Pestivirus infections in ruminants other than cattle

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Summary: Pestiviruses infect a wide range of domestic, captive and free-living ruminants. Among domestic livestock, Border disease virus is a well recognised cause of an important congenital disease of sheep in virtually all sheep-rearing countries of the world. The clinical signs, pathogenesis, diagnosis, epidemiology and control of this disease are described in detail. One natural outbreak of Border disease in domestic goats has been described and there is serological and virological evidence that pestiviruses occur widely in this species. A pestivirus has been isolated from a farmed red deer (Cervus elaphus) and there is serological evidence of a widespread low prevalence of infection among this new domestic species. Pestiviruses have been associated also with outbreaks of disease among captive ruminants in zoological collections.

Among free-living ruminants, pestiviruses have been recovered from dead roe deer (Capreolus capreolus), fallow deer (Dama dama), African buffalo (Syncerus caffer), giraffe (Giraffa camelopardalis) and wildebeest (Connochaetes spp.) but in all these instances the contribution of the virus to the cause of the disease was uncertain. Serological surveys have shown that many species of free-living ruminants in North America, Europe and Africa have varying prevalence rates of antibodies to pestiviruses.

KEYWORDS: Border disease - Border disease virus - Bovine virus diarrhoea virus - Control - Deer - Epidemiology - Free-living ruminants - Goats - Pathogenesis - Pestivirus - Ruminants - Sheep.

INTRODUCTION

The three pestiviruses, hog cholera (classical swine fever) virus (HCV), bovine virus diarrhoea (mucosal disease) virus (BVDV) and Border disease virus (BDV) were named after the important diseases they cause. The recognition of a serological relationship between viruses causing a systemic haemorrhagic disease of pigs, an enteric disease of cattle and a congenital disease of sheep remains a tribute to those responsible (19, 53, 58).

Pestiviruses appear to infect naturally only the even-toed ungulates belonging to the order Artiodactyla, within which there are 11 species of pig and 173 species of ruminant (80). Despite attempts to adapt pestiviruses to grow in a variety of other

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hosts, only rabbits appear to support virus replication (22). Recent reports of antibodies to pestiviruses in human sera (28, 59, 81) and pestivirus antigen in human stools (83) require further substantiation, but raise the possibility of human infection with a serologically related virus.

The success of pestiviruses, particularly in ruminants, is due to their ability to cross the placenta, invade the fetus and set up a persistent infection which continues into post-natal life. These persistently infected animals excrete virus continuously and spread infection wherever they go, sometimes for years (31, 49, 73).

This paper will address pestivirus infections in all ruminants, except cattle. After a brief consideration of the relationship between ruminant pestiviruses, the main features of Border disease (BD) in sheep will be described, followed by sections on BD in goats and pestivirus infections in other farmed, captive and free-living ruminants.

RELATIONSHIPS BETWEEN AND AMONG RUMINANT PESTIVIRUSES

Traditionally, pestiviruses isolated from pigs have been termed HCV, those from cattle BVDV and those from sheep and goats BDV, but as new information emerges the picture becomes more complex. Nevertheless, nearly all studies of the antigenic relationship between representative isolates of the three pestiviruses have readily differentiated HCV from the ruminant pestiviruses (31). The differentiation of bovine virus diarrhoea (BVD) and BD viruses is less clear-cut. Serological comparisons of more than 110 British isolates using polyclonal and monoclonal antisera indicate the presence of at least two antigenic groups of pestivirus, one of which predominates in cattle and one in sheep (21, 47). There is limited information on the antigenic relatedness between pestiviruses from other ruminants, although isolates from a giraffe and a fallow deer have been shown to be substantially different (16), and the fallow deer isolate is different from BVDV and BDV isolates (21).

The probability exists, therefore, that pestiviruses have evolved along with their own host species. Interspecies transmission is achieved easily experimentally (31) and it is prudent to believe that it will occur readily in domestic and free-living ruminants when permitted to do so by new husbandry practices or changes in population dynamics.

