

feedlots, processing delays might be expected. Stressors such as prolonged confinement, handling and certain infections such as BVD may depress the immune response of the animals (3, 6).

Vaccinations may be detrimental. Human study has shown a decrease in respiratory disease which corresponded to a decrease in the numbers of vaccinations performed on naval recruits (7). Thus it is important to ensure that the vaccines and bacterins administered to cattle are all really required.

The acquisition of salmonellosis in most animals is related to the infective dose and so attention should be focused on the cleanliness of pens at auction sales and of cattle liners. At the feedlot attention should be directed towards cleanliness of receiving pens, holding pens and hospital pens.

Feedlot veterinarians should be aware of the existence of multi-resistant salmonella species and of the conditions under which they cause disease. It is obviously very difficult to predict the future but in Europe, disease caused by these organisms has been increasing. Veterinarians should play a greater role in feedlot animal health. The feedlot manager should be advised on the number and the type of cattle to be admitted. Feedlot construction should be examined, particularly the number and type of receiving and hospital pens. Only the necessary vaccinations should take place. Finally, there will be much public health concern and enquiry if resistant feedlot salmonellae are found to cause human disease. In such a situation intervention similar to that applied to the poultry industry might be expected.

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BVD Vaccination Trial

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Introduction

BVD is an infection of cattle that occurs around the world. Evidence of BVD infection is found in 70-90% of the cattle population.¹ This level of infection is determined by random serological surveys and you can appreciate that if 70-90% of the cattle population is seropositive, then BVD virus is quite prevalent. BVD may be a source of economic loss to the cattle industry by causing a clinical or subclinical infection.

Clinical Infection

Clinical cases of BVD are either enteric or reproductive problems. The enteric form causes economic loss in animals that die of acute or chronic diarrhea. The reproductive form may contribute to losses by causing abortions, repeat breeders or the birth of malformed calves usually following a BVD infection in non-immune heifers.²

The magnitude of the economic losses due to the reproductive form has not been accurately determined and this should be done so the cost of the disease can be compared to the cost of control.

Subclinical Infection

Reports, which have not been substantiated by controlled studies, suggest that subclinical BVD infections may lead to economic losses.

This type of loss may be due to the fact that BVD is an immunosuppressive virus. BVD field virus is capable of decreasing the numbers of T & B lymphocytes in a calf, as well as impairing the functional capabilities of T & B cells and macrophages.³

This immune suppression following a mild or subclinical infection may occur and subsequently lead to secondary infections that impair growth rates in calves. This project was concerned with investigating the enteric and subclinical form of BVD infections. When considering the enteric form there are 2 epidemiological patterns involved; sporadic and enzootic.

Sporadic

These outbreaks occur from time to time in a herd and following the resolution of the disease, the herd remains free from clinical BVD for several years due to herd immunity.

After the total number of susceptible cattle increases due to the addition of susceptible replacements, i.e. calves, an outbreak may occur again.

Enzootic

This epidemiological pattern is much less common but it is being recognized more and more, especially in cow-calf operations.

With this pattern of diseases there are annual losses in the calf crop as they become susceptible with the waning of their colostrum derived antibodies. This persistence of disease may be due to a mature animal in the herd that is persistently infected, constantly shedding virus and challenging calves as they become susceptible.

The herd that we were involved with was a well managed cow-calf operation with about 100 purebred polled herford cows.

This was an open herd but there were very few new arrivals and all new arrivals were quarantined for 4-8 weeks before being mixed with the herd. The cows calved on pasture and the calves were identified with ear tags, given colostrum, vitamin E-Se, and their navels were dipped. Their health problem involved annual losses of 1 or 2 calves due to the enteric form of BVD. This emulates the enzootic epidemiological pattern that was described earlier.

The losses occurred in 1975, 1976 and 1977. The calves were about 4-5 months of age and usually were growing very well up until the day they became ill. The recurrent nature of this BVD problem led us to assume that BVD was enzootic in this herd, although infections were predominately subclinical, and therefore this herd was a good place to test a BVD vaccine.

