Incidence, epidemiology and control of bovine pestivirus infections and disease in Australia and New Zealand

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Summary: Pestivirus infection of cattle is widespread and common in both Australia and New Zealand. The majority of adult animals, of the order of 60%, carry antibody. Associated disease is almost entirely that resulting from infection in utero. This includes death of the conceptus, at any stage from conception through pregnancy, or, in those which are born as persistently infected carriers, mucosal disease, most commonly in a chronic form. Little or no disease is recognised as a result of the post-natal infection of non-pregnant animals and these appear to be of little consequence as spreaders of infection. Transmission and enzootic maintenance depend primarily on the persistently infected carriers that are immunotolerant after early in utero infection and range clinically from normal, or nearly normal, to overtly mucosal diseased. The expulsion of an infected conceptus, and associated discharges, also provides an effective source of infection. There is generally little active control attempted. Vaccines are not available in Australia and are not widely used in New Zealand. However, interest in control is growing in those areas of the industry, especially in breeding by artificial insemination and embryo transfer, where it is perceived that the pathogenic impact of the virus may be amplified.

KEYWORDS: Australia - Bovine virus diarrhoea - Cattle diseases - Control - Epidemiology - Mucosal disease - New Zealand - Persistent infection - Pestivirus - Transmission.

INCIDENCE

In Australia, mucosal disease (MD) was first reported as a clinico-pathological entity in 1957 (3). More extensive disease observed in South Australia was described in 1959 as a “mucosal type disease” (17) and considered to be part of a mucosal disease-virus diarrhoea complex, but there is no evidence of it being due to a pestivirus infection and an alternative aetiology was subsequently suggested (7). Antibody to pestivirus, neutralising strain C24V, in bovine sera was reported in 1962 (6). Virus was recovered from cases of MD in 1964 (7) when the disease was recognised to occur in acute and chronic forms. Mucosal disease, more commonly chronic than acute,
has since been widely diagnosed in all parts of the country. In these cases the degree of development of the various pathological facets has been quite variable (15), and the full clinico-pathological spectrum of MD appears to range from classical disease to virtual normality. This is consistent with the fact that some carriers, with the specific immunotolerance and persistent viraemic infection that are prerequisite to MD, may be clinically normal over what appears to be a normal life span. Conversely, some lesions of chronic MD may be demonstrable in persistently infected (PI) carriers which die of any other cause.

The picture is generally similar in New Zealand, where a severe MD-like syndrome was reported in 1961 (25). Although cytopathogenic agents were isolated, these were never confirmed as pestiviruses and prominent kidney lesions suggested an alternative aetiology. In 1966 neutralising antibodies to strain C60F were reported in cattle (5) and the following year a cytopathogenic strain of pestivirus was isolated from a clinical case of acute MD (11). Reports of serological evidence of bovine pestivirus infections followed (4, 22). The incidence rate of clinical disease has not been well defined. Between 50 and 100 pestivirus isolations are made annually at the Ruakura Animal Health Laboratory and about 90% of these are non-cytopathogenic strains. The majority (58%) are recovered from beef cattle (J. Howell, personal communication). Acute MD occurs (9) but the chronic form is more common. A number of veterinary case reports have associated the disease with foot lesions (19), polyarthritis (8), seasonal factors (most common in autumn and winter), pneumonia, concurrent parasitism and bacterial infections such as Dermatophilus and Salmonella (1).

Bovine virus diarrhoea (BVD), as a disease of high morbidity and low mortality following post-natal infection, has never been observed in Australia, nor have other defined clinical signs been recognised in that context. In New Zealand, it is considered that some animals may show transient diarrhoea, oral erosions, leucopaenia and anorexia after post-natal infection.

Natural MD recognised in this region may not be strictly comparable, or limited, to the disease that is induced experimentally by the superinfection of a PI animal with a cytopathogenic pestivirus. It has been noted that cytopathogenic strains tend to be associated with more acute and non-cytopathogenic with more chronic cases (W.A. Snowdon, personal communication) but there was no strict rule to the relationship (7). Subsequently, no definitive association between cytopathogenic virus and MD has been found in diagnostic experience. This may be due, in part at least, to the common diagnostic practice of attempting virus isolation from blood of sick animals in preference to gut or other tissues which are only available post mortem. Early experience in examining a wider range of tissues suggests that cytopathogenic viruses are then more frequently recoverable (P.D. Kirkland, personal communication). The New Zealand experience is that, from natural cases of MD, non-cytopathogenic virus is frequently isolated from the blood clot and cytopathogenic virus from a different tissue or source, such as a nasal swab, of the same animal.

