PEER REVIEWED

Case Study – Removal of Bovine Viral Diarrhea Virus (BVDV) Persistently Infected (PI) Animals from a United States Beef Herd: Effect on PI Animal Prevalence and BVDV Seroprevalence

Susan M. Cleveland, *MS*[1]; **Matthew A. Cleveland,** *MS*[2]; **Mo D. Salman,** *BVMS, MPVM, PhD*[1]; **Robert G. Mortimer,** *MS, DVM*[1]; **Hana Van Campen**, *DVM, PhD*[1]
[1]*Veterinary Teaching Hospital*
[2]*Department of Animal Sciences*
Colorado State University, Fort Collins, CO 80523

Abstract

This study was conducted to determine the effect of removing cattle persistently infected (PI) with bovine viral diarrhea virus (BVDV) on seroprevalence and prevalence of PI calves in an endemically infected beef herd. The prevalence of PI animals and the serum neutralizing (SN) titers of heifers, bulls and calves were determined for three cow-calf herds in a beef cattle ranch over a period of two years. In the first year, identified PI animals were removed from the herd within three weeks of identification. Five PI animals of 2921 (0.171%) cattle tested were identified in the first year. One PI animal was a yearling heifer and the remaining four were calves. Three of the four PI calves identified were removed prior to the breeding season, when the calves averaged two months of age. One PI animal was not identified until weaning at six months of age, and was present in the herd during the breeding season. The percentage of animals with high SN titers (≥1:512) were 20.6% (367/1784) to type I BVDV and 44.8% (799/1784) to type II BVDV. In year two, PI calves were not identified and the percentage of calves with high SN titers to BVDV type I and type II decreased to 5.91% (58/981) and 18.04% (177/981), respectively. Removal of PI animals in the first year reduced transmission of BVDV as evidenced by a significant decrease in the prevalence of high BVDV SN titers and the absence of PI animals the following year. These findings support the use of PI animal identification and removal as an effective BVDV control strategy in beef herds.

Résumé

Cette étude a été menée afin de déterminer l'effet de retirer le bétail infecté de façon persistante avec le virus de la diarrhée virale bovine (DVB) sur la séroprévalence et la prévalence de veaux infectés de façon persistante dans un troupeau de boucherie infecté endémiquement. La prévalence d'individus infectés de façon persistante et les titres d'anticorps neutralisants dans le sérum de taures, de taureaux et de veaux ont été examinés dans trois troupeaux vaches-veaux d'un élevage de bovins de boucherie sur une période de deux ans. Lors de la première année, les animaux identifiés comme étant infectés de façon persistante ont été retirés du troupeau dans les trois semaines suivant leur identification. Dans cette première année, on a identifié cinq animaux infectés de façon persistante parmi les 2921 animaux testés (0.171%). L'un de ces animaux était une taure de l'année alors que les quatre autres étaient des veaux. Trois de ces quatre veaux ont été retirés avant le début de la saison de reproduction quand ces animaux étaient âgés en moyenne de deux mois. Un des animaux infectés de façon persistante n'a pas été identifié avant son sevrage à six mois et a donc été présent dans le troupeau pendant la saison de reproduction. Le pourcentage d'animaux avec des titres neutralisants élevés (>= 1 :512) était de 20.6% (367/1784) pour le virus de la DVB de type I et de 44.8% (799/1784) pour le virus de la DVB de type II. Lors de la deuxième année, aucuns veaux infectés de façon persistante n'ont été identifiés et le pourcentage d'animaux avec des titres

neutralisants élevés est passé à 5.91% (58/981) pour le virus DVB de type I et à 18.04% (177/981) pour le virus DVB de type II. Le retrait des animaux infectés de façon persistante lors de la première année a réduit la transmission du virus DVB tel que démontré par la diminution significative de la prévalence de titres neutralisants élevés et l'absence d'animaux infectés de façon persistante lors de la deuxième année. Ces résultats supportent l'utilisation de la méthode d'identification et de retrait des animaux infectés de façon persistante comme stratégie de contrôle efficace du virus de la DVB dans les troupeaux de bovins de boucherie.

