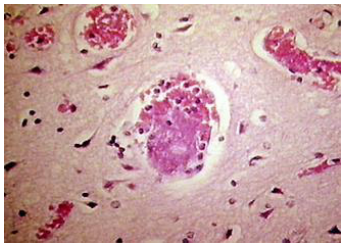
Disseminated Intravascular Coagulation (DIC)



DIC is a thrombohemorrhagic disorder characterized by primary thrombotic and secondary hemorrhagic diathesis causing multi-organ failure.

DIC is not a primary disease. It is a complication of a variety of diseases which cause pathologic activation of the extrinsic and/or intrinsic coagulation pathways. Thus, major mechanisms for triggering DIC include release of tissue factor or thrombo-plastic substances into the circulation (activation of extrinsic pathway of coagulation), injury to endothelial cells exposing subendothelial collagen (activation of intrinsic and extrinsic pathways of coagulation), systemic hypercoagulopathy (renal disease with loss of antithrombin III) and hepatic disease (decreased synthesis of coagulation factors and anticoagulants). Release of tissue factor and/or thromboplastic substances in the circulation is often triggered by sepsis, severe tissue destruction (trauma, burns), pancreatitis,

neoplasms (mucinous carcinomas, leukemia, lymphoma, hemangiosarcoma) and/or obstetric complications. Endothelial injury can initiate DIC by release of tissue factor and promoting platelet aggregation to exposed collagen. Causes of endothelial injury include vasculitis due to deposition of antigen-antibody complexes (lupus erythematosus, feline infectious peritonitis), temperature extremes (heat stroke, burns) and/or direct damage by trauma, toxins, rickettsiae, bacteria, or their toxins and/or viruses.

Endotoxemia (gram negative sepsis, intestinal infarction) is one of the most important and often fatal causes of DIC. In macrophages and monocytes, bacterial endotoxins stimulate increased synthesis and release of tissue factor and the proinflammatory mediators interleukin 1 (IL1) and TNFα, which in turn induce procoagulatory functions of endothelial cells such as increased expression of tissue factor and decreased expression of thrombomodulin. In addition, TNFα also upregulates expression of adhesion molecules on endothelial cells and, thus, promotes adhesion of leukocytes which may damage endothelial cells by release of proteolytic enzymes and oxygen-derived free radicals. Bacterial endotoxins can also directly activate factor XII.

Mucus released from some carcinomas acts as thromboplastic substance and directly activates factor X and, thus, is the common pathway of coagulation. In acute promyelocytic leukemia, granules of neoplastic leukocytes release thrombo-plastic substances. Tissue destruction (trauma, burns) causes release of thromboplastin in circulation. Release of proteolytic enzymes in the circulation and thus marked tissue destruction, is caused by pancreatitis. Thromboplastins derived from placenta, dead fetus and/or amniotic fluid may enter the maternal circulation in obstetric complications.

Activation of intravascular coagulation is followed by formation of thrombi within the microcirculation of multiple organs such as brain, renal glomeruli and/or pulmonary capillaries leading to microinfarction and tissue hypoxia. DIC is also called consumptive coagulopathy, since excessive intravascular coagulation leads to consumption of platelets and nonenzymatic coagulation factors. The sequelae to excessive intravascular coagulation are systemic hemorrhagic diathesis due to thrombocytopenia, reduced quantities of coagulation factors and initiation of fibrinolysis. Initiation of fibrinolysis is caused by a plasminogen activator, which cleaves plasminogen bound to fibrin to plasmin. Plasmin degradates fibrin to FDPs, which can bind to fibrinogen receptors on platelets and prevent further platelet aggregation. In addition, thrombin binding to thrombomodulin results in activation of protein C, which cleaves and inactivates plasminogen activator inhibitor and coagulation factors V and VIII.

Finally, DIC is characterized by concurrent thrombocytopenia and depletion of coagulation factors and antiboagulants such as antithrombin III, protein C and protein S. The formation of fibrin within the microcirculation causes microangiopathic hemolytic anemia with schistozyte and keratocyte formation due to damaged membranes of erythrocytes.

