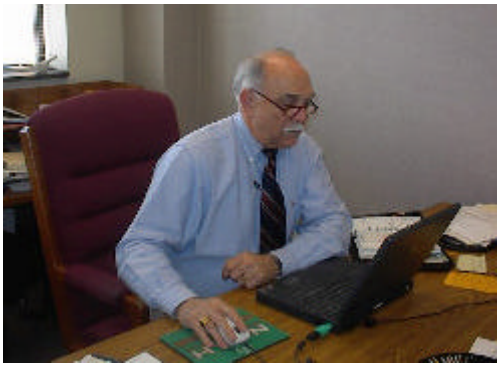
Spring 2001



FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

If anyone involved in an area of work associated with animal health ever has had question regarding the relevance of their efforts, this is a time when such thoughts should be very distant. The prominence of animal health and disease in the world has high visibility in our national news and in our everyday conversations. As veterinarians, we should keep ourselves as informed as possible as to what is going on in various countries of the world regarding diseases that could be a threat to our animal populations and we should be potential sources of accurate and relevant information regarding various diseases and disorders that have potential threat to our human or animal populations. The amount of misinformation and the number of uninformed individuals about some of these animal disease issues is astonishing and, in many instances, appalling.

The prominence of Foot and Mouth Disease (how many times have you heard it referred to as Hoof and Mouth?) and Bovine Spongiform Encephalopathy in the news gives us reason to increase our vigilance to detect either of these disorders should it find its way into our country by whatever means. This issue includes a release from USDA/APHIS regarding FMD that is succinct, pertinent and relevant. We continue to provide assistance to the national surveillance of our animal populations in identifying those national/internationally threatening diseases should they occur here.

We hope that this missive finds you enjoying beautiful spring weather; as I write this there is a light dusting of snow outside that I hope is the last for the year. As always, we look forward to assisting in the maintenance of the health of animals in Indiana. If there are changes we can make in pursuance of our mission, please let us know.

Foot and Mouth Disease

The following bulletin is provided by the USDA and is pertinent considering the ongoing outbreak of Foot and Mouth Disease (FMD) in Europe. Remember that cattle and swine are especially susceptible to FMD virus and that FMD is perhaps the most rapidly contagious disease known. In 1981, it even spread by wind currents across the English Channel from France to England. Because humans can transport FMD virus on clothing, shoes, food, or even by becoming infected themselves, FMD virus has the potential of spreading to the USA. Cattle or swine with an acute febrile disease that includes vesicle-formation and/or ulceration of the gums, tongue, interdigital skin, coronary bands, and teats are suspect. **DO NOT transport animals suspected of having FMD**. Instead, contact the Indiana State Veterinarian or the USDA Veterinarian in charge immediately.

from ***Veterinary Services, March 2001 Fact Sheet***

Foot-and-mouth disease (FMD) is a severe, highly communicable viral disease of cattle and swine. It also affects sheep, goats, deer, and other cloven-hoofed ruminants. FMD is not recognized as a zoonotic disease.

This country has been free of FMD since 1929, when the last of nine U.S. outbreaks was eradicated.

The disease is characterized by fever and blister-like lesions followed by erosions on the tongue and lips, in the mouth, on the teats, and between the hooves. Many affected animals recover, but the disease leaves them debilitated. It causes severe losses in the production of meat and milk.

Because it spreads widely and rapidly and because it has grave economic as well as clinical consequences, FMD is one of the animal diseases that livestock owners dread most.

What Causes It?

The disease is caused by a virus. The virus survives in lymph nodes and bone marrow at neutral pH, but destroyed in muscle when in pH<6.0, i.e., after rigor mortis. The virus can persist in contaminated fodder and the environment for up to one month, depending on the temperature and pH conditions.

There are at least seven separate types and many subtypes of the FMD virus. Immunity to one type does not protect an animal against other types.

