Acta vet. scand. 1998, *39,* 89-98.

The Duration of Antibodies Against Bovine Virus Diarrhoea Virus in Bulk Milk

By B. Fredriksen[1], T. Løken[2] and S. A. Ødegaard[1]

[1]Department of Reproduction and Forensic Medicine, and [2]Department of Large Animal Clinical Sciences, Norwegian College of Veterinary Medicine, Oslo, Norway.

Fredriksen B, Løken T, Ødegaard SA: The duration of antibodies against bovine virus diarrhoea virus in bulk milk. Acta vet. scand. 1998, 39, 89-98. – Fifty-eight dairy herds, suspected to be recently infected with bovine virus diarrhoea virus (BVDV) due to a rise in BVDV antibodies in bulk milk, were followed over a two-year period. In 34 (59%) of these 58 herds (Group 1), pooled milk samples from heifers or pooled blood samples from calves were negative for BVDV antibodies. In this group as many as 53 and 76% of the herds again had antibody-negative bulk milk one and two years after the positive sample, respectively. Of the remaining herds, 5 and 17 % had negative samples after one and 2 years respectively.

Possible explanations for the limited duration of antibodies against BVDV in bulk milk are discussed. In 65% of the herds in Group 1, animals had been purchased and introduced into the herd, or the herds had been exposed to other forms of contact representing a risk of infection with BVDV. In the remaining 35% of the herds in this group, no explanation for the rise in BVDV antibodies in the bulk milk could be found. In this study the introduction of seropositive animals into the herd, and infection inducing seroconversion in one or more animals as the only result, seem to be the most probable explanations for the rise in antibody levels observed in Group 1.

risk factors; transmission.

Introduction

Bovine virus diarrhoea virus (BVDV) in cattle is a member of the genus Pestivirus in the family Flaviviridae (*Collett et al.* 1989) as are border disease virus (BDV) in sheep and classical swine fever virus (CSFV) in pigs. Infection with BVDV in susceptible immunocompetent cattle most often cause a subclinical infection, resulting in production of neutralizing antibodies (*Howard* 1990). However, infection of pregnant animals may affect the foetus, and cause early reproductive failure (*Virakul et al.* 1988, *Houe & Meyling* 1991a, *McGowan et al.* 1993), abortions (*Casaro et al.* 1971, *Roeder et al.* 1986, *Carlsson et al.* 1989), congenital defects (*Done et al.* 1980), growth retardations (*Done et al.*1980, *Houe & Meyling* 1991a) and birth of dead or weak calves (*Lohr* 1983, *Woodard* 1994). If the infection occurs before the foetus becomes immunocompetent to the virus, which takes place at about 120 days of gestation, calves may be born which are immunotolerant to the agent and which remain persistently infected (PI) for the rest of their lives (*Done et al.* 1980, *McClurkin et al.* 1984). Since PI animals continue to excrete large amounts of virus as long as they live, they constitute the main source of new infections (*Roeder & Harkness* 1986, *Houe* 1992). This is especially evident when such animals are introduced into susceptible herds either as an infected foetus or as a live animal. The purchase of animals is therefore probably the most common cause of BVDV infection in previously uninfected

herds, although investigations have indicated that the number of infected herds may be higher than can be explained solely on the basis of the purchase of PI animals (*Houe & Palfi* 1993).

The distribution of BVDV is worldwide, and the prevalence of seropositive cattle is mostly reported to be from 50% to 90% (Baker 1987). In Norway, *Løken et al.* (1991) reported a prevalence of seropositive cattle of 18,5 %, seropositive cattle being found in 28 % of the investigated herds. In a control programme, 23 % of all dairy herds in Norway were found to have medium or high levels of antibodies against BVDV in bulk milk (*Waage et al.* 1994).

