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# Vets Hear New Concepts on Endotoxins in Cattle

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Several speakers at the April 2003 Academy of Veterinary Consultants meeting presented information covering several aspects of bacterial endotoxins and their effects on cattle.

Dr. Jim Cullor, from the University of California-Davis, said endotoxin release resulted in "mediator shock," which initiated a whole cytokine cascade. Among other changes, this cascade is characterized by an initial neutropenia and lymphopenia, followed approximately two days later by a neutrophilia. The severity of the initial reaction depended on the dose of endotoxin. Cullor presented the concept of "endotoxin stacking," where cumulative endotoxin from a variety of sources, bacterins or vaccines can create a reaction together. He saidthat the reaction to endotoxin from multiple sources usually had a more dramatic effect than the same amount of purified endotoxin from a single source. He pointed out that heat stress intensified endotoxic reactions, a vaccination program using multiple gram-negative bacterins administered during the summer would result in more endotoxic reactions than the identical program administered during the winter.

Cullor said that vaccine-induced endotoxic shock usually started to manifest itself two to three hours postvaccination, with the cattle becoming dull or depressed approximately 24 hours postvaccination. If pregnant, these cattle could abort within a few hours to three days later, especially if exposed during the first trimester. If exposed during the third trimester, calves could be born prematurely. If not pregnant, but with a corpus luteum between eight and 18 days of maturity, exposure could result in short cycling as the corpus luteum is lysed. Feedlot cattle could break with BRD five to six days postvaccination.

Dr. John Pollreisz of Pfizer Animal Health said endotoxins are not directly toxic, but initiate a cytokine cascade that produces observed symptoms. He said all endotoxin is not equal in its likelihood of reactivity. He listed several variables that influence the degree of reactivity, including host species genetic susceptibility, age of animal, priming or tolerance factors, bacterial species factors, whether the endotoxin is free or bound, adjuvant(s) used in a vaccine, and the presence of other irritants that might initiate a similar cascade in the absence of endotoxin.

> . . . heat stress intensified endotoxic reactions . . .

Pollreiz said cattle and pigs are the most susceptible domestic species. He indicated that the true incidence of endotoxic reaction is probably understated because many cases do not reach the stage where they are clinically observable. He classified bacterial agents by their tendency to result in an endotoxic reaction: highly reactive – *Campylobacter* (Vibrio), *Moraxella*, *E. coli* and *Salmonella*; moderately reactive – *Haemophilus*; less reactive – *Lepto*, *Mannheimia haemolytica*, *Pasteurella multocida*. He also gave some recommendations for combining gram-negative components in a vaccination program: No more than two gram-negatives at one time in dairy breeds; no more than three gram-negatives at one time in beef breeds. Five-way Lepto equals one gram-negative. If more than the allowable number of gram-negative organisms need to be included in the overall vaccination program, give a portion of the gram-negative vaccinations, then wait a week before giving the next portion. Heat, freezing or vigorous shaking can disrupt cellular components and release additional endotoxin from a gram-negative product. He strongly recommended swirling gram-negative bacterins when trying to keep particles in suspension, rather than pounding them like a bottle of penicillin.

Pollreisz offered tips on vaccination reactions: Record serial numbers at the time of vaccination. Intervene quickly if endotoxic reaction is suspected (NSAIDS, steroids, calcium IV). Send representative samples to the diagnostic lab early instead of trying to obtain them after the fact. Call vaccine manufacturers early; and call all of them, not just the ones you think

*Endotoxins, pg. 3*

## *Also in this issue*



*Thank you to the Pfizer Animal Health Group, Livestock Division, Cattle Products Group, for financial assistance in publishing this newsletter.*

# Prevention and Treatment of West Nile Virus in Horses

*Bonnie R. Rush, DVM, MS, DACVIM*
*Large Animal Hospital*

## Epidemiology:

West Nile virus (WNV) was first recognized in the United States in New York in 1999. Since its introduction, the virus has spread south and west across the country to include more than 40 states. WNV is considered an endemic disease of the United States and is predicted to maintain that status indefinitely.

