Winter 1998

FROM THE ASSISTANT DIRECTOR

Effective January 1, 1999, Dr. William VanAlstine will be assuming much of the leadership role as Assistant Director of the Animal Disease Diagnostic Laboratory. This change in leadership is necessary to allow me the time and opportunities necessary to fulfill my obligations as an Associate Professor of Veterinary Pathobiology.

I am very thankful to Drs. Thacker and Baumgardt, Deans Rebar and Lechtenburg for the opportunity to serve as a part of the administration of ADDL for the past 2 1/2 years. This has been a tremendously positive experience for me and I have thoroughly enjoyed the opportunity to serve the members of ADDL as well as our clients, including practicing veterinarians, animal owners and producers and the many citizens of the state of Indiana.

Dr. VanAlstine is a Diplomate of the ACVP who received his DVM degree from the University of Missouri in 1981 and his PhD degree from the Iowa State University in 1987. He is currently an Associate Professor in the Department of Veterinary Pathobiology, the Chief of Pathology Services of ADDL, as well as the Chairman of the Pathology Section in the Department of Veterinary Pathobiology. He is also a recent past-president of the American Association of Veterinary Laboratory Diagnosticians. He has been a faculty member here since 1987 and is well aware of the operations of this laboratory as well as the current issues facing ADDL.

I am certain Dr. VanAlstine will do an excellent job in maintaining the leadership of ADDL. I hope you will join me in welcoming Dr. VanAlstine to this position and give him all of your support.

As always, let us hear from you. We hope you enjoy this newsletter. Please notice that this newsletter contains our first index in the back. The idea of this index was derived from a reader's suggestion.

Equine Herpesvirus 1 Myeloencephalopathy

Multifocal hemorrhagic myeloencephalopathy with leptomeningeal vasculitis can be caused by Equine Herpesvirus Type 1 (EHV-1) and, although outbreaks are still rare, the frequency of occurrence has been increasing. The virus has a worldwide distribution and outbreaks can have up to 40% morbidity and 90% mortality.

There are four distinct herpesviruses known to cause disease in horses; EHV-1 (equine abortion virus), EHV-2 (equine cytomegalovirus), EHV-3 (equine coital exanthema virus) and EHV-4 (equine rhinopneumonitis virus). It was once thought that EHV-1 and 4 were a single virus able to produce early neonatal death, abortive, respiratory, and neurologic disease. However, it has recently been shown that there are two distinct viruses with only 20% corresponding DNA sequences and substantial antigenic site differences. Transmission of the virus occurs by direct contact, inhalation of contaminated aerosol secretions, and fomites. It can maintain environmental infectiveness for up to 14 days, and up to 42 days on suitable vectors, such as horse hair. The predisposition for the outbreak of the neurologic form of EHV-1 infection is not well understood, but often presents in association with outbreaks of abortion and respiratory disease. While any animal is capable of contracting the disease, pregnant mares in the first two trimesters of gestation seem to have an increased risk. After replicating in the respiratory epithelium and disseminating to lymphoid tissue, the virus is spread systemically via infected monocytes and lymphocytes. In the short duration of viremia, replication occurs in vascular endothelial cells. The vasculitis of arterioles in the brain and spinal cord are responsible for the clinical signs, which are often an acute onset of symmetrical ataxia and paresis. Neurologic signs progress rapidly for about 2 days, then stabilize. Because the lesions of the spinal cord are more likely to cause clinical signs, neurologic deficits are more predominant in the hindlimbs with many cases showing cauda equina syndrome (urinary incontinence, hypotonia of tail and anus).

A current theory surrounding EHV-1 myeloencephalopathy is the belief that the disease may be due to immune-complex induced vasculitis rather than direct viral infection. This is supported by the resemblance of the characteristic vascular lesions to type III hypersensitivity, and the presence of immune complexes correlating to high levels of antibody at the onset of clinical signs. This theory is by no means absolute, as various flaws exist in attempts to prove it experimentally.