Infections with BVDV cause huge economical losses (*Houe et al.* 1993), and the virus being, after mastitis, probably the most costly cause of illness in dairy cattle in many countries today. Several countries are now implementing control programmes. Identification of herds with an active BVDV infection is a crucial point in these programmes. Most often this means presence of one or more PI animals. Testing of individual animals is expencive and time-consuming. The use of tests that measure the antibody level in milk is an invaluable and unexpensive tool, if the herd has not been vaccinated against BVDV. *Niskanen* (1993) and *Alenius* (1992) showed that there was good correlation between the percentage of seropositive milking cows in a herd, and the level of BVDV antibodies in the bulk milk. However, a single examination of the antibody level in the bulk milk, does not give any indication of how recent the herd was infected (*Alenius* 1992), so examination of milk or blood from the young stock, is often used as additional tools, since antibodies in samples from this group of animals will indicate recent infection in the herd. An alternative way of identifying recently infected herds could be to use serial samples of bulk milk, and to concentrate on herds with an increase in antibody level, eventually with a set cut-off value.

In Norway a national programme with the ultimate goal of eliminating BVDV was launched in 1992. (*Løken & Krogsrud* 1993). In this programme, bulk milk from all dairy herds is examined for BVDV antibodies. All positive herds are followed up by the examination of pooled milk samples from heifers, and if antibodies are detected in these, also with examinations of pooled blood samples from calves. Only herds with positive test results on all these 3 samplings, are candidates for a full screening, with examination of individual blood samples from all animals. No BVDV vaccines is used in Norway, and consequently BVDV antibodies in blood and milk is always a result of natural infection.

The present study was performed to rule out the likelihood of active infection with BVDV in dairy herds in which the antibody status of the bulk milk had changed from negative to positive, using a simple cut-off line to distinguish between negative and positive herds. Explanations for a possible temporary increase in BVDV antibodies in bulk milk are discussed.

Materials and methods

Examinations for antibodies

All milk samples, stored at -20°C, were examined for antibodies against BVDV by an indirect enzyme-linked immunosorbent assay (ELISA) as described by *Niskanen et al.* (1989) (Svanovir®, Svanova Biotech, Sweden). The results were automatically read, and given as absorbance values at 450 nm. Bulk milk samples with values < 0.25 were characterised as negative for antibodies to BVDV, while those with a level ≥ 0.25 were characterised as positive.[1]

[1] A few of the bulk milk samples were analysed by the Norwegian Cattle Association (NRF). The remaining samples were analysed by The Veterinary Institue in Oslo, Sandnes, Trondheim or Harstad.

Table 1. Herds grouped according to findings of BVDV antibodies in pooled samples of milk from heifers and blood from calves at the start of the study. All herds had experienced an increase in antibody level in bulk milk previous to the presented results.

Heifer milk	Calf blood	Group
25 -	ND	Group 1
	9 -	n = 34
29 +	16 +	Group 2
	4 ND	n = 24
4 ND	4 +	

Heifer milk +/- = based on the level of BVDV antibodies in a pooled milk sample from 2-5 heifers.
Calf blood +/- = based on the level of BVDV antibodies in a pooled blood sample from 3-5 calves 8-12 months of age.
ND = Not done.

This was the cut-off line used by the control programme for further testing of the herds. Pooled milk samples from heifers were characterized as positive if the absorbance value was ≥ 0.1. This value was set lower than for bulk milk, because of the purpose of detecting even single seropositive animals. Pooled blood samples from calves were similarly examined for BVDV serum antibodies by an indirect ELISA (*Juntti et al.* 1987). The samples were characterized as positive for antibodies to BVDV when the absorbance value was ≥ 0.25.

Herds

The sampling frame consisted of about 1700 herds with accessible result of bulk milk testing (B_0), in the 18 month period preceeding the first national screening in 1993. This was mostly results of regional screenings performed by dairies or regional veterinarians, while about half of the samples origined from a pilot project in Rogaland county. Of the 1700 herds, 959 did have an antibody level in bulk milk characterized as negative. In 75 of these herds (7.8%), the bulk milk was positive for BVDV antibodies in the first screening of the control programme (B_1). These 75 herds that experienced a change in antibody status from negative to positive within a time period of 18 months, constituted the study group. According to the control programme, further antibody examination of a pooled milk sample from 2-5 heifers should be performed, and if this sample was positive, also of a pooled blood samples from 3-5 calves aged from 8 to 15 months. Four herds were closed down during the two-year period of the study, and from 13 herds neither heifer milk nor calf blood was available for testing. These herds are characterized as drop outs, and the final number of herds in the study is 58.

