



Living With IBR

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Three small letters can make one big impact, and in the case of IBR, they can close down export markets, deny our breeders access to top international bloodlines or simply impair health within the herd. In the third in our cattle health series by leading experts in their field, Dr Malcolm Banks answers some frequently asked questions.

What is IBR?

IBR or infectious bovine rhinotracheitis is an upper respiratory tract disease of cattle characterized by nasal and ocular discharges, conjunctivitis, fever, coughing, loss of appetite and apathy. Nasal abscesses and generalised nasal congestion may lead to forced, noisy breathing. It presents with a high infection rate in non-immune herds and may lead to abortion in pregnant cattle or even death in calves and occasional adults following a short illness. The genital diseases of infectious pustular vulvovaginitis (IPV) and infectious balanoposthitis (IPB) are a result of infection with the same organism as IBR but which present a different clinical picture, including the appearance of abscesses in the vagina or penis. Simultaneous IBR and IPV/IPB clinical signs may occur but only rarely. A reduction in milk yield and occasional cases of enteritis with diarrhoea may be seen following IBR infection. A further common complication to IBR infection is the occurrence of pneumonia caused by secondary bacterial pathogens. In addition, the presence of other viral respiratory pathogens such as para-influenza virus and respiratory syncytial virus may contribute to a respiratory disease syndrome which may persistently affect a herd, particularly in housed cattle. There is an encephalitic form of the disease but this is now known to be caused by a different virus which has never been seen in the UK and Ireland.

What causes IBR?

A herpesvirus named bovine herpesvirus 1 (BHV-1) is the causative agent of IBR, IPV and IPB. This virus is related to the common cold sore virus, herpes simplex and the virus of Aujeszky's disease of pigs.

The severity of disease caused by the IBR/IPV/IPB virus is largely a reflection of the virulence of the individual virus strain and the age and condition of the infected animal. The major determinant of the consequences of infection is the route of virus entry. Entry via the genitals predisposes to genital disease and similarly virus entry via the respiratory passages will produce 'head end' or respiratory effects. The one slightly confusing factor in this is that respiratory infections may also lead to abortion in pregnant animals. Generalised systemic infection affecting many organs in the body, although not common, occurs most frequently among non-immune calves and in these cases mortality is relatively high. Systemic infection may also be seen in animals which are suffering from an existing disease or which are otherwise immuno-deficient.

How much infection and disease is there in the UK?

According to several studies done at the VLA Weybridge, increased virulence strains introduced in the late '70s (see below) have now established a high level of infection in the national herd with a prevalence of around 70%. The present state of high immunity is reflected in the relatively low numbers of reports of severe clinical disease. In terms of occurrence over the last decade, reports of clinical disease have remained at about 250-400 pa. There is a seasonal variation in incidents (an incident is one outbreak of clinical disease) highest in winter months and lowest in summer months.

Bearing in mind that new tests and vaccines increase awareness of diseases, is there any evidence that the pattern of IBR is changing, or how long our national herd has been affected?

The virus was first isolated in Britain from an outbreak among store cattle in Oxfordshire in 1961. There is evidence that the relatively mild disease caused by these early virus strains was supplanted in the late '70s by more virulent, rapidly spreading strains causing more severe clinical disease and possibly emanating from imported cattle. These new strains have now established themselves as the dominant types of IBR virus in the UK. In terms of the percentage of IBR reactors within herds, there is some evidence from blood tests and the newly developed bulk milk tests that many herds fall into either a high or a low reactor rate category. At this time the factors which influence this phenomenon, also noted in Spain and Canada are unknown, but it is possible that viral strain differences and even husbandry practices may play a part.

Which animals are most likely to show signs of the disease?

The disease severity decreases with increasing age, from an acute, systemic often fatal disease in new born calves to an inapparent infection in adult cattle. Clearly, abortion is an obvious sequel to infection during gestation.

How do you know if an animal has IBR?

