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Spring 2003

FROM THE DIRECTOR
H. Leon Thacker, DVM, PhD

We hope this newsletter finds you enjoying spring weather and all the things that come with it. I am afraid that spring this year will bring with it mosquito-associated diseases including West Nile Virus infection of birds, horses, people and perhaps other animals. With the large number of horses that have been vaccinated in the state for WNV, I suspect and am hopeful that the number of equine cases will be less than we saw in 2002 but, as recommended and endorsed by veterinarians, epidemiologists, the Board of Animal Health and others, vaccination of horses for WNV is strongly suggested.

With the presence of Exotic Newcastle Disease in the southwest U.S. and the mobility of people and birds at this time, it is imperative that we be on the alert for the occurrence of this devastating disease of essentially all avian species in our state. As veterinarians you are asked to "keep your ear to the ground" for the occurrence of short term loss of large numbers of birds among a group or loss of birds with little or no clinical signs. Some birds may have clinical signs and marked post mortem lesions; however, the disease is so devastating and fulminating that birds may die rapidly without developing signs or lesions. If END enters our state, the damage from such introduction will be proportional to the time it takes to diagnose it, locate it, quarantine the area and limit its spread. If the disease is here for a long time before it is recognized, losses and cost of its eradication will be markedly increased

We are here to assist you with your diagnostic needs; we hope you will not hesitate to tell us if we should be doing something we aren't or should not be doing something we are.

PURDUE
UNIVERSITY

ANIMAL DISEASE DIAGNOSTIC LABORATORY

April 4, 2003

Dear ADDL Client,

The past several months have seen many changes in our nation, our state, and our laboratory. Among the paramount changes are the feelings and opinions of security in our nation, severe economic shortfall in our state, and loss of some of the key personnel in our laboratory, primarily to higher paying positions in industry. Over the past year, we have lost two senior diagnostic pathologists to industry and one diagnostic virologist to retirement. Funding for refilling two of these positions was lost to reduction in state funding to the laboratory.

The purpose of this mailing is to make you aware of an upcoming change in the operation of the ADDL. For as long as I can remember, the ADDL has been open for receiving accessions and for performing gross postmortem examinations of animals 8:00 to 12:00 noon on Saturday mornings. Due to the present reduction in our pathologist faculty, **the ADDL business hours will continue 8:00 AM to 5:00 PM Monday through Friday. However, effective May 1, 2003, the laboratory will not be open for business Saturday mornings. Emergency consultation or services will be available on as-needed basis through the same after-hours phone or pager access that is now in use.**

I am hopeful that this change from 'Open Saturday mornings' to 'Available for emergencies only' will be of very limited consequence as it has been the practice of most referring individuals heretofore that only necessary accessions were sent in on Saturday morning nonetheless. We were and are appreciative of this.

The ADDL phone # 765-494-7440 will continue to access after hours answering service. We at the ADDL hope to continue to offer you the best diagnostic service of which we are capable. Your indulgence of this change of operations is appreciated.

Yours truly,

H. Leon Thacker, DVM
Director

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FINAL DIAGNOSIS

Moldy Corn Poisoning (Equine Leucoencephalomalacia, Fumonisin Toxicity) in Horses

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

History: A 9-year-old quarterhorse gelding, from a herd of 6, became increasingly ataxic and weak over a period of 2 days. On the third day, it was found down, recumbent and "colicky". The referring veterinarian was called to the farm. After obtaining a thorough history and completing a physical examination, it was discovered that the horses were being fed cracked and moldy corn that was being scooped up from around a corn bin. The veterinarian treated the horse with banamine. The horse was euthanized after failing to respond to treatment.

Gross Findings: No disease-related gross lesions were present. The cut surface of the brain was normal in appearance.

Histologic Findings: There was multifocal liquefactive necrosis of the white matter with infiltration of large numbers of macrophages in the areas of necrosis.

Toxicology: HPLC analysis of the sample of moldy cracked corn revealed fumonisin B1 at 57.1 ppm and fumonisin B2 at 18.9 ppm for a total of (B1+B2) of 76 ppm.

Discussion: Equine leucoencephalomalacia is a generally fatal, rapidly progressing neurologic disease of horses (and other equids) caused by ingestion of fumonisin. It is characterized by liquefactive necrosis of the cerebral white matter. Liver lesions can also occur. Fumonisins are environmental toxins produced by the molds *Fusarium moniliforme (F. verticilloides), F. proliferatum,* and other *Fusarium* species that grow on agricultural commodities in the field or during storage. These mycotoxins have been found as common contaminants worldwide, mainly in corn. More than ten types of fumonisins have been isolated and characterized. Of these, fumonisin B_1 (FB_2) and fumonisin B_3 (FB_3) are the major fumonisins produced in nature. The most prevalent of these mycotoxins in contaminated corn is FB_1 which is believed to be the most toxic.