The herds were divided into 2 groups according to the results of analyses of the samples from heifers and calves (Table 1). Group 1 included all herds with negative heifer milk, and the herds that had positive heifer milk but negative blood sample from calves. Group 2 included all the herds with positive calf blood samples, and 4 herds where this blood sample was not available, but where the heifer milk was positive. In both groups, bulk milk samples were examined for BVDV antibodies one (B_2), and two (B_3) years after the study started. In one herd, individual milk samples from all milking cows were examined for BVDV antibodies shortly after B_1.

The number of cows per herd was available in 46 (79%) of the herds, ranging from 4.2 to 28.8 between the herds. The average number of dairy cows per herd was 12,3 and 13.8 in group 1 and 2 respectively.

Questionnaires

Questionnaires were submitted to all herds in Group 1, to investigate factors which could possibly influence the level of BVDV antibodies in bulk milk. The questionnaire included data about purchase of animals, sharing of pasture, and other forms of contact with herds suspected to be sources of BVDV infection (Table 2).

Table 2. Results of questionnaire from herds with BVDV antibody-negative pooled samples of milk from heifers or blood from calves (Group 1), regarding herd management practices during the 18 months before the antibody-positive bulk milk sample.

Risk factors	Number of herds with the risk factor present/ total number of herds (%)
Introduction of one or more animal into the herd[1]	13/28 (46)
Newly calved heifer - bought when calf[2]	2/20 (10)
Shared pasture[1]	6/28 (21)
Pasture with shared border to other herds[2]	2/20 (10)
Visiting animals[2]	1/20 (5)
Regulary indirect contact with other herds (exemplified as other farmers visiting the barn)[2]	2/20 (10)
None of the risk factors present[2]	7/20 (35)

1: Answered by 85% of the cattle owners
2: Answered by 58% of the cattle owners

Results

Occurence of antibodies

In Group 1, 18 (53%) of 34 bulk milk samples (B2) were negative for antibodies one year after the positive sample, and 25 (76%) of 33 after 2 years (B_3) (Table 3). Three herds were negative at B_2 but positive again at B_3. In Group 2, only one of 22 (5%) bulk milk samples were negative after one year, and 4 of 24 (17%) after 2 years. The absorbance values of antibodies in the bulk milk in Groups 1 and 2 were not significantly different at B_0, but were significantly higher in Group 2 than in Group 1 at the next 3 samplings (Mann-Whitney test) (Fig. 1). In Group 2, the mean antibody level remained high in both years, while in Group 1, it decreased during the whole study period, with negative values (abs < 0.25) at the last 2 samplings.

Data given in the questionnaires

Twenty (59%) of the 34 farmers in Group 1 answered all the questions on the questionnaire, while a further 8 (24%), answered only the questions about animal trading and grazing. An overall total of 28 (82%) then answered these latter questions. In 13 (46%) of these 28 herds (Table 2), one or more animals had been intro-

Table 3. Antibodies against BVDV in bulk milk in 3 groups of dairy herds in which such antibodies were demonstrated at the start of the study, but not in bulk milk less than 18 months earlier, respectively one (B_2) and 2 (B_3) years after the start of the study.

Group	B_2	B_3
Group 1 (n = 34)	18 (53%) negative/16 (47%) positive	25 (76%) negative/8 (24%) positive/1 ND
Group 2 (n = 24)	1 (5%) negative/21 (95%) positive/2 ND	4 (17%) negative/20 (83%) positive
Total (N = 58)	19 (34%) negative/37 (66%) positive/2 ND	29 (51%) negative/28 (49%) positive/1 ND

ND = Not done
For definition of Groups 1 and 2, see Table 1.

