

# LINWOOD VETERINARY SERVICES PROFESSIONAL CORPORATION

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## April 2005 Newsletter

### DAIRY

As you read this newsletter the first warm rays of spring sunshine are upon us. Spring truly is just around the corner – a welcome relief after another long winter. With spring comes an increased risk of watery mastitis as yards and barns become wet, and a proliferation of mastitis – causing E. coli bacteria. In order to reduce and minimize the risk of this terrible disease please consider the following:

1. Keep cows clean and dry. The organism responsible for watery mastitis thrives on wet and dirty environments. Eliminating such conditions goes a long way toward disease prevention.
2. Vaccinate cows with environmental mastitis vaccines. These products will reduce the severity of E. coli mastitis. This can be done as either a whole-herd approach, or by vaccinating animals pre-freshening. Example products are J-Vac or Enviracor.

Call the office to learn more.

As an aid to helping keep the stalls dry to reduce your incidence of mastitis, consider using Stalosan-F. This is a phosphorus based product that helps dry up the stall without drying up the cow's teats. It is cost-competitive with lime; a 25kg bag is enough for 100 stalls per month. Call for more information. Good stall management is still required.

### COW/CALF

Finally, confessions from another veterinarian in a land far away called Alberta. If you receive "Cattleman's Magazine", turn to page 30 to read about other producers who struggle with scours. You have heard us for the last several years go on and on about colostrum and nutrition, this is important but in the event of an outbreak remember: "The Solution to Pollution is Dilution". Many farmers are not readily set up to deal with this before dry weather and pasture are available, hence my push for tonnes and tonnes of straw. In essence we are trying to bury the cow's manure and the calf's manure beneath the straw. The next approach is to have the cow due to calve separated from the cows that have calved and ideally cows that are far away from calving. Pregnancy checking early in the fall and removing the bull are two very good management practices which help record more accurate due dates. Veterinarians are far more accurate when pregnancy diagnosing under 90 days in comparison to over 90 days.

The Sandhill's Calving System from Nebraska goes one step further: cow/calf pairs that have calved within one to two weeks of each other are housed/pastured together. Cow/calf pairs are only co-mingled once the youngest calf is four weeks old. If year after year scours eats up your profits, then re-think the design/lay-out of your farm. Or, push out calving season to May/June and tighten up the calving period. Yes, some calves can live in manure and get away with it if you lack the right combination of scour bugs and weather. However, once the right combination of scour bugs come together you need to ensure that you are doing everything right and you have to reduce the challenge. The Boy Scout's motto is correct: "**Be Prepared**".

### BEEF

Due to the uncertainty surrounding the continued closure of the U.S. border to Canadian cattle, Pfizer Animal Health has extended the BSE Canadian Cattle Health Credit Program for 2 more months. The programs will end On May 31, 2005. All eligible products purchased between August 1<sup>st</sup>, 2004 and May 31<sup>st</sup>, 2005 qualify for rebate, provided that Dectomax was used at the fully recommended label dosage.

Eligible Products: Bovi-Shield 4, Cattle-Master 4, Resvac 4/Somnubac, One Shot, Bovishield FP and PreGuard FP. If you have any questions or require further information, please call the clinic.

## **SWINE**

### **Swine A.I. Update:**

Please do not store semen in a refrigerator. The fridge is too cold! The ideal temperature for semen storage is 60 - 65°F. If you do not have a Koolatron, store your semen in the basement of the house. A.I. rods should only be used once. After the breeding is finished throw the rod away. We recently have received positive feedback from customers concerning the use of "Breeding Buddy", as an aid for artificial insemination in swine. The Breeding Buddy is a horseshoe shaped clamp. The clamp pressure on the sow's back is similar to the boar's front legs during natural mating. This pressure helps to stimulate uterine contractions and ensures proper semen draw into the uterine horns. Some producers also use the Breeding Buddy for heat detection. In the presence of a boar, place the Breeding Buddy on the back of the sow just ahead of the rump area. If the sow/gilt is in heat she will lock up and stand rigid. If she is not in heat, she will continue to move around and be restless. If you would like some more information call the Clinic and ask for Brian Misener.

The information and approaches to handling PRRS are the focus points of our swine industry. We have had many producers inquiring about serum therapy (vaccination). Articles in some major swine magazines, about this approach, have resulted in these questions. Any time a new idea is presented, that may help us to battle PRRS, the industry bandwagon takes off. Some ideas prove to be helpful others prove to be harmful. Serum therapy is the most radical intervention that we have heard of to date.