Antemortem diagnosis of EHV-1 myeloencephalopathy is often unsuccessful. Cerebrospinal fluid evaluation often reveals xanthochromia (due to denatured heme pigment), elevated protein (>80 mg/dl, and as high as 300 mg/dl), yet a normal nucleated cell count (<8 cells/ul). It should be noted that EHV-1 can alter the blood brain barrier and when serum concentration of equine protozoal myeloencephalitis (EPM) antibody is high there may be a passage of EPM antibodies into the CSF, causing a horse to falsely test positive to EPM. IgG index and albumin quotient assessments can be used to detect such invalid diagnoses. Isolation of the virus from CSF can be difficult due to the short viremic stage, and isolation from other tissues such as blood (collected with sodium citrate) or respiratory tract (collected by nasopharyngeal swab) may be helpful. Polymerase chain reaction (PCR) technique of tissue samples shows promise for increased sensitivity for diagnosis compared to virus isolation. Paired serum virus antibody titer taken at onset and 2-3 weeks later is diagnostic with a fourfold or greater increase. Also, a single serum titer of approximately 1:400 may be indicative of the disease. Postmortem histopathology of horses suspected of EHV-1 myeloencephalopathy should include brain, spinal cord, spleen, thyroid and lung. Again, virus isolation is often unsuccessful. Immunofluorescent antibody testing of brain and spinal cord can be more sensitive, but

false-negatives have occurred. Another promising technique is indirect immuno-peroxidase method using light microscopy of various tissues of affected animals.

Since there is no specific antiviral treatment, supportive care is the main therapy. Because of the suspected immune-mediated component, corticosteroids may be helpful as well as non-steroidal anti-inflammatories. Precaution should be used when cauda equina syndrome is present to prevent a ruptured bladder or constipation due to eliminatory dysfunction. Rectal examination, fecal softeners and urinary catheterization may be indicated. Prognosis corresponds to clinical signs, and progression to recumbency usually has a high mortality rate with survivors taking months to years to recover. If recumbency does not present in the first 3-5 days, prognosis is improved, but recovery may take several months with no guarantee of return to normal gait or eliminatory functions.

- by Lisa Wisz, Class of 1999
- edited by Jason Baldwin, DVM

Canine Mast Cell Tumors

Mast cell tumors comprise 20-25% of all cutaneous and subcutaneous tumors in dogs, making them the most prevalent canine skin tumor. Mast cell tumors may occur in the intestines, liver, spleen, and bone marrow at a much lower prevalence. Mast cell tumors most frequently develop in dogs at an average age of eight years.

The clinical signs of mast cell tumors are variable and dependent on the location and grade of the tumor. Mast cell tumors can be found on all areas of the skin. Mast cell tumors most commonly appear as small raised nodular masses that vary from firm to solid on palpation, but no typical morphology of mast cell tumors exists. Mast cell tumors can imitate the appearance of most other skin tumors, making diagnosis based on physical exam difficult. Most mast cells are solitary tumors, but approximately 10% are multicentric or infiltrative.

Other clinical signs of mast cell tumors relate to the release of mediators stored in their intracellular granules or cytoplasm. These mediators include histamine, heparin, proteolytic enzymes, and other cytokines. The most common sign is gastric ulceration via histamine, which can manifest as vomiting, diarrhea, or anemia. Similarly, local ulceration near the cutaneous neoplasm may develop as a result of inflammation and edema. Delayed wound healing may be caused by the proteolytic enzymes and the activation of macrophages via histamine to release fibroblastic suppressor factor. In addition, coagulation abnormalities due to heparin release or hypotensive shock due to a massive histamine release may occur.

A diagnosis of a mast cell tumor can be accomplished through cytological examination of a fine needle aspirate of the tumor. Cytology of a typical mast cell tumor will reveal a large number of discrete round cells with abundant, small, uniform, basophilic cytoplasmic granules located intracellularly and extracellulary. The granules may stain poorly with Diff Quick® stains. The nucleus will appear round to oval, but may be hidden in heavily granulated cells. Mast cells can vary greatly in appearance, and some tumors may contain agranular mast cells. Varying numbers of eosinophils and neutrophils may also be scattered throughout the smear.

Preliminary staging of the tumor should include palpation of the local lymph nodes, which are the most common sites of metastasis. The liver and spleen should also be palpated because hepatomegaly and splenomegaly are common with disseminated mast cell neoplasia. Cytology of the regional lymph nodes and bone marrow, as well as an enlarged liver or spleen, can reveal increased numbers of mast cells

signifying metastasis. Greater than 10 mast cells per 1000 nucleated cells in the bone marrow is indicative of neoplastic infiltration. .