What is recognised as chronic MD may be comparable to what has elsewhere been termed ‘chronic BVD’. However, we see no clear line of demarcation between the acute and chronic forms of MD. For this reason, and also because no acute BVD is recognised, the option of referring to disease, due to persistent infection by non-cytopathogenic virus only, as ‘chronic BVD’ is not apposite. Both acute and chronic forms of MD have early in utero infection as an essential prerequisite.
Other consequences of early in utero infection have been observed as field cases (2, 21) and/or demonstrated after controlled natural infection with local strains of virus (18). These include early embryonic death, mummification, abortion, stillbirth and perinatal mortality, congenital abnormalities of the eye and central nervous system, and illthrift. The last two syndromes are seen with or without classical clinical signs and lesions of MD. However, in the south coastal regions of Western Australia, congenital abnormalities, stillbirth and illthrift are seen as the most common manifestations of disease while chronic MD is uncommon, perhaps because young stock, particularly steers, are turned off for slaughter before clinical signs develop (R. Reuter, personal communication).

The incidence of post-natal infection, as reflected by the prevalence of animals with serum antibody, is high and comparable to that described in others parts of the world where cattle industries are well developed. In Australia, no study on a national scale has been made since that published by St. George et al. in 1967 (23), when previous data (6, 7) were confirmed and neutralising activity against the C24V strain was found in 60% of individual sera and 90% of herds overall. Higher rates were found in the more northern areas and where herds are larger, although lesser figures (average within-herd prevalence of approx. 40%) for antibody prevalence in north Queensland have been found in a recent sample of 1,632 animals on ten properties (A. Janmaat, personal communication). More recent casual experience and observations would suggest that the 1967 figures would still be about right in the 1980's and, in testing of Western Australian cattle in 1985 for export purposes, approximately 60% were found to have antibody (T.M. Ellis, personal communication). In South Australia, from specimens submitted over approximately two years, including all of 1988, for the diagnosis of herd problems (mostly abortion), antibody was found in 76% (of 70) herds and 45% (of 777) individual animals (A.M. Pointon, personal communication). In the larger of the New Zealand serological studies (4), 34% of cattle were found to have antibody neutralising the local Bovax strain (11). However, an analysis of serological testing on diagnostic sera over ten years (G.W. Horner, unpublished data) showed 58% of sera to have neutralising antibody, and this is probably a more accurate indication of the current status of the New Zealand cattle population.

In herds in which pestivirus infection is enzootic, antibody prevalence, excluding PI carriers, often reaches 100% in groups which include carriers. Antibody-free groups in such herds are usually those that have, for one reason or another, been raised in isolation from carriers. Within-herd and between-herd antibody prevalence estimates therefore depend largely on the definition of what constitutes a 'herd', whether by management, property or ownership.

**EPIDEMIOLOGY**

Most of the understanding of the epidemiology of pestivirus infections and disease relates to cattle held under extensive pastoral conditions, usually beef animals which are subject to minimal handling under relatively simple management regimes. Relevant information is derived from diagnostic and subsequent investigations, and is therefore liable to be biased to those circumstances that allow overt disease to develop and be recognised. Quiescent enzootic infections, and epidemiological parameters that
favour that state, are therefore likely to be under-represented. More attention is now being directed towards the situation in dairy cattle (12) with the expectation that it will be more complex because of a number of factors. These include more handling, non-seasonal breeding and in some areas, disposal of male calves and the hand-rearing of replacement heifer calves and, particularly, the feeding of pooled milk to calves. The pooled milk might be expected often to contain both virus from a PI cow and neutralising antibody from her herdmates, while many of the calves to which it is fed will have some, decaying, maternal antibody. Under these circumstances, sources of infection and patterns of spread are likely to be different from those understood under management for beef production. Potent exposure of calves to infection while they carry marginal amounts of maternal protection is likely to compromise the sensitivity of serological tests subsequently used.