West Nile virus is a flavivirus, and is antigenically similar to Japanese encephalitis complex and St. Louis encephalitis. The natural life cycle of the virus involves birds (reservoir host) and various species of mosquitoes. Birds produce a large number of virus particles in circulating blood, which allows them to serve as amplifying hosts of WNV. Humans and horses are considered aberrant, dead-end hosts. Many other mammals demonstrate evidence of exposure to WNV (seroconversion), however, clinical disease is not evident except in isolated cases associated with immunocompromise. The incubation period in horses is approximately 3 to 14 days. Following the bite of an infected mosquito, the virus multiplies at low levels in the horse's bloodstream and crosses the blood-brain barrier to infect the brain. Viral replication produces central nervous system (CNS) swelling and inflammation. Horses may be affected at any age, and there is no breed or gender predilection.

## Clinical Signs:

The clinical signs of WNV infection in horses are characteristic, and wax and wane dramatically at the onset of disease. The severity of disease ranges from mild, transient neurologic deficits to flaccid paralysis and recumbency. The most common early signs of infection include twitching of the muzzle and ears, frequent chewing, hyperresponsiveness, hyperaesthesia, aggression, and fine muscle fasiculations. Horses then develop progressive ataxia, weakness, and listlessness.

One characterstic behavior in WNV affected horses is a narcoleptic-like maneuver whereby the horse appears asleep and drops in the forelimbs until the chest is nearly on the ground, while maintaining the hindlimbs in a standing posture. Severely affected horses may develop paresis or paralysis of the limbs, seizures, mania, disorientation, coma, or death. Some horses may present as a single limb, non-weight-bearing lameness.

The progression of clinical signs is rapid. Unlike other viral encephalitides, fever is not generally observed. Other clinical signs that may be observed in horses with WNV include impaired vision, colic (autonomic disruption), inability to urinate (autonomic disruption), head tilt, and facial-nerve paralysis. Inability to urinate is observed in approximately 30 percent of the cases, and these horses appear to have an upper motor neuron bladder dysfunction. Urethral sphincter tone is exaggerated and catheterization of the bladder is challenging. As horses begin to recover, 7 to 10 days after the onset of clinical signs, they may develop a characteristic hypermetric gait that persists for 2 to 5 days. The most difficult diseases to differentiate from WNV based on clinical signs are rabies, eastern/western encephalitis, and equine protozoal myeloencephalitis.

## Diagnosis:

It is important to obtain a definitive diagnosis of WNV infection to document the number of cases in a region and rule out diseases with similar clinical signs (rabies, EEE/WEE, and EPM). Diagnosis of WNV is determined by detection of antibody against WNV in serum by IgM capture ELISA. Horses typically have a sharp rise in IgM antibody against WNV for 30 days after exposure. A positive test result indicates recent exposure. Vaccination does not appear to interfere with testing. False negative diagnosis may occur if the individual is tested early in the disease process or if the horse fails to mount a typical immune response to the virus. Retesting serum by IgM capture ELISA is recommended for horses with clinical signs typical of WNV but negative test results.

## Treatment:

Treatment of WNV is directed towards reducing edema and inflammation in the central nervous system. Flunixin meglumine (1.1 mg/kg, IV, BID) is administered for analgesia and anti-inflammatory activity. Dimethyl sulfoxide (1 gm/kg, 10% soln, IV) is recommended to reduce cerebral edema twice daily for the first 3 days, followed by once daily until improvement is noted. Dexamethasone (50 to 100 mg, IV) is administered judiciously and may be used for rapidly progressive cases or horses that present recumbent. It is unclear if the immunosuppressive effects of dexamethasone interfere with antibody production (and diagnostic testing) or prolong the course of disease. Interferon-alpha (rHuIFNa-2b) has direct antiviral activity and is an early endogenous antiviral defense. Interferon is administered at 3 million units intravenously once daily for 3 to 5 days. Adverse effects such as fever, malaise, and inappetence were not observed at this dose.

In many instances, horses will recover from WNV infection but have sustained a life-threatening injury or condition during a neurologic episode (fractured leg, luxated joint, laminitis, skull fracture). Therefore, supportive care to protect the horse from self-inflicted trauma is crucial to the management of WNV encephalitis. Sedation, sling support, protective leg bandages, and a helmet are recommended for horses with moderate to severe signs of WNV. Detomidine, chloral hydrate, diazepam, and butorphanol are effective sedatives for most cases. Acepromazine should be avoided due to the risk of seizure in some horses with WNV infection.

An indwelling urinary catheter is required in some patients (male and female) due to urinary retention (UMN bladder). Bladder decompression should be maintained to ensure return of detrussor tone upon recovery. Horses with indwelling urinary catheters should receive prophylactic antibiotics, such as trimethoprim-sulfamethoxazole (15 mg/kg, BID, PO).