We are using serum vaccination on some farms but it is not the "answer" to controlling PRRS. The term vaccination is not accurate. This program is literally putting live virulent field strain PRRS into a bottle and injecting pigs with it. The most accurate name for this program is planned, timed exposure to disease. The concept of timed exposure is not new. We have strongly recommended gilt acclimatization programs for years. The point of gilt acclimatization is timing exposure of the gilts to PRRS is such a way as to not make them terribly sick but to allow them to go viremic and stop shedding virus before they are placed in the breeding herd.

Many gilts acclimatization programs have been very successful without using serum vaccination but some have not. One of the main reasons for acclimatization failures is lack of viral exposure. Serum vaccination is a tool that can be used to ensure viral exposure. We feel that we can use this tool effectively and safely in gilt acclimatization programs where viral exposure has been a problem.

Serum vaccination is not an intervention to be used in the breeding herd. You will cause abortions in pregnant animals if you intentionally given them PRRS virus!

We have developed a new program for herds unfortunate enough to have an acute outbreak of PRRS. The results to date are excellent. The program is expensive but very effective. We are very excited about this intervention program. It combines Pulmotil use and serum vaccination. When dealing with the frustration of PRRS it is rewarding to have some optimistic focus on new intervention strategies.

## **EQUINE**

The following information on West Nile vaccination is from an excellent article from the AAEP. We think it is worth keeping as a reference guide.

### **West Nile Virus Vaccination Guidelines**

Developed by the American Association of Equine Practitioners

West Nile Virus (WNV) infection was first diagnosed in horses in the United States in 1999 and is now an important consideration in the differential diagnosis of horses presenting with signs of neurologic disease in all areas of North America. West Nile virus, a flavivirus, was first identified as a cause of infection and fatal encephalomyelitis (inflammation of the spinal cord and brain) in horses and people in Egypt, Uganda and France in the early 1960's. Further epizootics of disease in horses have occurred in Morocco in 1996, Italy in 1998, France in 2000 and the United States from 1999 to the present. West Nile virus is now considered to be endemic in all areas of North America.

The flaviviruses, like the other encephalomyelitis viruses, are transmitted by mosquitoes, and infrequently by other bloodsucking insects, to horses, human beings and a number of other mammals from avian hosts, which serve as natural reservoirs for these viruses in nature. Horses and humans are considered to be dead-end hosts of the West Nile virus and, therefore, do not contribute to the transmission cycle. The virus is not directly contagious from infected horse to horse or horse to human. Similarly, indirect transmission via mosquitoes from infected horses is highly unlikely because horses do not experience significant viremia (i.e. they have negligible amounts of virus circulating in their blood).

The incubation period for West Nile virus in horses appears to be 3 to 15 days. Clinical signs of WNV infection in horses may include fever, ataxia (stumbling or incoordination) depression or apprehension, stupor, behavioral changes, weakness of limbs, partial paralysis, droopy lip, teeth grinding, muscle twitching, fasciculation and tremors, difficulty rising, recumbency (inability to rise), convulsions, blindness, colic and intermittent lameness, or death. The mortality rate for horses exhibiting clinical signs of West Nile virus infection is approximately 33%. Data has supported that 40% of horses that survive the acute illness caused by WNV still exhibit residual effects, such as gait and behavioral abnormalities that were attributed to the illness by owners, 6 months past diagnosis.

The variable clinical signs associated with WNV infection necessitate inclusion of many neurological disorders in the differential diagnoses. These include: rabies; equine protozoal myeloencephalitis (EPM); equine herpes virus-1; botulism; eastern, western and Venezuelan encephalomyelitis (EEE, WEE, VEE); heat stress; trauma; bacterial meningitis; cervical vertebral myelopathy (wobbler syndrome); myelocnecephalopathy; and equine degenerative myelopathy.

Serologic tests used to diagnose WNV include plaque reduction neutralization (PRNT), virus neutralization, hemagglutination inhibition, complement fixation, ELISA and antigen (IgM and IgG) capture ELISA. Virus can also be identified in central nervous system tissue using techniques such as virus isolation, PCR and immunohistochemistry. The Igm-capture ELISA is currently the most reliable test for confirmation of recent exposure to West Nile Virus in a horse exhibiting clinical signs. Horses exposed to WNV typically develop a sharp rise in West Nile virus-specific IgM antibody that persists for 4-6 weeks after infection. Little IgM is demonstrated in horses that are recently vaccinated. The antibody measured by the PRNT is stimulated both by vaccinated and recent exposure, making this test difficult to interpret in the suspect horse.