Maintenance of pestiviruses ultimately depends on transplacental infection of fetuses within the first few months of pregnancy, to produce immunotolerant PI carriers. Fetal infection can follow either PI (congenital) or transient (post-natal) infection of the dam, but the resulting epidemiological patterns of disease may be rather different. The PI cow may produce a number of PI animals over a number of years whereas, particularly in seasonally bred herds, effective horizontal infections tend to occur in clusters, producing clusters of PI animals which later become clusters of cases of MD. Horizontal transmission from a transient infection is most unlikely to occur under grazing conditions or even with the levels of close housing and husbandry that occur in Australia. Consequently, serial horizontal transfers are of little importance in maintaining the virus and it is likely that enzootic infection in, and status of, a herd will usually depend on the presence within that herd of one or more PI animals. Even then, transmission rates under grazing conditions may be quite low, of the order of 1% per day (14). This is illustrated by observations made on animals found to be in contact with a PI carrier (10) when, after at least three months, only four of nine previously susceptible animals had developed antibody. However, the transmission rate can be greatly increased by closer contact, such as yarding for short periods. For example, 8 of 13 (62%) susceptible animals were transiently infected after they had been yarded overnight with one PI carrier (14). A similar result was achieved using similar conditions to establish natural infections for experimental purposes (18). Valid generalisations about the importance of the PI carrier as the major source of infection do not preclude that less common and less effective mechanisms may obtain occasionally. For example, in one herd which was otherwise serologically free of pestivirus activity over two years, two calves of one cow each developed active antibody. The cow had circulating precipitating antibody and no virus was recovered from a wide variety of secretions, including milk, excretions or swabs from mucosal surfaces. Nevertheless, the conclusion that this systemically immune cow could, somehow through the intimacy of nursing, infect its own calves but not others, is difficult to refute (I.R. Littlejohns, unpublished data).

The prevalence of persistent infections has not been established over large numbers and is likely to vary considerably over the diverse conditions under which cattle are managed. An overall estimate, again based on casual experience and observations, of the order of 1% is suggested. Prevalences of 2% to 5%, of PI animals which may be clinically normal over periods of years, have been found in individual herds. Higher figures, usually recognised retrospectively after an incidence of MD, may be reached when there has been previous epidemic exposure of pregnant susceptible animals. Preliminary results have been reported for a survey of maiden dairy heifers in
43 herds in the Hunter Valley of New South Wales (12). Further results from this work (D.J. Kennedy, personal communication) indicated an overall prevalence of 0.8% (4/514). Testing of healthy cattle for export certification at the Central Animal Health Laboratory, Wallaceville, has shown that 1.8% (16/876) were viraemic. It is probable that most of these were PI animals.

It is suggested that the prevalence of the PI state may be sex-related. Of 14 PI animals detected without selection bias in three herds, 12 were male (exact $p < 0.01$ compared to 1:1 ratio). This would imply an interesting possibility of there being a higher in utero mortality rate among female than male fetuses. Impressions from diagnostic experience over the years would suggest that the incidence of MD may also be sex-related, with the majority of MD cases being steers. However, a recent dissection of 159 diagnostic virus isolations, for which the sex of the case animal was known, at the Central Veterinary Laboratory, Glenfield, found only a modest bias with a male/female ratio of 92:67 (exact $p = 0.03$ compared to 1:1 ratio). The sex ratios of both persistent infection and disease deserve to be investigated further.

It is recognised that the PI prevalence will decrease with age and, hence, is at its highest in fetuses. As several hundred or more fetuses contribute to each batch of fetal calf serum produced commercially, it is statistically unlikely that any will be free of pestivirus. It is also likely that any pool which includes serum of a pestiviraemic fetus will also include homologous antibody, from a herdmate which was infected after achieving immunocompetence, thereby increasing the difficulty of virus detection. In studies on samples representing different stages of processing, pestiviruses were recovered more frequently from those taken after more handling, presumably indicating carry-over contamination from reused equipment (A. Janmaat, personal communication). Results from these studies also give strong indirect evidence of virus presence being extensively camouflaged by the concurrent presence of antibody. The hazards of using fetal calf serum and, to a lesser extent, any bovine serum, in work with pestiviruses is now generally appreciated. An even broader scope to the problem is suggested by the recovery of pestivirus from horse serum used in cell culture medium, which was presumed to be due also to contamination from shared processing equipment (G.W. Horner, unpublished data).

