

Managing BVD.

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What is BVD and what does it do?

In cows, BVD virus infection causes many reproductive problems including reduced conception rates ([McGowan et al., 1993](#)), abortion, persistent life-long infection of the fetus, or fetal deformities ([Grooms, 2006](#)), and late gestation infection can result in small weak calves and affect the future reproduction of the foetuses ([Munoz-Zanzi et al., 2004](#)). BVD virus also causes immune suppression increasing the rate and severity of mastitis and other infections ([Niskanen et al., 1995](#); [Waage, 2000](#)). Somatic cell counts can be increased due to BVD virus exposure ([Beaudeau et al., 2005](#)), and milk production has been shown to decrease with exposure to BVD virus in individuals ([Moerman et al., 1994](#)) and Heuer *et al.* (2007) found 5% lower average production in New Zealand dairy herds with evidence of recent infection (high bulk tank antibody level).

Persistently infected (PI) animals come from fetal infection in the first 4 months (125d) of pregnancy ([Grooms, 2006](#)). These PIs tend to be ill-thrifty ([Hessman et al., 2009](#)) and have a high (50% per year) death and culling rate ([Houe, 1993](#)). Voges *et al.* (2006 non-peer-reviewed) reported a case series of PI cows involved in another trial in New Zealand where the PIs grew 18% more slowly and had 22% poorer general health. Three quarters survived to enter the milking herd but they produced half as much milk as expected based on their genetic potential, and much less than their peers, and did not survive well in the herd despite no culling for production under the conditions of the trial (8% alive at 4.5 years compared to 71% of their non-PI full sibling peers).

Calves are affected more severely by transient BVD virus infection with increased severity of other diseases with concurrent BVD infection ([Castrucci et al., 1992](#); [Brodersen and Kelling, 1998](#)). The antibody response ([Elvander et al., 1998](#)), and the number of immune cells in the blood ([Ganheim et al., 2005](#)) both drop with BVD virus infection and even more so with other infections at the same time. BVD virus infection is associated with higher risk of death and lower weaning weights ([Waldner and Kennedy, 2008](#)), and Hessman *et al.* (2009) found calves with direct contact with PI calves had a 20% lower average daily weight gain despite vaccination.

The effect of BVD virus on bulls can be dramatic as well. Although PI bulls can have acceptable semen quality, they often have poor fertility ([Bielanski and Loewen, 1994](#)). Even transient infection can have a marked effect on semen quality ([Paton et al., 1989](#); [Kirkland et al., 1997](#)), as well as being a source of infection for cows ([Paton et al., 1989](#); [Brownlie et al., 2000](#); [Givens et al., 2002](#)).

BVD virus infection is most often spread by direct contact with a PI ([Houe, 1999](#)). Transiently infected cows shed virus for a few days ([Brownlie et al., 1987](#)), but there is some debate whether it is enough to be infectious ([Meyling and Jensen, 1988](#); [Moerman et al., 1993](#); [Niskanen et al., 2002](#); [Moen et al., 2005](#)). Infection can be spread by indirect contact through equipment ([Gunn, 1993](#)), and through interactions such as rectal examination ([Langree et al., 1994](#)). Airborne transmission is possible under some circumstances ([Mars et al., 1999](#)) but it is unclear how important this is under New Zealand conditions.

What can you do about it?

BVD control is all about PIs. Since PIs are main source of infection, finding and removing PIs, stopping them from coming into the herd, and preventing them from being created should always be the focus of BVD control. Without exposure to PIs, the rest of the stock will not suffer the effects of infection.

There are 3 categories of activities or approaches to controlling BVD infection. The test and cull approach involves testing blood, ear notch, or milk samples for virus to identify possible PI animals, and then removing any confirmed PIs from the herd. Since PIs are the main source of infection, removing the PIs is usually a rapidly successful way to stop the spread and the effects of BVD infection. If money is spent on clearing infection, it makes sense to take steps to prevent the reintroduction of infection so on-going screening of introductions is often part of any BVD control program.

The other main approach to controlling infection is through vaccination. There is some debate about how effective vaccination is at preventing any possible consequences, but it is likely that vaccination alone will not be 100% effective. It is however a very useful tool to greatly reduce if not prevent new generations of PIs being formed. The main goal of vaccination in terms of BVD control should be to protect the fetus and thereby cut off the supply of new PIs. As a bonus, vaccination will mostly stop the reproductive losses (abortions and reduced conception rates) as well.