The prognosis for WNV depends on the severity of clinical signs. The survival rate in horses that remain standing (or transiently dog-sit) is approximately 80 to 90 percent. Horses that become recumbent and cannot be supported with the use of a sling have a survival rate of 30 percent or less. Overall survival at KSU in 2002 was 83 percent.

## Prevention:

A vaccine is available for administration to horses for the prevention of WNV infection. The vaccine has been determined to be safe and effective for preventing viremia. An initial vaccine is administered, followed by a booster 3 to 6 weeks later. A single vaccination appears ineffective for prevention. Preliminary data indicates protection may begin 30 days after the booster vaccination. Annual vaccination is recommended 30 days before mosquito season, followed by a booster vaccination in late June to July, to stimulate maximal immunity for peak WNV season in early September. Foals from vaccinated mares should begin their vaccination program at 3 months of age, and should be followed with two booster vaccines. Foals from unvaccinated mares should be vaccinated at 1, 2, 3, and 6 months of age. Vaccine recommendations may change as information becomes available regarding efficacy, duration of protection, and immunity following natural infection.

# Reporting a Foreign Animal Disease

*Reprinted with permission from the American Association of Swine Veterinarians, 2003*

If you suspect a foreign animal disease (FAD), would you know what steps to take to obtain a diagnosis? As a veterinarian, you and your clients are the first line of defense. Recognizing that a disease could be the result of the introduction of a foreign animal disease pathogen is the first step in initiating an investigation.

The clinical signs and gross lesions associated with a FAD may be subtle and resemble many common disease conditions. Mildly virulent strains of classical swine fever (hog cholera), for example, may appear with clinical signs resembling SIV or PRRS infections with little or no gross lesions, or the lesions may be masked by those of concurrent diseases. Descriptions and pictures of foreign animal diseases can be found at *http://www.vet.uga.edu/vpp/gray_book/index.htm* and *http://www.aphis.usda.gov/vs/ep/fad_training/bibpage.htm*.

It is important to obtain a complete and thorough disease history regarding onset, recent foreign travel by employees or visitors, feed sources (i.e., garbage feeders), other species of animals exhibiting similar or abnormal clinical signs, efficacy of prior treatments, recent animal movements (introductions and shipments), and consumption of foreign foods by employees, etc. Answers to these and similar questions may increase your suspicion of a FAD even in the absence of abnormal clinical signs or lesions.

If you suspect a FAD, it is important to contact the federal area veterinarian in charge (AVIC) or state veterinarian immediately. Sending samples to diagnostic labs yourself can contaminate those labs and spread the disease.

## If you suspect a FAD. . .

1. Do not leave the farm unless absolutely necessary, and then only after a thorough disinfection.
2. Contact the AVIC in your state or the state veterinarian's office.

- A list of the federal AVIC contact numbers can be found at *http://www.aphis.usda.gov/vs/area_offices.htm*
- A list of state veterinarian contact numbers can be found at *http://www.usaha.org/whoswho.htm#nass*

3. The AVIC will dispatch a Foreign Animal Disease Diagnostician (FADD) to investigate within 24 hours of notification.
4. The FADD will visit the premises, assess the situation, collect and submit laboratory samples, execute disease control actions if necessary, and file a report with the AVIC.
5. The AVIC will assign a priority level to the lab submissions that will govern the response of the federal lab(s).
6. The AVIC, in consultation with the FADD, state veterinarian and USDA emergency programs, may take further action.
7. Lab results will be reported to the AVIC, who will notify the state veterinarian and the FADD. The FADD will notify the practitioner.

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*Endotoxins, from pg. 1*

might pay. Have realistic expectations and create the same in your clients. Work together with all participants. Understand the science of endotoxins.

Dr. Dan Scruggs, from Mississippi State University, said endotoxins initiate an acute phase response in animals, and the individual animal's response depends on the extent to which the animal's system is challenged. Low-level challenges can be detoxified in the liver with no adverse response. Moderate challenges activate a higher level of response, and healthy animals usually survive. Severe challenges activate an even higher level of response and pose a serious threat to most animals.

He said oral endotoxin is almost always denatured in the liver, regardless of dosage, whereas inhaled or systemically-acquired endotoxin may cause major problems. He said the guide to measuring endotoxin effects includes pull rates, mortality, fever, anorexia and outward appearance.