The frequency with which overt MD occurs in PI carriers is not known. In New South Wales approximately 100 diagnostic pestivirus isolations are made annually, mainly from cases of MD. They represent only about 0.5% of an estimated 20,000 PI animals born annually (estimated as 1% of a total of approx. 2.10^6 calves born). This probably reflects a low rate of disease-reporting more than it does a low incidence of disease among PI animals.

Although the incidence of post-natal infections, based on antibody prevalence, must be high at some stage of life, there is very little detail available on the circumstances under which these usually occur. The extent to which they may enhance the effects of other disease under conditions of intensive management and/or stress is not known. Only a very small proportion of the cattle populations of Australia and New Zealand are raised under intensive conditions.

Related pestiviruses occur in sheep and goats and, on serological evidence, in pigs, buffalo and deer, including large feral populations of all of these species except sheep. The prevalence of C24V neutralising antibody in sheep in Australia in 1967-68 was approximately 10% of adult animals and 13% of flocks (24), implying a within-flock prevalence of the order of 75%. In contrast to the geographic distribution of reacting
cattle, the prevalence of antibody in sheep increased from north to south. However, lower prevalences were found in a survey conducted in the south-west of Western Australia in 1985, when pestivirus group precipitating antibody was found in 31/1226 (2.5%) sheep, and C24V neutralising antibody in 10/679 (1.5%) (T.M. Ellis, personal communication). The comparison of antibody prevalences in sheep and cattle (approx. 60%) in this area of Western Australia, where the two species would have considerable contact, would imply that transmission between species would not commonly occur under field conditions. In New Zealand neutralising antibody is not uncommon in sheep (19% of 1013 sera tested at Ruakura) but these titres are probably due mainly to infections with ovine strains of pestivirus since clinical hairy shaker disease (Border disease) is common in some, especially southern, areas. Similarly, pestivirus antibody has been detected in deer (9% of 907 sera) and goats (4% of 539 sera) (G.W. Horner, unpublished data).

The question of pestivirus infection of Australian marsupials needs to be examined further. Sera from 2 of 44 Bennett’s wallabies neutralised C24V virus but those of 29 other marsupials, including 7 forester kangaroos, 10 pademelons, 2 potoroos, 3 brush possums, one brown bandicoot and 6 Tasmanian devils did not (20).

The PI animal, as a source of infection, includes the infected conceptus of a normal cow, now systemically immune. Its expulsion, together with associated discharges, at any stage up to and including the birth of a viable PI calf, presents a potent source of virus. A cluster of new infections has been observed to occur in heifers after herdmates had been infected following successful joining, and some returned to service at 21 days. This suggests that virus excretion, presumably from the reproductive tract, was associated with early embryonic death and consequent vaginal discharges (M.R. McGowan, P.D. Kirkland and I.R. Littlejohns, unpublished data). This has not been regarded as transmission from the transiently infected parent. Discharges at and after parturition of a PI calf are almost certainly highly infectious but the period over which discharges might remain so is not known.

Transmission rates are also greatly increased by handling, particularly when procedures such as the use of nosegrips or shared needles for bleeding or inoculations are involved. Human operatives, including veterinarians, may be effective vectors of infection. The importance of close contact and handling in facilitating pestivirus transmission cannot be overstated. For such generalisation, however, the nature or 'quality' of the contact may be as important as the apparent 'quantity'. On occasions, even when there has been apparent close contact, transmission rates have been inexplicably low (A. Janmaat, personal communication).

When infection of the fetus is derived from a PI dam, a familial incidence is established which may extend over at least several generations (13, 14). This may be responsible for a low incidence of disease within a herd over a number of years. However, because a proportion of the individuals within viraemic families will develop disease and die, and those which survive for a time are generally not very successful breeders, the families are at a survival disadvantage compared to the rest of the herd and tend to die out. Survival of the virus in a population therefore depends on the alternative mechanism whereby horizontal transmission results in transient asymptomatic infection of a pregnant female followed by transplacental infection of its fetus. It is believed that self-cure of individual herds may occur if horizontal transmission to pregnant females is precluded.
Infection of pregnant beef females most commonly occurs when heifers have been raised in isolation from the adult herd, within which there is one or more carriers, and are then mixed with these older animals at or after joining. When heifers are mated separately, for example when bulls of particular conformation are preferred, then their isolation and susceptibility may be maintained for a further year and their exposure to effective infection may be deferred by one year. Other circumstances which may allow susceptible cows of any age to become infected are those which necessitate emergency movement and agistment of stock, typically occurrences of drought, flood or fire. Production of PI animals may then occur in numbers, resulting in a high incidence or ‘outbreak’ of MD some 12-30 months after the emergency, that is, allowing 5-9 months in utero plus 6-24 months of age.

