DAIRY VETERINARY NEWSLETTER

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BVD is Still a Major Bovine Disease, Carried by Some Interesting Fomites as Well

Bovine Viral Diarrhea (BVD) virus has been recognized as an important pathogen of bovines and other ruminants for over 70 years. Today, many people in the dairy industry including veterinarians tend to think of it as a pathogen that has largely been eliminated as a major cause of disease in well managed dairy herds. I have been told by some dairy veterinarians that in their practices, none of their client herds have any BVD infected animals. (I won't say that this cannot be true, but some data below will cast doubt on whether this is likely to be the case for many herds.)

The assumption is that testing such as the antigen capture ELISA (most commonly performed on ear notch skin biopsy samples) to identify and cull Persistently Infected (PI) animals, and anti-BVD vaccinations including killed or modified live vaccines, usually in combination products that immunize against other pathogens such as IBR, PI_3, BRSV and Leptospirosis, for example have virtually eliminated BVD.

BVD data from the Utah Veterinary Diagnostic Laboratory

Nevertheless, data from a recent 5-year period from nearly 9000 bovine samples of all types submitted to the Utah Veterinary Diagnostic Laboratory (UVDL) shows that 1.2% of the specimens were diagnosed BVD-positive by laboratory testing. Most of the BVD tests performed were antigen capture (ear notch) ELISA, serum ELISA, or PCR on various tissues. If the breed of the animal was known (more common from necropsy specimens), dairy breeds were 3.3% BVD-positive and beef breeds were 1.6% BVD-positive. If the age of the animal from which the specimen came was known (primarily from necropsies), the percentages of BVD-positive results were: fetuses 8.3%, calves 5.0%, immature animals 2.2%, adults > 2 years old 5.3%. Whenever BVD was diagnosed at necropsy, it was considered to have contributed directly to the death of the animal (including abortion in the case of a fetus), as either the primary cause or a major contributory cause of death. In addition to lesions observed at necropsy, there was laboratory confirmation, mainly using PCR.

It must also be kept in mind that BVD is a notoriously difficult virus to detect in tissues of dead animals,

often not surviving well to be detected by tests such as PCR. Our pathologists often strongly suspect BVD based upon lesions, either gross or microscopic, in cases where the virus cannot be definitively diagnosed by laboratory methods. Such cases were not counted as being diagnosed BVD-positive. Taken together, the evidence suggests that BVD is still an important cause of abortions, stillbirths, and deaths of bovines of all ages, proportionally higher in dairy animals than beef animals, possibly because of more close housing of dairy cattle.

It is well recognized that PI animals shed large amounts of BVD virus, nearly continuously. Major means of spread of BVD are via inhalation from respiratory tract infected animals and ingestion or muzzle contamination from feces of animals with BVD, especially those with diarrhea. It can also be spread via milk, urine and semen.

Fomites that can carry BVD virus

However, many inanimate objects can act as fomites to spread BVD as well. An October 2014 article in Drovers Cattle Network by John Maday summarized this, as did a USDA Veterinary Services Info Sheet regarding BVD Virus in December 2007.
http://www.aphis.usda.gov/animal_health/emergingissues/downloads/bvdinfosheet.pdf

Some fomites found to carry BVD virus include:

- Rectal palpation sleeves (PI cow palpated, same sleeve used on subsequent BVD-negative cows)
- Housing pens (PI calf in pen, removed, 2 hours later BVD-negative calves placed in pen)
- Vaccine bottle stoppers (PI nasal fluid on stopper, dried, vaccinated BVD-negative calves)
- All three of the transmission studies above resulted in BVD virus isolation from naïve animals
- Latex as in latex gloves (21% survival of live BVD virus for 48 hours)
- Water (16% survival of live BVD virus for 48 hours)
- Nose tongs
- Milk nipple bottles
- Halters
- Balling guns (This and the 5 fomites immediately above have no documented transmission studies published that I can find. I asked one of the members of USU's Anti-Viral Group about this. He said that as infectious as BVD virus is, if it survived on fomites long enough to contact a naïve animal in any way that could result in ingestion or inhalation, he would expect a new infection to result.)

These infectious contact surfaces just add to the picture that removal of PI BVD animals and vaccination of the remaining herd against BVD are vitally important to the control of the disease. The BVD virus has not been completely eliminated as an important pathogen in dairy of beef cattle.

DHIA SCC Data from Every State - How does Utah Compare in SCC?

Many people in the dairy industry including veterinarians are familiar with the annual NAHMS (National Animal Health Monitoring System) data on somatic cell counts from U.S. dairy farms that is released annually. However, the majority of federal milk marketing orders, and the considerable regions of the U.S. that are not in a federal order contribute no data to that report. Large geographical gaps are present in that data every year simply because data is not available from much of the country.

Dairy Herd Improvement Association data is biased toward somewhat more progressive farms and for approximately 50 years the SCC values in milk of cows in DHIA herds (as well as prevalence of mastitis as defined by milk culture) have been lower than the national average. The DHIA data is very interesting, though because it is provided for all 50 states. Somatic cell count data from DHIA for 2013 was recently reported by Dave Natzke in the January 2015 issue of Dairy Herd Management.

The report includes much interesting data, including the fact that 2013 was the first year during which the DHIA mean SCC was < 200,000/ml, coming in at 199,000/ml. It was not clear whether this was the average of the bulk tank SCC samples collected on test days, or the mean of all of the individual cows' SCC values. SCC, mean cows per herd, daily milk yield, and percentage of test days above various SCC thresholds such as 400,000/ml and 750,000/ml are reported. Utah had 180,000/ml mean SCC, 211 lactating cows/herd, 71 lb daily milk, and 92.6% of test days below 400,000/ml SCC.

Utah's mean SCC of 180,000/ml was lower than that for these other Western states:

State	SCC (DHIA herds)
North Dakota	297,000
South Dakota	221,000
Montana	184,000
Arizona	201,000
Colorado	212,000
Kansas	249,000
Nebraska	224,000

While these other Western states' SCC means were lower than Utah's:

State	SCC (DHIA herds)
Wyoming	174,000
Idaho	174,000
California	174,000
Oregon	166,000
Washington	162,000
Nevada	150,000
New Mexico	147,000

SCC's for much of the rest of the U.S. ranged between 207,000/ml and 359,000/ml with the highest cell counts

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reported in the Southeastern U.S. The Northeast U.S., Connecticut, Maine, Massachusetts, New Hampshire, New York, Rhode Island, and Vermont, had SCC ranging from 157,000/ml to 199,000/ml, making it the lowest SCC region of the country.

The national DHIA means for 2000 to 2013 are shown in the article, and the SCC has steadily declined from 233,000/ml to the most recent 199,000/ml. However, the decrease from 2012 to 2013 was only from 200,000/ml to 199,000/ml. The rate of decline in SCC may naturally be biologically limited, and the pace of decrease may continue to be slower. Perhaps a future subject in this newsletter can be the sometimes controversial question of what level of SCC is too low for cows to resist severe clinical mastitis, a level that is too low for the welfare of the cows and the overall benefit to the industry and to consumers. However, according to much of the evidence, we have not gotten that low (at least in terms of average) yet. Milk quality and udder health continues to improve, and all involved in the dairy industry deserve congratulations for their part in that.

Please let us know your comments and also suggestions for future topics. I can be reached at (435) 760-3731 (Cell), (435) 797-1899 M-Tues, (435) 797-7120 W-F or **David.Wilson@usu.edu**.

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