# Testing Bulls Using In-Pouch® System

*Peter J. Chenoweth, Large Animal Hospital*

Collecting preputial samples from bulls for detection of trichomoniasis has long been a routine diagnostic procedure. This procedure generally entails using a plastic pipette to obtain aspirated material from the fornix region. This material is then placed into an appropriate transport/culture media for subsequent examination. The inoculated media should be stored at $35^0$ to $37^0$C until microscopic examination for *T.foetus*. A practitioner may conduct the examination, or it may be sent to a diagnostic laboratory where it is commonly examined after incubation for both two and five days.

A transport/culture system that has gained wide acceptance is the In-pouch® system. This represents a self-contained, micro-aerophilic culture system containing a buffered proprietary media that inhibits bacterial and fungal growth. Under good conditions, its sensitivity in detecting *T. foetus* from cultured bovine preputial smegma is approximately 90 percent. However, success depends on a number of factors, and sensitivity has been reported as low as 70 percent. One complication that can compromise the test is contamination and overgrowth of the sample. This is not an uncommon complication for samples submitted to the K-State Diagnostic Laboratory. To minimize this problem, the following guidelines are suggested.

### Storage – Before Use

1. Store at room temperature ($15^0$ to $25^0$C), vertically and in the dark.
2. Respect recommended shelf-life of 1 year from date of manufacture.
3. Do not freeze.
4. Do not use if medium is cloudy or contains precipitate.

### Collection Regime

1. Minimize contamination from the bull and environment.
2. Insert a small sample (0.5 to 1.00 cc). Avoid rinse fluids and urine because maintenance of pH is important.
3. Delete air from upper pouch before closing.
4. Store upright to concentrate cellular material.

# Equine Leukoencephalomalacia Linked to Contaminated Corn

*John M. Ragsdale, DVM, and Brad M. DeBey, DVM, PhD.,*
*Diagnostic Medicine/Pathobiology*

This spring, a herd of approximately 30 horses experienced morbidity and mortality with clinical signs and macroscopic and microscopic lesions diagnostic of equine leukoencephalomalacia (ELEM). Five horses died and two others showed clinical signs and no weight gain. The clinical signs the horses exhibited before death include blindness, aggressiveness, ataxia, seizures, recumbency, thrashing and coma. The horses that died did so in a matter of hours after first showing clinical signs. All of the horses were being fed the same diet of prairie hay, grass, alfalfa pellets and sweet feed, which contained corn and corn screenings.

Three of the dead horses were submitted to the Kansas State University Veterinary Diagnostic Laboratory for postmortem examination. Gross lesions were mostly limited to the brain and included a subtle yellow discoloration to malacia and liquefaction of the white matter of one or both cerebral hemispheres *(Figures 1 and 2)*. Microscopically, the lesions were necrosis, perivascular edema, and perivascular neutrophilic, eosinophilic, and lymphocytic inflammation in the white matter. The sweet feed was tested and was positive for fumonisin.

Leukoencephalomalacia, or necrosis of the white matter of the cerebrum, is a disease of equids that results from feeding corn contaminated with a mycotoxin produced by *Fusarium* species. Of the *Fusarium* species, *Fusarium moniliforme* is the more common fungus to affect corn and produce the mycotoxin, but *Fusarium proliferatum* also has been found to produce mycotoxin. Three mycotoxins are produced by *F. moniliforme* and *F. proliferatum*: fumonisin $B_1$, fumonisin $B_2$, and fumonisin $B_3$. Both fumonisin $B_1$ and fumonisin $B_2$ are of equivalent toxicity to horses, but fumonisin $B_1$ is reported to be produced in quantities three times that of fumonisin $B_2$. Fumonisin $B_3$ is much less toxic than the other two fumonisins.

Leukoencephalomalacia usually occurs in epizootics that result from horses being fed contaminated corn, often corn screenings. The disease is usually seasonal occurring in the fall, winter and early spring. *Fusarium* species colonizes the corn during a time of stress to the corn such as a drought followed by a period of wet weather, which allows the fungus to proliferate in the corn. The mycotoxin is produced by the fungus when the corn is in the field and not while the corn is in storage.