The preferred treatment for a mast cell tumor confined to the dermis with no nodal involvement is complete excision with a wide margin of at least 3 cm. Although most tumors palpate as discrete masses, most mast cell tumors are not discrete but surrounded by small numbers of neoplastic cells. Histologic examination of the tissue removed is imperative to confirm the diagnosis, grade the tumor, and evaluate completeness of excision. If histology reveals a poor margin of excision, a second aggressive surgery is the treatment of choice. If a proper margin of excision cannot be obtained, treatment usually involves surgery to remove as much of the tumor as possible followed by radiation or chemotherapy. No protocol has been agreed upon or proven to be markedly effective for malignant tumors, multiple tumors, and tumors that cannot be excised. A treatment plan for these tumors usually involves corticosteroids as well as chemotherapeutic drugs and radiation therapy. Palliative treatment with cimetidine and histamine blockers can help prevent gastric ulceration and some of the other secondary effects of the tumor.

Mast cell tumors are graded histologically based upon the number of granules, mitotic index, and cellular characteristics of malignancy. The grading scale divides mast cells into three groups. The distribution within these groups is: 40% differentiated, 40% intermediately differentiated, and 20% undifferentiated. Prognosis is excellent for a solitary well differentiated tumor that can be easily excised with good surgical margins.

- by Matt Renninger, Class of 1999
- edited by Janice Lacey, DVM

Feline Heartworm Disease

The first reported case of *Dirofilaria immitis* in a cat was in 1922 in Virginia. This cat had 2 adult worms in the right ventricle. Research into feline heartworm disease has grown significantly since then, and is recognized as a potentially life threatening disease.

Feline heartworm disease is very important clinically because even a light infection is capable of producing severe, life threatening disease. The regional prevalence of heartworm infections in domestic cats tends to parallel canine heartworm infections but at a lower rate. Cats are less easily infected by heartworms. It has been shown that dogs are the preferred host and there are indications that cats are not the ideal hosts.

The life span of the parasite is shorter in cats and infections tend to be self-limiting after two to three years. Cats also have a greater tendency to spontaneously eliminate the infection or die from the infection. Another reason cats are proven not to be ideal hosts is the aberrant migration of fourth stage larvae which is more likely to occur in cats than in dogs. Large numbers of ectopic heartworms have been found in the body cavities and central nervous systems of cats.

Other important differences in feline heartworm disease exist. Unlike dogs, circulating microfilariae are seldom found in cats. If found in cats, the average time needed for the worms to produce the larvae is 8 months post-infection with microfilariae persisting for one month. Cats generally cannot tolerate as many adult heartworms as dogs. Dogs can potentially maintain a burden of several hundred worms whereas many cats can die from fewer than 10 worms.

The lesions caused by heartworms is slightly different in cats than in dogs. While cats can develop interstitial lung disease like dogs, there is also an extensive alveolar type II cell hyperplasia in cats. These parenchymal lesions may have an important role in the pathogenesis of acute respiratory distress 4-9

months post-infection. It is thought that cats mount a more intense immune and inflammatory response to the adult heartworms which coincides with the pathogenesis.

Clinical signs are frequently non-specific and could correlate with many other disease processes. The most common chronic signs are tachypnea, coughing, and anorexia. Abnormal lung sounds may be heard but heart mumurs are rare. Intermittent vomiting that is not associated with eating is another common sign. There is also a peracute syndrome that can be seen with feline heartworm disease. The signs associated with this may include acute respiratory distress, ataxia, collapse, seizures, hemoptysis, and sometimes sudden death.

Diagnostic testing for cats is more difficult than in dogs. Microfilariae can be detected with the modified Knott's test, however since cats only have microfilariae for a short time, a negative test does not necessarily exclude the diagnosis of heartworms. Reasons why the results of an ELISA antigen test may produce a false negative result include: immaturity of parasites, too few female worms to produce detectable levels of antigen, or infection by solely male worms.

Thoracic radiographs may help confirm a positive heartworm test. Radiographic evidence of feline heartworm disease includes enlargement of lobar and peripheral branches of pulmonary artery. This sign may be limited to the right caudal lobar artery where heartworms are most commonly found. Unlike dogs, right-sided cardiomegaly is seldom seen. There can be main pulmonary artery enlargement in cats but it is obscured by the cardiac silhouette. Radiology along with angiography and echocardiography are primarily used to confirm a tentative diagnosis of feline heartworm disease rather than as screening tools.

Treatment of feline heartworm disease must be based on the clinical signs of the individual cat, not solely on the basis of a positive Knotts or antigen test. If the cat displays no overt signs of heartworm disease it is best to allow time for spontaneous elimination of the parasites. These cases can be monitored every 6 to 12 months for worsening of radiographic signs.