Introduction

Bovine viral diarrhea virus (BVDV) is an economically important pathogen of cattle worldwide. The transmission of BVDV vertically from dam to fetus and horizontally from animal to animal compounds the difficulty in controlling these infections. *In utero* infection of the fetus during the first trimester of pregnancy can result in the birth of a persistently infected (PI) calf, which is the primary transmitter of BVDV.[1] Fetal infections also result in significant calf losses during BVDV epidemics as a result of abortions, infertility, stillbirths and neonatal calf mortality.[23]

Multiple studies have documented the prevalence of PI animals and BVDV seroprevalence in dairy herds.[3,11,16] Sorensen *et al*[23] showed that with one PI animal present in a naïve dairy herd, transmission of BVDV could proceed at a rate where 20% of the susceptible animals become infected per week. In vaccinated and unvaccinated BVDV endemic dairy herds, the PI animal prevalence can range from 1-2% with 60-90% of seropositive animals in the herd.[11,12] In previous surveys of US beef herds, PI animal prevalence ranged from 0.1-2%.[10,25] The seroprevalence in herds where one PI animal was present ranged from 15-80%.[8,10,15,18]

There are important differences between dairy and beef herd management practices that may impact BVDV spread and infection. Dairy cattle are normally confined within close proximity to one another, which might result in more contacts between susceptible and PI cattle, possibly increasing the rate of BVDV infection. Dairy cattle are also bred year round, allowing PI animals to be introduced to the herd continuously, potentially increasing exposure rates. Because most beef cattle are bred synchronously, there is a limited time period when exposure to BVDV can result in a PI fetus. In addition, beef cattle are generally grazed on large pastures where contact with other cattle may be reduced, thus slowing the spread of BVDV.[24] The purpose of this study was to determine the efficacy of PI animal removal as a BVDV control method in United States (US) beef herds.

Materials and Methods

Herd Selection and Management

An endemically BVDV-infected beef herd was recruited for this study. Ranch managers had noted excess calf mortality from abortions and respiratory disease for several years. Herd BVDV infection was previously diagnosed on the basis of elevated serum neutralizing (SN) titers in heifers and the isolation of BVDV from calves' tissues.

Cattle were managed in five groups consisting of three cow/calf herds, one replacement heifer herd and one bull herd. First-calf heifers (herd A) were bred from June to July and calved from February to April the following year. Cows in herds B and C were bred from June to August and calved from April to May the following year. Calves were uniquely identified at birth with ear tags. The identification of the dam, the date of birth and weight of calves were recorded. Calves were branded between April and June, and weaned in September. At weaning, calves were weighed and calf losses from branding to weaning were recorded. Heifers were selected for replacement at weaning based on weaning weight and general thriftiness. Other weaned calves were sold between 8 and 10 months of age.

Cows were estrus-synchronized, bred by artificial insemination and followed by breeding with bulls. All herds were grazed separately after breeding. Throughout the year, the herds were rotated to different pastures every two to six days. Animals in herd A could have contact across fence lines with animals in herd C. In addition, animals in herds B and C could potentially have contact across fence lines with neighboring cattle and one another.

Calves were vaccinated at branding with an inactivated vaccine composed of infectious bovine rhinotracheitis virus (IBR), type I and type II BVDV, parainfluenza-3 (PI3) and bovine respiratory syncytial virus (BRSV). Calves received a second vaccination with the same product at weaning. Each year cows and bulls received an inactivated vaccine at breeding containing IBR and type I and type II BVDV.

Sample Selection and Collection

In the first year, blood samples were collected from all replacement heifers (n=919) and breeding bulls (n=82). Calves from three cow/calf herds were bled once at branding (4-6 weeks of age) and again at weaning (4-6 months of age). At branding, there were 561 calves in herd A, 623 in herd B and 736 in herd C. At weaning, herd A contained 540 calves, herd B had 571 and herd C contained 673. Decreases in calf numbers were due to calf mortalities and the sale of cow/calf pairs prior to weaning (55 cow/calf pairs from herd B and 62 pairs from herd C).

In the second year, blood samples were collected from calves at branding from herd A (n=532), herd B (n=479) and herd C (n=585). At weaning, 522 calves in herd A, 631 in herd B and 580 calves in herd C were tested. The replacement heifer and breeding bull herds tested in the first year were not retested the second year.