The final approach to BVD control can be described as management changes where actions are taken to reduce the risk of new introduction of infection or maintenance of infection once present. This includes things like doing artificial breeding right through so that bulls are not required (untested or unvaccinated bulls are an important potential risk to the herd), and choosing not to buy in any cows since cows and their calves are an important potential route for BVD infection to enter a herd. Improving boundaries through permanent double fencing border paddocks, or even outriggers or temporary hotwires is one of the most important management-type control measures.

These control activities can be applied systematically to each of the potential risks to the herd. All risks to the herd can be categorised as bulls, bought cows and their calves, neighbours stock, new born calves, and people (mostly people's equipment and clothing). A summary of how control approaches can be applied to these risks is shown in Table 1.

Bulls should always be tested and vaccinated before contact with the herd. Ideally the vaccination should be done 2 months before required for service because transient infection can cause dramatically reduced fertility in bulls for up to 2 months after infection, but that can be difficult to manage and the greatest risk of exposure is probably during transport and mixing with new stock so vaccination shortly before sale is more common. If a herd doesn't use bulls, obviously this risk does not apply.

Many herds in New Zealand buy cows (40% per year in an unpublished study by the author). These cows themselves are a potential risk to the herds they enter, and since many enter the herd while pregnant, their fetus is a potential risk once it is born as it may be persistently infected already. These PI fetuses are commonly referred to as a Trojan calf from the Trojan horse story. All bought cows should be tested to ensure they are not PI, and any calves that may be kept should be tested as well. Due to the high apparent mismothering rate in most New Zealand herds, any herd with bought cows should either test all replacement calves even if not intending to retain calves from bought cows, or calve bought cows separately and either bobby or test their calves. Obviously if cows are not bought, this risk does not apply.

Contact over the fence with neighbours stock is an important risk and seems to be a common way for infection to be introduced onto a farm. To minimise this risk, boundary fences should be strong and prevent stock breaking through (especially calves), but should also prevent nose to nose contact. This can be achieved with double fencing, or possibly with outriggers. Another option may be to put up a temporary hotwire along the boundary whenever neighbours stock may be present. Also remember

that races are at least as important as paddocks and probably greatly increase the chance of contact if you or your neighbour's race run along a boundary. Spread through the air from nearby stock may be an issue even if direct contact cannot occur but that cannot easily be prevented and it is unclear how commonly this occurs. Further control beyond trying to prevent contact should therefore be considered. Vaccinating the herd will minimise the impact of any contact that does occur and will usually prevent fetuses becoming PI following any contact. Testing all replacement calves is a way to catch any breakdown before it becomes a disaster. By finding and culling any PI calves before they can cause further damage in the herd, annual calf screening is an effective way to control this risk.

Many potential sources of exposure such as neighbours stock or indirect contact through equipment will affect only a few cows at a times. Those few individuals may suffer consequences such as aborting, but the main issue for the herd is if any end up with a PI fetus which is then kept as a replacement. Many breakdowns are only discovered after a PI calf is born on the property and causes a major outbreak in the herd. It is useful to consider calves as a risk to the herd and try to minimise contact between cows and calves, especially after the start of mating when a new generation of PIs could be made. Heifers and any other cows grazing off the home farm during the first 4 months of pregnancy are at particularly high risk of having a PI calf. Testing replacement calves every season is a great way to protect the herd from this and other risks. Vaccinating the herd, or at risk groups such as carry over cows and heifers will reduce the risk, and calving any at risk groups separately and not keeping their calves may be useful options.

The final risk to the herd involves indirect contact with PIs through people moving between herds or mobs and carrying infectious material. The virus is very fragile and does not survive for very long outside a host (hours to days), but PIs shed huge amounts of virus in all secretions including faeces, urine, saliva, and nasal secretions. Any equipment or gear that has been in contact with stock or stock waste should be cleaned before coming into contact with other stock. Basic hygiene is likely sufficient to destroy the virus but gross contamination can be effective at transferring infection. This applies to other farmers, vets, scanners, AI technicians, embryo transfer techs, and stock transporters or other vehicles that could transfer any fresh material from one farm to another. Expect and encourage best practice from people coming onto your property and minimise situations where cows (especially pregnant cows) may be exposed to any contamination. Testing all replacement calves each year will catch any minor breakdown before it turns into a disaster, and vaccinating the herd will minimise this risk as well.