Objectives

The objectives of this study were:

- 1) To compare the weight gain between vaccinated and unvaccinated calves. This allowed us to test the claim that the weight gain of calves is improved if you minimize the effect of subclinical infection by vaccinating the calves.
- 2) To do a comprehensive study of the titre dynamics in both groups of calves.
- 3) To monitor and record any health problems in order to compare the general health status between vaccinated and unvaccinated calves.

Methods

The calves were born on pasture and calves of similar age were put into groups and raised up to 6 weeks of age under the owner's usual management regime. At this time we implemented our vaccination regime. The calves were handled at 6, 12 and 24 weeks of age. About one half of the calves were vaccinated and the rest were left as controls. They were all weighed, measured for height and girth and bled for BVD virus neutralizing antibody detection.

Three weeks later, at 9, 15 and 27 weeks, all of the calves were rebled to provide convalescent serum samples. Both groups of calves were pastured together throughout the trial.

The first vaccination at 6 weeks of age is earlier than most recommended procedures. We chose to vaccinate this early in order to protect any calves that received little or no BVD antibody from their dam's colostrum. The calves were vaccinated 3 times to ensure that each vaccinated calf received at least one immunization when its passive antibody

levels had decreased to a point low enough that it did not interfere with the active immunization. In the case of BVD these titres must decline to levels less than 1/100.⁴

The vaccine that was used was a commercial, modified live BVD virus vaccine with the NADL strain of BVD virus of porcine kidney tissue culture origin. It was a monovalent vaccine.

Independent investigations report that calves vaccinated with such a vaccine do not shed BVD vaccine virus⁵ and ensured that the vaccinated calves did not shed and thus challenge the control calves.

The vaccine was stored and reconstituted properly to protect the level of the antigen. The vaccine was titrated and found to contain an adequate amount of virus.

Care was taken not to spill or slop the vaccine around the farm so that the controls would not be exposed to vaccine virus by this route.

Results

The statistical analysis supports the null hypothesis that there is no significant difference in weight gain between vaccinated and unvaccinated calves in this study. (See Table I).

		Weight Data	
		# of calves	Gain
1978	vaccinated males	26	1.84 lb/day
	control males	16	1.92
	vaccinated female	22	1.77
	control females	16	1.54
1979	vaccinated males	20	2.07 lb/day
	control males	16	2.26
	vaccinated female	19	2.02
	control females	16	2.04

The weight gains in 1979 appear to be higher than 1972 and this may be attributed to the owner spending a lot of money on semen in order to improve the growth rate of the calves by selecting a herd sire proven to improve gain in the calves.

Titre Patterns

Figure 1 and Figure 2 show the pattern of BVD titres in the calf crops of 1978 and 1979. The "expected" decline of passive antibody is shown to point out what the pattern of decay is in a calf with passive antibody titre of 1/128. The half life of BVD antibody from colostrum is about 21 days, so if the animal is not challenged with vaccine virus or field virus, the passive antibody would decline to zero in about 12 weeks.⁴

The pattern for the vaccinated and control calves is parallel to the "expected" pattern for the first 12 weeks. This suggests that passive antibody was too high to allow the immunized calves to seroconvert until they were older.

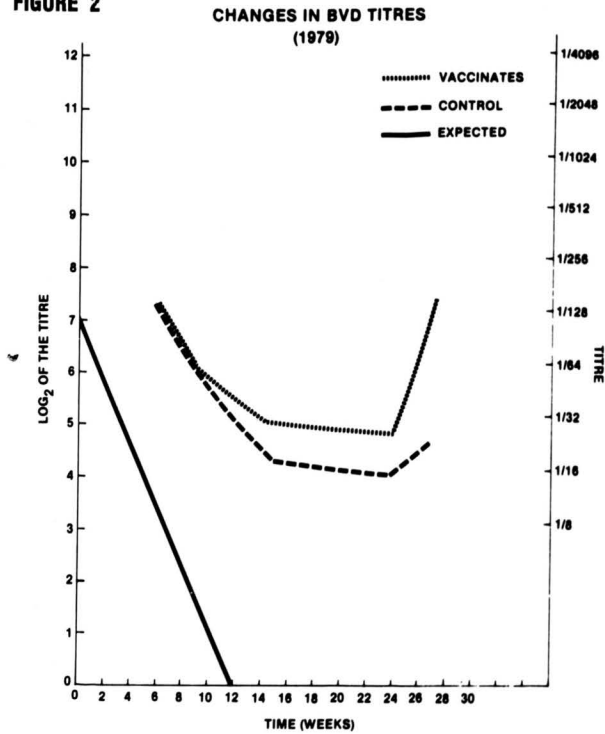
With the second immunization at 12 weeks of age and the third at 24 weeks of age, it is clear that titres increased in the vaccinated calves.