How It Spreads

FMD viruses can spread by animals, people, or materials that bring the virus into physical contact with susceptible animals. An outbreak can occur when:

- people wearing contaminated clothes or footwear or using contaminated equipment pass the virus to susceptible animals
- animals carrying the virus are introduced into susceptible herds
- contaminated facilities are used to hold susceptible animals
- contaminated vehicles are used to move susceptible animals
- raw or improperly cooked garbage containing infected meat or animal products is fed to susceptible animals
- susceptible animals are exposed to materials such as hay, feedstuffs, hides, or biologics contaminated with the virus.
- susceptible animals drink common source contaminated water
- a susceptible cow is inseminated by semen from an infected bull.

Signs

Vesicles (blisters) followed by erosions in the mouth or on the feet and the resulting excessive salivating or lameness are the best known signs of the disease. Often blisters may not be observed because they easily rupture, leading to erosions.

Some of these other signs may appear in affected animals during an FMD outbreak.

- Temperatures rise markedly, then usually fall in 2-3 days.
- Ruptured vesicles discharge either clear or cloudy fluid and leave raw, eroded areas surrounded by ragged fragments of loose tissue
- Sticky, foamy, stringy saliva is produced
- Consumption of feed is reduced because of painful tongue and mouth lesions.
- Lameness with reluctance to move is often observed

- Abortions often occur
- Milk flow of infected cows drops abruptly.
- Conception rates may be low

Meat animals do not normally regain lost weight for many months. Recovered cows seldom produce milk at their former rates. FMD can lead to myocarditis (inflammation of the muscular walls of the heart) and death, especially in newborn animals.

Confusion with Other Diseases

FMD can be confused with several similar, but less harmful, diseases such as vesicular stomatitis, bluetongue, bovine viral diarrhea, and foot rot in cattle, vesicular exanthema of swine, and swine vesicular disease. Whenever mouth or feet blisters or other typical signs are observed and reported, laboratory tests must be completed to determine whether the disease causing them is FMD.

Where FMD Occurs

While the disease is widespread around the world, North America, Central America, Australia, New Zealand, Chile and some countries in Europe are considered free of FMD. Various types of FMD virus have been identified in Africa, South, South America, Asia, and part of Europe.

Prevention and Control

FMD is one of the most difficult animal infections to control. Because the disease occurs in many parts of the world, there is always a chance of its accidental introduction into the United States.

Animals and animal byproducts from areas known to be infected are prohibited entry into this country.

Livestock animals in this country are highly susceptible to FMD viruses. If an outbreak occurred in the United States, this disease could spread rapidly to all sections of the country by routine livestock movements unless it was detected early and eradicated immediately.

If FMD were to spread unchecked, the economic impact could reach billions of dollars in the first year alone. Deer and wildlife populations could become infected rapidly and could be a source for re-infection of livestock.

What You Can Do

You can support U.S. efforts against FMD by:

- Watching for excessive salivating, lameness, and other signs of FMD in your herd; and
- Immediately reporting any unusual or suspicious signs of disease to your veterinarian, to State or Federal animal disease control officials, or to your county agricultural agent.

Your participation is vital. Both the early recognition of disease signs and the prompt notification of veterinary officials are essential if eradication is to be carried out successfully. Your warning may prevent FMD from becoming established in the United States or, if it does spread, reduce the time and money needed to wipe it out.

For more information about FMD, contact

USDA, APHIS, Veterinary Services
Emergency Programs
4700 River Road, Unit 41
Riverdale, MD 20737-1231
Telephone: (301) 734-8073
Fax: (301) 734-7817

The APHIS Emergency Operations Center
(800) 940-6524
email: emoc@aphis.usda.gov

In Indiana, contact:

Office of the State Veterinarian
805 Beachway Drive, Suite 50
Indianapolis IN 46224
Telephone (317) 227-0300
Fax (317) 227-0330

USDA, APHIS
Area Veterinarian in Charge
6960 Corporate Drive
Indianapolis IN 46278
Telephone: (317) 290-3300
Fax: (317) 290-3311

FINAL DIAGNOSIS

Skull malformation
Vitamin A deficiency

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

Signalment: A 1-1/2 year old, 280 pound. captive, intact male African lion

Clinical history:

The lion was presented with a history of progressive ataxia and generalized weakness. Bloodwork showed elevated amylase, CPK, canine distemper antibody titers of 1:8 and negative antigen. Initial response to dexamethasone was short-lived. Tensilon test was negative. A muscle biopsy was normal. The lion continued to eat well, with a normal mental status, but remained ataxic. Two months after initial presentation, the lion had an acute onset of recumbency and tonic-clonic rigidity. The owners requested euthanasia.