The main risks to the herd can be summarised with "in, out, and over" – that is stock coming into the farm, stock that are off the farm in the first 4 months of pregnancy, and over the fence contact with neighbours stock.

So in short, testing replacement calves or vaccinating the herd are the main options for control of BVD and whatever else you do, bulls, bought cows, and calves from bought cows should be tested for virus and bulls should be vaccinated before arrival.

Simulation Model

Because BVD is fairly complex in the way it behaves in a herd, the effects it can have on cows, and how it moves between herds, there's no way to directly measure all the effects of BVD in one grand study and put a number on what it costs let alone including various control options. New Zealand is also quite different to many other places in the world with our very seasonal calving, predominantly pasture fed systems. There are many sources of information which paint part of the picture of what BVD can do and it's easy to get the impression that BVD is bad and causes substantial losses, but if farmers are to invest in control measures, there should be a reasonable case for an economic

benefit. It's just too complex with too many interactions for anyone to hold it all in their head and calculate what the least cost approach would be with any confidence. That was the reason for building a suitably complex simulation model that puts all the information together and crunches the numbers for us.

The concept of the model is fairly intuitive. It simulates each cow in a herd with a list of statuses which can change each day as events occur. For example calves and heifers grow at age and genetic potential appropriate rates, cows have heats & are mated, then either conceive or return to oestrus 18 to 24 days later. If they conceive, then the pregnancy advances (unless they abort) and 282 days later (give or take 2 weeks), they calve and a new animal either enters the herd, or is sold. Then cows have an anoestrus period of variable length before starting to cycle again and can be mated depending on the stage of the season.

Cows go through a season producing milk at the rate appropriate to their age, individual potential (genetic potential, individual variation around their potential, and the herd's variation from average to account for management effects), and time since calving following a typical lactation curve. The production can be interrupted or depressed due to the effects of disease or treatment withholding period. Cows can die due to disease or misadventure, most often around calving. Discretionary culling is based on a herd-specific rank weighting system of production, age, mastitis history, and "other", while non-pregnant cows are either culled or carried over depending on a herd-specific carryover rate and the culling score (the best cows according to the herd's rating system are carried over). Replacement cows can be bought if planned, or if there were not enough replacements coming through to account for the enforced losses.

This herd simulation system was examined and tweaked until measures such as replacement rate, production patterns, culling, 3 week submission rate, 6 week in calf rate, empty rate, age distribution, the probability of buying any cows and the number bought all matched the pattern seen in New Zealand. A disease simulation and tracking system was added to this system where many of the effects of BVD infection could happen and the effect on the herd and the cost recorded.

Animals move through mobs including new born pen; larger calf mob and management such as rotational calf grazing or a few to a paddock; heifers grazing off farm; dry cows (on or off farm); springing cows; colostrum cows; sick cows; and milking cows (can be up to 3 milking cow mobs). Contacts between cows within a mob, between mobs on the farm, and contacts with neighbours stock are drawn each day if there are any infected individuals, and new infections can result. Many of the BVD disease effects mentioned earlier occur in the model at rates and severity consistent with those reported in research papers and the costs are summed at the herd level.

Each time the model is run, it creates a herd based on many values which are drawn from patterns that match New Zealand herds. Things like number of neighbours for example are defined as a number between zero and about 10 with the most common being 2 neighbours (fits questionnaire data). One of 15 potential BVD control strategies is selected for the herd at creation and applied right through that simulation. The BVD infection status and immunity level, the herd size, and the region are drawn from a pool of about 3,800 herds that did bulk tank testing with LIC in the 2011 season. Once a herd is created, it is simulated for 10 years and a summary of the results stored. This is repeated about 10,000 times to get a data set of around 10,000 simulated herds to analyse.