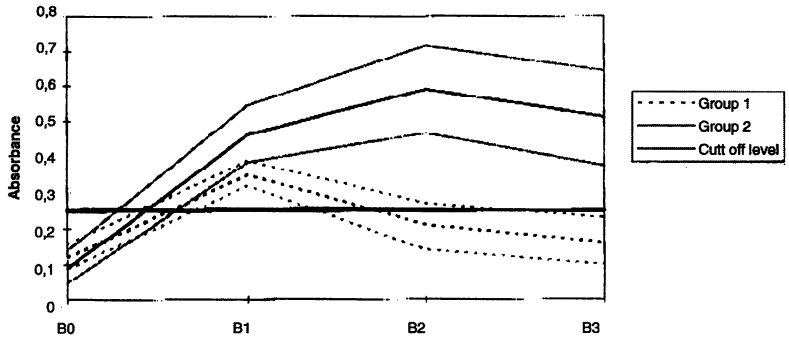

Figure 1. Mean level of BVDV antibodies in bulk milk (ELISA, absorbance at 450nm), with 95% confidence intervals, less than 18 months before the start of the study (B_0), at the start of the study (B_1), and one year (B_2) and two years (B_3) later. For definition of Groups 1 and 2 see Table 1.

duced into the herd during the last 18 months before the positive bulk milk sample (B_1). In one herd a single heifer was shown to be antibody positive while all the other dairy cows were negative. The animal in question was bought-in as a young calf, and contributed about 10% to the herd bulk milk at the time of sampling.

Six herds grazed common pasture with other herds, while 2 herds had pasture with a common border to pasture grazed by herds which were, or were suspected to be infected with BVDV. In one herd, a cow from another herd had been housed for a short period, and in 2 herds there had been regular indirect contact with herds suspected to be infected with BVDV. In 7 of the herds for which complete data were available, none of the mentioned risk factors were present.

Discussion

The fact that a bulk milk sample from a herd is tested negative for BVDV antibodies one year, but tests positive the next year, as in all the present herds, generally indicates that several milking cows have seroconverted due to an effective source of infection in the herd. The most effective BVDV source is one or more PI animal(s) in, or in close contact with, the herd (*Meyling et al.* 1990). In such cases antibody levels in the bulk milk would be expected to increase considerably after only a few months, and to remain high for several years. Unexpectedly, in this study, however, the bulk milk from 34% of the herds tested negatively for antibodies to BVDV one year after the primary positive test, and as many as 51% tested negative after two years (Table 3).

There are strong indications that infection with BVDV induces prolonged production of antibodies which are mostly of lifelong duration (*Duffell & Harkness* 1985). On the other hand, such long-lasting antibody levels could be a result of reinfections, as PI animals are usually present in the herd for a long time. The results of some studies, however indicate that immunity after natural infection is not lifelong. A decrease in the serum antibody titres being found after a few months, was subsequently increasing again in some animals (*Howard* 1990). On the other hand there is no reported evidence indicating that antibody production induced after a BVDV infection is of so short duration as to provide an explanation for the only temporary

increase in antibody levels in the bulk milk found in nearly half of the herds in the present study.

With regard to the 29 herds in which the bulk milk became negative for BVDV antibodies again after one or 2 years, as well as the additional 8 herds in Group 1 in which pooled milk samples from heifers or pooled blood samples from calves were negative, it is unlikely that any PI animal had been present in the herd during the first year after the positive bulk milk sample. A PI animal that is together with the rest of the herd will infect most of the susceptible animals in the course of a few months (*Houe & Meyling* 1991b, *Wentink et al.* 1991). Certainly, the young animals with no antibodies in samples of milk or blood, could not have been raised in close contact with PI animals.

There are several possible explanations for the temporary increase of BVDV antibodies in the bulk milk. In some of the herds, there was probably an active infection in one or a few animals only. This could result from the introduction of a transiently infected animal into the herd. Such transient infection have been shown to occur (*Barber & Nettleton* 1993, *Moermann et al.* 1993). In other herds, direct contact could have taken place between animals at pasture, including contact over the fence, which has been shown to be an important route of infection (*Roeder & Harkness* 1986). Such direct contact between PI animals and susceptible animals is the most common route of BVDV infection (*Duffell & Harkness* 1985). In Norway, it is a common practice for 2 or more herds to graze a common pasture. In the present study 21% of the herds in Group 1 shared pasture with other herds, while another 10% were on pasture with a common boundary to pastures grazed by other herds. Sheep infected with BDV have also been shown to infect cattle and vice versa (*Gibbons et al.* 1974, *Carlsson* 1991, *Carlsson & Belák* 1994). BVDV in cattle and BDV in sheep are closely related (*Wensvoort et al.* 1989), and should probably be regarded as different strains of the same virus. Independent of route the infection results in one or more seropositive animals in the herd(s), which in turn might well result in the the bulk milk becoming positive for BVDV antibodies.