Necropsy Findings:

The atlanto-occipital joint was immobile due to complete fusion of the atlas to the occipital bone, and had a slight deviation to the left of the spinal axis. The dens of the axis was curved dorsally and to the left, causing pressure on the ventral portions of the cervical spinal cord, which was slightly flattened. The caudal portions of the calvarium were markedly thick, the occipital bone being 3 cm thick and the parietal bone being 2 cm thick. The osseous tentorium cerebelli, normally a thin ossified membrane, was 1 cm thick and 1.5 cm long, forming a hard knob compressing the cerebellum and portions of the cerebrum. The vermis of the cerebellum was coned and protruded into the foramen magnum. Small amounts of blood surrounded the brain stem at the level of the cerebellar herniation.

Histopathology:

Lesions were primarily observed in sections of cranial bones and spinal cord. The bone changes were characterized by marked proliferation of bone, which consisted of increased numbers of loosely spaced, irregular spicules of lamellar and, to a lesser extent, woven bone, and little evidence of bone resorption. Spinal cord changes consisted of Wallerian-type degeneration, which was most prominent in the white matter of the ventral funiculi. The axon sheaths were ballooned and either contained swollen, pale eosinophilic axons, or a few macrophages and no axons. There was multifocal dissecting interstitial astrocytosis in degenerate areas.

Morphologic diagnoses:

1) Atlanto-occipital malformation with ankylosis
2) Hypertrophic osteopathy in occipital bone, parietal bone, and osseous tentorium cerebelli
3) Degenerative myelopathy due to compression

Ancillary tests:

Selenium (0.38 ppm) and vitamin E (20.48 ppm) levels in the liver, and 25-OH-vitamin D3 (nMol/L) levels in the kidney were within the normal reference range for dogs. However, the vitamin A levels in the liver were undetectable (0.0 micrograms/g).

Discussion:

Similar cranial bone lesions have been described in captive African lions in Europe, South Africa, Australia, Israel and Florida. It has been attributed in most cases to vitamin A deficiency, although a genetic or familial basis has

also been suggested. Vitamin A sustains development and differentiation of epithelial structures and bone. Vitamin A stimulates osteoclast activity, causing them to increase acid phosphatase content and resorb bone and, for this reason, excesses and deficiencies of this vitamin have an impact in bone development. Vitamin A excess is characterized by injury to growth cartilage, osteoporosis, and developmental exostosis. In kittens, for example, the occipital bone may be so thin that the cerebellum can be crushed by manual pressure to the overlying skin, and osteophyte formation may lead to a deforming cervical spondylosis. On the other hand, vitamin A deficiency in pigs, cattle, and dogs has been associated with thickening of cranial bones, especially the tentorium cerebelli. The pathogenesis of these skeletal changes is related to altered patterns of drift in bones that are growing during the period of deficiency. Normally, osteoclasts are responsive to vitamin A and, in the cranium of deficient animals, there is inadequate resorption of endosteal bone. Bone is often produced in sites where resorption should be occurring.

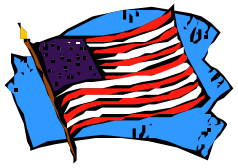
The liver vitamin A levels in this case were undetectable and, in other reports, they ranged from undetectable to 48 micrograms/g of tissue, compared to a value of 1,636 micrograms/g reported as a control in a normal, 2-year-old wild lion. According to the National Research Council of the USA, liver vitamin A levels in fully grown animals should be 300 micrograms/g or greater, and a value of less than 40 micrograms/g should be considered a dietary deficiency. Felids have a high vitamin A requirement compared to other species, and deficiencies are frequently encountered in captive cats. Frequent vitamin A supplementation, or addition of bovine liver (which contains large amounts of vitamin A) to the diet, may be necessary to prevent these abnormalities in lions. However, the nutritional requirements for many zoo animals have not been completely determined and care must be taken to prevent vitamin deficiencies or excesses.