These results were analysed with a linear regression model where total cost (effects of BVD infection plus any costs associated with control) was dependent on herd size and control strategy. The control options investigated (Table 2) were:

1. No control
2. Bull test only (single virus test before the breeding season)
3. Bull test and vaccination only
4. Clear infection and bull testing. Annual bulk tank testing with a search and removal of any persistently infected animals (PIs) if required.
5. Clear infection and test any new arrivals. Same as 4 but also includes bull vaccination and virus testing any purchased stock (only adult cows or pregnant heifers were purchased in this model).
6. Test all new arrivals. Includes 3 and testing all new introductions but no PI search and cull if any enter by another route.
7. Test in and test replacement calves. Same as 6 but also screens new born calves each year.
8. Clear, test in and calves. Same as 7 but also includes a PI search and cull from the herd if required.
9. Vaccination build up. Only the first calving cows are vaccinated in the first season, then for the next 2 seasons only previously vaccinated and first calving cows are vaccinated, then the whole herd is vaccinated after that. Calves and heifers are also vaccinated from the first season and bulls are tested and vaccinated each year.
10. Vaccinate first calving cows. Calves, heifers, and first calving cows are vaccinated, and bulls are tested and vaccinated.
11. Vaccinate the herd. Same as 10 but the whole herd is vaccinated each year.
12. Clear and vaccinate the herd. Same as 11 but a PI search and cull is also conducted whenever bulk tank testing indicates that there is infection present in the herd.
13. Clear, test in and vaccinate the herd. Same as 12 but any purchased cows are also tested before entry.
14. Full biosecurity. Includes 8 and calves and heifers are vaccinated but cows are not vaccinated. Also the boundary is double fenced or temporary hotwires are used as needed to prevent over the fence contact (with a chance to fail depending on the type of boundary control).
15. Full biosecurity and herd vaccination. Same as 14 but the herd is also vaccinated each year.

Results

Full Biosecurity (option 14) which included clearing infection, testing all incoming stock, vaccination of all except lactating cows and improved border fencing was the most cost effective approach resulting in an average NZ\$63,384 benefit over 10 years for an average sized herd (n = 406 cows). The next best option was to clear infection when required, test all incoming stock, and vaccinate the herd (option 13, \$53,229), closely followed by clear, test all incoming stock and screen replacement calves each year (8, \$53,019). The rest of the results can be seen in Table 1, and calculated total cost for an average herd by control type can be seen in Figure 1.

In simulated herd seasons with no BVD control (n=15,830), the 6 week in-calf rate for herds with virus in the bulk tank pre-mating in that season (n=1,970) ranged from 43% (5% quartile) to 77%

(95% quartile), with a median of 61.2%, while herds with no virus detected pre-mating (n=13,860) ranged from 48% to 80% (5% and 95% quartile) with a median of 66.8%. The final non-pregnant rate in herds with virus in the bulk tank pre-mating ranged from 4.9% to 30.2% (5% and 95% quartile) with a median of 13.2%, and herds with no virus in the bulk tank pre-mating ranged from 3.7% to 23.7% (5% and 95% quartile) with a median 9.5%. Some herds that tested negative pre-mating would have introduced infection later such as with a PI bull, and some herds that were positive pre-mating would have naturally cleared infection shortly after testing such as when a PI cow dies or is culled due to very low production around the start of mating.

All control options significantly decreased the cost of BVD in the regression model at $p < 0.001$ compared to no control, with a positive net benefit for the farm. The average cost of BVD was \$10,000 per year for the average herd (\$35,435 intercept and \$159 per cow over 10 years), or \$24.63 per cow and year with substantial variation between years when infection is present.

Generally, implementing more measures resulted in lower overall costs despite increasing control costs, except that vaccinating the entire herd (13) could be considered an alternative to testing replacement calves plus boundary controls (14) since both together (15) saved less than either one. Testing replacements was the superior choice on average although there are likely situations where vaccination would be superior, even if just for logistical reasons, and both are much better than no control.

Conclusions

The simulation model demonstrated that all, and any, control measures for BVD were financially attractive and better than no control. The most cost-effective strategies included either vaccinating the herd or screening incoming replacements with a virus test. Calf testing or herd vaccination appeared in all of the most cost-effective strategies and should be routinely undertaken. Additionally, the six most cost-effective strategies included ongoing monitoring of bulk tank milk for the presence of virus with identification and removal of virus positive cows and bull testing and vaccination, and 6 of the 7 most cost-effective strategies including screening cows entering the herd.