Clinical central nervous system disease in horses is initially characterized by failure to respond to voice commands, depression, inappetance and separation from the herd progressing to dementia, ataxia, paresis, agitation, hyperesthesia, aggression, blindness, head pressing, circling, hyperexcitability, facial paralysis, recumbency, seizures, coma and death. The onset of clinical signs after feeding fumonisin-contaminated corn can vary widely from days to weeks. The progression of clinical signs to death can also vary from hours to weeks. There is some variability in an individual horse's susceptibility to fumonisin; a few horses do not develop clinical signs after receiving repeated doses of the mycotoxin that kill other horses.

Another manifestation of fumonisin toxicosis in horses is liver disease. With large doses of fumonisin, horses become icteric with elevated alkaline phosphatase, aspartate aminotransferase, lactate dehydrogenase, gamma-glutamyl transpeptidase and bilirubin. Horses can develop liver failure, chronic liver disease due to fibrosis, hepatic encephalopathy and death. Death usually occurs within hours to days in horses with signs of hepatic necrosis.

There is no adequate antemortem test for ELEM. Most diagnoses are made using the epizootic nature of the disease, clinical signs and postmortem lesions. Serum chemistries may have elevated liver enzymes and increased creatine phosphokinase activity. The complete blood count may show an increase in the hemoglobin concentration, packed cell volume, or hematocrit due to dehydration. Occasionally, a horse with fumonisin toxicity will be anemic. Testing of the feed for the presence of fumonisin can be diagnostic, but the feed sample evaluated may not reflect that total fumonisin dosage the horses had previously received.

Most horses with ELEM or hepatic necro-

*Leukoencephalomalacia, pg. 5*

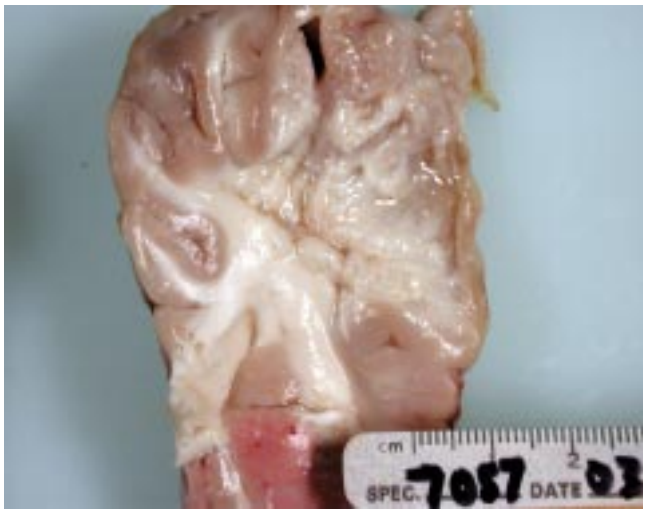


*Figure 1. Formalin-fixed saggital section of the cerebrum. Severe leukoencephalomalacia in a 1-year-old male quarter horse. Only the white matter is affected.*

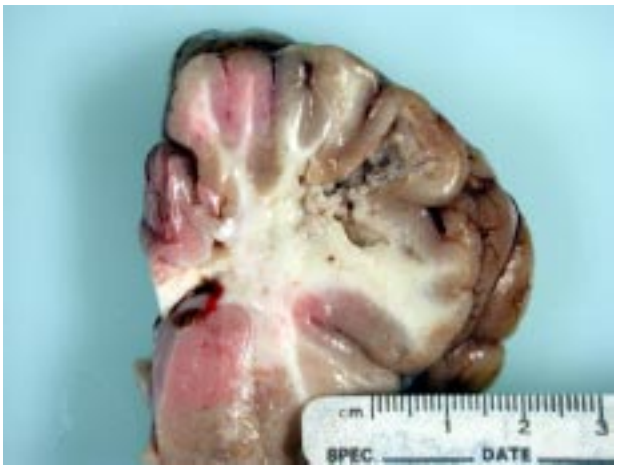


*Figure 2. Formalin-fixed saggital section of the cerebrum. Less severe leukoencephalomalcia in 2-year-old male quarter horse. The white matter at the gray-white matter conjunction of the corona radiata is affected.*

*Leukoencephalomalacia, from pg. 4*

sis due to fumonisin toxicosis die. A few survive, but have lingering neurologic deficits. Treatment with dimethyl sulfoxide, dexamethasone, nonsteroidal anti-inflammatory drugs (flunixin meglumine) and thiamine has been successful in very few horses.