- by Marlon Rebelatto, DVM
ADDL Graduate Student

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

May 28, 2001	Memorial Day
July 4, 2001	Independence Day
September 3, 2001	Labor Day

Please plan accordingly.
No samples can be accepted on these days.



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Proventricular Dilatation Syndrome

Proventricular dilatation syndrome (PDS), also known as splancnic neuropathy, myenteric ganglioneuritis, neuropathic gastric dilatation or "Macaw wasting disease", is characterized by the progressive dilation and dysfunction of the proventriculus of affected psitticines. Affected birds exhibit signs of progressive weight loss, crop stasis with regurgitation, weakness, passage of undigested feedstuffs in the feces, and a lack of response to antibiotic and supportive therapy. Some birds may occasionally exhibit neurological signs such as weakness, ataxia, lameness, and the inability to perch. Proventricular dilatation syndrome occurs most frequently in the cockatoo and macaw, but has been diagnosed in other species of psitticines. There appears to be no sex or age predilection for PDS.

Diagnosis is generally based on exclusion. Proventricular dilatation syndrome can mimic many other diseases such as foreign body or feed impaction, lead or zinc poisoning, gastric neoplasia, proventriculitis, or koilin defects. Hemogram and serum biochemistry profiles are generally unremarkable with PDS; however, radiographs are an excellent diagnostic tool. The proventriculus and ventriculus are often found to be extremely dilated and filled with digesta, gas and fluid. Contrast radiography can also be performed to delineate organs, rule out foreign bodies and assess gastrointestinal transit time, which is prolonged in PDS and is often the first presenting clinical sign.

Histological examination of the proventriculus, ventriculus, crop and autonomic ganglia reveal lymphoplasmacytic infiltration of the nerves, ganglia and neuropil. As the disease progresses, a nonsuppurative encephalitis, myelitis and radiculoneuritis can often be identified and may possibly occur before gastrointestinal signs are noted. Definitive diagnosis is based on histological examination of the proventriculus and gizzard that has lymphoplasmacytic ganglioneuritis. This is often not performed antemorten due to the difficulty of the procedure and the associated postsurgical complications. Recent studies show that approximately 65-75% of affected birds have crop lesions; therefore, full-thickness crop biopsies are considered a worthwhile diagnostic procedure. The crop often shows the characteristic lymphoplasmacytic ganglioneuritis as seen in the proventriculus. It is considered that demonstration of classical lesions in the crop is 100% specific for PDS since no other known diseases cause these lesions and lesions do not occur in the crop without occurring in the proventriculus as well.

The causative agent of PDS is currently unknown. It has been hypothesized that PDS is viral in origin, and recently this has been supported by the isolation of virus-like particles from tissue and fecal samples of affected birds. These include paramyxovirus, adenovirus and an unidentified cytopathic-type virus. The types of histological lesions seen in the

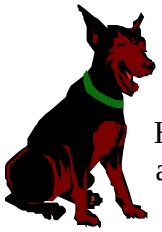
crop, proventriculus and ventriculus point towards an infectious organism as the causative agent.

Treatment is generally unrewarding and the prognosis is poor. Symptomatic treatment with cisapride or metachlopramide and parenteral fluids may increase gastrointestinal transit time and correct hydration. Antifungal and antibacterial agents are often needed to treat organismal overgrowth associated with the gut stasis. Feeding small, regular amounts can decrease the chances of crop and proventricular overload and decrease regurgitation. Duodenal or ventricular feeding tubes which bypass the proventriculus have been used with some success; however, maintenance expense and quality of life often become issues. Owners often elect euthanasia as their bird becomes progressively debilitated.

-by Sasha Burczewski, Class of 2001
-edited by Tsang Long Lin, DVM, PhD, Chief of Avian Pathology

Cooperative Extension Service Fund

The Extension Education and Training Fund, a resource to assist individuals returning to Purdue for advanced degrees in preparation for a career in the Cooperative Extension Service, has been established. Anyone wishing to donate to this fund may do so by writing a check payable to "Purdue Foundation" and noting "Extension Education and Training Fund #704 1140 0012" on the memo line. Any questions may be directed to Floyd Branson, Assistant Director, Cooperative Extension Service, at 765.494.8489.