Many pestiviruses readily infect alternative hosts (including sheep, goats, deer, buffalo and pigs) experimentally, so that cross-infections, either of cattle by a virus which is normally maintained in another host species, or of cattle by a virus of bovine origin which has infected an alternative host, can potentially play a part in the epidemiology of bovine pestivirus infections. Thirty three of 41 sheep were infected by a yearling carrier heifer over 147 days when they were grazed together in a small paddock with shared trough watering and supplementary feeding (T.M. Jessep and I.R. Littlejohns, unpublished data). This intimacy of contact between species may be uncommon in the field and no information is available on the extent to which pestiviruses are exchanged between host species under natural conditions. Despite the differences, previously mentioned, in the geographic distributions of higher prevalences of antibody in sheep and cattle, a weak association \( p = 0.12 \) was found between the presence of cattle on a property and the detection of antibody in sheep (24).

Artificial breeding techniques are also capable of contributing to the spread of pestiviruses. At least two occurrences of multiple cases of infection and disease have been diagnosed following embryo transfers (13, details unpublished; P.D. Kirkland, K.G. Hart, A. Moyle and E. Rogan, unpublished data). In the first case, pestivirus was recovered from a stored sample of the fetal calf serum which had been used in the flushing fluid. However, the management of recipient cows would also have allowed for infection to be passed between animals at the critical time of pregnancy. Either or both of those factors may have contributed to the final outcome. Artificial insemination has not been found to be responsible for problems but the potential for transmission via semen exists. Other factors attendant on the act of insemination, including close yarding and intimate handling by inseminators, are also relevant at this critical time. At present, few precautions are taken with regard to pestivirus infection, either transient or persistent, in collecting centres in Australia. The introduction of control measures is planned in New South Wales, and in screening stock already held, a young PI bull has recently been detected in one centre. Its semen appeared to be of normal quality but contained more than \( 10^7 \) TCID\(_{50}\) of virus per straw. This material had been distributed to approximately 100 herds for progeny appraisal and the consequences of its use are now to be assessed (P.D. Kirkland, personal communication). In New Zealand there are no formal controls applied to artificial breeding establishments but it is common practice for semen collections to be checked for freedom from pestiviruses at regular intervals.

Vaccinations have not been widely recognised as causing pestivirus transmission, perhaps largely because live virus vaccines have been little used on cattle. However, the risks implicit in the use of vaccines of animal or cell culture origin, which have
not been treated to inactivate pestiviruses, are now appreciated. General handling procedures which are part of any vaccination procedure, including the multiple use of hypodermic equipment, may facilitate pestivirus transmission if PI and susceptible animals are handled together.

**CONTROL**

Mucosal disease is notifiable in several States of Australia, viz. Queensland, Victoria, Western Australia and the Northern Territory. This appears to be primarily for awareness, especially for health certification purposes and overview of its differential diagnosis from exotic disease. Pestivirus infection *per se* is not notifiable and no State of Australia has a control policy in regard to it. National quarantine measures relating to animals and animal products have in the past been incompletely effective, as MD has been diagnosed in an imported bull (P.D. Kirkland, personal communication). It can be expected that more attention will be given to this risk in the future. No pestivirus vaccines are available or registered for use in Australia. Control of disease is therefore based on management, and the detail is at the discretion of individual herd managers and their veterinary advisers. Various options in this direction have been suggested (16) and include measures to eliminate carriers from herds, to maximise opportunities for breeding females to contact PI carriers, identified or not, outside of the critical period just prior to and during early pregnancy and, conversely, to minimise possibilities of contact within that period. In the south coastal region of Western Australia, separation of heifers from cow herds until mating is seen as a major factor which allows the critical persistent infections to occur. Management which allows young stock to be exposed to infection before pregnancy has been successful in the control of individual farm problems (R. Reuter, personal communication). In one government-owned herd in New South Wales, use has been made, over several years, of controlled exposure of heifers. At least one month before joining, neat serum from a PI herdmate is inoculated intranasally. Alternatively, serum diluted up to 1/1000 can be inoculated subcutaneously (L.G. Cook, T.M. Jessep and I.R. Littlejohns, unpublished data). This procedure has been successful in inducing seroconversion and in interrupting the sequence of PI births and disease in the herd. In South Australia, cows to be used as recipients in one embryo transfer operation are inoculated intramuscularly with cytopathogenic virus at least four weeks prior to use. The results obtained in this programme are the subject of ongoing study (A.M. Pointon, personal communication).