The extent of contamination of raw corn with fumonisins varies with geographic location, agronomic and storage practices, and the vulnerability of the plants to fungal invasion during all phases of growth, storage, and processing. The levels of fumonisins in raw corn are also influenced by environmental factors such as temperature, humidity, and rainfall during pre-harvest and harvest periods. High levels of fumonisins are associated with hot and dry weather, followed by periods of high humidity. High levels of fumonisins may also occur in raw corn that has been damaged by insects. Further, fumonisin levels in raw corn can increase under improper storage conditions. For example, optimal growth of fumonisin-producing molds that lead to increased levels of fumonisins in raw corn can occur when the moisture content of harvested raw corn during storage is 18-23%.

Horses, along with rabbits, are the species most sensitive to the toxic effects of fumonisin. Corn and corn by-products used in rations of horses and rabbits should contain less than 5 ppm (FB_1 + FB_2 + FB_3) and comprise no more than 20% of the dry weight of the total ration. The total ration should contain less than 1 ppm (FB_1+FB_2 + FB_3). Horses should never be fed corn screenings or moldy, damaged corn. Catfish and swine are together as intermediate in sensitivity. Ruminants, mink and poultry are more resistant than horses, rabbits, catfish and swine to fumonisin.

Onset of clinical signs can occur from 1-21 weeks after beginning eating feeds containing fumonisin, but generally occur within 2-9 weeks. Time of onset depends on the concentration of fumonisins in the feed. Clinical signs of fumonisin poisoning in horses are usually related to liquefactive necrosis of the white matter of the brain and include progressive ataxia, depression, anorexia, delirium, aimless wandering,

recumbency, coma and death. Death can occur from 12 hours – 1 week after onset of clinical signs. If leucoencephalomalacia is suspected, gunshot should not be used for euthanasia as this may render tissues unsuitable for postmortem examination. At necropsy, lesions in the cerebral cortex can range from none to multifocal areas of hemorrhage and necrosis, to the presence of large cavitations of liquefactive necrosis. Histologically, there are multifocal areas of liquefactive necrosis within the cerebral cortex with infiltration of macrophages. Differential diagnoses should include rabies, equine encephalomyelitis, equine herpesvirus, botulism, head trauma, hepatoencephalopathy, and bacterial meningoencephalitis.

Summary: Several cases of equine leucoencephalomalacia have been presented to the ADDL since the beginning of the year. Horse owners should be aware of the dangers of feeding horses mold-dmaaged corn, waste corn and corn screenings. If fumonisin contamination of corn and/or feed is suspected, please send a minimum of ¼ pound (100 g) of a representative sample to the Toxicology Section, ADDL. Horses suspected of having died of leucoencephalomalacia should undergo a complete necropsy to establish a definitive diagnosis taking appropriate precautions remembering that rabies would be among the differential diagnoses.

-by Dr. Steve Hooser, ADDL Toxicologist
Dr. Duane Murphy, ADDL Pathologist

Reference: Carson TL and Poppenga RH: 2002. Equine leucoencephalomalacia. The 5-Minute Veterinary Consult (Brown CM and Bertone J, eds.) 624-625.

American Canine Hepatozoonosis

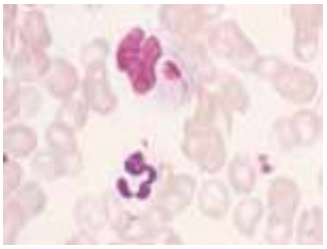

Photo from article by Lucia Helena O'Dwyer: 2001. Some aspects of hepato-zoonosis. Clinica Veterinaria. 31: 34-40.

Hepatozoonosis is an emerging disease in the United States caused by the protozoan *Hepatozoon. Hepatozoon* species have been found to infect a wide range of carnivorous hosts including domestic dogs, jackals, coyotes, foxes, hyenas, domestic cats, bobcats, lions, leopards, and cheetahs. The causative agent of hepatozoonosis in the United States is a newly recognized species, *Hepatozoon americanum*, whereas in other parts of the world, *Hepatozoon canis* is the primary agent. The features of American hepatozoonosis greatly contrast non-American infections. The clinical presentation of dogs with hepatozoonosis in the US is much more aggressive than that of infected dogs from other parts of the world, indicating that *Hepatozoon americanum* is more pathogenic. Non-American infections are often subclinical and seem to be limited to the immunosuppressed. Most cases of domestic canine hepatozoonosis in the United States are diagnosed in the region from Texas to Georgia. *H. americanum* infection has also been identified in coyotes in Oklahoma.

The exact life cycle and transmission for *H. americanum* has not been completely elucidated. Most portrayals are based on extrapolations from that which is known of *H. canis.* The tick vector is the definitive host. The vector for *H. canis* appears to be primarily the brown dog tick, *Rhipicephalus sanguineus*. Evidence suggests the Gulf Coast tick, *Amblyomma maculatum*, as the vector involved in transmission of *H. americanum* in the United States. Although some reports have demonstrated the ability of dogs to become infected with *H. americanum* after ingesting an infected *R.*

sanguineus, transmission from an infected dog to the tick has not been documented.