The multitude of pathogens in circulation which may cause respiratory or reproductive problems means that a laboratory diagnosis is the only certain way of knowing if an animal has IBR, but there are some clues from the clinical presentation and the history of the herd, particularly with respect to previous outbreaks and incoming stock movements. Although juveniles may show the full range of clinical signs indicated above, in adults the clinical signs may be restricted to mild depression and inappetance. Increased frequency of urination and tail swishing are an indication of genital inflammation and irritation which may be caused by IBR virus. Congestion and hyperaemia of the nasal linings and surrounding soft tissue contributes to the familiar red-nose appearance, particularly among calves.

Detection of virus from nasal or genital swabs or tissue samples is a good way of confirming infection, although as question eight below indicates, these results should be interpreted carefully. If the herd was known to be free of IBR, positive blood tests are nearly always diagnostic. However, if the herd had a history of IBR, samples of blood collected from a cohort of animals (including the aborting animal) at the time of the clinical signs and again two to three weeks later will improve the accuracy of the diagnosis.

Can animals carry the infection without being obviously clinically affected?

When an animal recovers from an IBR infection the virus is cleared from all parts of the body except particular sites in the nervous system. These sites are usually linked to the site of first infection, so with IBR the site is in nervous tissue in the head, and in IPV/IPB the site is in nervous tissue in the pelvic region of the spine. The virus may remain in this dormant or latent form for the remainder of the life of the host animal but may also on occasions break out into an infective form again. Although at this time the animal may show few or no clinical signs, it may be shedding virus in nasal or vaginal secretions or in semen. This cycle of non-infective latency interspersed with periodic infectivity is a feature of many herpesviruses. The factors which cause the virus to break out from latency are many and complex but usually involve some form of stress such as movement, calving or infection with another disease agent. A good example of the latter type of stress may be seen as a sequel to abortion caused by another disease agent. Often in these cases the IBR virus shedding detected

by nasal or vaginal swabs taken shortly after the abortion was caused by, and was not the cause of, the abortion.

Can animals other than cattle carry IBR?

Most large ruminants including goats, sheep and deer may be infected with IBR but in all these animals the disease is much milder than in cattle. Sheep and goats have been shown to be capable of carrying a latent infection of IBR (see above) and reactivation to an infective state has been shown experimentally. Whether the virus would persist in a sheep or goat herd when it had been cleared from any associated cattle herd is unknown. Until this is determined it is wise in an IBR free herd to avoid direct contact between cattle and sheep or goats unless the latter have been tested IBR negative. Pigs may be infected with IBR although this is quite rare and is clinically mild in most cases. Although rabbits have been infected experimentally there is no evidence that the wild rabbit population in Britain is infected or is in any way implicated in the persistence or transmission of IBR.

How do cattle catch IBR?

The main route by which cattle contract IBR is via the mouth and nose following contact with virus contained in nasal, oral or, less commonly, vaginal secretions. Virus in the secreted mucous may remain infective for several days carried either on the infected animal or carried on people, bedding or implements. Insects and rodents are not considered to be of any significance in the transmission of IBR. During coition, virus may be transferred in the semen or in vaginal secretions to the bull and in rare cases transmission may be by ingestion of milk from an infected dam. Transmission by inhalation of aerosols containing virus does not often occur beyond a range of a few metres.

Can you treat the disease successfully in sick animals?

There is no established antiviral treatment regime for IBR once the clinical signs have appeared. In most cases the animals return to normal within one to two weeks and vaccination will help reduce spread of the virus to other animals. Secondary bacterial infections may be controlled by appropriate antibiotics.

Does treatment eradicate infection?

Vaccination will reduce but not prevent entry of the virus and will not remove the virus from an already infected animal. However, in a way which is not fully understood, vaccination appears to reduce the frequency with which a virus breaks out from latency. Moreover, the duration of excretion and concentration of virus excreted during a breakout from latency is much reduced by vaccination.

Does your understanding of the infective process help to construct control programmes?

One of the main principles of the infective process which help shape control and eradication strategies is the latency phenomenon referred to above. Infected animals must be considered a potential source of infection for the remainder of their lives.

In the context of this question it must also be remembered that whilst vaccination (and colostral immunity) should prevent the occurrence of the clinical signs of infection, it will not necessarily prevent the virus from entering the animal and establishing a sub-clinical infection which may later break out into the infectious form, again in the absence of clinical signs.