Risk of exposure and geographic distribution of West Nile virus vary from year to year with changes in distribution of insect vectors and reservoirs of the virus. Because of the unpredictable nature of those factors and the effects of the disease, it is recommended that all horses in North America be immunized against West Nile virus.

Preventive management practices may minimize the risk of the spread and transmission of West Nile virus from infected mosquitoes. Reduction of mosquito numbers and exposure can be achieved by reducing or eliminating any stagnant or standing water in your area, removing old tires, keeping horses in the barns from dusk to dawn (prime mosquito feeding times), setting out mosquito traps, keeping air moving with fans, and removing organic debris (muck) promptly. Chemical controls include the use of topical anti-mosquito repellent agents approved for the horse and use of mosquito dunks in areas of standing water.

Vaccination is the primary method of reducing the risk of infection from West Nile virus to the horse but clinical disease is not fully prevented. Vaccination with one of the commercially available licensed vaccines is recommended

for all horses residing in those areas of North America where the disease occurs. Of the licensed vaccines currently available, one is monovalent or multivalent inactivated and the other is a live canarypox vector vaccine. These available vaccines have been tested with a challenge model and have been proven to be effective as an aid in the prevention of viremia in experimentally infected vaccinated horses compared to nonvaccinated control horses for as long as 12 months after primary vaccination with two doses of vaccine. Following the label instructions, primary vaccination of previously non-vaccinated horses involves administration of 2 doses of vaccine 3 to 6 weeks apart. In endemic areas, boosters are required or warranted according to local conditions conducive to disease risk. Vaccinate semi-annually or more frequently (every 4 months), depending on risk. Annual revaccination is best completed in the spring, prior to the onset of peak insect vector season.

While neither of the licensed vaccines is labeled for administration to pregnant mares at this time, it is recommended that mares be ideally vaccinated before breeding when possible. However, practitioners have vaccinated thousands of pregnant mares due to the risk associated with pregnant mares getting the disease from infected mosquitoes. It has been accepted practice by many veterinarians to administer vaccines to pregnant mares on the assumption that the risk of adverse consequences of WNV infection outweighs any reported adverse effects of use of vaccines in pregnant mares. Booster vaccination of pregnant mares 4 to 6 weeks before foaling provides augmented passive colostral protection to their foals, lasting for 3-4 months.

Primary vaccination of foals from vaccinated mares should be started at 3-4 months of age in order to avoid interference from colostral antibodies. Foals from non-vaccinated mares may be vaccinated earlier than three months of age as they may not have colostral interference issues; however, data on which to base more specific recommendations for foals from non-vaccinated mares is not sufficient at this time. Foals should be revaccinated at 1 year of age (in the Spring of the year following their birth) to ensure adequate protection. Because of the high mortality associated with West Nile virus, it is recommended that foals born in areas where there is a high risk of exposure to West Nile virus should receive an initial series of three (3) doses of vaccine against West Nile beginning at 3 months of age and at 4 to 6 week intervals. The third dose may be at an interval of 8-10 weeks if desired, followed by a fourth dose at 1 year of age.

Many veterinarians in Southern states where mosquitoes are active year-round, prefer to vaccinate horses semi-annually or more frequently to help ensure uniform protection throughout the year, although this practice is not specifically recommended by manufacturers of vaccines. Horses that have been naturally infected with the disease should be vaccinated one year after the acute illness. Although the ability of horses to become re-infected with WNV is unknown at this time, horses that have been naturally infected with WNV may be included in a routine vaccination program as previously described.

#### **West Nile Virus Vaccination Schedule**

<b>Foals/Weanlings</b>	<b>Yearlings</b>	<b>Performance Horses</b>	<b>Pleasure Horses</b>	<b>Broodmares</b>	<b>Comments</b>
First dose: 3 to 4 months. Second dose: 1 month later (plus 3 <sup>rd</sup> dose at 6 months in endemic areas)	Annual booster, prior to expected risk. Vaccinate semi-annually or more frequently (every 4 months), depending on risk.	Annual booster, prior to expected risk. Vaccinate semi-annually or more frequently (every 4 months), depending on risk.	Annual booster, prior to expected risk. Vaccinate semi-annually or more frequently (every 4 months), depending on risk.	Annual, 4 to 6 weeks prepartum (see full text in guidelines)	Annual booster is after primary series. In endemic areas, booster as required to warranted due to local conditions conducive to disease risk. Vaccinate semi-annually or more frequently (every 4 months), depending on risk.

Note: As with the administration of all medications, the label and product insert should be read before the administration of all vaccines.

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