BORDER DISEASE OF SHEEP

Introduction

Border disease is a congenital disease of sheep first reported from the Border region between England and Wales (33). Since then BD has been recorded in Scotland (6), New Zealand (46), Ireland (30), Australia (1), USA (34), Switzerland (18), Greece (66), Netherlands (68), Canada (57), Norway (43), Federal Republic of Germany (41), Syria (82), France (15) and Sweden (2). In addition, there is serological evidence that
BD is present in sheep in Nigeria (67) and the German Democratic Republic (39) and there is a strong likelihood that sheep flocks in other countries will be shown to be infected by future investigators.

Virtually all field isolates of BDV are non-cytopathic in cell cultures, although cytopathic strains have been described (37, 74). While the non-cytopathic biotypes cause congenital disease and persistent infection, cytopathic isolates have been recovered from sheep dying of a 'mucosal disease-like' syndrome (7, 27).

Review articles on BD include a monograph (8), a general review (70), an update on clinical disease (48) and chapters in two books (4, 31).

Clinical disease

One or more of the following signs may indicate the presence of BD in a flock:

1. An excessive number of abortions and barren ewes and the birth of small weak lambs (Fig. 1).

![A group of hand-reared one-week-old lambs affected with Border disease. All were small and only one was able to stand unaided. At one month old, 3 of the 4 lambs were sufficiently recovered to move about freely.](FIG. 1)
2. A number of lambs being born with abnormal body conformation, tremor and/or fleece changes sometimes with abnormal pigmentation (Fig. 2). The fleece changes are due to long hairs rising above the fleece to form a 'halo' effect especially along the neck and back (Fig. 3). This effect is most evident in smooth-coated breeds and is much less obvious in the coarse-fleeced breeds such as the Scottish Blackface. Affected lambs have been termed 'hairy-shaker' or 'fuzzy lambs' in English-speaking countries, and 'pellous' or 'bourrus' in France.

**FIG. 2**

**Characteristic stance of a BD affected lamb**

Hind legs splayed to reduce swaying of hindquarters.
Tail swinging from side to side
Border disease lamb with hairy fleece

Long hairs rising above the rest of the fleece form a 'halo' effect, especially over the back of the neck.

3. A group of older lambs, especially around weaning time, in which some have died and others are scouring and/or ill-thriven.

4. Occasionally other unusual clinical signs are associated with infection. In one outbreak in France, lambs showed an unusual atrophic lesion of the diaphragm which resulted in constriction between the thorax and abdomen (64).

Pathogenesis

Infection of non-pregnant sheep

Infection of normal healthy sheep with virtually all BDV isolates is short-lived and mostly sub-clinical. Mild pyrexia and transient lymphopaenia coincide with viraemia, but with the production of neutralising antibodies 2 to 3 weeks after infection these signs disappear.

One French isolate of BDV, however, has been shown to produce profound leucopaenia and death in 50% of 3- to 5-month-old lambs. This unusually pathogenic
strain of BDV was recovered from a case of Syndrome ‘X’ [also called Aveyron disease, ovine leucopaenic enterocolitis or petega ovina (sheep plague)] first reported in December 1983 in the Aveyron region of France among sheep reared intensively for the production of milk used in the manufacture of Roquefort cheese. This new disease killed 1,500 ewes and 24,000 lambs in 1984. The principal symptoms were severe depression, pyrexia and diarrhoea and at post-mortem examination haemorrhages were seen at many sites and were consistently present in the caecum, colon and mesenteric lymph nodes. Some recovered ewes later aborted or gave birth to weak shaking lambs with poor viability. The incidence of the disease fell sharply in 1985 and has remained low and although its cause was never determined conclusively, characteristics of the syndrome suggested it had a viral aetiology. Of the 5 viral agents recovered, only the BD virus isolate (AV2 strain) has a strong claim for having made a major contribution to causing syndrome ‘X’ (17).