Vestibular Dysfunction in Old Dogs

Head tilt, nystagmus, rolling and circling are common signs of vestibular dysfunction. It is important to differentiate a central vestibular disease (brain stem vestibular nuclei and floculonodular lobe of the cerebellum) from peripheral vestibular disease (inner ear receptors and vestibular nerve), since the causes and prognoses are different for each disease. Idiopathic peripheral disorders are common in old dogs. These disorders produce an acute onset of severe vestibular signs, but have a good prognosis for recovery. Therefore, it is very important to differentiate these from other vestibular diseases that require specific treatments and could have a poor prognosis.

An acute onset of vestibular signs in an older dog should be examined carefully. Complete history, physical, and neurological examination are keys to diagnosis. The exposure to certain medications is also important. Metronidazole in doses greater than 30 mg/kg could cause signs of central vestibular disease. Prognosis in this case is excellent. The animal usually recovers as soon as the drug is withdrawn.

During the physical and neurological examinations, special attention should be given to the ear canals, tympanic membranes, osseous bullae, pharynx and temporomandibular joints. The cranial nerves examination should be carefully performed since it is often one of the key points in diagnosis.

The first step, after confirming a vestibular dysfunction, is to differentiate

central from peripheral vestibular disease.

Clinical signs of central vestibular disease include horizontal, rotary or vertical nystagmus that may change direction with different positions of the head; hemiparesis, ataxia, ipsilateral postural reaction deficits, tendency to roll in one direction, depression or other changes in mentation, head tremor and hypermetria. Any of cranial nerves V through XII can be affected in a central vestibular disease. Horner's syndrome is not a characteristic of central vestibular dysfunction.

Clinical signs of peripheral vestibular diseases include ipsilateral head tilt, falling, rolling, nystagmus and walking in tight circles. Nystagmus in peripheral vestibular disease always has the quick phase in a direction away from the side of the head tilt. Besides the vestibulocochlear nerve (VIII), the only other nerves affected are the facial (VII) and the sympathetic: Horner's syndrome and facial paralysis are often seen with peripheral vestibular disorder.

Idiopathic peripheral disorders in dogs are characterized by an acute onset of severe head tilt and rolling or falling to the side. During the first 24-48 hours, the animal is often disoriented and may require sedation. After 72 hours, the nystagmus often resolves and the animal is able to ambulate. The head tilt may persist for a couple of weeks or even be permanent. Because this syndrome has erroneously been called "stroke" or brain stem infarct or hemorrhage, a complete neurologic examination should be performed to rule out brain stem involvement. All ancillary procedures including complete blood count and chemistry screen, cerebrospinal fluid analysis, and skull radiographs are normal in idiopathic peripheral vestibular disorders. No lesions are found in the vestibular system on necropsy. The etiology is unknown and there is no specific treatment.

This syndrome usually affects old animals and the initial clinical signs can be severe. Often, affected dogs are euthanized soon after onset of clinical signs due to the mistaken belief that the neurological disorder is irreversible. However, because idiopathic vestibular syndrome carries an excellent prognosis, a complete neurologic examination should be performed in any acutely affected animal with vestibular deficits.

-by Claudia Orso, ECFVG Student
-edited by Karen Tucker-Gillum, DVM, ADDL Graduate student

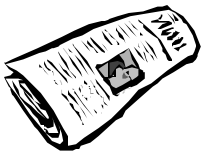
ON THE ROAD

Drs. Randy White and Tariq Qureshi (VPB) attended the annual Whirling Disease Symposium in Salt Lake City, Utah, February, 2001.

Dr. Stephen Hooser attended the Symposium on Reproductive and Developmental Biology and Toxicology in Tucson, Arizona, November, 2000.

Dr. Tom Bryan attended scientific sessions of the United States Poultry Association annual meeting and the International Poultry Trade Show in Atlanta, Georgia, January, 2001.