Is vaccination an appropriate part of control?

If used with an appropriate husbandry and disease security policy, vaccination is a valuable way of reducing the clinical and economic effects of IBR infection, including growth check and milk drop. It must be remembered that although control is possible by application of the above strategy it does bring with it a continuing requirement to maintain complete and adequate vaccine cover, particularly in areas of endemic IBR. Cost-benefit appraisals have clearly demonstrated the value of a well regulated vaccination policy.

In general, live vaccines confer a broader, more long lasting immunity but may have some slight risks not associated with killed vaccines. In particular, they may sometimes spread from vaccinated to unvaccinated animals and cause unexpected positive blood test results. Furthermore, live vaccines may very occasionally be contaminated with another virus; there is a recent instance of BVD being spread in this way in an IBR vaccine.

Are the tests for the infective agent completely sensitive, or is it possible to have animals carrying the disease organism which test clear?

There is evidence that a small number of animals, particularly newborns having suckled IBR-immune dams, may be infected and may not show any evidence of the infection either by symptoms or by a blood test. When this colostral antibody disappears, the animal may test negative and yet be carrying latent virus, which may subsequently become infective. Dutch workers estimate the occurrence of these SNC or seronegative carriers may be as high as 1 in 50 but evidence of this phenomenon in the UK is scarce.

Is eradication from the disease from a herd, a region or a country achievable?

Eradication of the disease has been achieved on a national basis by Switzerland, Denmark, Austria, and Sweden; Finland has remained free of IBR. Eradication schemes in Holland, France, Belgium and Germany are in various stages of development and execution, all supported by a combination of industry and state resources.

States which are free of IBR will be able to apply to Brussels to require pre-import testing of stock and semen from states where IBR is still present. In many of these schemes a marker vaccine (gE marker) which enables discrimination between vaccinated and virus infected animals by a blood test is a central feature. However, recent technical problems mean that these vaccines are not yet fully established in the marketplace. Eradication of IBR without marker vaccines is possible but this demands strict disease security and is clearly more difficult and protracted where there are high numbers of infected herds.

What is the risk of reintroduction of the disease in a 'clear' herd?

The biggest single risk factor to a free herd is the introduction of new stock from external sources which are not proven clear of IBR. These animals may not show any clinical signs but may excrete virus which could spread rapidly in an IBR free herd. There is also evidence from Holland that transported latently infected stock suffer more severe clinical disease following reactivation of virus than similar animals which are not moved. Direct contact with cattle of unknown or positive IBR status should be avoided at all times, whether indoors or out.

Does the disease have any human implications?

No. Although related to the cold sore virus of humans, there is no evidence to suggest that IBR virus may infect humans.

What is the role of the bull or AI semen in transmitting the infection?

The infection may be transmitted from the semen of an infected bull following natural or artificial insemination. Following the first infection of the bull the virus may be shed in semen for as long as 60 days. This period is much longer than previously thought and results from the application of the more sensitive PCR tests on semen. As a result of the lifelong latency of the virus described above, in theory at least an infected bull has the potential to shed virus in semen at periodic intervals for the rest of its life. The frequency, duration and amount of virus re-excretion in semen varies between infected bulls and although vaccination appears to

reduce this frequency, the mechanisms which control re-excretion are poorly understood and may be influenced by the strain of virus and the age, condition and possibly genetic background of the bull.

It follows from the above that control and eradication of IBR amongst bulls and in particular those bulls in AI centres is of paramount importance in reducing the spread of IBR. Clearly, careful selection and frequent monitoring allied to appropriate husbandry and disease security measures are key elements in any disease control or eradication programme. Consequently, since 1988, bulls may only be moved on to EU approved AI centres if they are seronegative for IBR.

Until the 31 December 1998, IBR positive bulls which were already on centre when the semen directive came into force were still permitted to be used for EU trade provided the semen passed the virus isolation test; this concession has now been withdrawn and semen may only be traded between member states if the bull was IBR negative when he entered the centre. This explains the withdrawal of several popular bulls from the EU market at this time.

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