The introduction of seropositive animals into a herd can result in a positive bulk milk sample, even if such animals do not represent a risk of infection. Forty-six percent of the farmers in Group 1 had introduced one or more animals into the herd during the last 18 months before the positive bulk milk sample. In dairy herds, a positive relationship between the sero-prevalence for BVDV and the level of BVDV antibodies in bulk milk has been demonstrated (*Niskanen et al.* 1991, *Alenius* 1992, *Niskanen* 1993). However, it has also been shown that the presence of just a few animals with high serotitres can result in high levels of antibodies in the bulk milk, this sometimes leading to an overestimation of the number of seropositive animals, especially in small herds. The average herd size in Norway is small, only 12.8 dairy cows/herd (1995), so that even a small number of seropositive animals in the herd may give a positive bulk milk sample. In one of the herds, a single seropositive heifer, contributing about 10% of the bulk milk, was the cause of an antibody-positive bulk milk sample.

Some animals in the herd may not be in lactation when the bulk milk is sampled. Variation in respect to whether seropositive animals are dry or lactating at the time of sampling could explain why one bulk milk sample is negative while the next one is positive, even if the herd consists of identical animals on both occasions. This effect will be highest in small herds, particularly if seasonal calving is practiced.

Indirect transmission of BVDV from PI animals has been a subject for speculation. In this study, 2 of the farmers suspected indirect con-

tact with other herds to be a possible source of infection. People and vehicles might be the source of infection in some cases, via contaminated working clothes, footwear, hypodermic needles or other veterinary equipment (*Pritchard* 1963, *Roeder & Harkness* 1986, *Bolin* 1990, *Gunn* 1993). In such cases, it is most likely that only a small number of animals, or even a single animal will be infected. Semen has also been shown to be a possible source of BVDV infection (*Meyling & Jensen* 1988, *Meyling et al.* 1990, *Kommisrud et al.* 1996). In Norway, all donor bulls used for artificial insemination have to be negative when tested for persistent BVDV infection. However, some bulls seroconvert during semen production, and such acutely infected bulls have been shown to excrete low levels of virus in semen for 4-6 days (*Kirkland et al.* 1991). Other indirect routes of transmission, such as embryo transfer (*Liess et al.* 1987), vaccination (*Lohr et al.* 1983, *Liess et al.* 1984) and vector borne transmission (*Tarry et al.* 1991), are much less probable in the present study.

It might also be possible that variation in the strain of virus could be responsible for differences in the duration of antibody production, and also in the initial antibody titres induced by the infection.

Virus in milk from PI animals might neutralize antibodies in the bulk milk (*Niskanen* 1995). In such cases, the measured antibody level will be too low, and in extreme cases this could result in false negative samples. This being so, it is therefore possible that some herds were incorrectly included in this study, if the first negative bulk milk sample was in fact a false negative. Nevertheless, such herds, in which one or more PI animals were present, would not be expected to show a decreasing antibody level in the bulk milk during the subsequent 2 years, and the inclusion of such herds would therefore not have influenced the results of the study. It is also theoretically possible that some of the negative B_2 or B_3 were such false negatives, and further testing of pooled milk samples from heifers would have been necessary to show that this was not so.

It is of course essential that results of the antibody testing are correct. Both the sensitivity and specificity of the method used for determination of antibodies in this study are high (*Juntti et al.* 1987, *Niskanen et al.* 1989), so the majority of the test results should be fairly correct. However, there will always be the possibility of samples being mixed, and of false analytical results. Because the criteria for inclusion of a herd in this study was that a bulk milk sample should first be negative and then positive, and because a simple cut-off level was used to distinguish between negative and positive samples, some herds showing only a slight increase in antibody level in the bulk milk were included. Alternatively, one could have chosen to include only herds showing an increase in antibody level from below 0.25 to higher than, for instance, 0.40. In this way many of the herds with negative bulk milk both one and 2 years later would have been excluded, but so would a considerable number of herds with an antibody level higher than 0.40 at one of the later samplings. This alternative would have resulted in a lot of infected herds being excluded.