Dr. Randy White spoke at the Veterinary Medicine in Aquaculture meeting and Midwest Perspective Roundtable in Middleton, Wisconsin, March, 2001.



ADDL STAFF NEWS

We will be saying good-bye to three of our graduate students later this spring as they have completed their programs at ADDL and will move on to new challenges.

From left to right, they are

- **Dr. Victoria Owiredu-Laast** who will begin a PhD program at Johns Hopkins Hospital In Baltimore, Maryland.
- **Dr. Lavun Anonthayanontha** who has accepted a position at Antech in Phoenix, Arizona
- **Dr. Jan Lacey** who has accepted a position as diagnostic pathologist at Texas A&M University, College Station, Texas.

Please join us in wishing them well.

Ellen Stevenson, daughter of **Dr. Greg Stevenson**, ADDL pathologist, was recently named Tippecanoe County's Junior Miss. She was crowned by last year's winner **Mindy Thacker**, daughter of ADDL Director **Leon Thacker.**

Editor's note: This is the first of a two-part article that is being reprinted with the written permission from the *Compendium on Continuing Education For the Practicing Veterinarian.* This article was originally printed in this journal in November, 2000, Vol 22 (11), pages S160-166

Performing Diagnostic Procedures on Salmonid Fishes

by

Melvin Randall White, DVM, PhD

Aquaculture is a rapidly developing agribusiness. The culture and harvest of salmonids by private industries for food consumption and exportation represent a $79 million industry in the United States.[1] This article provides guidelines on how to perform diagnostic techniques and properly collect tissue samples from salmonids. In addition, various diagnostic techniques can help distinguish disease processes unique to salmonids.

DISEASE EVALUATION

Obtaining a Disease History

Important information can be learned about an aquaculture facility by asking the proper questions while compiling a disease history. Although many questions may be directly related to a particular disease, basic information regarding the facility's operating procedures should be obtained to reveal possible environmental concerns or problems that are personnel related.

- When was the current disease or problem noticed, and what steps have been taken to correct it?

- What clinical signs have been noticed?
- When were new fish last introduced to the system?
- What changes, if any, have occurred with respect to nutrition?
- What changes, if any, have occurred with respect to water quality?
- What changes, if any, have occurred with respect to the physical environment (e.g., tanks, ponds, aeration equipment, feeders)?
- Have there been any personnel changes?

Environmental Criteria

Water quality is the most important environmental parameter for salmonid producers. Environmental abnormalities can be categorized into acute and chronic problems. With acute water problems, at least one of the parameters is severely affected and poses a life-threatening condition to the fish; high mortality will result if the situation is not corrected immediately. A chronic water problem acts as a stressor and not as the initiating cause of mortality.

Although water-quality parameters vary according to environment and geographic locality, several guidelines are available.[2-4] Dissolved oxygen (O_2), temperature, ammonia, and nitrite should be monitored at least once or twice daily. The dissolved O_2 of the water must be evaluated onsite using a portable O_2 meter or a commercially available test kit. Parameters that should be evaluated at least once a week include pH, hardness, and alkalinity.

In ponds, dissolved O_2 is a critical water-quality parameter. Usually ponds are aerated, but they also depend on the photosynthetic activity of aquatic plants and algae to produce dissolved O_2. The respiration of these plants consumes dissolved O_2, however, potentially resulting in an "O_2 debt." In artificial aquatic settings (e.g., aquaculture raceways), O_2 is added by using mechanical means to agitate the water or by "bubbling" atmospheric gases or purified O_2 into the water. The loss of stratification of ponds or an acute algal bloom die-off can cause the dissolved O_2 content of pond water to become acutely lowered. A vicious cycle occurs whereby the decaying plant life consumes O_2 and at the same time the decreased viable algal mass cannot produce as much O_2. In aquatic environments in which mechanical aerators or agitators are used to provide dissolved O_2 to the water, equipment failure and/or power outages are common causes of acutely decreased dissolved O_2.