Blood was collected from cattle by jugular or tail venipuncture into 5 ml purple-topped (EDTA) blood collection (Vacutainer®) tubes. Plasma was separated and stored at 39.2°F (4°C) until completion of virus isolation and was then stored at -4°F (-20°C).

Virological Examination

Plasma samples from replacement heifers, breeding bulls and calves at branding and weaning were assayed for BVDV antigen by microtiter virus isolation (MTVI) ELISA using previously described methods.[21] A sample was considered positive for BVDV if specific antigen staining was observed in the cytoplasm of BT cells examined by light microscopy. Blood samples from dams of BVDV-positive calves were collected and tested for the presence of virus by MTVI.

Reverse-Transcriptase Polymerase Chain Reaction (RT-PCR)

If a sample was positive by MTVI, the buffy coat was collected for reverse-transcriptase polymerase chain reaction (RT-PCR) as previously described.[20] A second blood sample taken three weeks after the initial sample was also assayed to confirm persistent infection. Contaminating red blood cells were lysed in tris-NH_4Cl buffer (0.85% w/v NH_4CL in 0.1M Tris) at 39.2°F (4°C) for 10 minutes. The Tris-NH_4Cl mixture was centrifuged at 1500 rpm for 10 minutes, the supernatant was decanted and the cells were washed with 500 ml of Hank's balanced salt solution (HBSS) and centrifuged a second time at 1500 rpm for 10 minutes to remove residual Tris-NH_4Cl. Total RNA was extracted from buffy coat cells (Rneasy Mini Kit, Qiagen, Inc., Valencia, CA), as well as from plasma and cell culture media. Diethylpyrocarbonate (DEPC) water was used as a negative control while RNA extracted from Singer (type Ia), NY-1 (type Ib), and 125c (type II) infected cell cultures were used as positive BVDV controls. PCR products were separated on a 1.5% agarose gel containing 3.3% ethidium bromide at 90V for approximately 30 minutes. Further genotyping of the type I BVDV strains into type Ia and type Ib was performed as previously described.[19]

Serum Neutralization Assay

Plasma from calves collected at weaning as well as samples collected from bulls and replacement heifers were assayed for antibody titers to BVDV types I and II using standard serum neutralization (SN) methods.[5] Antibody titers were determined for all animals sampled in year one. In year two, SN titers were determined for 50% of the calves.

Data Analyses

Basic statistics were computed and a Chi-square analysis was performed using the FREQ procedure in SAS.[22] Fisher's exact one-sided test was used, adjusting for multiple comparisons, to determine if the probability of observing an animal with a high SN titer for type I and type II BVDV in each herd (A, B, C) exceeded the probability in each other herd. The SN titer data were then transformed and analyzed using a generalized linear model to compare geometric means, adjusting for multiple comparisons, between the three herds. A statistical level of 0.05 was considered significant.

Results

In the first year, blood samples from 401 calves in herd A were available shortly after birth. Two animals

Table 1. BVDV PI screening of replacement heifers, bulls and calves in three cow/calf herds (A, B and C) by MTVI and RT-PCR.[a]

Year 1	Total samples	PIs	PI prevalence	Genotype
Cow/calf Herd A	561	2	0.36%	Type Ib
Herd B	623	2	0.32%	Type II
Herd C	736	0	0.00%	n/a
Replacement heifers	919	1	0.11%	Type II
Breeding bulls	82	0	0.00%	n/a
Year 2	**Total samples**	**PIs**	**PI prevalence**	**Genotype**
Cow/calf Herd A	532	0	0.00%	n/a
Herd B	585	0	0.00%	n/a
Herd C	479	0	0.00%	n/a

[a]Replacement heifers and breeding bulls were not tested in year two because they were tested in year 1.

were identified as persistently infected with a ncp type Ib BVDV by MTVI-ELISA and RT-PCR. An additional 164 calves from herd A were screened at branding (six weeks of age) and were negative for BVDV (Table 1). Two PI calves were found in herd B; one at branding and the second detected at weaning. Both were infected with ncp type II BVDV. The viremic calf identified at weaning was tested previously at branding, but gave a negative result by MTVI, presumably due to maternal antibody interference.[4] Serum neutralization titers of this calf at branding were 1:128 to type I BVDV and 1:64 to type II BVDV. At weaning, the calf had BVDV antibody titers <1:8 for type I and type II, which is consistent with a decline in maternal antibodies and persistent BVDV infection.