The tick becomes infected by ingesting a blood meal, containing monocytes or neutrophils laden with isogamonts from an infected vertebrate host. Syngamy occurs in the gut of the tick, producing a zygote which penetrates the tick gut wall. Sporogony occurs in the haemocoel where an oocyst is formed containing multiple sporozoites. The intermediate vertebrate host must ingest the tick to become infected since the sporozoites apparently do not migrate to salivary tissue in the tick. Sporozoites penetrate the intestinal wall of the intermediate host and undergo schizogony, forming schizonts, and then cysts within mononuclear phagocyte or endothelial cells of the spleen, bone marrow, lungs, liver, lymph node, or muscle. When the schizonts rupture, an inflammatory reaction is initiated. In completion of the life cycle, gamonts are produced that infect circulating leukocytes. A paratenic prey host in which only cysts are formed has not been documented with *H. americanum*, and the feeding of encysted meat to carnivorous hosts has not produced infection; however, such paratenic hosts have been identified with other *Hepatozoon* species.

Immunosuppression seems to be an important determinant of susceptibility for infection with *Hepatozoon* species. Concurrent infection, debilitating disease, immunosuppressant drugs, and young age seem to influence clinical manifestation. It is unclear if clinical hepatozoonosis occurs only in the immunosuppressed, or if infection elicits immunosuppression in the host, predisposing to concurrent infection.

American hepatozoonosis typically presents as severe clinical disease. A majority of the clinical syndrome is composed of clinical signs related to chronic inflammatory disease. Many patients present with recurrent fever, lethargy, depression, and weight loss. Muscular disease is also apparent on presentation. Schizogony of the hepatozoon causes a marked pyogranulomatous polymyositis which results in stiffness, lameness, hyperesthesia, and muscle atrophy. Clinical signs fail to resolve with antibiotics. Bloody diarrhea, related to intestinal penetration by the sporozoites, may be documented soon after exposure. A generalized lymphadenomegaly may also be present.

Clinical pathological changes are often typified by a marked, mature neutrophilic leukocytosis. A mild nonregenerative normocytic, normochromic anemia is often observed. An eosinophilia or thrombocytosis may be evident. Hypoglycemia, low urea nitrogen, hypoalbuminemia, and hyperglobulinemia are other common findings.

Electromyography may indicate a generalized polymyopathy. Radiography of the appendicular skeleton may reveal disseminated periosteal bone proliferation mostly involving the diaphysis of long bones. Histopathological examination of the osseous lesions displays changes that closely resemble those of hypertrophic osteopathy. New spicules of bone forming in the periosteum are oriented perpendicular to the cortex without producing cortical destruction. The pathogenesis of such changes is not well understood. Changes do not seem to be associated with the presence of parasites or inflammation in the adjacent skeletal muscle.

Immune-complex hypersensitivity resulting in fatal vasculitis or glomerulonephritis is a potential sequela of infection. A protein-losing nephropathy may be diagnosed in dogs with glomerulonephritis.

Definitive diagnosis is made by microscopic observation of the organism. Gamonts may be detected in neutrophils and monocytes on peripheral blood smears, although this finding is more typical of *Hepatozoon canis* infection. Successful diagnosis is usually achieved via muscle biopsy where developing organisms are usually abundant. Histopathological changes often consist of pyogranulomatous myositis, muscular necrosis, and muscular atrophy. Hepatozoon cysts, or zoites contained within acute granulomas, may be identified interspersed between the muscle fibers. Lesions similar to those found in

skeletal muscle may also be found in cardiac muscle and smooth muscle of the intestine. Lymph node aspirates often yield reactive lymphoid hyperplasia, but organisms are very rarely found. Bone marrow aspirates may indicate granulocytic hyperplasia and erythroid hypoplasia, and also rarely yield organisms. Pyogranulomas and *H. americanum* cysts may also be found in pancreas, lymph nodes, kidney, spleen, and lung.

Since *Hepatozoon americanum* causes a relatively low level of parasitemia in comparison to *H. canis*, antibody production rate may be lower than expected and therefore cause higher numbers of false negatives when tested serologically. Studies have not been performed to identify the prevalence of uninfected or subclinically infected seropositive dogs. Future studies are needed to evaluate the clinical efficacy of serological diagnosis.

-by Michelle Dennis, Class of 2002
-edited by Dr. Mika Tanabe, ADDL Instructor

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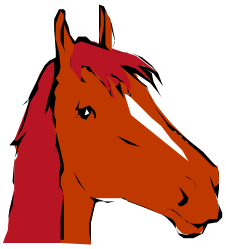

Strangles

Introduction: Strangles is an infectious, transmissible, world-wide disease of horses, donkeys and mules. It continues to rank among the three most significant respiratory diseases of horses. Its widespread distribution is favored by its highly contagious mode of spread and a mobile horse population.