Water in ponds commonly stratifies in the late spring and early summer because of temperature fluctuations and the difference between the density of warm and cold water. As the water temperature increases, and in the absence of water agitation, the pond stratifies by forming an upper layer of warm water (epilimnion) and a deeper layer of cold water (hypolimnion). Loss of stratification by mechanical agitation or from a severe thunderstorm often results in rapid O_2 depletion. A dilutional effect results as the large volume of O_2-poor water in the hypolimnion mixes with the O_2-rich surface waters, thereby decreasing the total dissolved O_2 content of the pond.

For salmonids raised in raceways, O_2 may be added to the aquatic environment when water flows over splashboards. Raceways are rectangular-shaped tanks; water is commonly added to the tank at one end and drained at the opposite end. The raceways are usually sloped to enable

water to flow over the splashboards, thus creating enough turbulence to increase the dissolved O_2 content. Mechanical failure with the splashboards or the fouling of these devices with extraneous or excessive amounts of organic material can lead to decreased dissolved O_2 content.

Ammonia is excreted by both fish and plants[2] and rapidly metabolized into nitrite by *Nitrosomonas* bacteria. *Nitrobacter* species then converts the nitrite to nitrate. Of the three compounds, nitrate is the least toxic to fish. Ammonia is present in the water in ionized (NH^+_4) and un-ionized (NH_3) forms. The ratio of ionized and un-ionized ammonia is pH dependent; more acidic water favors the less toxic ionized ammonia, whereas basic water favors the more toxic un-ionized ammonia. Acute ammonia toxicity can occur from a sudden die-off of fish or plants or if a large amount of fish food is inadvertently introduced into the system. In these situations, considerable decomposing protein is released into the water, resulting in a concomitant increase of ammonia followed by nitrite.

Acute ammonia toxicity often occurs when fish are introduced into a new water system with inadequate amounts of *Nitrosomonas* bacteria to convert the ammonia to nitrite. In the aquaculture industry, this is known as new tank syndrome. Fish can also be affected by acute ammonia toxicity with a sudden die-off of *Nitrosomonas* bacteria in the biofilter of recirculating systems if chemicals that kill these bacteria are introduced into the water system. Although salmonids were not raised in recirculating systems in the past, these systems are now being used successfully.

The clinical signs of acute ammonia toxicity are nondiagnostic; therefore, careful monitoring of the water quality is needed to prevent it. Acute ammonia toxicity results in systemic acidosis. Although ammonia may act as a false neurotransmitter, ammonia toxicity primarily results from inhibition of the citric acid cycle caused by the blockage of oxaloacetate with resultant anaerobic glycolysis.[5]

Nitrite is another important water-quality parameter that must be constantly monitored. Acute nitrite toxicosis has been called *brown blood disease* because of the rapid oxidation of hemoglobin, which occurs when nitrite diffuses across the gill epithelium of fish. This oxidation results in methemoglobin formation, which causes brown discoloration of the blood.[2] Although this condition is observed in catfish raised for production, it is not a common problem in salmonids.

The pH as well as the hardness and alkalinity of water should be monitored periodically to denote trends or changes in parameters that may result in chronic water problems. The pH of water is the measurement of the hydrogen ion content expressed as the negative logarithm of the hydrogen ion concentration; thus the pH measures the acidity or alkalinity of water. The alkalinity is a determination of the buffering capacity of water as measured by the amount of bicarbonate (HCO^-_3) and/or carbonate (CO^-_3) in milligrams per liter (mg/l) in the water. The hardness of water is the measurement of divalent metal cations (e.g., calcium, iron, zinc, magnesium). The majority of the cations are calcium. Chronic poor water quality reportedly can diminish the growth, feed efficiency, and feed conversion rates of fish.

(Part two of this article, Diagnostic Techniques and Summary, will be concluded in the next newsletter.)