In the Norwegian control programme, the criterium of an antibody positive pooled blood sample from calves is used as a supplement to the antibody positive bulk milk sample to characterize the herds as probably being currently infected with BVDV (*Waage et al.* 1994). The results of the present study indicate that this seems a reasonable approach as 20 of 24 of such herds still had bulk milk positive for antibodies 2 years after the primary positive sample, while using only the change from negative to positive bulk milk, only half of the herds still had antibody positive bulk milk after 2 years.

Aknowledgements

We are grateful to the Norwegian Control Programme for BVD/MD represented by its leader Ola Nyberg, for kindly letting us use data from the programme. We also appreciate being allowed to use data from Dag Lindheim's pilot project in Rogaland county, as well as the results of antibody testing done by the Norwegian Cattle Association (NRF), Hamar, concerning the year prior to the start of the national programme.

References

Alenius S: Bovint virusdiarrévirus hos nötkreatur. Symptom, diagnostik, profylax och bekämpande [Bovine virus diarrhoea virus (BVDV) in cattle]. Svensk Vet.-Tidn. 1992, *44,* 51-62.

Baker JC: Bovine viral diarrhea virus: A rewiew. J. Amer. vet. med. Ass. 1987, *190,* 1449-1458.

Barber DML, Nettleton PF: Investigations into bovine viral diarrhoea virus in a dairy herd. Vet. Rec. 1993, *133,* 549-550.

Bolin SR: Control of bovine virus diarrhoea virus. Rev. sci. tech. Off. int. Epiz. 1990, 9, 163-171.

Carlsson U: Border disease in sheep caused by transmission of virus from cattle persistently infected with bovine virus diarrhoea virus. Vet. Rec. 1991, 128, 145-147.

Carlsson U, Belák K: Border disease virus transmitted to sheep and cattle by a persistently infected ewe: Epidemiology and Control. Acta vet. scand. 1994, *35,* 79-88.

Carlsson U, Fredriksson G, Alenius S, Kindahl H: Bovine virus diarrhoea virus, a cause of early pregnancy failure in the cow. J. vet. Med. A, 1989, *36,* 15-23.

Casaro APE, Kendrick JW, Kennedy PC: Response of the bovine fetus to bovine viral diarrhea-mucosal disease virus. Amer. J. vet. Res. 1971, *32,* 1543-1562.

Collett MS, Moennig V, Horzinek MC: Recent advances in pestivirus research. J. gen. Virol. 1989, *70,* 253-266.

Done JT, Terlecki S, Richardson C, Harkness JW, Sands JJ, Patterson DSP, Sweasey D, Shaw IG, Winkler CE, Duffell SJ: Bovine virus diarrhoea-mucosal disease virus: Pathogenicity for the fetal calf following maternal infection. Vet. Rec. 1980, *106,* 473-479.

Duffell SJ, Harkness JW: Bovine virus diarrhoea-mucosal disease infection in cattle. Vet. Rec. 1985, *117,* 240-245.

Gibbons DF, Winkler CE, Shaw IG, Terlecki S, Richardson C, Done JT: Pathogenicity of border disease agent for the bovine foetus. Brit. vet. J. 1974, *130,* 357-360.

Gunn HM: Role of fomites and flies in the transmission of bovine virus diarrhoea virus. Vet. Rec. 1993, *132,* 584-585.

Houe H: Bovin virusdiarré (BVD) II. Epidemiologi. (Bovine virus diarrhoea (BVD) II. Epidemiology). Dansk vet. Tidsskrift, 1992, *75,* 321-326.

Houe H, Meyling A: Surveillance of cattle herds for bovine virus diarrhoea virus (BVDV)-infection using data on reproduction and calf mortality. Arch. Virol. 1991a, Suppl 3, 157-164.

Houe H, Meyling A: Prevalence of bovine virus diarrhoea (BVD) in 19 Danish dairy herds and estimation of incidence of infection in early pregnancy. Prev. vet. Med. 1991b, 11, 9-16.