Infected cats with radiographic signs of pulmonary interstitial lung disease may benefit from a diminishing dose of prednisone beginning with a dose of 2 mg/kg per day and gradually reducing it to 0.5 mg/kg every other day by two weeks and then discontinuing after an additional two weeks. At the end of treatment the cat should be assessed radiographically. This treatment may be repeated periodically in cats with recurrent respiratory signs.

In cats that are not controlled by the above regime and are stable clinically, adulticide treatment could be initiated with this acetarsamide at 2.2 mg/kg intravenously twice a day for 2 days. Post treatment cats should be in caged confinement and under close observation for 3 to 4 weeks. Owners should be warned that a possible side effect of treatment is potentially fatal pulmonary thromboembolism.

As always, prevention is the best method of controlling this potentially fatal disease in cats. Chemoprophylaxis with a monthly dose of ivermectin at 24 mg/kg is recommended to cat owners. Even though the heartworm antigen test sensitivity is low, it is good medical practice to test for feline heartworm disease before giving chemoprophylaxis for the first time, if at least 8 months have passed since there was an opportunity for infection.

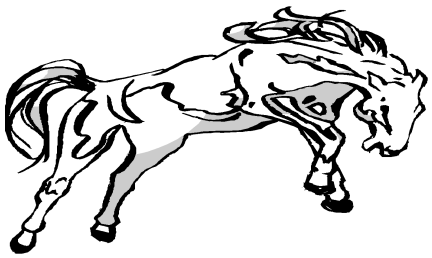
- by Jennifer Keenan, Class of 1999
- edited by Victoria Owiredu-Laast, DVM

Vesicular Stomatitis in the Horse

Vesicular stomatitis is a contagious disease caused by a rhabdovirus. The two major serotypes are New Jersey and Indiana. Aside from horses, the virus may affect swine, cattle, goats, sheep, llamas, wild animals and humans. The lesions include vesicles up to 2 cm in diameter on the mouth and lips and less frequently on the hooves, prepuce and teats. These vesicles quickly rupture, leaving large painful erosions and ulcers. Horses may display ptyalism, pyrexia and anorexia. The rhabdovirus is believed to be spread via insect bite and direct contact, and may have morbidity of 5-10% in affected herds. Incubation time is 1-3 days and the course of the disease is 1-2 weeks, with solid immunity possible for up to 6 months. Diagnosis of vesicular stomatitis is based on history, signs, serology and virus isolation. Histopathology reveals non-specific hyperplasia and edema (intra- and intercellular) of the epidermis, reticular degeneration, spongiotic microvesicles, focal necrosis and superficial and deep perivascular dermatitis. Due to the rapid course of the disease, treatment is usually not indicated. However, mild septic mouthwashes and softened pelleted feed may be used as palliative treatment for the painful ulcers. Of high clinical significance is its similarity to foot-and-mouth disease, thus making vesicular stomatitis reportable.

NOTE: At the time this article was written, areas in Colorado were still under quarantine for vesicular stomatitis in horses.

- by Lisa Wisz, Class of 1999
- edited by Jason Baldwin, DVM



Serpulina pilosicoli: What We Know and What We Do Not

What We Know

The organism - There are 5 distinct *Serpulina sp*. known to infect swine. Two species are pathogenic. *Serpulina hyodysenteriae* (formerly *Treponema hyodysenteriae*) causes swine dysentery. *Serpulina pilosicoli* (formerly *Anguillina coli*) causes intestinal spirochetosis. Three additional species are nonpathogenic i.e. *Serpulina innocens* (formerly *Treponema innocens*), *intermedia* and *murdochii*. Some strains of *S. intermedia* have been associated with diarrhea; however, inoculation studies in pigs have not consistently reproduced disease.

Serpulina pilosicoli can be differentiated from other *Serpulina sp*. by PCR or by culture (weak β-hemolysis) followed by biochemical testing, i.e. indole negative, hippurate hydrolysis positive and lack of β-glucosidase activity in the API-ZYM profile. BJ medium that is most commonly used to culture *S. hyodysenteriae* in diagnostic laboratories is slightly inhibitory when used for isolation of *S. pilosicoli* due to the moderate sensitivity of *S. pilosicoli* to 2 of the included antibiotics, i.e. rifampicin and spiramycin. Culture of *S. pilosicoli* is most sensitive with a modified BJ media that does not contain rifampicin or spiramycin. Currently, the most sensitive diagnostic protocol involves culture on modified BJ media, followed by screening of suspect colonies using the hippurate hydroysis test and confirmation of positives by PCR testing.