Currently, the U.S. Food and Drug Administration recommends that the total ration for horses should contain less than 1 parts per million (ppm) of total fumonisins. The corn used in equine feeds should contain less than 5 ppm of total fumonisins and comprise less than 20 percent of the total diet. This information can be found at *http://www.cfsan.fda.gov/~dms/fumongu2.html* and *http://www.cfsan.fda.gov/~dms/fumonbg4.html*. Some authors recommend to never feed corn screenings to horses.

In conclusion, equine leukoencephalomalacia is a disease of horses caused by the mycotoxin fumonisin found in corn, often corn screenings. The central nervous system signs, caused by edema and necrosis of the white matter of the brain, can be rapidly progressive. Equine leukoen-cephalomalacia has a grave prognosis. The disease is best prevented by not feeding fumonisin-contaminated corn to horses and other equids.

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# *Lepto in Cattle: Old Disease, New Opportunity*

*Larry C. Hollis, D.V.M., M.Ag*

At the May 2003 conference at Kansas State University on Investigating Pregnancy Wastage in Cattle Herds, Dr. Carole Bolin gave an update on leptospirosis. Bolin's report changes a lot of the perceptions we may have had about the disease – both the causative agent responsible for most of the problems seen in cattle herds and the nature of the clinical signs of Lepto infection.

Bolin said *Lepto hardjo*, not *Lepto pomona*, is the most common cause of leptospirosis in cattle. However, the *Lepto hardjo* she referred to is not the one contained in any of our popular five-way Lepto vaccines. The hardjo strain used in all current U.S. vaccines is *Leptospira interrogans serovar Hardjo* (type hardjoprajitno), a strain that causes leptospirosis primarily in the United Kingdom. The primary hardjo strain that causes leptospirosis in the United States and the rest of the world is *Lepto borgpetersenii* serovar Hardjo (type hardjo-bovis).

Bolin also described the pathogenesis and epidemiology of hardjo-bovis in cattle herds. Cattle are the maintenance host for hardjo-bovis. She said hardjo-bovis can enter the body through any mucosal surface and infect the kidneys and reproductive tracts of both males and females. It can cause classic Lepto signs such as late-term abortions, stillbirths, weak calves, and retained placentae. However, abortion storms are usually seen only upon initial infection in naïve cows.

Once established in a herd, Hardjo-bovis abortions are usually sporadic. Hardjo-bovis infected animals typically develop a renal carrier state and become chronic urinary shedders. They also develop a persistent infection of the reproductive tract, allowing the infection to act like a venereal disease and be mechanically transmitted. Hardjo-bovis more commonly occurs as a subclinical infection, where it is manifested as infertility (low conception rates, early embryonal deaths, delayed return to heat, etc.) or results in clinically normal, infected calves.

When infection becomes endemic within a herd or region, it is common to have 30 to 40 percent of the animals infected and shedding leptospires in their urine. A prevalence conducted study in four states showed that hardjo-bovis was present in 36 to 91 percent of surveyed dairies. The incidence in beef herds is unknown, but is suspected to range from 2 to 20 pecent. The survival of leptospires is greater in lush grass pastures where the organism is protected from ultraviolet light.

Because of the subclinical nature of hardjo-bovis, it is necessary to determine if this species is established in a herd. Diagnosis of leptospirosis depends on a good clinical and vaccination history, as well as laboratory testing. Bolin emphasized that coordination between the diagnostic laboratory and veterinarians is necessary to maximize the chances of making a diagnosis.

Diagnostic tests for leptospirosis can be divided into two categories: tests designed to detect antibodies against the organism and tests designed to detect the organism itself or its DNA in tissues or body fluids. There are advantages and disadvantages to each of the diagnostic procedures, and often a combination of tests is necessary to establish the diagnosis.

Diagnostic tests for leptospirosis include:

Serologic tests: the microscopic agglutination test (MAT) is the most common. Detection of a high antibody titer may be adequate to establish a diagnosis in some instances, but other times, especially with serovar hardjo-bovis, there may be a poor antibody response to infection, making interpretation difficult. Previous vaccination can also complicate interpretation.

Detection of leptospires: the organisms are difficult to culture in the laboratory and other techniques are usually necessary to detect the organism. Other tests to detect the organism include fluorescent antibody (FA) and polymerase chain reaction (PCR). The FA test can be used on tissues or body fluids, but it is not serovar specific. This test is rapid, inexpensive, and fairly sensitive, but sensitivity decreases with autolysis. The PCR test detects leptospires' DNA in tissue and body fluids. In general, PCR is more sensitive on urine than on tissues. This test is very sensitive and specific, but also is not serovar specific and is subject to some false positives.