All PI animals were removed from the herd within three weeks of confirmation by RT-PCR. The dams of suspected PI calves were negative for BVDV. PI calves were not detected in herd C or in the bull herd (Table 1). The replacement heifers had been tested previously for PI status by skin biopsy (ear notch) immunohistochemistry, but were re-tested. A PI heifer was identified by MTVI and removed from the replacement heifer herd shortly after testing. The PI animal prevalence for the total animals on the ranch was 0.171% (5/2921). Herd A, B and the replacement heifers had PI animal prevalences of 0.36, 0.32 and 0.11%, respectively (Table 1). The second year, calves in herds A, B and C were tested at branding and weaning and no PI calves were identified.

BVDV Serology: Year 1

Serum neutralizing titers to type I and II BVDV were determined for blood samples collected at weaning, when calves were between four and six months of age. Serum neutralization (SN) titers ≥1:512 were considered to be the result of BVDV infection due to infectious contact with a PI animal versus due to residual maternal antibodies at this age.[20] The seroprevalence of calves with SN titers ≥1:512 to type I in all three cow/calf herds was 20.6% and 44.8% to type II BVDV (Figure 1). Comparison of the geometric mean titers (GMT) showed calves from all three cow/calf herds had significantly more individuals with high titers to type II than to type I BVDV (Figure 1). Replacement heifers and bulls also had more individuals with higher titers to type II than type I (data not shown).

A within-herd comparison showed significantly fewer animals had infectious contact with a PI animal in herd A than did herds B or C, and that the difference between animals with high SN titers in herd B was not significantly different than herd C (Figure 1).

BVDV Serology: Year 2

The BVDV seroprevalence of the cattle in year two was 5.91% (58/981) to type I BVDV and 18.04% (177/981) to type II BVDV. Comparison of GMT between the herds showed herds A and B had similar type I BVDV GMT, while herd C had a higher GMT. For type II BVDV, herd A had a lower titer than B, which had a lower titer than herd C ($P<0.05$). When compared to the GMT from year one, the seroprevalence of the three herds in year two was statistically lower.

Discussion

BVDV eradication programs have been implemented in several European countries.[2,6,13] However, the focus of these programs has been European dairy herds. The successful application of the same BVDV control measures to extensively managed beef herds in the western US is uncertain. In endemically infected beef herds, the constant presence of PI animals results in high herd immunity where many cows have been exposed to BVDV and have potentially developed sufficient immunity to prevent fetal infection.[7] When only a few susceptible animals are left in a herd, the probability of the birth of PI calves decreases. Consequently, the probability of infectious contact with a PI animal decreases. Ironically, as the proportion of immune cows in the herd increases, the proportion of susceptible replacement heifers born to them and retained in the herd also increases. In subsequent years, the proportion of susceptible female cattle increases as does the probability of *in utero* infection leading to the birth of a PI calf increases. The presence of susceptible females that can give birth to PI calves allows BVDV infection to continue in a herd.

The two PI calves identified in herd A in year one were removed before six weeks of age, therefore, the chance of a susceptible cow having infectious contact with PI animals during pregnancy was eliminated. Reduced BVDV transmission in herd A is supported by the lower percentage of calves with high SN titers at weaning compared to calves in herd B in year one. In

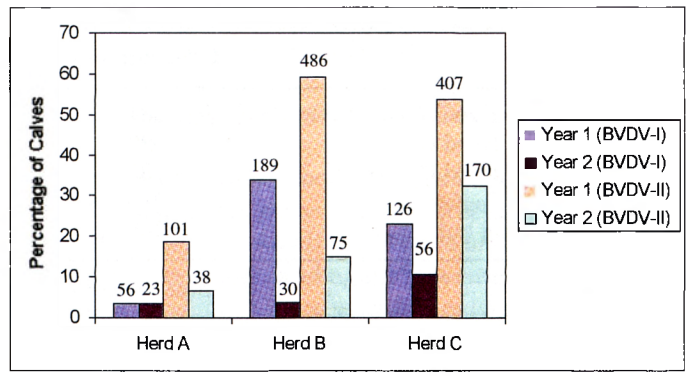


Figure 1. Percentage of calves from three cow/calf herds (A, B and C) with high SN titers (≥512): year 1 and 2. Numbers indicate GMT.