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Tables and figures

Table 1: BVD risk control options

RISKS TO HERD	TEST & CULL	VACCINATE	MANAGEMENT
Bulls	bulls	bulls	AB only
Bought cows	cow / bulk tank	~	don't buy in cows
Trojan calves	calves	~	calve separately & don't keep calves
Neighbours	calves	herd	border fence or manage
Calves	calves	herd	manage calf-cow contact

from cows away in 1st 4 months pregnancy	calves	before leave	isolate cows or don't keep
People (vets, scanners, AI, ET, transporters)	calves	herd	best practice / no contact

Table 2: Linear regression model of marginal total cost over 10 years without control or with various control options starting in year 1.

Code	Variable	Control options for the control type variable										n	Total cost (NZ\$)	sd ³
		Monitor	PI hunt	Test bought	Test bull	Vac ¹ bull	Vac ¹ calf	Vac ¹ heifer	Vac ¹ herd	Calf test	Border fencing ²			
	Intercept											12,570	\$ 35,435	
	n cows (mean 406)												\$ 159	
	Control type													
14	Full biosecurity	}	}	}	}	}	}	}	}	}	}	752	-\$ 63,384	23,038
12	Clear test in and vac ¹	}	}	}	}	}	}	}	}	}	}	776	-\$ 53,229	37,292
8	Clear test in and clv	}	}	}	}	}	}	}	}	}	}	788	-\$ 53,019	36,692
11	Clear and vac ¹	}	}	}	}	}	}	}	}	}	}	795	-\$ 50,899	32,073
15	Vac ¹ and test	}	}	}	}	}	}	}	}	}	}	804	-\$ 50,517	33,031
3	Clear and test in	}	}	}	}	}	}	}	}	}	}	789	-\$ 47,913	48,988
7	Test in and clv	}	}	}	}	}	}	}	}	}	}	752	-\$ 45,274	53,080
2	Clear only	}	}	}	}	}	}	}	}	}	}	821	-\$ 42,299	57,651
10	Vac ¹ first calver	}	}	}	}	}	}	}	1st clv	}	}	810	-\$ 28,355	102,892
9	Vac ¹ build up	}	}	}	}	}	}	}	}	}	}	762	-\$ 26,625	96,943
5	Bull test and vac.	}	}	}	}	}	}	}	}	}	}	784	-\$ 23,037	105,383
13	Vac ¹	}	}	}	}	}	}	}	}	}	}	790	-\$ 22,079	100,542
6	Test in	}	}	}	}	}	}	}	}	}	}	781	-\$ 21,080	98,010
4	Bull test only	}	}	}	}	}	}	}	}	}	}	783	-\$ 11,400	117,668
1	None											1,583	Ref	118,806