Clinical features: DIC may present as acute, subacute or chronic disease. Whereas acute and subacute cases have clinical symptoms of multiorgan involvement such as respiratory signs (dyspnea, cyanosis), neurological signs (convulsions, coma), renal symptoms (oliguria, acute renal failure), chronic DIC usually causes only minor clinical signs or is subclinical. DIC is based on evaluation of blood parameters. Hemorrhagic diathesis predominates commonly in acute DIC and thrombotic complications are the main feature of subacute or chronic DIC. Acute DIC may develop primarily (e.g. caused by sepsis), or secondary to decompensation of chronic DIC. Acute DIC is often fatal with multiorgan failure and circulatory collapse.
Acute DIC is more commonly observed in dogs and is rare in cats.

Diagnosis: Diagnostics for DIC include concurrent prolongation of coagulation profiles (APTT, PT) and thrombocytopenia. In addition, there may be an increase in fibrin degradation products (FDPs), increase or decrease of fibrinogen, decreased antithrombin III, regenerative hemolytic anemia with schistocyte and keratocyte formation, neutrophilia with left shift or neutropenia and hypoalbuminemia. Increased FDPs may be observed in DIC, but is also seen in inflammatory diseases without concurrent DIC. Most commonly, fibrinogen is decreased in DIC; however, increased fibrinogen levels do not rule out DIC since fibrinogen is an acute phase protein synthesized in the liver and is often elevated in inflammatory conditions. Regenerative hemolytic anemia results from fibrin deposition in microcirculation and/or internal bleeding. Neutrophilia with left shift is a sequela to inflammation and/or marked tissue necrosis. Overwhelming inflammation may cause neutropenia. Inflammation may be the cause of DIC or develop secondarily in the course of DIC due to organ damage and immuno-suppression. Hypoalbuminemia may be a sequela to inflammation and/or excessive external bleeding.

-by Young Choi, ECFVG Student
-edited by Dr. Sandra Schoeniger, ADDL Graduate Student

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Purdue ADDL and Heeke ADDL will be closed on the following University holidays in 2005.

Martin Luther King Day…….	Jan 17
Memorial Day…….	May 30
Independence Day……	July 4
Labor Day……..	Sep 5
Thanksgiving…….	Nov 24,25
Christmas……..	Dec 23,26
New Year……..	Dec 30

ADDL NEWS

Our congratulations to Drs. Theresa Boulineau, Ingeborg Langohr, Alok Sharma, and Sandra Schoeniger, ADDL graduate students who recently became diplomates of the American College of Veterinary Pathologists

Murine Respiratory Mycoplasmosis Infection in Rats

Both wild and laboratory rats and, to a lesser degree, mice are the natural hosts of murine respiratory mycoplasmosis (MRM). Hamsters, guinea pigs, and rabbits may carry the causative bacteria, *Mycoplasma pulmonis*, but do not develop lesions *Mycoplasma pulmonis* is transmitted horizontally by direct contact by aerosol and vertically by *in utero* transmission. Venereal transmission may be possible. Once inside the host, the bacteria damages host cells by causing dysfunction of the cilia of respiratory and genital tract epithelial cells. In the respiratory tract, *M. pulmonis* preferentially colonizes the nasal passages and middle ears. The bacteria competes for host cell nutrients and metabolites and may also produce toxic peroxides and nonspecific mitogens Clinical signs are most commonly observed in older animals and MRM may be asymptomatic in young animals. Signs include rales and dyspnea, snuffling and chattering, ocular and nasal discharge, rubbing of eyes, and head tilt. In severe cases, weight loss and reduced fertility may occur. The severity of disease is dependent upon the interaction of host, pathogen and environmental factors. The hosts' age, strain, immune status, and presence of concurrent infections can exacerbate the disease. Additionally, dietary deficiencies of vitamins A and E may add to the severity of disease. More than 40 strains of *M. pulmonis* exist, which vary in virulence. Temperature, humidity, and

intracage ammonia levels are important environmental factors,.

