Screening of cattle herds for BVD virus

Bovine virus diarrhoea (BVD) is caused by a pestivirus which is antigenically related to pestiviruses causing disease in sheep (hairy shaker disease), pigs (swine fever) and other ruminants such as deer and goats.

Over recent years the complex pathogenesis of BVD has been largely resolved.[1,2] The key to spread and control of the disease is the persistently infected carrier animal. These are animals which have been infected *in utero* within the first few months of pregnancy by non-cytopathic BVD virus. Often these animals are clinically normal although they remain seronegative to tests for BVD and shed large quantities of virus in all secretions and excretions. When super-infected with a homologous strain of cytopathic BVD virus, these persistently infected animals are the ones that develop mucosal disease, either acute or chronic. Chronic disease, especially in beef breeds, is the most common form of the disease seen in New Zealand.[1] When other cattle are infected post-natally, they experience transient low grade disease which is often subclinical. Usually, post-natal infections are only significant if they occur in seronegative pregnant animals. In such cases the virus will cross the placenta to produce a new generation of persistently infected animals. The most obvious losses caused by BVD virus are the cases of mucosal disease, but these may only be the tip of the iceberg. The other effects of congenital infections, such as abortions, foetal resorption and mummification, congenital defects and the birth of weak, under-sized calves, may be less obvious but more significant financially to the farmer.

The BVD virus is widespread in New Zealand cattle, with about 60% having antibodies against it.[3]

Laboratory tests for herd screening

Practical systems for testing large numbers of cattle for BVD virus have been developed over the last few years. A combined serum neutralisation test/immunoperoxidase assay (SNT/IP) has been developed to give the virus and antibody status of animals relatively quickly and for a reasonable price. The test is carried out on serum. More recently, a BVD antigen-capture ELISA has been developed and is currently being evaluated. It shows great promise as a rapid method of detection of persistently infected animals. However, the assay requires tissue rather than serum. Buffy coat, blood clot, spleen and lymph node are suitable tissues for the ELISA.

BVD infected herd histories

The severity of BVD infection in individual herds, and the value of herd screening, was illustrated in two herds recently.

Herd A: A farmer had been buying calves from a Waikato farm for beef production for several years without problems. In 1990 40 calves were purchased and many were noticed to be ill-thrifty and were unresponsive to extra anthelmintic or other treatments. They progressively lost condition and when a year old, began to die. Veterinary examination revealed oral and interdigital lesions suggestive of chronic mucosal disease. After nine had died and two had been fattened and sent to a slaughterhouse, the remaining 19 were tested in the SNT/IP test. Six of them were BVD virus positive and antibody negative while 11 were virus negative and antibody positive. The remaining two were virus negative and antibody negative, but could have been persistently infected animals with low levels of virus not detected by the test. The farmer had predicted, from the physical appearance of the animals, which were likely to be persistently infected animals. The healthy, robust animals were the seropositive, virus negative animals. It is therefore probable that 17/40 (42.5%) of the animals purchased in 1990 were persistently infected carriers. This indicates that the dams of these calves had been exposed to BVD virus while at a critical stage of pregnancy on the farm of origin.

Herd B: On a Canterbury beef farm two cases of confirmed mucosal disease and a number of unexplained sudden deaths had occurred in a herd of rising two year old (R2) bulls over a two-month-period. The heifers of the same age were described as being in very poor condition, but no deaths had occurred.

When the R2 and sire herd bulls were tested by SNT/IP, 15 (all R2 bulls) were BVD virus positive and antibody negative, five were virus and antibody negative and 57 were virus negative, antibody positive. All 15 of the virus positive animals were culled together with four of the virus and antibody negative animals. The remaining bull, one of the sire herd, was retested with the R2 replacement heifers and the R1 bulls. In this group there were 10 virus positive and antibody negative animals, two virus and antibody negative, and 115 virus negative and antibody positive animals. The retested bull was now antibody positive, indicating it had seroconverted. Of the 10 confirmed persistently infected animals, seven were R2 heifers which had been suspected clinically to be carriers because of their poor condition.

If the confirmed cases of mucosal disease are included, this means 27/209 (12.9%) of the animals tested in this herd were persistently infected carriers. It is suspected that BVD infection was introduced into the herd during mating or early pregnancy in 1988/89. None of the sire bulls could be incriminated, but bulls from another station had been used and may have been responsible.

The findings from these two cases illustrate the serious consequences of BVD virus infection in some herds. Where unexplained, unresponsive cases of ill-thrift are occurring in young stock, a herd test for BVD virus should be considered, especially if a case of mucosal disease has been diagnosed.

Control measures

These are based around identification of persistently infected animals. Many persistently infected animals will be ill-thrifty, although some are clinically normal and may survive for several years, producing calves that will also be persistently infected. Cases of mucosal disease will also occur in this group, usually when the animals are between six months and two years of age.

Once identified, persistently infected animals can be culled or they can be used to infect young stock with BVD virus before breeding. Contact of persistently infected animals with heifers and other seronegative cows during the critical period just prior to, and during, early pregnancy must be avoided. Serum from persistently infected animals has also been used successfully to immunise susceptible cattle, in the absence of suitable BVD vaccines.

The creation of a virus-free herd seems attractive, but there are pitfalls. Once all persistently infected animals are removed, the herd's immunity will decrease over a number of years until all animals are seronegative. This means there is potential for a major breakdown if the herd cannot be managed as a closed herd or one in which all replacements are virus tested. Even then, there is the risk of infection from neighbouring farms, by fomites and from other ruminants such as sheep, deer and goats.

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References

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