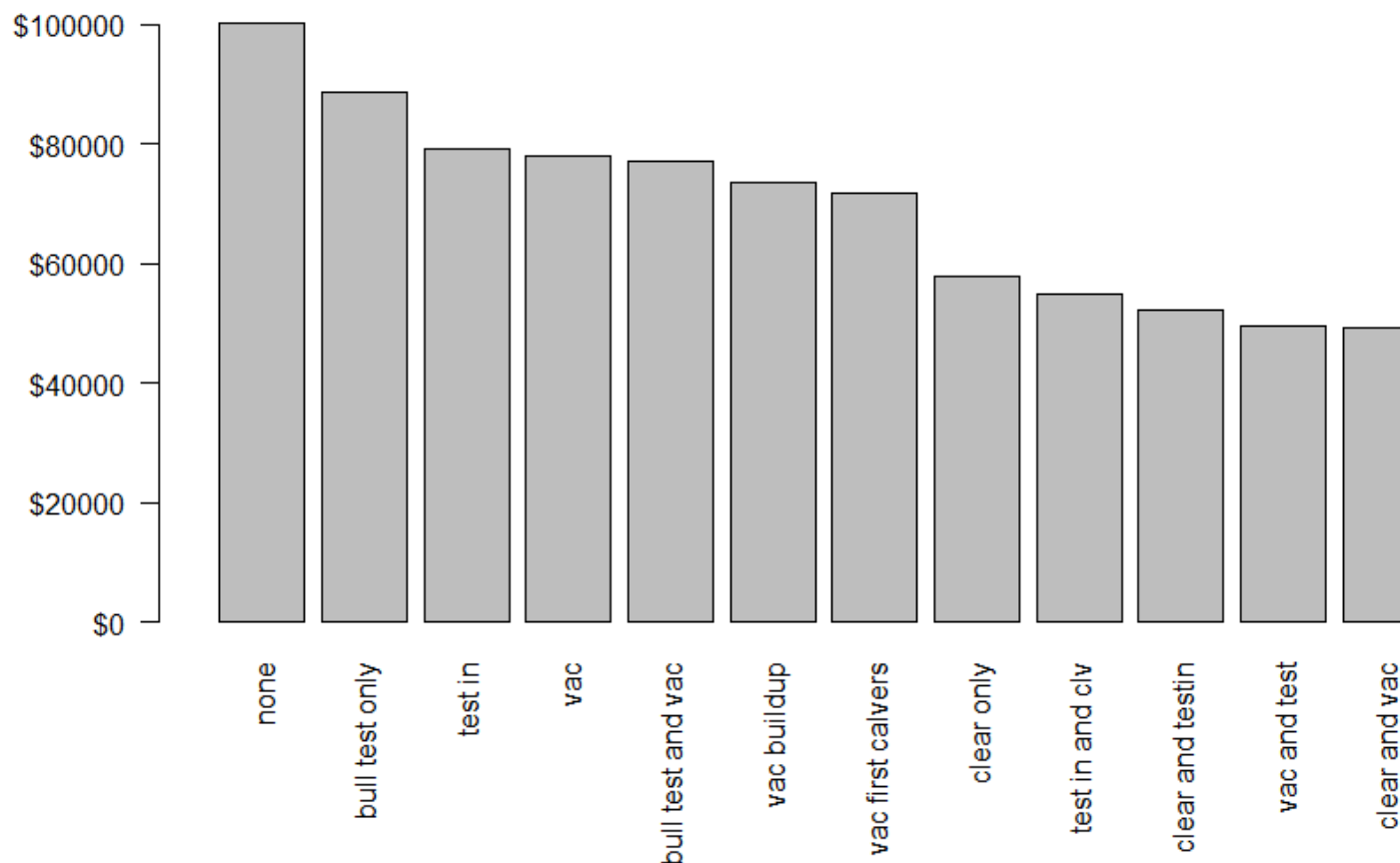
Interpretation: The total discounted cost of no control in a 406 cow herd is $\$35,435 + 406 \times \$159 + 0 = \$99,989/10$ years, while the total cost for the average herd using a full biosecurity approach would be $\$35,435 + 406 \times \$159 + -\$63,384 = \$36,605$ (see Figure 1). All p -values < 0.001 .

¹ Vaccination

² Either double fence, or use temporary hotwires around boundary with neighbours (50% chance of either being drawn)

³ Standard deviation of total cost in the data (not the model). This includes variation due to herd size but gives some indication of variability of the cost between herds and with different control types.

Figure 1: Average total discounted cost (NZ\$) over 10 years without control or with various control options starting in year 1 for a 406 cow herd estimated from the regression model from Table 1.



References

- BEAUDEAU, F., FOURICHON, C., ROBERT, A., JOLY, A. & SEEGER, H. 2005. Bulk milk somatic cell counts and bovine viral diarrhoea virus (BVDV) infection in 7252 dairy herds in Brittany (western France). *Preventive Veterinary Medicine*, 72, 163--167. Available: <Go to ISI>://000233944900023
- BIELANSKI, A. & LOEWEN, K. 1994. In-Vitro Fertilization of Bovine Oocytes with Semen from Bulls Persistently Infected with Bovine Viral Diarrhea Virus. *Animal Reproduction Science*, 35, 183--189. Available: <Go to ISI>://A1994NN25000004
- BRODERSEN, B. W. & KELLING, C. L. 1998. Effect of concurrent experimentally induced bovine respiratory syncytial virus and bovine viral diarrhoea virus infection on respiratory tract and enteric diseases in calves. *American Journal of Veterinary Research*, 59, 1423--1430. Available: <Go to ISI>://000076814700026
- BROWNLIE, J., CLARKE, M. C., HOWARD, C. J. & POCOCCO, D. H. 1987. PATHOGENESIS AND EPIDEMIOLOGY OF BOVINE VIRUS DIARRHEA VIRUS-INFECTION OF CATTLE. *Annales De Recherches Veterinaires*, 18, 157-166. Available: <Go to ISI>://A1987H894700007
- BROWNLIE, J., THOMPSON, I. & CURWEN, A. 2000. Bovine virus diarrhoea virus - strategic decisions for diagnosis and control. *In Practice*, 22, 176-+. Available: <Go to ISI>://000086523000002.
- CASTRUCCI, G., FERRARI, M., TRALDI, V. & TARTAGLIONE, E. 1992. Effects in Calves of Mixed Infections with Bovine Viral Diarrhoea Virus and Several Other Bovine Viruses. *Comparative Immunology Microbiology and Infectious Diseases*, 15, 261--270. Available: <Go to ISI>://A1992JV64100005