Diagnosis of MRM is dependent on cultural isolation of *M. pulmonis*. The lower sensitivity and specificity of commercially available ELISA test kits make them less desirable. Gross and histopathologic lesions are commonly present but are not diagnostic. Gross lesions of the upper airways include suppurative rhinitis, otitis media, laryngitis, and tracheitis. In the lung, suppurative bronchopneumonia, atelactasis, bronchiectasis and abscesses can occur. When widespread, bronchiectasis and abscesses lead to the "cobblestone" lung appearance commonly seen in endstage disease. Histopathologic lesions include suppurative exudates in airways. Microscopic lesions have a neutrophilic response, accumulation of plasma cells and lymphocytes, and include epithelial hyperplasia and metaplasia.

Murine respiratory mycoplasmosis must be differentiated from other bacterial pneumonias such as infection with *Coryne-bacterium kutscheri, Streptococcus*, cilia-associated respiratory (CAR) *Bacillus* infection, and mycotic pneumonia. Concurrent infections with Sendai virus, pneumonia virus of mice, and other viruses are common.

M. pulmonis infection is a serious complication of research with rats due to its effects on the immune, respiratory, and reproductive systems. It is also a primary causes of early mortality in affected colonies. However, the use of SPF rats has limited its prevalence. Treatment with tetracycline or tylosin may suppress clinical signs in pet rats, but caesarian derivation and barrier maintenance, along with rigorous testing, is necessary in research facilities.

- by Sarah Kanagy, Class of 2005
- edited by Dr. Leon Thacker, ADDL Director

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Enteric Salmonellosis in Horses

Introduction: *Salmonellae* are facultative anaerobic, non-spore forming, gram-negative rods of the family *Entero-bacteriaceae*. There are more than 2200 serovars of *Salmonellae* and all are considered possible pathogens for adult horses. The most commonly isolated serovars are *Salmonella typhimurium, S. agona, S. anatum, S. krefeld, S. newport,* and *S. saint-paul.* There are no host-adaped serovars for horses.

Epidemiology: The transmission of *Salmonella* occurs most often by the fecal-oral route, although infection may also take place through the mucous membranes of the eyes and the nose via aerosol droplets. The most common sources of infection are contaminated feed and water and carrier birds, rodents, horses and other farm animal species that excrete the bacteria. Reported prevalence of infection with *Salmonella* in horses has been variable, ranging from less than 1% to 70%. Variability in prevalence can be explained by differences in methods of detection and populations studied. For

example, prevalence will be higher among horses with diarrhea than among apparently healthy horses, and the prevalence will be lower when microbiological culture of feces is used to detect infected horses than when more sensitive methods, such as polymerase chain reaction (PCR) test or culture of intestinal or paraintestinal organs, are used. Risk factors that might enhance the fecal excretion of *Salmonella* organisms and occasionally precipitate overt clinical disease in carrier animals include transportation, crowding, abrupt change in diet, intensive physical activity, antimicrobial treatment, surgery and gastrointestinal tract disorders. The three most important factors that influence whether a horse becomes ill after exposure to *Salmonella* are the infective dose of the bacteria, the inherent virulence of the bacteria, and the inherent susceptibility of the host. The infective dose is determined by the amount of bacteria shed in the feces and the environmental conditions that favor (or hinder) the proliferation of the bacteria. *Salmonellae* possess an array of virulence factors that confer attributes of mucosal adhesion and invasion; secretion of electrolytes and water into the intestinal lumen (enterotoxin-mediated effect), activation of focal immune responses including the recruitment of inflammatory cells and release of their mediators, local cytotoxic effects; and systemic responses (attributable to an endotoxin). The host susceptibility to *Salmonella* infection is influenced by the composition of the normal intestinal microflora and the competence of local and systemic immune surveillance. Factors that can negatively affect host susceptibility include antimicrobial administration, crowding, or intensive physical activity.