One other pestivirus isolate has been shown to cause disease in 4- to 5-month-old lambs. This isolate, recovered in the Netherlands, was a pestivirus contaminant of a live HCV vaccine. The contaminant was probably of sheep origin since its most likely source was the secondary lamb kidney cells used to prepare the vaccine. Lambs given the contaminated vaccine developed fever, prolonged leucopaenia, anorexia, conjunctivitis, nasal discharge, pale conjunctivae, dyspnoea and diarrhoea and 4 of 8 lambs died (78).

**Infection of pregnant ewes**

The most serious consequences of BDV infection occur when the virus infects susceptible ewes during pregnancy. The ewes show no clinical signs but virus spreads rapidly to the placenta and crosses to the fetus within one week of infection. The immune response of the ewe quickly eliminates all virus from the maternal tissues but it has no effect in the fetus where virus can persist.

**Fetal infection**

The outcome of the fetal infection depends on the strain and dose of virus, the breed of the fetus and its ability to repair damage but most important is the stage of fetal development at which infection occurs. The age at which the fetus gains immunological competence is critical in determining the distribution and persistence of virus, which in turn influences the extent of fetal damage. The ovine fetus can first respond to an antigenic stimulus between approximately 60 and 80 days of gestation. The possible fates of fetuses before, during or after this crucial period are summarised in Figure 4.

The most dangerous time for a fetus to become infected is in the first 60 days of gestation. Virus replication is uncontrolled and the death of the fetus is likely. Death may occur rapidly leading to resorption or the unnoticed abortion of small fetuses or may not occur until weeks or months after infection when the subsequent abortion or stillbirths are obvious. It is also possible for one fetus to die in early gestation and be found mummified at the birth of its surviving twin. In lambs surviving infection in early gestation virus is widespread in virtually all organs. Typically there is no evidence of any inflammatory reaction. The principal pathological findings are myelin deficiency in the CNS which accounts for the tremor and an increase in the number of primary hair follicles causing ‘hairiness’. The low pathogenicity of some virus strains, however, means that some lambs can be born persistently infected with virus without showing any clinical signs and with only minimal pathological
Diagram showing the likely virological and serological status of lambs at birth following \emph{in utero} infection with BDV during early, mid or late gestation.
lesions (14). Precolostral blood samples from ‘hairy-shaker’ and other persistently infected lambs contain readily detectable amounts of infectious BDV. Such lambs are tolerant to the virus and have a persistent infection usually for life (69).

If fetal infection occurs when the immune system is beginning to develop (60-80 days) the outcome is unpredictable. Some lambs will be born viraemic and persistently infected without detectable antibody in precolostral blood. Others will be born virus negative and antibody positive. Infection at this stage can result also in widespread inflammatory lesions in the CNS leading to cerebral cavitation and cerebellar dysplasia; the alternative pathology of BD (5). Lambs thus affected frequently have severe nervous symptoms and major locomotor disturbances and have a high concentration of serum antibody to BDV (61).

Fetal infection after 80 days gestation is met by an immune system capable of eliminating the virus. Fetal death is rare and virtually all lambs will be born apparently normal. They will be free of virus but have demonstrable antibody.

Persistently infected (PI) lambs

Persistent infections with BDV can be established only if lambs are infected during their first 80 days of intra-uterine life. At birth, clinically affected lambs have a low chance of survival. Many die early in life while survivors have a poor growth rate and an increased susceptibility to other diseases. Less severely affected lambs can be reared with careful nursing but death may occur at any age. As lambs mature, the nervous signs gradually diminish but may recur at times of stress. ‘Halo’ hairs are soon lost from the birth-coat which is replaced by a coarse fleece. Whilst some affected lambs grow poorly and die young, others mature normally and can survive as persistent excretors of virus for years.