Etiology: The causal agent, *Streptococcus equi* subspecies *equi*, is a gram-positive, beta-hemolytic coccobacillus organism. *S. equi* is highly host adapted to equids and demonstrates no genetic or phenotypic variation although there is variation in virulence related to factors such as hyaluronic acid capsule, the M-like proteins SeM and SzPSe, streptolysin S, and pyrogenic superantigenic exotoxins.

Epizootiology: An obligate parasite of equids, *S. equi* relies on its host for survival and interepizootic maintenance. Strangles

may affect animals of all ages, but it is most common in horses less than two years old (except foals under four months of age, which are usually protected by colostrum-derived passive immunity). Although the majority of animals with strangles are subsequently immune, some may contract the disease a second, or even third, time. Horses that have recovered from the clinical disease may have persistent infection of *S. equi* in the pharynx and guttural pouches for many months and are an important source of infection. Horses with clinically inapparent disease, such as some cases of guttural pouch empyema, may shed the organism for over three years.

Strangles is highly contagious, with transmission occurring by the oral and nasal routes. Communal drinking sources, population density, and mobility are important risk factors. *S. equi* may survive for several weeks in water troughs, but dies quickly in soil and on pasture. It will remain viable in frozen discharges; otherwise survival requires moisture and protection from sunlight.

Pathogenesis: Following entry into a new host, *S. equi* attaches primarily to the cells on the tonsillar crypts and the ventral surface of the soft palate. The organism slowly multiplies in the lymph node. Subsequent migration of neutrophils into the lymph nodes causes swelling and abscessation. Nasal shedding usually begins 4-7 days after infection. Resistance to phagocytosis mediated by a combination of the hyaluronic acid capsule and SeM protein is the key feature of *S. equi* virulence.

Clinical findings: After an incubation period of 1-3 weeks, the disease develops suddenly with complete anorexia, depression, fever, and a serous nasal discharge which rapidly becomes copious and mucopurulent. Retropharyngeal lymph node enlargement may cause obstruction of the oro- and nasopharynx with subsequent dyspnea and dysphagia. Death by asphyxiation may occur at this time in severe cases.

An atypical form of the disease can occur in older animals with residual immunity to *S. equi*, which is characterized by a transient fever, profuse nasal discharge, and anorexia. Lymphadenopathy is seen in approximately half of the affected horses.

Sequelae and complications: Complications occur in about 20% of the cases. The most common fatal complication is the development of suppurative necrotic bronchopneumonia secondary to the aspiration of pus from internal ruptured abscesses or metastatic infection of the lungs. Guttural pouch infection with empyema may also result from rupture of abscesses in the retropharyngeal lymph node. Metastatic infection, also known as "bastard strangles", results in the formation of abscesses in any organ or body site, but most commonly in the lungs, mesenteric lymph nodes, liver, spleen, kidneys and brain. Purpura hemorrhagica may occur as sequelae of *S. equi* infection as well.

Necropsy findings: In the rare fatalities that occur, necropsy examination usually reveals suppuration in internal organs, especially in liver, spleen, lungs, pleura, and peritoneum. When the latter is involved, it is usually due to extension from abscesses in the mesenteric lymph nodes.

Diagnosis: Culture of nasal swabs, nasal washes or pus from abscesses is essential for confirming the presence of *S. equi*. However, culture may fail to detect the organism during the incubation period, in early clinical phases, and in the guttural pouch carriage in apparently normal horses following recovery from strangles. PCR, combined with culture, increases the carrier detection rate while serology is not very useful in the detection of *S. equi* infection.

Hyperfibrinogenemia is characteristic of both the acute and chronic disease. Leukocytosis with neutrophilia and hyperproteinemia attributable to a polyclonal gammaglobulinemia is characteristic of metastatic and chronic abscessation.

Differential diagnosis: Strangles should be differentiated clinically from other upper respiratory tract diseases of horses. Chronic weight loss due to metastatic infection should be differentiated from equine

infectious anemia, parasitism, inadequate nutrition, and neoplasia.

Treatment and control: There is considerable debate about the antibiotic treatment of strangles. It has been suggested that antibiotic treatment of horses with strangles is contraindicated because it promotes the development of metastatic infection. Since there is no evidence to support this contention, horses with strangles should be treated with therapeutic doses of an appropriate antibiotic, such as procaine penicillin, for a period of time sufficient to effect a cure. The very contagious nature of strangles requires rigorous control measures.

Newly arrived animals, including nurse mares, should be observed for signs of strangles for three weeks before admission to the resident population. Rectal temperature should be monitored twice daily. Horses with elevated temperatures should have nasopharyngeal or guttural pouch swabs cultured. Affected horses should be promptly isolated. All potential fomites, including pails, brooms and grooming brushes, should be thoroughly cleaned and disinfected. Nasopharyngeal swabs or washes from recovered animals should be cultured or tested by PCR to demonstrate cessation of nasal shedding.