In addition to swine, *S. pilosicoli* also infects humans, non-human primates, dogs and several species of birds. Strains of *S. pilosicoli* can colonize laboratory mice with fecal shedding for up to 30 days, suggesting the potential for rodents to act as resevoirs of infection for swine. Likewise, birds, dogs and humans are also potential resevoirs for swine. The pathogenic potential of swine strains of *S. pilosicoli* for humans is unknown, but zoonotic potential exists.

The disease - Intestinal spirochetosis is a non-fatal large intestinal disease caused by *S. pilosicoli* that has been described in field studies of affected swine herds and in inoculation studies in which disease was reproduced.

Clinical disease occurs in weaned pigs primarily 8-16 weeks of age, usually commencing 7-14 days after moving and mixing of pigs. This is consistent with the reported incubation period in inoculation studies of 3-16 days. Typically, 5-15% of pigs are affected and affected individuals exhibit diarrhea and poor growth for 2-3 weeks. Some pigs may develop chronic diarrhea and exhibit poor growth for longer periods of time. Clinical signs typically are present in a group of pigs for 3-6 weeks. Affected individuals may require up to 28 additional days to reach a slaughter weight of 210 pounds. Mortality rarely exceeds 1-2%. Economic loss is primarily due to poor feed conversions and effects of uneven growth rates on pig-flows and market uniformity. The diarrheic feces are usually first soft and wet ("wet cement" consistency) and then later change to a watery consistency and are gray to brown with a small amount of mucous ("oily" sheen). During recovery or in chronic cases, feces may contain thick tags of mucous. Occasionally, flecks of blood may also be present. Affected pigs generally remain alert and active, but appetite is depressed and pigs may show abdominal discomfort and/or may appear gaunt and develop rough hair coats.

Gross lesions of intestinal spirochetosis are usually subtle. Pigs are variably gaunt and have rough hair coats. The spiral colon is flaccid, enlarged and contains abundant watery content with variable amounts of mucous and occasionally some blood. The colonic mesentery and serosa may be thickened by edema in acute cases and the serosa may be thickened by fibrin or fibrous connective tissue in chronic cases (serositis). Colonic lymph nodes are sometimes enlarged. Mucosal lesions are most common and severe in the mid-spiral regions of the spiral colon followed by the proximal spiral colon. The cecal mucosa is either not involved or has mild lesions. The colonic and cecal mucosa in affected areas may be congested (reddened) and thickened by edema fluid forming prominent ridges. Mucosal erosions occur in variable numbers. With few erosions, the mucosa appears relatively normal (glistening) with a few scattered adherent feed particles. With many erosions, the mucosa appears granular and fibrin exudation admixed with necrotic cellular debris may result in multifocal fibrinonecrotic tags or plaques. Variable amounts of mucous, and occasionally blood, may be in contents and on the mucosal surface. Mucosal lesions are mild compared to classic lesions of swine dysentery or salmonellosis. To see small erosions, i.e. adherent feed particles or small areas of fibrinonecrotic debris, the mucosa should be gently washed free of contents with flowing water. Scraping contents from the mucosa with a knife should be avoided since this will often destroy many of the mucosal lesions and alter some microscopic lesions.

Microscopically, there is a mild to moderately severe multifocal to diffuse superficial erosive colitis. A variable amount of fibrinonecrotic debris is on the luminal surface in areas of erosion. The mucosa is variably thickened by an increased depth of crypts (crypt hyperplasia), edema of the lamina propria and increased numbers of lymphocytes and plasma cells in the lamina propria and, to a lesser degree, the submucosa. Goblet cell hyperplasia is common and may cause distention of crypts with mucous. A lesions unique to *S. pilosicoli* is end-on attachment of the bacterial cells to the apical margin of mature epithelial cells on the colonic luminal surface creating a "false brush border" appearance. Unfortunately, this lesion is present only in the early stages of infection and is thus present in a minority of diagnostic cases and cannot be used as a reliable diagnostic tool. Large serpentine spirochetes typical of *Serpulina sp.* are more commonly present admixed with other bacteria in adherent fibrinonecrotic debris, in the superficial lamina propria and in the crypts. Unfortunately, *S. pilosicoli* cannot be differentiated from other

Serpulina sp. based on morphology at a light microscopic level. Apart from the unique, but inconsistent, lesion of end-on attachment by S. pilosicoli, the microscopic lesions of intestinal spirochetosis are relatively nonspecific and can be mimicked by lesions of salmonellosis, mild swine dysentery or allergy to certain types of pelleted diets.