- ELVANDER, M., BAULE, C., PERSSON, M., EGYED, L., BALLAGI-PORDANY, A., BELAK, S. & ALENIUS, S. 1998. An experimental study of a concurrent primary infection with bovine respiratory syncytial virus (BRSV) and bovine viral diarrhoea virus (BVDV) in calves. *Acta Veterinaria Scandinavica*, 39, 251--264. Available: <Go to ISI>://000076227600010
- GANHEIM, C., JOHANNISSON, A., OHAGEN, P. & WALLER, K. P. 2005. Changes in peripheral blood leucocyte counts and subpopulations after experimental infection with BVDV and/or *Mannheimia haemolytica*. *Journal of Veterinary Medicine Series B-Infectious Diseases and Veterinary Public Health*, 52, 380--385. Available: <Go to ISI>://000233204400003
- GIVENS, M. D., HEATH, A. M., BROCK, K. V., BRODERSEN, B. W., CARSON, R. L. & STRINGFELLOW, D. A. Detection of bovine viral diarrhoea virus in semen obtained after inoculation of seronegative postpubertal bulls. Conference on Detecting and Controlling Bovine Viral Diarrhoea Virus Infections, Apr 04 2002 Ames, Iowa. Amer Veterinary Medical Assoc, 428-434.
- GROOMS, D. L. 2006. Reproductive losses caused by bovine viral diarrhoea virus and leptospirosis. *Theriogenology*, 66, 624--628. Available: <Go to ISI>://000239522000019
- GUNN, H. M. 1993. ROLE OF FOMITES AND FLIES IN THE TRANSMISSION OF BOVINE VIRAL DIARRHEA VIRUS. *Veterinary Record*, 132, 584-585. Available: <Go to ISI>://A1993LG25900006
- HESSMAN, B. E., FULTON, R. W., SJEKLOCHA, D. B., MURPHY, T. A., RIDPATH, J. F. & PAYTON, M. E. 2009. Evaluation of economic effects and the health and performance of the general cattle population after exposure to cattle persistently infected with bovine viral diarrhoea virus in a starter feedlot. *American Journal of Veterinary Research*, 70, 73-85. Available: <Go to ISI>://000262168400009
- HEUER, C., HEALY, A. & ZERBINI, C. 2007. Economic effects of exposure to bovine viral diarrhoea virus on dairy herds in New Zealand. *{JOURNAL OF DAIRY SCIENCE}*, {90}, {5428-5438}.
- HOUE, H. 1993. Survivorship of Animals Persistently Infected with Bovine Virus Diarrhoea Virus (Bvdv). *Preventive Veterinary Medicine*, 15, 275--283. Available: <Go to ISI>://A1993KZ09200004
- HOUE, H. 1999. Epidemiological features and economical importance of bovine virus diarrhoea virus (BVDV) infections. *Veterinary Microbiology*, 64, 89--107. Available: <Go to ISI>://000078438400002
- KIRKLAND, P. D., MCGOWAN, M. R., MACKINTOSH, S. G. & MOYLE, A. 1997. Insemination of cattle with semen from a bull transiently infected with pestivirus. *Veterinary Record*, 140, 124-127. Available: <Go to ISI>://A1997WG92500013
- LANGREE, J. R., VATN, T., KOMMISRUUD, E. & LOKEN, T. 1994. TRANSMISSION OF BOVINE VIRAL DIARRHEA VIRUS BY RECTAL EXAMINATION. *Veterinary Record*, 135, 412-413. Available: <Go to ISI>://A1994PP34200011
- MARS, M. H., BRUSCHKE, C. J. M. & VAN OIRSCHOT, J. T. 1999. Airborne transmission of BHV1, BRSV, and BVDV among cattle is possible under experimental conditions. *Veterinary Microbiology*, 66, 197--207. Available: <Go to ISI>://000079897800003
- MCGOWAN, M. R., KIRKLAND, P. D., RICHARDS, S. G. & LITTLEJOHNS, I. R. 1993. INCREASED REPRODUCTIVE LOSSES IN CATTLE INFECTED WITH BOVINE PESTIVIRUS AROUND THE TIME OF INSEMINATION. *{VETERINARY RECORD}*, {133}, {39-43}.
- MEYLING, A. & JENSEN, A. M. 1988. TRANSMISSION OF BOVINE VIRUS DIARRHEA VIRUS (BVDV) BY ARTIFICIAL-INSEMINATION (AI) WITH SEMEN FROM A PERSISTENTLY-INFECTED BULL. *Veterinary Microbiology*, 17, 97-105. Available: <Go to ISI>://A1988P909100001
- MOEN, A., SOL, J. & SAMPIMON, O. 2005. Indication of transmission of BVDV in the absence of persistently infected (PI) animals. *Preventive Veterinary Medicine*, 72, 93--98. Available: <Go to ISI>://000233944900012
- MOERMAN, A., STRAVER, P. J., DEJONG, M. C. M., QUAK, J., BAANVINGER, T. & VANOIRSCHOT, J. T. 1993. A LONG-TERM EPIDEMIOLOGIC-STUDY OF BOVINE VIRAL DIARRHEA INFECTIONS IN A LARGE HERD OF DAIRY-CATTLE. *Veterinary Record*, 132, 622-626. Available: <Go to ISI>://A1993LJ56700002