Most PI lambs will receive colostral antibody to BDV and for the first three months of life can be serum antibody positive and viraemic. The virus can be difficult to detect in serum during this time but can be recovered readily from leucocytes. Lambs become seronegative as the colostral antibody concentration wanes. Virus persists in most tissues and the lambs remain a potent source of infection.

Within groups of PI lambs some animals can suffer from chronic wasting, others can develop excessive ocular and nasal discharges sometimes with respiratory distress, while others develop an intractable scour and die within 2 to 4 weeks. Necropsy of these scouring lambs often reveals gross thickening of the distal ileum, caecum and colon resulting from focal hyperplastic enteropathy. Cytopathic BDV can be recovered from the gut of these lambs and this syndrome has several similarities with bovine mucosal disease (7, 27).

PI sheep surviving to breeding age often have reduced fertility but PI ewes have produced infected lambs in successive pregnancies (10, 79). Also virus-containing semen from a PI ram can produce PI offspring, with the early embryo becoming infected from virus replicating in the uterine epithelium (26).

Diagnosis

The diagnosis of BD on clinical grounds will present little difficulty if typical ‘hairy-shaker’ lambs are born. Often however, laboratory confirmation will be necessary since swayback, ‘daft’ lamb disease, bacterial meningo-encephalitis, focal symmetrical encephalomalacia and hypothermia may have to be considered in the differential
diagnosis of affected lambs. The specimens required by the laboratory to confirm BD are summarised in Table I. As placentas and fetuses aborted due to BDV infection have no distinguishing characteristics, laboratory confirmation will be required to differentiate BD from the other known infectious causes of ovine abortion. All animals in suspected groups should be blood sampled in order to detect the antibody negative, virus positive persistently infected sheep, whereas to determine the presence and extent of BDV infection in a flock, 10% of animals from different age groups should be tested for antibody. The most commonly used laboratory methods for demonstration of virus are detection of viral antigen in cryostat sections of tissues using immunofluorescence (IF), and virus isolation in susceptible ovine cell cultures, e.g. fetal lamb kidney or muscle cells, with detection of the non-cytopathic virus by IF or immunoperoxidase (IP) tests. Serum neutralisation or ELISA methods are used for detecting specific antibody. Histopathology on brain and spinal cord is also useful if skilled interpretation is available.

**TABLE I**

*Specimens to be collected for the laboratory confirmation of BD*

<table>
<thead>
<tr>
<th>Clinical manifestation</th>
<th>Live animal</th>
<th>Dead animal</th>
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</thead>
<tbody>
<tr>
<td>‘Hairy-shaker’ or weak lambs; also poorly thriving or scouring lambs</td>
<td>Whole blood (clotted and heparinised) for virus isolation and serology from both the lamb and its dam</td>
<td>Thyroid, kidney, brain, spleen, gut and lymph nodes: fresh for antigen detection and in virus transport medium for virus isolation. Heart blood for serology. Brain and spinal cord in calcium formol saline for histopathology</td>
</tr>
<tr>
<td>Abortion</td>
<td>Blood from dam for serology and virus isolation</td>
<td>Tissues as above plus placenta for antigen detection and virus isolation. Brain and spinal cord in calcium formol saline for histopathology</td>
</tr>
</tbody>
</table>

**Epidemiology**

Sheep-to-sheep contact is the principal way in which BDV is spread, and the most potent source of virus is the persistent excretor. Bought-in PI sheep have been shown to introduce BDV into a susceptible flock and cause a serious outbreak of BD, and many other outbreaks of BD have a history of introduction of new stock at the beginning of the breeding season (14, 76). More intensive husbandry, particularly housing during early pregnancy, increases the risk of an explosive outbreak of BD.