It is interesting to note that the titre pattern for the control calves also started to increase at 12 weeks. In fact 40% of the control calves seroconverted. Evidently a large number of unvaccinated calves were mounting an immune response, presumably to field virus, since vaccine virus was not supposed to be shed from the vaccinated calves. The calves underwent a natural immunization process.

Health Problems

Over two years, 4 calves died of BVD, and three of these had been vaccinated. All of them were growing well at the time that they developed clinical signs. Table 2 shows the titres of 2 calves that died of BVD. K 268 was a 1978 calf that

FIGURE 2



BVD Titres of the Calves That Died of BVD

Table 2

Calf #	6 weeks	9 weeks	12 weeks	15 weeks	24 weeks	27 weeks
K 268 (Vaccinate)	64	<8	<8	<8	-	-
L 365 (Control)	<8	<8	<8	<8	<8	<8

not seroconvert after being vaccinated at 6 or 12 weeks of age. Within a week of the second vaccination the calf developed clinical signs of BVD and died 2 weeks later. This calf did not develop any detectable BVD antibodies at any time in its life.

Calf L 365 was a control calf and it did not develop antibodies to BVD while it was on the trial. This calf became ill at 30 weeks of age and died shortly after, still seronegative to BVD.

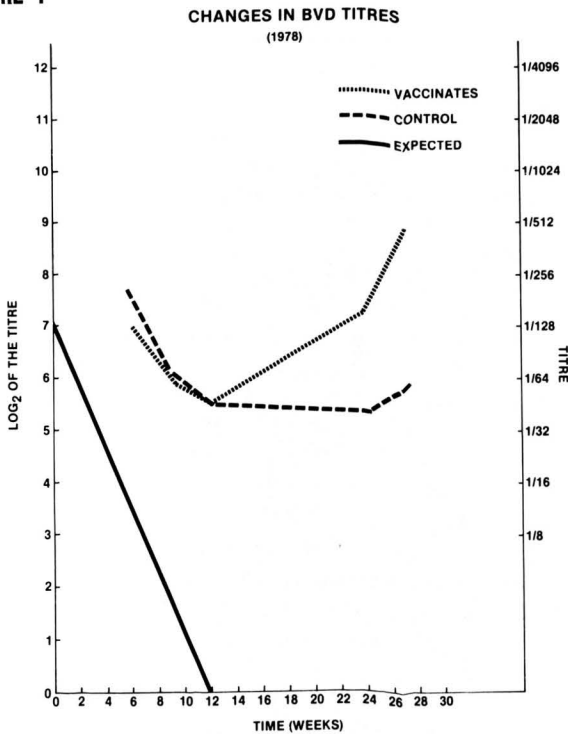
Table 3 shows the titres of the 2 other calves that died of BVD. K 251 and K 252 are unusual in that both responded

BVD Titres of the Calves That Died of BVD

Table 3

Calf #	6 weeks	9 weeks	12 weeks	15 weeks	24 weeks	27 weeks
K 251 (Vaccinate)	<8	1024	>2048	>2048	-	-
K 252 (Vaccinate)	<8	2048	256	512	-	-

FIGURE 1



was vaccinated and L 365 was a 1979 calf that was a control.

Both of these calves displayed the typical titre pattern reported in the literature for calves that die of BVD.^{3 6} Both were apparently incapable of mounting an immune response to BVD.