Salmonellae can persist in the environment for protracted periods and have been recovered from contaminated soil after more than 300 days and from water after 9 months. The organisms are very resistant and very adaptable to environmental conditions, surviving for protracted periods in feed. *Salmonellae* can be killed by desiccation and exposure to sunlight, but can survive in dried fecal matter for as long as 30 months. Freezing will not kill the bacteria, particularly if they are in food or other organic matter. For instance, *Salmonella* has been isolated from infected ice cream after more than two years.

Fecal shedding of the bacteria: Assuming that the *Salmonella* cultured from the feces of a horse with diarrhea was the actual cause of the disease, fecal shedding of the bacteria may persist for days to weeks in the animal. Some horses remain consistently positive on fecal culture while they shed the bacteria, but a negative culture does not rule out intermittent or lower level of fecal shedding of *Salmonella.* In the latter situations, a PCR test for *Salmonella* may detect persistent fecal shedding of the organism because this test is more sensitive than is the bacteriologic culture. In most cases, the amount of bacteria shed in the manure of convalescing horses will be relatively small and will not pose a serious threat to other animals. However, if the serovar of *Salmonella* is especially virulent or is able to survive and proliferate in the environment, persistent fecal shedding of the bacteria could pose a risk to other animals or humans.

The surrounding environment is at greatest risk for *Salmonella* contamination from a diarrheic horse because bacteria will spread over a relatively large area and will be able to “hide” in cracks or stalls, on the surface of water buckets or automatic watering devices, and in bedding materials that may be spread by wind or careless foot traffic. If the horse’s manure is formed, however, its removal will facilitate the elimination of pathogenic bacteria from the environment. In such cases, with judicious isolation measures, exposure to other animals and humans to *Salmonella* infection is reduced.

Clinical findings: Enteric salmonellosis is characterized by an acute colitis that results in profuse diarrhea and, occasionally, abdominal pain. Horses with enteric salmonellosis often have signs compatible with endotoxemia, and suffer from cardio-vascular shock and coagulopathies. Horses

are usually febrile, tachycardic, moderately to severely obtunded, and dehydrated. Other clinical signs include fever and leukopenia, colic and proximal enteritis with gastric reflux, dark red or purple mucous membrane, abdominal pain, and abdominal distention. Diarrhea is usually profuse and watery, the feces being often malodorous and variable in color from green to black. Based on clinical and experimental data, it has been suggested that infection with *Salmonella* produces one of the following eight clinical syndromes in adult horses: peracute diarrhea, acute diarrhea, chronic diarrhea, mild colic without diarrhea, moderate colic with subsequent diarrhea, severe colic with subsequent diarrhea, colic with persistent gastric reflux, and asymptomatic infection. Categorizing the disease into one of these syndromes is not vital, but it is important to recognize the salmonellosis in horses can develop without diarrhea. Complications in animals with enteric salmonellosis include laminitis, bacteremia and septicemia, renal failure, thrombophlebitis, disseminated intra-vascular coagulation, hepatitis, and fungal pneumonia secondary to compromise of the intestinal barrier and the immune system.

Diagnosis: Bacteriologic culture of the bacteria is the only way to make a definitive etiological diagnosis of salmonellosis and of determining the serovar. Multiple fecal cultures for *Salmonella* should be performed on all horses with diarrhea. It is recommended that at least 3-5 fecal samples be submitted for culture to enhance the chances of isolating *Salmonella.* Formed fecal samples are more likely to result in a positive culture from infected horses. A 5-10 gram fecal sample should be submitted for culture. Recently, detection of *Salmonella* using PCR (a highly sensitive and specific test for the detection of *Salmonella* in fecal samples from horses) has been described as being more rapid and more sensitive than microbiologic culture. Microbiological culture of a punch biopsy of the rectal mucosa or, if the animal does not survive, of the wall of the cecum, large colon and ileum, mesenteric lymph nodes, and spleen may increase the numbers of isolations of *Salmonella* when compared to the feces.