Immunity and vaccination: Foals that receive adequate high quality colostrums from exposed or vaccinated mares have serum and nasopharyngeal mucosal immunoglobulins that provide resistance to *S. equi* infection. The efficacy of vaccination of adult horses with *S. equi* bacterins or M protein extracts is controversial. A common vaccination protocol involves the administration of an M protein vaccine intramuscularly for an initial course of 3 injections at 2-week intervals, with further administration of the vaccine every 6 months in animals at increased risk of contracting the disease. On breeding farms, the vaccination of mares during the last 4-6 weeks of gestation and of the foals at 2-3 months of age may reduce the incidence of the disease.

The intramuscular vaccine frequently causes swelling and pain at the injection site. Injection into the cervical muscles may cause the horse to be unable to lower its head to eat and drink for several days. Injection into the pectoral muscles is preferred for this reason. Purpura hemorrhagica has been reported associated with administration of the *S. equi* vaccine.

An intranasal vaccine of an avirulent, live strain of *S. equi* has recently been reported and appears useful. However, its efficacy in field situations, safety in the face of an outbreak, in pregnant mares, incidence of adverse effects, and risk of reversion to virulence have not been reported.

-by Dhana Natarajan, ECFVG Student
-edited by Dr. Ingeborg Langohr, ADDL Graduate Student

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ON THE ROAD

Dr. Leon Thacker attended the annual Indiana Veterinary Medical Association meeting in Indianapolis, IN, January, 2003.

Dr. Steve Hooser and ADDL graduate student **Christina Wilson** attended the Society of Toxicology annual meeting in Salt Lake City, Utah, March, 2003.

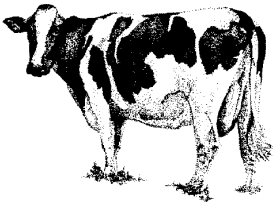
Drs. Zheko Kounev and **Tom Bryan** attended the World Poultry Exposition and United States Egg and Poultry Association meeting in Atlanta, Georgia, January, 2003.

Dr. Ching Ching Wu attended a Global Antibiotic Resistance Panel meeting in Puerto Vallarto, Mexico, January, 2003.

Dr. Ching Ching Wu presented generic compound interpretive criteria at the NCCLS Veterinary Antimicrobial Susceptibility Testing meeting and chaired the National Committee on Clinical Laboratory Standards in Tampa, Florida, January, 2003.

ADDL NEWS

Christina Wilson, ADDL graduate student, was named student representative to the Society of Toxicology at their annual meeting in Salt Lake City in February, 2003 and will serve as co-chair for the Student Advisory Committee. Ms. Wilson will represent the Midwest district of the SOT.



Hepatic Lipidosis in Dairy Cattle

Hepatic lipidosis, commonly referred to as "fatty liver syndrome," is a multifactorial syndrome of peri-parturient dairy cows. It occurs most frequently in production situations that commingle dry cows and lactating cows in a single group. These cattle are likely to become over-conditioned late in lactation or during their dry period. Mortality can be as high as 25% without aggressive treatment and correction of concurrent diseases.

In over-conditioned cattle, fatty acids synthesized in the liver are stored as triglycerides in adipose tissue at extra-hepatic sites. With an increased energy demand not met by a parallel increase in intake, triglycerides in adipose tissue are converted to glycerol and NEFAs (non-estrified fatty acids), which are bound to albumin in the blood. The albumin bound NEFAs can be used as sources of energy by the mammary glands, liver, spleen, and muscle. The liver receives much of the NEFAs due to its large blood supply and efficiency at extracting these substances. In the liver, NEFAs are re-estrified back into triglycerides and remain in the liver until they can be oxidized or repackaged in an envelope of cholesterol, phospholipid, and protein. Repackaging and export from the liver is a very slow process in cattle. Cattle with hepatic lipidosis have smaller amounts of the packaging materials, further slowing hepatic output. It is likely that glucose availability influences the course of the disease, with high glucose availability favoring fatty liver syndrome and the opposite favoring ketosis.

Development of fatty liver syndrome can happen very quickly. Triglycerides present can increase from 5% to more than 25% in 48 hours under extreme conditions of fat mobilization. A prepartum decline in DMI (Dry Matter Intake) appears to increase the NEFAs in the serum at this time. This is

most severe in obese cows, those in stressful environments, or those under nutritional stress. Endocrine changes that occur at parturition may contribute to the decrease in DMI. It has been shown that force-feeding to prevent a DMI drop will reduce the amount of hepatic triglycerides accumulated at day 1 postpartum.

Cows that develop fatty liver syndrome tend to be obese or well-conditioned with high amounts of omental and/or subcutaneous fat. They show nonspecific signs of illness, including depression, anorexia, weight loss, and weakness that may lead to recumbency. Decreased milk production and rumen motility are also evident. Metritis, retained fetal membranes, mastitis, milk fever, and displaced abomasum are some concurrent diseases that can be present with fatty liver syndrome. The number of days to first ovulation following calving has been shown to be increased with greater amounts of triglycerides within the liver.

