The Nature of Bovine Virus Diarrhea in Cattle

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The School of Veterinary Medicine at the University of California Davis recently hosted a conference on the nature of Bovine Virus Diarrhea Virus. The meeting was attended by scientist actively conducting research on BVDV, practicing veterinarians, livestock producers and other interested in the latest knowledge on this virus. Here are some of the more significant facts that came out of the meeting:

Types of BVDV

BVDV isolates are typed in several ways. There are two **bio**types of BVDV. The biotypes are defined by their effect on growing cells cultured in the laboratory. The more common biotype is known as non-cytopathic (NCP) and it has no effect on the growing laboratory cells. NCP is felt to be responsible for animals with persistent infections, PI's. The less common biotype is cytopathic (CP), because it causes damage to cells grown in the laboratory. The biotype does not determine how the virus acts in cattle. The modified live virus (MLV) vaccines are made using CP biotype. BVDV is also classified by **geno**type using newer genetic mapping technologies. There are two main genotypes – BVDV1 and BVDV2. Of the two, BVDV2 has caused more virulent disease than BVDV1 in recent years. Beyond the broad genotypes 1 and 2, there are several additional classifications within each genotype. For example there is BVDV1a or 1b and BVDV2a or 2b and each includes several different strains of virus. Within each of the genotypes, each virus is either NCP or CP.

More subtypes to BVDV1 and BVDV2 are being found in infected cattle. The reason that this is important is because there may be only moderate or little cross-protection when cattle are vaccinated with one strain and infected with another strain.

Different areas of the US and for that matter internationally, have different distributions of the different types of BVD both by biotype and genotypes. It also seems to be true that the various types of livestock such as dairy, pastured beef and feedlots have different types of BVD that are more commonly isolated than other types. BVD is can be found with other pathogens. For instance in feedlots, BVD might be found with Mycoplasma whereas in dairies, Neospora and BVD can be found together.

Vaccinations

It should be noted that no BVD vaccine can be expected to give complete 100% protection from all forms of BVD infections. Incomplete protection may result even when using MLV vaccines containing both types 1 and 2.

It is very important to follow the label directions for BVDV vaccines. Failure to follow the directions, particularly with killed products, may lead to a false sense of security about the vaccination status of cattle. The primary series of vaccinations is very important for later response to booster or infection challenge. A combination of killed BVD vaccine in calves prior to breeding followed by MLV BVD vaccine can be used to provide adequate protective immunity.

To control respiratory disease in feedlots caused by BVD, the vaccine probably should contain BVDV 2a virus as well as BVDV 1a. Vaccination should take place at least 2 weeks before the cattle enter the feedlot. MLV vaccines are suggested and cattle should also be vaccinated against M. haemolytic and P multocida. BoviSheild Gold, Pyramid 5 and Titanium all have BVDV types 1 and 2.

Most BVDV infections in nature occur by the oral or nasal route. A comparison of oral and subcutaneous route of vaccination was made using MLV BVDV1. The oral route provided faster immune response compared to subcutaneous route. Both vaccination routes were protective against challenge given 28 days after inoculation. Even though not in the test vaccine, titers to BVDV2 were also detected in the vaccinated animals.

It has been suggested that a vaccine titer of at 1:16 will prevent fever and drop in white blood cell and that a titer of 1:256 will prevent shedding of the virus as well. Some MLV bivalent (BVD1 and BVD2) vaccines have been shown to prevent fetal infections and thereby prevent PI's. Some monovalent (BVD1) have also been shown to reduce fetal infections. Most agree that MLV vaccine is needed to protect the fetus.

In order to avoid suppression of estrus, MLV BVDV vaccines should given at least 30 days prior to beginning of breeding period. When MLV vaccines are given close to estrus, the vaccine may alter the estrogen response and thereby influence the estral behavior.

Clinical disease in cows

The severity of BVD in cattle depends on the viral type, the immune status of the cattle, the reproductive status of the cattle, the presence of other pathogens and the age of the animal. The primary result of BVD infections is a decrease in immune system capability due to reduction in the white blood cells. This is usually called immunosuppression. Much of this is due to suppression of the macrophages, neutrophils and lymphocytes. These cells are among the first to respond to infection. Virus can localize in tissues and remain present for an extended period of time, perhaps for the life of the animal.