the first year, two PI calves were identified in herd B, one at branding and the other at weaning. If the PI calves had remained in herd A through weaning, as was the case with herd B, a higher percentage of calves with SN titers >1:512 to BVDV would be expected as a result of continued transmission within the cohort.[4,8] PI calves were not identified in herd C; however, the percentage of calves with high SN titers was similar to herd B, suggesting the presence of a PI calf or transient BVDV transmission by acutely infected animals.

Sixty-two cow/calf pairs from herd C were sold between branding and weaning. Maternal antibodies may have prevented the identification of a PI calf in this herd when it was sampled at branding. Alternatively, BVDV transmission has been reported in herds where no PI animals were detected.[17] Acutely infected cattle may have spread BVDV to herd mates in herd C, leading to a high proportion of animals with elevated SN titers.

Three of the PI animals identified were infected with a type II BVDV strain (herd B and replacement heifers). Both of these herds had higher type II antibody titers than type I, indicating a higher rate of infection with type II BVDV. The percentage of animals with high titers (≥1:512) to type I was most likely due to cross-reaction of type II antibodies with the type I virus used in the SN assay.[9,14] Of all calves with high SN titers, 92.45% had titers to type II BVDV greater than or equal to the type I titer (data not shown). Two type I PI calves were identified in herd A; however, significantly more calves had high antibody titers to type II (18.52%) than to type I (3.52%) ($P<0.05$). There may have been some cross-reaction of type I antibodies with type II virus, but herd A was also pastured next to herd C on at least one occasion between branding and weaning. If herd C had contained a type II PI calf or acutely infected animals the virus may have spread to calves in herd A, resulting in high type II SN titers.

No PI calves were identified in year two and the seroprevalence rates (5.91% type I, 18.04% type II BVDV) were significantly lower than in year one. The significant decrease in the number of animals with high SN titers supports the hypothesis that removal of PI animals can reduce the spread of BVDV.

Conclusions

Seroprevalence and PI animal prevalence data from an endemically infected beef herd provides evidence that removal of PI animals from a beef herd prior to breeding is effective at reducing the transmission of BVDV and eliminating PI animals from a beef herd. The impact of BVDV infection and eradication efforts on production values has not been investigated. It is hypothesized that removal of PI animals will positively affect herd health in the long term. In this study, herd health and animal performance at the time of testing did not appear to be affected by the presence of PI animals. This may have been further complicated by the application of drought management strategies at the same time. The economical benefits of this control strategy to producers will require cost-benefit analysis of herd production data and diagnostic testing.

Acknowledgments

The authors would like to acknowledge the technical assistance of Anita Schiebel and Kathi Wilson for skillful help and instruction with virus isolation and serum neutralization assays. This study was supported by grants from the College of Veterinary Medicine and Biomedical Sciences at Colorado State University and the USDA:CSREES funded Program of Economically Important Infectious Animal Diseases at Colorado State University.