Houe H, Palfi V: Estimation of herd incidence of infection with bovine virus diarhoea virus (BVDV) in herds previously without animals persistently infected with BVDV. Acta vet. scand. 1993, *34,* 133-137.

Houe H, Pedersen KM, Meyling A: A computerized spread sheet model for calculating total annual national losses due to bovine viral diarrhoea virus infection in dairy herds and sensitivity analysis of selected parameters.

Proceedings of the 2nd Symposium on Ruminant Pestiviruses, Annecy, 1993, 179-184.

Howard CJ: Immunological responses to bovine virus diarrhoea virus infections. Rev. sci. tech. Off. int. Epiz. 1990, *9,* 95-103.

Juntti N, Larsson B, Fossum C: The use of monoclonal antibodies in enzyme linked immunosorbent assay for detection of antibodies to bovine viral diarrhoea virus. J. vet. Med. B, 1987, *34,* 356-363.

Kirkland PD, Richards SG, Rothwell JT, Stanley DF: Replication of bovine viral diarrhoea virus in the bovine reproductive tract and excretion of the virus in semen during acute and chronic infections. Vet. Rec. 1991, *128,* 587-590.

Kommisrud E, Vatn T, Lang-Ree JR, Lɕken T: Bovine virus diarrhoea virus in semen from acutely infected bulls. Acta vet. scand. 1996, *37,* 41-47.

Liess B, Orban S, Frey H-R, Trautwein G, Wiefel W, Blindow H: Studies on transplacental transmissibility of a Bovine Virus Diarrhoea (BVD) vaccine virus in cattle. Zbl. Vet. Med. B, 1984, *31,* 669-681.

Liess B, Frey H-R, Grambow H, Stahl C: Embryo-

transfer und BVD-virusinfection bei Rindern. (Embryotransfer and BVD-virusinfection in cattle). Dtsch. tierärztl. Wschr. 1987, *94,* 506-508.

Lohr CH, Evermann JF, Ward AC: Investigation of dams and their offspring inoculated with a vaccine contaminated by bovine viral diarrhea virus. Vet. Med. /Small Animal Clinician, 1983, 1263-1266.

L¢ken T, Krogsrud J, Larsen IL: Pestivirus infections in Norway. Serological investigations in cattle, sheep and pigs. Acta vet. scand. 1991, *32,* 27-34.

Løken T, Krogsrud J: Programme for making the Norwegian cattle free from pestivirus. Proceedings of the 2nd Symposium on Ruminant Pestiviruses, Annecy, 1993, 179-184.

McClurcin AW, Littledike ET, Cutlip RC, Frank GH, Coria MF, Bolin SR: Production of cattle immunotolerant to bovine viral diarrhea virus. Canad. J. comp. Med. 1984, *48,* 156-161.

McGowan MR, Kirkland PD, Richards SG, Littlejohns IR: Increased reproductive losses in cattle infected with bovine pestivirus around the time of insemination. Vet. Rec. 1993, *133,* 39-43.

Meyling A, Houe H, Jensen AM: Epidemiology of bovine virus diarrhoea virus. Rev. sci. tech. Off. int. Epiz. 1990, *9,* 75-93.

Meyling A, Jensen AM: Transmission of bovine virus diarrhoea virus (BVDV) by artificial insemination (AI) with semen from a persistently-infected bull. Vet. Microbiol. 1988, *17,* 97-105.

Moermann A, Straver PJ, de Jong MCM, Quak J, Baanvinger Th, van Oirschot JT: A long term epidemiological study of bovine viral diarrhoea infections in a large herd of dairy cattle. Vet. Rec. 1993, *132,* 622-626.

Niskanen R: Relationship between the levels of antibodies to bovine viral diarrhoea virus in bulk tank milk and the prevalence of cows exposed to the virus. Vet. Rec. 1993, *133,* 341-344.

Niskanen, R: Bovine Virus Diarrhoea Virus Infections in Dairy Cattle with emphasis on diagnosis, epidemiology and control. Swedish University of Agricultural Sciences. Thesis. 1995. pp. 52.