K 268 had a passive antibody titre of 1/64 at 6 weeks of age but this calf was apparently immunoincompetent as it did

strongly to the first vaccination and developed high titres that, according to the literature, should be protective.⁶ The calves developed clinical signs at about 15 weeks of age or 3 weeks after their second vaccination.

The failure of the antibodies to be protective may mean that there are different strains of BVD with major antigenic differences but this has not been reported in the literature as all strains of BVD appear to cross react antigenically to some degree with one another. The diagnosis was confirmed by the pathology department at OVC on the basis of clinical signs, gross and histopathology that were all typical of BVD. The other explanation for the failure of the antibodies may be that a protective immune response to BVD is more complicated than just the development of neutralizing antibodies prior to a challenge with BVD virus.

This leads to the question whether one should vaccinate to prevent the enteric form of BVD. If the main concern is to prevent losses caused by the enteric form of BVD then it is important to remember that animals that die of BVD are usually immunoincompetent and unlikely to respond to BVD vaccine antigen if they are unable to respond to field virus. In addition to this, there are reports of outbreaks of BVD following vaccination for BVD and since, according to our work, the titres induced by vaccination are not always protective, one should be cautious using vaccines containing live BVD virus.

Conclusion

In conclusion, there was no benefit in weight gain in calves that were vaccinated for BVD over those left as controls, at least not in this particular study.

Calves were capable of responding to the vaccine once their passive antibody titres declined to levels less than 1/100. While over 90% of the vaccinated calves seroconverted, 40% of the control calves were able to actively produce BVD antibodies without being vaccinated.

Finally, BVD virus neutralizing antibodies are not always effective in protecting against disease. The vaccination program made no difference in the incidence of clinical BVD in both the vaccinated and control calves.

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Herd Health in Free Stalls:

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I practice in the Caslow valley. I have probably the same kinds of dairy clients that you have. Most of them came from Holland, they know how to grow grass really well. We grow very little cornsilage, we grow mostly grass and production on these fields is about eight to nine pounds dry matter to the acre that's well above 10% protein, probably in the neighborhood of fourteen or fifteen percent protein. We use mostly bunker silos. These barns are drive-through barns and this particular barn holds about two hundred cows, a hundred cows on each side divided into groups of fifty. The cows are in one part, another part is the drive-through, the trucks can drive on both sides. There is a row of free stalls on one side and another row of free stalls on the other side. The hay storage is above the feeding alley. So the hay truck can drive through and put the hay off on either side. One barn has tie stalls or they have tie ups, so herd health is a piece of cake in this herd, they have about an eighty cow herd, I can go in twice a month. It takes us about fifteen or twenty minutes and the cows are tied up, any of you that particularly want plans for this type of thing I can probably get them, for many of you appreciate the tie up. For those who haven't seen them before, the cow puts her head in and ties herself. So about four-thirty or five o'clock in the evening this herd is finished except for the part they feed the hay, I arrive about fifteen minutes after that and we can do reproductive herd health in a matter of fifteen or twenty minutes. Piece of cake! IN this barn with these free stalls, I don't really need to show many pictures of free stalls for people to appreciate what free stalls are. But this particular herd, for example, has about four stalls where they tie up cows for AI and in order for me to adopt to my clients in the valley, in order for what I want to do, which is reproductive herd health, I adapted to their situation of not having restraint to do reproductive herd health. So I adapted to their situation of me wanting to do reproductive herd health in these free stalls. I've been driving around the valley for thw last month trying to figure out where I can get a picture of a barn with the cows all tied up, I couldn't find one, I haven't got one left, not one tie stall barn. I've got those tie ups, you know in the free stall barns, but I haven't got a tie stall barn left. And so I was trained to do reproductive herd health where I could put the carry-all down in the alley way between the stanchion barns and I could go to do rectals on the cows and decide what I wanted to treat them with and go back to the carry all, load things up and then go back and treat. But maybe you can appreciate the fact that if you're going to treat these cows in free stalls then it's a matter of chasing them up in the free stall the first time. And some of these cows don't appreciate being chased up a second time so that if in fact you get her in the free stall the first time your utmost is to treat the cow there while she's in the stall before