Differential diagnoses: In adult animals suffering from acute clinical salmonellosis with diarrhea, colitis X, *Clostridium perfringens* type A and Potomac horse fever (caused by *Ehrlichia risticii*) should be considered as differential diagnoses. Clinical signs and lesions of colitis X are very similar to those of enteric salmonellosis, and the distinction between the two conditions is dependent on culturing the organism from the content or the wall of the intestinal tract. A history of recent transportation is also often helpful in suggesting the diagnosis of salmonellosis in adult horses. Chronic diarrhea due to salmonellosis in horses may resemble parasitism, granulomatous enteritis, or alimentary lymphosarcoma.

Treatment: In most cases of enteric salmonellosis, aggressive treatment facilitates resolution of the severe diarrhea and associated metabolic disorders within 7-10 days of the onset of illness. Intravenous administration of polyionic fluids is required to replace fluid and electrolyte losses and to augment preload in horses with poor venous return. Plasma may help to alleviate the hypoproteinemia, which develops as a consequence of the enteropathy. Parenteral nutritional support is indicated to provide adequate calories and amino acids during the most debilitating periods of the disease. The use of antimicrobial drugs for adult horses with salmonellosis remains controversial. The use of antimicrobials such as chloramphenicol, trimethoprim-sulfona-mide, gentamicin, and cephalosporin has not appeared to accelerate resolution of signs of colitis. Ceftiofur can be used successfully (2-4 mg/kg IV q12h). Blood samples should be monitored for electrolyte and acid-base status, and the fluid therapy plan should be adjusted according to any changes detected. Horses that have diarrhea for more than 10 days are unlikely to survive since they often have extensive

loss of colonic mucosa and chronic severe inflammation within the wall of the colon.

Control and Prevention: Management practices should lessen exposure of horses to possible sources of infection. Measures do not have to be laborious and expensive. Removing potentially contaminated fecal material is the most important measure. Other important practices include thorough cleaning of areas where fecal contamination is likely as well as the prevention of mechanical distribution of contaminated material. Cleaning must include the removal of organic debris which can be accomplished with several products specifically designed for that task. Areas that require particular attention are stalls (including water buckets or automatic watering systems), drains, cracks in floors and walls, stall implements, nasogastric tubes and stomach pumps. Extensive use of disinfectants may not be necessary if cleaning measures are adequate. People handling sick horses should wash their hands thoroughly for at least 30 seconds with a disinfectant soap and take precautions to avoid wearing contaminated clothing from stall to stall. Immuno-suppressed people should not provide care to horses with salmonellosis. Personnel entering the stall should wear disposable plastic boots. Footbaths that contain disinfectants are probably not effective since they quickly accumulate organic material that negates the disinfecting potential of the footbath. A *Salmonella* vaccine is available, but its effectiveness has not yet been demonstrated in clinical cases. Because immunity to *Salmonella* is complex, effective and practical immunotherapy may be difficult to attain. Good management practices to minimize stressful situation that may precipitate the disease, such as changes in diet and overcrowding, may be the most effective methods of prevention.

Isolation of affected animals: An affected horse should be isolated from other animals for 10-14 days after returning from the veterinary clinic or hospital. This typically includes stall confinement which is often indicated for a horse convalescing from colitis. When turnout is appropriate, the horse should be confined to an isolated paddock. Some people will elect to continue to isolate a horse as long as it remains positive for *Salmonella* on fecal culture. An animal which has had enteric salmonellosis should not be returned to contact with other horses until five consecutive fecal samples have proved negative for the organism. Among horses that have recovered from salmonellosis, approximately 2/3 will have ceased shedding after 1 month and approximately 90% will have ceased shedding after 4 months.

- by Briardo Reich, EVFVG student
- edited by Dr. Ingeborg Langohr

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On the Road

Drs. Leon Thacker, Greg Stevenson, Duane Murphy, Bob Everson, Steve Hooser, Ching Ching Wu, Jose Ramos-Vara, Peg Miller and **Steve Vollmer** and **Linda Hendrickson** attended the annual American Association of Veterinary Laboratory Diagnosticians annual meeting in Greensboro, NC, October, 2004

Dr. Jose Ramos-Vara and Virology Lab Supervisor **Mary Woodruff** attended an Immunohistochemistry workshop in Ames, Iowa, December, 2004.