- MOERMAN, A., STRAVER, P. L., DEJONG, M. C. M., QUAK, J., BAANVINGER, T. & VANOIRSCHOT, J. T. 1994. Clinical Consequences of a Bovine Virus Diarrhea Virus-Infection in a Dairy-Herd - a Longitudinal-Study. *Veterinary Quarterly*, 16, 115--119. Available: <Go to ISI>://A1994PC70500011
- MUNOZ-ZANZI, C. A., THURMOND, M. C. & HIETALA, S. K. 2004. Effect of bovine viral diarrhea virus infection on fertility of dairy heifers. *Theriogenology*, 61, 1085--1099. Available: <Go to ISI>://000220010700009
- NISKANEN, R., EMANUELSON, U., SUNDBERG, J., LARSSON, B. & ALENIUS, S. 1995. Effects of Infection with Bovine Virus Diarrhea Virus on Health and Reproductive-Performance in 213 Dairy Herds in One County in Sweden. *Preventive Veterinary Medicine*, 23, 229--237. Available: <Go to ISI>://A1995RG59200010
- NISKANEN, R., LINDBERG, A. & TRAVEN, M. 2002. Failure to spread bovine virus diarrhoea virus infection from primarily infected calves despite concurrent infection with bovine coronavirus. *Veterinary Journal*, 163, 251--259. Available: <Go to ISI>://000176646600007
- PATON, D. J., GOODEY, R., BROCKMAN, S. & WOOD, L. 1989. EVALUATION OF THE QUALITY AND VIROLOGICAL STATUS OF SEMEN FROM BULLS ACUTELY INFECTED WITH BVDV. *Veterinary Record*, 124, 63-64. Available: <Go to ISI>://A1989R993800006
- VOGES, H., YOUNG, S. & NASH, M. 2006. Direct adverse effects of persistent BVDv infection in dairy heifers - a retrospective case control study. *Vetscript (non-peer-reviewed)*, 19, 22-25.
- WAAGE, S. 2000. Influence of new infection with bovine virus diarrhoea virus on udder health in Norwegian dairy cows. *Preventive Veterinary Medicine*, 43, 123-135. Available: <Go to ISI>://000084742300006
- WALDNER, C. L. & KENNEDY, R. I. 2008. Associations between health and productivity in cow-calf beef herds and persistent infection with bovine viral diarrhea virus, antibodies against bovine viral diarrhea virus, or antibodies against infectious bovine rhinotracheitis virus in calves. *American Journal of Veterinary Research*, 69, 916-927. Available: <Go to ISI>://000257295000015