Most diagnostic tests are poor indicators of hepatic lipidosis. The most common clinical pathological abnormalities are ketonuria, hypoglycemia, and increased serum free fatty acids. Liver derived enzymes, such as GGT and AST, are usually higher than in a dry cow, but are still within normal limits. Due to decreased functional liver mass, the triglycerides and cholesterol (mostly lipoproteins) may be decreased. Serum bile acids, in one study, were found to be an unreliable indicator of hepatic fatty degeneration.

A liver biopsy is the most accurate and reliable way to confirm and assess the degree of fatty degeneration of the liver antemortem. A mild to moderate amount of fat can be present in the liver of most post-parturient high producing dairy cows without evidence of disease. The amount of fatty degeneration can be quantitated via histopathology or flotation in copper sulfate solutions of varying specific gravities. It has been shown that there is little correlation between the amount of fatty degeneration and clinical signs until it is marked and the liver will float in distilled water (SG=1).

On necropsy, fatty livers are usually yellow or tan. Fatty degeneration is especially prominent in the centrilobular and intermediate areas of hepatic lobules, and a pronounced reticular pattern, on both serosal and cut surfaces, can usually be observed. A white discoloration may be evident in the abdominal fat due to accelerated lipolysis.

The main goal in treatment of fatty liver syndrome is the elimination of the negative energy balance. This includes administration of both glucose and insulin twice daily. Corticosteroids can be used for treating the ketosis that may be present, but only for a short period of time. Corticosteroids will increase appetite, reduce milk production (to help alleviate the demand for energy), and stimulate synthesis of glucose from the stores present. Vitamin E and selenium have been found to be low in cattle with fatty liver, and supplementation is recommended. Transfaunation of rumen fluid from a normal cow may increase production of volatile fatty acids used for glucose precursors.

The best policy regarding fatty liver syndrome is prevention. Goals of a good prevention program include the prevention of obesity in cattle late in their lactation and during their dry period. It is important to maintain a good breeding program so that there are appropriate dry period lengths (45-60 days). The ration should be balanced according to maintenance and pregnancy requirements to maintain, not put on, condition. At 2-4 weeks before calving, the grain content in the ration should be slowly increased to acclimate the rumen flora to the fresh cow ration.

Fatty liver syndrome can be prevented with proper management. In situations in which management is poor, fatty livers, in addition to the other problems mentioned, are likely to be much more prevalent. With good management decisions, the owner will be much happier with the efficiency and productivity of the herd.

-by Lynne Catania, Class of 2002

-edited by Dr. Matt Renninger, VPB Graduate Student

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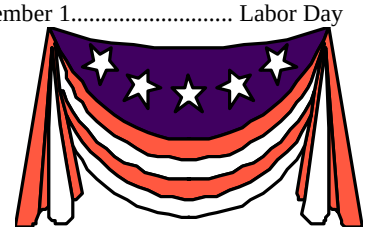
Purdue ADDL and Heeke ADDL
will be closed
on the following University holidays

May 26.....Memorial Day
July 4..... Independence Day
September 1..... Labor Day



Guidelines for Submitting Bovine Ear Notches for FA testing for Persistently Infected BVD

1. Ear notches should be taken with a sharp adult-sized pig ear notching tool.

- Baby pig ear notchers, punches and other cutting and punching tools are not recommended; the sample they provide is too small for an accurate test.
- Dull notchers can damage the sample.

2. Ear notches need to be fresh and from healthy ears.

- Avoid testing scabby or frostbitten ears.
- DO NOT put sample in formalin.
- Samples should be fresh and chilled, NOT FROZEN.

3. Package individually in tubes or vials.

- Number tubes 1-..... and with animal identification to match information on the accession form.

4. If submitting more than 50 ear notches, please give prior notice (765-494-7440) at least 24 hours to allow for fastest possible processing of samples.
-by Virology Laboratory Technicians

...before submitting serology samples

The following information is a general review for submitting samples to ADDL/Serology.

1. Samples hand-carried to ADDL by owners must be sealed using the veterinarian's label or tape bearing the veterinarian's signature.

2. A health certificate is the only necessary test record for 4-H exhibition. Do not submit duplicate test charts.

3. Use of BD-Vacutainer or Monoject tubes is preferred. EDTA-treated tubes will not be accepted.

4. Each tube must be identified with a tube number; additional identification is desirable.

5. Tube numbers and numbers on charts must match and be in consecutive order. Tubes should be packaged in consecutive order as well.

6. Clear serum is preferable to whole blood.

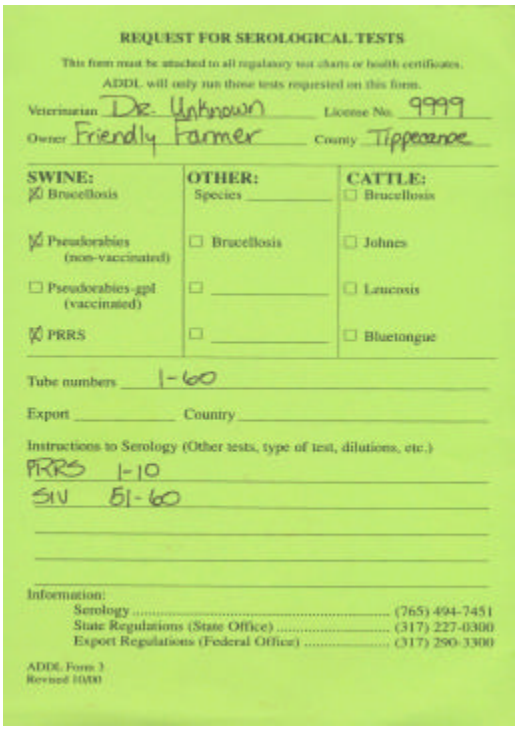
7. Tests for Pseudorabies will be performed routinely. Turnaround time will be 8-10 days (may be slightly longer at peak 4-H testing periods).