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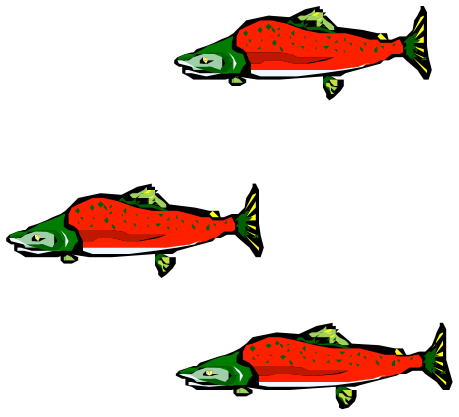

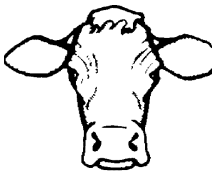

Bovine Neosporosis: A Review

In the past decade, the protozoan parasite *Neospora caninum* has become increasingly recognized as an important cause of reproductive failure in dairy and beef cattle. The parasite is widespread in the United States, but the majority of case reports have come from California, where *Neospora caninum* is considered to be the number one cause of abortion in dairy cattle. In Indiana, neosporosis has been sporadically reported as a cause of abortion and neonatal mortality in cattle; however, the current seroprevalence of *Neospora caninum* in Indiana cattle is unknown.

Neospora caninum is a protozoan parasite which closely resembles *Toxoplasma gondii.* Wild or domestic canids are the natural definitive host for *Neospora caninum*. Infected animals may shed large numbers of oocysts in their feces, which may then be ingested by intermediate hosts such as cattle. Sheep, goats, horses, deer, and other animals are also suitable intermediate hosts. There is no direct transmission between cows; however, the parasite can be maintained by vertical transmission of the organism from the dam to the fetus in utero. Vertical transmission, which may occur over several generations, is a major factor contributing to the persistence of *Neospora* to their offspring. At birth, congenitally infected calves may have neurologic signs, be underweight, unable to rise, or have no clinical signs. Congenitally infected calves have a higher rate of abortion, particularly during their first pregnancy, and a high rate of vertical transmission to their offspring.

Neospora abortion typically occurs in mid gestation with a mean age of 5.5 months (range 3.5-8 months) and may occur throughout the year. This is different from most causes of bovine abortion which occur during late gestation. Abortion storms may occur with multiple abortions over a period of 1-2 months. The aborted fetuses are typically autolyzed and usually do not have characteristic gross lesions. If present, lesions are subtle, consisting of pale white foci in the skeletal muscle and heart, and there may be fluid in the pleural or peritoneal cavities. The dams typically do not have clinical signs.

Neosporosis can be diagnosed by serology and/or histopathology. One or more aborted fetuses, the placenta, and serum from the dam should be submitted to the Animal Disease Diagnostic Laboratory at Purdue University. Important organs from which to identify the organism via histopathology and fluorescent antibody (FA) testing include the brain, heart, liver, and skeletal muscle. The most characteristic lesion in the fetus is focal encephalitis, characterized by necrosis and nonsuppurative inflammation. Although most aborted fetuses are autolyzed, *Neospora* organisms and/or the characteristic lesions may be found in the brain. The brain should be fixed in 10% formalin and submitted for histopathology. A herd diagnosis can be achieved by serology. By comparing the *Neospora* status of aborting versus nonaborting cows, it will become clear if seropositivity correlates with the tendency to abort. During abortion storms, it is best to collect blood immediately from all animals at risk and then post abortion blood samples should be taken later. This technique is better for evaluating seroconversion to any abortifacient than paired serology, using samples collected at abortion and 3 weeks later, because most abortions occur several weeks after acute infection.

There is no effective treatment for *Neospora* infection in cattle. Research is being conducted to develop a vaccine to help prevent abortion in cattle; however, none are commercially available at this time. Culling seropositive cattle and their infected offspring has been advocated for the control of Neosporosis in herds with a low prevalence of infection. However, this approach would not be cost effective in high prevalence herds. Control may be attained by using seronegative animals as replacement heifers. Valuable seropositive cows can be used as breeding stock by embryo transfer to seronegative recipients. Since dogs are the natural definitive host for *Neospora caninum*, control programs should start with preventing access by dogs to infectious material such as dead calves, aborted fetuses, stillborn calves, and fetal membranes. Furthermore, cattle feed and water should be protected from contamination by dog feces.

- by Scott Storandt, Class of 2001
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