In New Zealand, MD was notifiable in the 1960's due to its resemblance to serious exotic diseases. Once its aetiology was defined and it was established that the responsible virus was widespread, it was removed from the list of notifiable diseases. A live attenuated vaccine (Bovax), developed from a local cytopathogenic isolate (11), was available for a number of years until economic reasons led to its withdrawal. It produced good levels of neutralising antibody and no undesirable side effects were documented. Several imported dead vaccines have recently become available.
ACKNOWLEDGMENTS

Apart from colleagues acknowledged in the text, the authors are grateful to M. Flanagan for his careful and constructive criticism of the draft of this contribution.

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Résumé : L'infection bovine à pestivirus est fréquente et largement répandue en Australie comme en Nouvelle-Zélande. Les animaux adultes sont porteurs d'anticorps dans leur majorité (60 % environ). La maladie correspondante résulte presque toujours d'une contamination in utero. Elle se traduit soit par la mort de l'embryon ou du fœtus, à n'importe quel stade de la gestation à partir de la conception, soit, chez ceux qui naissent et sont des porteurs infectés de manière persistante, par la maladie des muqueuses, le plus souvent sous forme chronique. On observe peu ou pas de cas de maladie à la suite d'une contamination post-natale de vaches non gestantes ; leur rôle dans la propagation de l'infection semble limité. La transmission et le maintien du virus sous forme enzootique reposent essentiellement sur les porteurs infectés de manière persistante ; ceux-ci, après une contamination précoce in utero, sont immunotolérants ; leur état clinique peut varier d'un état normal, ou presque, à la maladie des muqueuses manifeste. L'expulsion d'un fœtus infecté et des liquides associés représente aussi une source de contamination importante. En général, peu de mesures de prophylaxie active sont entreprises. Il n'y a pas de vaccins disponibles en Australie ; leur utilisation en Nouvelle-Zélande est limitée. Toutefois, la prophylaxie suscite un intérêt grandissant parmi les professionnels de l'élevage, notamment dans le domaine de la reproduction par insémination artificielle ou par transfert d'embryons, où l'on pense que l'impact pathogène du virus pourrait être amplifié.


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INCIDENCIA, EPIDEMIOLOGÍA Y PROFILAXIS DE LAS INFECCIONES Y ENFERMEDADES POR PESTIVIRUS BOVINO EN AUSTRALIA Y NUEVA ZELANDA. – I.R Littlejohns y G.W. Horner.

Resumen: La infección bovina por pestivirus es frecuente y muy difundida tanto en Australia como en Nueva Zelanda. Los animales adultos son portadores de anticuerpos en su mayoría (aproximadamente 60%). La enfermedad correspondiente es casi siempre resultado de una contaminación in utero y da lugar, o bien a la muerte del embrión o del feto, cualquiera que sea el estadio de la gestación a partir de la concepción, o bien, en los animales que nacen y son portadores infectados de manera persistente, a la enfermedad mucosa, muy a menudo en forma crónica. Se observan pocos o ningún caso de
enfermedad como consecuencia de una contaminación posnatal de vacas no gestantes; su papel en la propagación de la infección parece limitado. La transmisión y mantenimiento del virus en forma enzootica se basan esencialmente en los portadores infectados de manera persistente. Tras una contaminación precoz in utero, éstos son inmunotolerantes y su estado clínico puede variar entre uno normal, o casi, y la enfermedad mucosa manifiesta. La expulsión de un feto infectado y de los líquidos asociados representa también una fuente de contaminación importante. En general se han tomado pocas medidas de profilaxis activa. No hay vacunas disponibles en Australia y su uso en Nueva Zelanda es limitado. Sin embargo, la profilaxis suscita un interés creciente entre los profesionales de la cría, en particular entre quienes trabajan en la reproducción por inseminación artificial o por transferencia de embriones, situaciones en que se piensa que el impacto patógeno del virus podría crecer.


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REFERENCES