8, Brucellosis tests are performed daily (M-F). Turnaround time is 2-4 days.

9. Swine samples for both Brucellosis and Pseudorabies will be tested first for Brucellosis, followed by Pseudorabies. Turnaround time will be 10-14 days.

10. All regulatory charts must include submitting veterinarian's signature and complete animal identification. Unsigned charts will cause delay in results.

11. Request for Serological Test (buckslip) must be completed and **stapled** to all regulatory test charts. ADDL will run only those tests requested on this form.



REQUEST FOR SEROLOGICAL TESTS

This form must be attached to all regulatory test charts or health certificates.
ADDL will only run those tests requested on this form.

Veterinarian Dr. Unknown License No. 9999
Owner Friendly Farmer County Tippecanoe

SWINE:	OTHER:	CATTLE:
☒ Brucellosis	Species ____	☐ Brucellosis
☒ Pseudorabies (non-vaccinated)	☐ Brucellosis	☐ Johnes
☐ Pseudorabies-gpl (vaccinated)	☐ ____	☐ Leucosis
☒ PRRS	☐ ____	☐ Bluetongue

Tube numbers 1-60
Export ____ Country ____
Instructions to Serology (Other tests, type of test, dilutions, etc.)
PRRS 1-10
SIV 51-60

Information:
Serology ..... (765) 494-7451
State Regulations (State Office) ..... (317) 227-0300
Export Regulations (Federal Office) ..... (317) 290-3300

ADDL Form 3
Revised 10/00

12. Only one buckslip is needed for each individual herd owner or farm; do not attach to continuation charts.

Please note
EIA tests charts do not require a buckslip. Request for method of test can be made directlv on the chart.

13. ADDL will not release results to owners.

If you have questions, contact ADDL/Serology at 765-494-7451 prior to submitting samples.
-by Karen Crane, Serology Laboratory Supervisor

Percent of Micro-organisms that are Resistant to Selected Antibiotics for 2002

Antibiotic	Canine										Equine												Feline							
	E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus		Staph. intermedius		E. Coli		Salmonella sp.		Staph. aureus		Staph. epidermidis		Strep. equi		Strep. zooepidemicus		E. Coli		Enterococcus sp.		Pse. aeruginosa		Staph. aureus
	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June
Amikacin	1	1	18	38	0	2	0	4	0	0	0	0	0	0	0	0	0	0	71	100	65	100	0	0	0	71	0	0	0
Amoxycillin/Clauvulinic acid	24	18	9	19	93	95	31	4	0	2	8	27	25	20	43	50	38	0	0	0	0	0	11	14	33	43	100	100	0
Ampicillin	48	48	9	23	100	95	54	46	44	42	36	50	25	40	71	50	38	50	0	0	0	5	44	38	33	43	100	100	100
Cefazolin	23	18	64	62	100	98	23	4	0	0	12	27	25	20	43	50	25	0	0	0	0	5	0	10	100	100	100	100	0
Cefotaxime	13	7	9	33	7	10	23	4	0	0	0	0	0	0	43	50	25	0	0	0	0	5	0	5	33	57	0	20	0
Cefoxitin	24	17	91	81	100	95	23	4	0	0	8	19	25	20	43	50	25	0	0	0	0	5	0	0	67	86	100	100	0
Ceftiofur	20	16	73	82	100	98	23	4	0	0	0	12	13	20	57	50	25	0	0	0	0	5	0	5	100	100	100	80	0
Cephalothin	17	23	36	57	100	95	23	4	0	0	12	31	25	20	43	50	25	0	0	0	0	5	22	19	67	86	100	100	0
Chloramphenicol	15	16	0	0	67	80	0	0	3	0	4	38	13	40	0	0	13	0	0	0	0	0	0	5	33	0	67	60	0
Ciprofloxacin	16	16	0	10	13	17	15	8	3	2	4	4	0	0	14	0	13	0	0	0	0	0	6	10	0	43	0	0	0
Clindamycin	99	100	82	86	100	95	0	8	15	9	100	100	100	100	0	0	0	0	14	0	0	5	100	100	100	86	100	100	0
Enrofloxacin	16	18	27	45	27	55	15	17	3	2	4	4	0	0	14	0	13	0	0	0	0	0	6	14	33	57	0	40	50
Erythromycin	99	100	0	23	100	95	8	17	15	9	100	100	100	100	29	50	0	50	14	0	0	5	100	100	67	43	100	100	0
Gentamicin 500 microgm/ml	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	14	0	0	50
Gentamicin	9	11	9	32	0	10	8	1	3	0	16	31	0	0	14	50	0	0	0	100	20	55	0	19	67	57	0	0	0
Sulphadimethoxine/Ormetoprim	29	29	0	10	100	95	8	13	15	9	40	62	0	40	43	50	13	50	0	0	0	5	11	19	0	14	100	100	0
Oxacillin + 2% NaCl	99	100	91	76	93	95	23	4	0	0	100	100	100	100	43	50	0	0	0	0	0	0	100	100	100	86	100	100	0
Penicillin	99	100	9	27	100	98	46	42	44	38	100	100	100	100	71	50	25	50	0	0	0	5	100	100	33	43	100	100	100
Rifampin	97	97	36	33	100	95	0	0	0	0	100	100	100	100	0	0	25	0	0	0	0	0	94	100	33	29	100	100	0
Tetracycline	31	33	36	52	33	27	8	17	12	16	36	42	13	20	29	50	0	50	0	0	30	50	17	24	100	86	33	40	50
Ticarcillin	43	42	18	24	13	7	46	42	44	38	32	42	13	20	71	50	38	50	0	0	0	5	33	29	33	71	0	40	100
Tribrissen	29	29	0	10	33	29	15	42	32	47	40	62	0	40	43	50	25	50	0	0	0	9	11	14	0	14	33	40	0
Vancomycin	99	100	0	10	100	98	0	0	3	0	100	100	100	100	0	0	13	0	0	0	0	0	100	100	0	0	100	100	0
number of isolates	74	90	11	21	15	41	13	24	33	45	25	26	8	5	7	2	7	2	7	1	20	22	18	21	3	7	3	5	3