If hardjo-bovis is found in a herd, the carrier state has been found to be removable with antibiotic therapy, such as 20 mg/kg of long-acting 200 mg/ml oxytetracycline.

If a herd is free from hardjo-bovis, and it is essential that the producer keep the disease out, a vaccine (Spirovac®) is currently being marketed that appears to do an excellent job of preventing establishment of the carrier state. This new vaccine is monovalent, therefore it is also recommended that one of the current five-way Lepto products also be used to protect against infection with *L. Pomona* and *L. grippotyphosa*. Dr. Bolin indicated infections with *L. canicola*, *L. icterohaemorrhagiae* or *L. Bratislava* are rare in cattle.

# Backyard Flocks May Increase Risk of Exotic Newcastle Disease

*Larry C. Hollis, D.V.M., M.Ag*

Do you have any fighting-cock raisers or other backyard, hobbyist or exhibition bird flocks in your area? If so, there is a substantial risk that Exotic Newcastle Disease (END) may be brought into your area if these raisers buy and sell birds, participate in their favorite pastimes, or visit other flocks while looking to purchase birds. All bird raisers are at risk of bringing the infection back to their flock if they travel into one of the areas where this disease has recently been found.

Exotic Newcastle Disease was confirmed in a backyard flock of chickens last October in the southern California area. It has spread beyond backyard flocks to affect 22 commercial egg-laying operations. The disease has been confined to backyard flocks since the beginning of the year: Jan. 16, in the Las Vegas, Nev.; Feb. 4, near Yuma, Ariz.; and April 9, in the El Paso, Texas, area. Counties in and surrounding outbreaks have been placed under federal quarantine, and depopulation efforts have been initiated. To date, more than 3.5 million birds have been destroyed in an effort to control the spread of the disease. The USDA is providing fair market payment for birds that must be destroyed during this disease outbreak.

Exotic Newcastle Disease is a highly contagious foreign-origin virus affecting birds and poultry. END usually has a two- to 15-day incubation period, and infected birds or poultry may exhibit signs of respiratory distress, including gasping or coughing. The virus also affects the central nervous system, causing infected birds to become paralyzed, develop muscle tremors, or twist their necks. In some flocks, the disease may strike quickly, and the only sign is death. Mortality is up to 90 percent of exposed birds. This disease does not affect human health, nor does it affect the safety of poultry products or eggs.

Officials in quarantined areas are asking for full cooperation from bird and poultry owners. They ask owners not to move birds from or within the quarantined areas. They recommend all bird owners keep birds in isolation on their own property and ensure that no birds are introduced onto their property during the quarantine period. The quarantines will last until state and federal animal health officials are certain the disease has been eradicated.

Basic biosecurity is critical on individual premises during the quarantine period. Boots should be cleaned before entering bird pens. Owners should understand that contaminated manure can be picked up on footwear at the feed store, coffee shop or a neighbor's place. Using a bleach solution or a commercial disinfectant to spray or dip shoes is recommended. Wearing clean clothes when working with the birds is also recommended.

If you are asked to investigate a flock of poultry or game birds that are experiencing high mortality, Exotic Newcastle Disease should be a consideration. Necropsy lesions may include one or more of the following: fibrinohemorrhagic, diptheritic or necrotic stomatitis, pharyngitis, esophagitis, laryngitis or tracheitis, hemorrhages in the proventriculus, or necrohemorrhagic enteritis. If any of these lesions are detected, pursue additional testing.

Dr. Kevin Varner and the USDA office in Topeka have participated in the federal eradication efforts in southern California and are familiar with the disease. If you suspect an owner has END in his flock, please contact Dr. Varner at (785) 235-2365. END is a federally-reportable disease.

## Continuing Education

July 31 – August 2
Fourth Annual Merck-Merial Veterinary Scholars Symposium

August 12 – 13
Integrating Biosecurity Practices Into Livestock Production Management

October 3 – 4
13th Annual Fall Equine Conference on Equine Practice Management

November 8 – 9
12th Annual Midwestern Exotic Animal Medicine Conference

December 6
12th Annual Emergency Medicine Conference on Opthamology

For the most complete, up-to-date, conference information visit our Web site at: *www.vet.ksu.edu* and click on Continuing Education, or contact: Linda M. Johnson, Ph.D. at 785-532-5696 or *johnson@vet.ksu.edu*

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