Niskanen R, Alenius S, Larsson B, Jacobsson S: Determination of level of antibodies to bovine virus diarrhoea virus (BVDV) in bulk tank milk as a tool in the diagnosis and prophylaxis of BVDV infections in dairy herds. Arch. Virol. 1991, Suppl 3, 245-251.

Niskanen R, Alenius S, Larsson B, Juntti N: Evaluation of an enzymed-linked immunosorbent assay for detection of antibodies to bovine virus diarrhoea virus in milk. J. vet. Med. B, 1989, *36,* 113-118.

Pritchard WR: The bovine viral diarrhea-mucosal disease complex. Advanc. vet. Sci. 1963, *8,* 1-47.

Roeder PL, Harkness JW: BVD virus infection: Prospects for control. Vet. Rec. 1986, *118,* 143-147.

Roeder PL, Jeffrey M, Cranwell MP: Pestivirus fetopathogenicity in cattle: Changing sequelae with fetal maturation. Vet. Rec. 1986, *118,* 44-48.

Tarry DW, Bernal L, Edwards S: Transmission of bovine virus diarrhoea virus by blood feeding flies. Vet. Rec. 1991, *128,* 82-84.

Virakul P, Fahning ML, Joo HS, Zemjanis R: Fertility of cows challenged with a cytopathic strain of bovine viral diarrhea virus during an outbreak of spontaneous infection with a noncytopathic strain. Theriogenology, 1988, *29,* 441-449.

Waage S, Krogsrud J, Nyberg O: The Norwegian programme for eradication of bovine viral diarrhoea/mucosal disease. Proceedings XVIII World Buiatric Congress, August 29-September 2, 1994 Bologna, Italy, pp. 773-776.

Wensvoort G, Terpstra C, de Kluyver EP: Characterization of porcine and some ruminant pestiviruses by cross-neutralization. Vet. Microbiol. 1989, *20,* 291-306.

Wentink GH, van Exsel ACA, de Goey I, van Lieshout JAH: Spread of bovine virus diarrhoea virus in a herd of heifers calves. Vet. quarterly, 1991, *14,* 233-236.

Woodard LF: BVD virus associated with outbreaks of abortion, stillbirths, and weak calves. Vet. Med. 1994, *89,* 379-384.

Sammendrag

Varigheten av antistoffer mot bovin virusdiaré-virus i tankmelk

På grunnlag av stigning i nivået av antistoffer mot bovin virusdiaré-virus (BVDV) i tankmelk, ble femtiåtte melkekubesetninger antatt å være nyinfiserte med BVDV, og fulgt i en toårsperiode. I 34 (59%) av besetningene (Gruppe 1), var samlemelkprøver fra kviger eller samleblodprøver fra ungdyr negative for antistoffer mot BVDV. I denne gruppen hadde hele 53 og 76% av besetningene igjen antistoff-negativ tankmelk etter henholdsvis ett og 2 år. Av de resterende besetninger var det henholdsvis 5 og 17% som hadde negative prøver etter ett og 2 år. Mulige forklaringer til den kortvarige stigningen av antistoffer mot BVDV i tankmelk er diskutert. I 65%

av besetningene i Gruppe 1, var nye dyr blitt kjøpt inn, eller besetningene hadde på annen måte blitt utsatt for kontakt som kunne representere risiko for smitte med BVDV. I de resterende 35% av besetningene i denne gruppen ble det ikke funnet noen forklaring på den observerte stigningen av BVDV-antistoffer i tankmelken. I denne undersøkelsen ser introduksjon av seropositive dyr i besetningen, og infeksjon som induserer serokonversjon hos et enkelt eller et fåtall dyr som eneste konsekvens, ut til å være de mest sansynlige forklaringene på den observerte stigningen av antistoffnivået i tankmelk i Gruppe 1.

(Received January 10, 1997, accepted August 15, 1997).

Reprints may be obtained from: Bente Fredriksen, Department of Reproduction and Forensic Medicine, Norwegian College of Veterinary Medicine, P.O. Box 8146, Dep., N-0033 Oslo, Norway. E-mail: bente.fredriksen@veths.no, tel: +47 22 96 48 85, fax: +47 22 59 70 83.