With clinical disease, the virus is present in the blood from about day 3 to 11 after the virus has entered the animal. Clinical signs usually begin at about day 9 to 13 after the initial infection. Three types of clinical disease are recognized: 1) Mucosal disease where both cytopathic and non-cytopathic virus is present; 2) severe acute BVD and 3) hemorrhagic syndrome. Along with clinical disease there is also non-clinical or inapparent disease.

Typical effects from BVDV infection are determined by the pregnancy status of the cow. Infections prior to about 45 days of gestation may result in early embryonic death. Infection between 45 and 130 of gestation may produce fetal death or persistently infected calves may result. Congenital defects such as cerebellar hypoplasia, contracted tendons, hydranencephaly and hydrocephalus may form when infection takes place between 100 and 150 days of gestation. Abortions may occur with infection from 100 to 270 days of pregnancy. This is also the time frame for congenital infections to occur. Still births occur with infections during the last week or so of pregnancy.

BVDV can affect estrogen production of the cow and may cause infertility in this manner. This may effect the estral cycles of the cow due to altered estrogen production.

Clinical disease in calves

Calves that are congenitally infected with BVD between 140-270 days of gestation are born with titer but later become negative. Their titers last longer than those produced by colostral antibodies from the dam. The CI calves have higher morbidity (2.3x) than normal calves. The higher the titer in the CI calves, the greater the morbidity. They also have greater number of days to 1st breeding compared to non-CI calves.

Colostral antibodies passed on from the vaccinated or exposed cow to the calf and usually decline to a titer of <1:64 by 75-90 days of age in the calf. Vaccination will usually produce titers of up to 1:256. Titers from vaccination will decline to less than 1:512 within 3 months of vaccination. Titers of >1:512 after 3 months following vaccination usually suggest infection. Calves born with congenital infection will have titer of >1:8. About 10% of the calves that are born maybe congenitally infected.

BVDV type 2 fetuses were found to weigh less and have shorter CR length than BVDV negative fetuses. No effect on the fetus was found with BVDV type 1.

Critical periods for new BVDV infections are in calves 3-6 months of age when colostral titers are gone and at 9-10 months when ovaries and testicles are becoming active.

Clinical disease in bulls

Three types of BVD infections have been documented in bulls. 1) PI bulls continually shed a lot of virus in their semen and will infect most cows that they breed as well as their calves. About 1% of bulls are PI's. The virus from these bulls can survive freezing and acts as a source of infection for cows during insemination. They have variable fertility. 2) Acute infected bulls shed much less than PI bulls after about 10 days post infection. They can breed cows satisfactorily. About 5% of the cows they breed will have PI calves. 3) Bulls can also develop persistent testicular infections and consistently shed virus in their semen. These bulls will be sero-positive. AI semen from these bulls will infect cows. This has been seen with localization within the seminal vesicles of bulls. These bulls will shed virus in their semen.

PI animals

PI cows can respond to vaccination and without any signs of disease. Most BVD infections take place at 90-100 days of age in dairy calves. Few BVD infections probably take place on calf ranches if calve have received adequate colostrums. Not all PI animals die young!

PI animals might possible be used to "vaccinate" other animals within the herd. This might be possible with pre-breeding age heifers to simulate immunity prior to breeding.

Diagnostics

Skin samples such as ear notches can be tested for BVDV. In a large diagnostic laboratory, about 1% of the ear notches examined showed PI infections. Testing 20% of a herd should detect PI's if more than one is present. PI animals serve as source of infection for their herd mates. In some laboratories, variations of the ear notches can be used to suggest acute infection instead of PI status. Other tests such as the ELISA or PCR can also be used to find the PI animals.

Control Measures

It is important to remove aborted fetuses and placentas as they may serve as a source of virus when licked or eaten by other cattle. The BVDV can easily spread over the fence from one pen to another.

Summary

If nothing else is clear, it should be that BVD is a very complex disease that is difficult to control in all it various presentations. Vaccination is clearly indicated and should be given as directed by the manufacture's label at least 30 days prior to breeding or entry into a new population of cattle. Appropriate biosecurity measures along with vaccination will aid in the spread of the BVD virus between livestock. Consult your veterinarian for detailed information of sampling for diagnose and to develop a comprehensive BVD control program.