References

1. Baker JC: The clinical manifestations of bovine viral diarrhea infection. *Vet Clin North Am Food Animal Pract* 11:425-445, 1995.
2. Barber DML, Nettleton PF: Investigations into bovine viral diarrhoea virus in a dairy herd. *Vet Rec* 133:549-550, 1993.
3. Bolin SR, McClurkin AW, Coria MF: Frequency of persistent bovine viral diarrhea virus infection in selected cattle herds. *Am J Vet Res* 56:2385-2387, 1985.
4. Brock KV, Grooms DL, Ridpath J, *et al*: Changes in levels of viremia in cattle persistently infected with bovine viral diarrhea virus. *J Vet Diag Invest*10:22-26, 1998.
5. Carbrey EA, Brown LN, Chow TL, *et al*: Recommended standard laboratory techniques for diagnostic infectious bovine rhinotracheitis, bovine virus diarrhea, and shipping fever (parainfluenza-3). *Proc United States Animal Health Assoc,* 1971, pp 629-648.
6. Ferrari G, Scicluna MT, Bonvincini D, *et al*: Bovine virus diarrhoea (BVD) control programme in an area in Rome province (Italy). *Vet Micro* 64:237-245, 1999.
7. Fredriksen B, Sandvik T, Loken T, *et al*: Level and duration of serum antibodies in cattle infected experimentally and naturally with bovine virus diarrhoea virus. *Vet Rec* 144:111-114, 1999.
8. Grooms DL, Kaiser L, Walz PH, *et al*: Study of cattle persistently infected with bovine viral diarrhea virus that lack detectable virus in serum. *J Am Vet Med Assoc* 219:629-631, 2001.
9. Hamers C, Di Valentin E, Lecomte C, *et al*: Virus neutralising antibodies against 22 bovine viral diarrhea virus isolates in vaccinated calves. *Vet Journal* 163:61-67, 2002.
10. Houe H: Epidemiologic features and economical importance of bovine virus diarrhoea virus (BVDV) infections. *Vet Micro* 64:89-107, 1999.
11. Houe H, Baker JC, Meas RK, *et al*: Prevalence of cattle persistently infected with bovine viral diarrhea virus in 20 dairy herds in two counties in central Michigan and comparison of prevalence of antibody-positive cattle among herds with different infection and vaccination status. *J Vet Diagn Invest* 7:321-326, 1995.
12. Houe H, Meyling A: Prevalence of bovine virus diarrhoea (BVD) in 19 Danish dairy herds and estimation of incidence of infection in early pregnancy. *Prevent Vet Med* 11:9-16, 1991.
13. Houe H, Palfi V: Attempts at preventing further spread of bovine virus diarrhoea virus (BVDV) infection in 5 Danish dairy herds in which BVDV had been isolated. *Acta Veterinaria Scandinavica* 34:139-144, 1993.

14. Jones L, Van Campen H, Xu ZC, *et al*: Comparison of neutralizing antibodies to type 1a, 1b and 2 bovine viral diarrhea virus from experimentally infected and vaccinated cattle. *Bov Pract* 35:137-140, 2001.
15. Kelling CL, Stine LC, Rump KK, *et al*: Investigation of bovine viral diarrhea virus infections in a range beef cattle herd. *J Am Vet Med Assoc* 197:589-593, 1990.
16. Mainar-Jaime RC, Berzal-Herranz B, Arias P: Epidemiological pattern and risk factors associated with bovine viral-diarrhoea virus (BVDV) infection in a non-vaccinated dairy-cattle population from the Asturias region of Spain. *Prevent Vet Med* 52:63-73, 2001.
17. Moerman A, Straver PJ, de Jong MCM, *et al*: A long term epidemiological study of bovine viral diarrhoea infections in a large herd of dairy cattle. *Vet Rec* 132:622-626, 1993.
18. Paisley LG, Wells S, Schmitt BJ: Prevalence of bovine viral diarrhea antibodies in 256 U.S. cow-calf operations: a survey. *Theriogenology* 46:1313-1323, 1996.
19. Ridpath JF, Bolin SR: Differentiation of types 1a, 1b and 2 bovine viral diarrhea virus (BVDV) by PCR. *Molecular and Cellular Probes* 12:101-106, 1998.
20. Ridpath JF, Bolin SR, Dubovi EJ: Segregation of bovine viral diarrhea virus into genotypes. *Virology* 205:66-74, 1994.
21. Saliki JT, Fulton RW, Hull SR, *et al*: Microtiter virus isolation and enzyme immunoassays for detection of bovine viral diarrhea virus in cattle serum. *J Clin Micro* 35:803-807, 1997.
22. SAS. *SAS/STAT Guide for personal computers.* 8 ed. Cary, N.C., SAS Institute, Inc, 2001.
23. Sorensen JT, Enevoldsen E: Dynamic stochastic simulation as a tool for studying bovine virus diarrhea virus infections at the herd level. *Vet Res* 94:25:317-321, 1994.
24. Taylor LF, Janzen ED, VanDonkersgoed J: Losses over a 2-year period associated with fetal infection with the bovine viral diarrhea virus in a beef cow-calf herd in Saskatchewan. *Can Vet J* 38:23-28, 1997.
25. Wentink GH, van Exsel CA, de Goey I, *et al*: Spread of bovine virus diarrhoea virus in a herd of heifer calves. *Vet Quarterly* 13:233-236, 1991.
26. Wittum TE, Grotelueschen DM, Brock RV, *et al*: Persistent bovine viral diarrhea virus infection in US beef herds. *Prevent Vet Med* 49:83-94, 2001.