Knowledge of the epidemiology/ economics of intestinal spirochetosis is limited, based on few inoculation studies and field epidemiologic studies. Infection with *S. pilosicoli* has been reported in swine in nearly every country with a significant swine industry. The proportion of infected swine herds in the U.S. is unknown. In a limited study of diarrheic pigs on 10 grower sites in a single U.S. swine production company, *S. pilosicoli* was isolated in 50%. In a study of 85 swine herds with a history of colitis in the U.K., *S. pilosicoli* was detected in 52% and was the sole pathogen detected in 33%. In Finland, one study of 894 farms demonstrated *S. pilosicoli* in 18% and in another study of 50 finishing sites that were stocked from "LSO 2000 quality chain" health-status farrowing sites, *S. pilosicoli* was detected in 28%. Transmission of *S. pilosicoli* is considered to be exclusively fecal-oral. The greatest risk factor for infection of negative pigs is exposure to fresh feces from shedding carrier pigs. *Serpulina pilosicoli*, like *S. hyodysteriae*, survives in anaerobic lagoons and in moist fecal matter; hence open flush gutters, inadequate cleaning of contaminated pens/facilities, contaminated truck/trailers, etc. are all significant risk factors for infection. Other species known to sometimes carry *S. pilosicoli*, including humans, dogs, birds, and possibly mice may pose some biosecurity risk to negative herds. Bird-proofing of buildings and rodent control are recommended as prudent preventative measures.

Not all pigs that are infected with *S. pilosicoli* develop diarrhea. In oral inoculation studies, nearly all pigs become colonized by *S. pilosicoli*, but only 1/3-2/3 develop diarrhea. Pigs remain colonized and shed *S. pilosicoli* in feces for up to 6 weeks. Pelleted feed increases the risk of diarrhea in *S. pilosicoli* infected pigs. When the ration is changed from pellets to meal, the proportion of diarrheic pigs decreases.

Diagnosis - A definitive diagnosis of intestinal spirochetosis requires the demonstration of typical colonic lesions, confirmation of infection by *S. pilosicoli* and elimination of other causes of colitis in swine. Other diseases that should be excluded by testing are salmonellosis, swine dysentery, proliferative enteritis, whipworm infestation, and possibly yersiniosis caused by *Yersinia pseudotuberculosis*. Tests available for detection of infection with *S. pilosicoli* include culture and/or PCR. Fecal samples may be used to confirm infection and predict prevalence in populations; however, sensitivity for both tests is not as high in fecal samples as when testing is on colonic mucosal samples. Duhamel estimated that the sensitivity of fecal culture for *S. pilosicoli* is approximately 80% under ideal conditions, i.e. shedding large numbers of organisms with no antibiotics in the feed. Under most field conditions, the sensitivity would probably be lower. Fecal samples should be collected on swabs and immersed in Ames transport medium with activated charcoal, chilled and shipped overnight to the laboratory for testing. *In-situ* hybridization testing for *S. pilosicoli* done on formalin-fixed sections of colon has been described experimentally as a sensitive method for the diagnosis of intestinal spirochetosis, but is not yet available in diagnostic laboratories in the U.S.

Treatment - *Serpulina pilosicoli* is generally sensitive to the same antibiotics as is *S. hyodysteriae* and variable clinical response of intestinal spirochetosis to treatment is described. For 19 U.S. strains, all were susceptible to carbadox and tiamulin, 47% were susceptible to gentamycin and 42% were susceptible to lincomycin. Most schemes for control of intestinal spirochetosis combine therapeutic levels of antimicrobials during the first few weeks in grower buildings with sanitation, i.e. cleaning and disinfecting pens/buildings between groups of pigs. It is assumed, but not proven, that schemes

combining treatment and sanitation for the elimination of *S. hyodysenteriae* would also be effective against *S. pilosicoli*. It is unknown whether elimination of *S. pilosicoli* would be cost-effective.

What We Do Not Know

As is true of many diseases, especially recently recognized or emerging diseases, some of the most practical and important questions remain unanswered.

1. What is the prevalence of *S. pilosicoli* infection in the U.S. swine herd?
2. In an infected herd, what proportion of pigs become infected when?
3. What is the cost of subclinical infection?
4. What is the cost of clinical disease?
5. What are the best methods of treatment, control or elimination and are they cost-effective?
6. Is infection with other enteric agents additive or synergistic?
7. In diarrheal disease with multiple concurrent pathogenic agents, what is the relative contribution of each?

- by Greg Stevenson, DVM, PhD

References for all articles are available upon request.

HAPPY HOLIDAYS

FROM ADDL

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