Percent of Micro-organisms that are Resistant to Selected Antibiotics for 2002

Antibiotic	Beef								Dairy						Swine							
	E. coli		Past. Haemolitica		Past. Multocida		Salmonella sp.		E. coli		Staph. aureus		Salmonella sp.		APP		E. coli		Salmonella sp.		Strep. suis	
	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.	Jan.-June	July-Dec.
Ampicillin	49	46	0	0	0	0	29	43	56	62	63	17	66	80	15	6	64	64	53	41	1	3
Apramycin	7	12	20	22	40	67	0	29	39	35	na	na	21	26	2	13	21	20	10	9	33	41
Ceftiofur	20	19	0	0	0	0	29	43	25	26	4	0	45	50	2	0	8	10	8	6	7	8
Chlortetracycline	74	69	20	0	0	0	57	43	74	91	na	na	66	85	11	10	99	86	84	76	90	96
Clindamycin	100	100	100	100	100	100	100	100	94	97	na	na	97	100	8	19	99	100	100	100	89	82
Enrofloxacin	14	12	0	0	0	0	0	0	20	12	na	na	0	0	2	0	0	0	0	0	2	3
Erythromycin	100	100	20	0	60	0	100	100	95	98	na	0	97	100	0	3	99	99	100	100	91	80
Florphenicol	100	100	20	11	60	0	100	100	94	98	na	na	97	98	2	0	99	95	96	97	68	67
Gentamicin	19	27	20	0	60	0	0	29	53	59	na	na	24	35	2	0	19	21	10	15	2	5
Neomycin	31	42	80	44	80	67	43	43	65	71	na	na	59	67	9	3	46	43	24	15	60	63
Oxytetracycline	76	69	40	33	60	50	57	43	74	92	na	na	66	85	32	35	99	88	84	76	92	97
Penicillin	100	100	20	44	0	17	100	100	95	98	33	17	97	100	72	90	99	100	100	100	8	12
Sulphadimethoxine	69	58	60	44	60	83	57	57	64	73	70	92	76	83	15	10	74	71	66	71	63	62
Spectinomycin	36	38	40	78	80	50	86	100	63	76	na	na	66	93	13	0	65	55	90	100	19	29
Sulphachloropyridazine	69	58	20	11	80	83	57	43	75	89	na	na	79	83	25	35	74	72	64	68	66	60
Sulphathiazole	69	58	80	56	80	83	57	43	76	89	na	na	79	83	21	23	75	73	64	68	67	65
Tiamulin	100	100	100	78	60	83	100	100	93	98	na	na	97	100	15	0	99	100	100	100	22	25
Tilmicosin	98	100	0	0	60	0	100	100	92	96	na	na	97	100	4	0	98	98	100	100	88	77
Triple Sulfa	38	42	20	0	0	0	0	43	57	61	na	na	28	37	2	0	14	18	12	12	3	6
Tylosin	100	100	100	89	60	83	100	100	94	98	na	na	97	100	na	na	99	100	100	100	na	na
number of isolates	67	26	5	9	5	6	7	7	112	85	27	12	26	46	53	31	188	96	51	34	123	117