Abstract

Effects of Neonatal Calf Oral Rehydration Therapy Solutions on Milk Clotting Time

Nappert G., Spennick H.
Cattle Practice 11(4):285-288, 2003

The effect of 50 commercially formulated neonatal calf oral rehydration therapy solutions on clotting time of milk *in vitro* has been investigated. Rennet was used as the clotting agent. Electrolyte solutions that contained large amounts of bicarbonate, and/or citrate (>40 mEq/L), and/or glucose had negative effects on milk clotting time. Isotonic oral electrolyte solutions that contained mainly acetate and propionate did not interfere with milk clotting time if low amounts of citrate (10 mEq/L) were included. As our knowledge of the nutritional requirements of the calf's digestive tract has become more precise, the feeding of diarrhoeic calves with whole cows milk in combination with these respective metabolisable-base oral electrolyte solutions to correct or prevent dehydration and metaboilic acidosis has been recommended world-wide.

PYRAMID® 5
with MetaStim®

Serious Protection

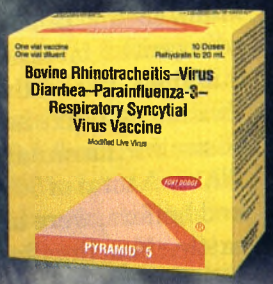
Type I & II BVD Protection

Two specifically selected strains offering broad protection and increased immune response.

Proven Safety

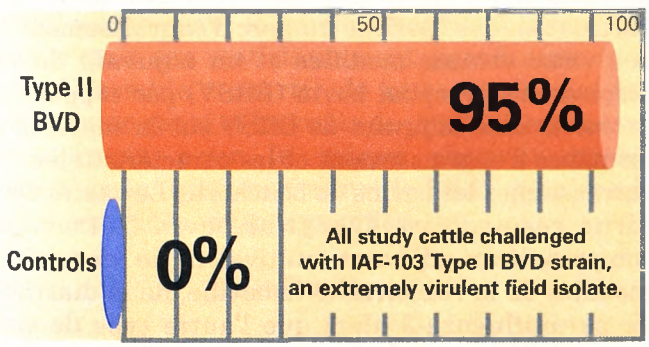
Licensed by the USDA for Sub-Q administration and proven safe when used according to label directions.

Stimulating Results Against BVD



Type II Respiratory Challenge Results[1]

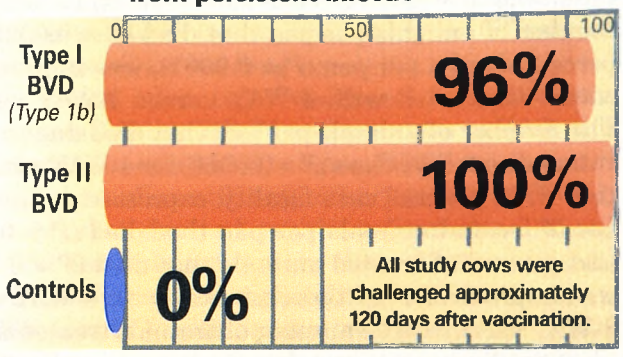
Protection from Disease
(sum of all clinical parameters)



***95%** of PYRAMID 5 vaccinated calves showed no clinical Type II BVD signs following virulent challenge.*

BVD Fetal Challenge Results[1]

% fetuses protected from persistent infection



***Up to 100%** fetal protection against Type I and Type II BVD challenges.*

1. Data on file.

FORT DODGE®

PYRAMID® 5
with MetaStim®