Under experimental conditions pestiviruses from other species can also cause BD in sheep so that, among domestic animals, cattle in particular but also goats and possibly pigs represent potential sources of infection. Among free-living ruminants pestiviruses have been isolated from red, roe and fallow deer and serological surveys in Europe, North America and Africa have shown that many species have detectable antibodies to pestiviruses (see section below). Therefore, where sheep are grazed extensively in contact with free-living ruminants the possibility of infection from these other species cannot be excluded.
One other possible source of BDV infection which cannot be ignored is a modified live vaccine (MLV) contaminated with a pestivirus. All MLV's produced in ovine, bovine or porcine cell cultures, or in medium supplemented with serum from these species run the risk of being contaminated with a pestivirus (72). Screening techniques must include IF or IP tests to detect non-cytopathic viruses. Both sheep pox and orf virus vaccines administered to sheep have been incriminated as vectors of BDV infection (41, 66).

Control

Countries importing high quality breeding stock should ensure that they originate from flocks with no serological evidence of BD. Seronegativity of individual animals from infected flocks is not acceptable since such sheep could be seronegative, virus positive carriers. In countries where the disease is endemic, the introduction of replacement breeding stock onto farms with no history of BD needs careful consideration. Ideally, replacement females should be home-bred and purchased rams tested to ensure they are not persistently infected. Where females are bought-in the feasibility of testing them should also be considered. Newly purchased females should always be mated and kept separate from the rest of the flock until lambing time.

In a flock which has recently had a sporadic outbreak of BD, the entire lamb crop and the sheep suspected of or shown by blood tests to have introduced infection must be removed from the farm before the start of the next breeding season. Disposal by slaughter is the only way of preventing further spread of disease. Control of infection in endemically infected flocks by the identification and disposal of persistently infected sheep may not be practicable. In such a flock, control of disease can be achieved by deliberately exposing all breeding stock to known persistently infected lambs. This natural exposure of breeding stock should be done when they are not pregnant and at least two months before they are mated. The rate of virus spread will depend on the ratio of PI to susceptible animals and the closeness of their contact. Close herding indoors for at least 3 weeks is recommended for effective spread of BDV.

It is very likely that the vaccination of female breeding stock several months before tupping will ultimately play a major role in the control of BD. There are currently no vaccines available against BD and there is a need to develop one to protect sheep against both the BD virus which predominates in them and the antigenically distinguishable viruses which have been recovered from sheep but which are more closely related to BVD virus isolates from cattle (21, 47, 75). In the meantime, in countries where BVDV vaccines are licensed for use in cattle, their use in sheep could be considered. Such vaccines, however, should be critically evaluated to see if they afford protection against local isolates of BDV.

BORDER DISEASE OF GOATS

There is serological evidence that BD occurs in goats in Africa (12, 67), North America (25, 36), Australia (24) and Europe (44). There appears to have been only a single case of spontaneous clinical BD (44), although pestiviruses have been isolated from aborted goat fetuses (52, 56) and the lung of a kid which died at 4 months of age (23).
Experimental infections of pregnant goats with BDV produces severe placentitis and clinical and pathological signs in the offspring similar to severe BD in sheep (9, 32, 42). Goats also appear very susceptible to HCV: intravenous inoculation of 13 pregnant goats with virulent HCV between 64 and 84 days of gestation resulted in all their offspring becoming infected. Six goats kept until parturition produced 6 normal and HCV antibody positive, one apparently normal but dead, one mummified and 3 oedematous kids (65).

PESTIVIRUS INFECTION IN FARMED RED DEER

The only other domestic ruminant from which a pestivirus has been recovered is a penned red deer calf which died approximately 24 hours after first being seen unwell. It was considered that the non-cytopathic virus was unlikely to have been responsible for the symptoms and fatal outcome of this illness but that it may have been a contributory factor (50).

The experimental infection of healthy red deer calves with BVD and BD viruses results in seroconversion without obvious disease (45; Nettleton, unpublished findings). A serological survey of 371 British farmed deer revealed 6% had antibodies to pestiviruses (Nettleton, unpublished findings).

PESTIVIRUS INFECTION OF RUMINANTS IN ZOOLOGICAL COLLECTIONS

Outbreaks of disease with similarities to mucosal disease in cattle have been described in captive ruminants in zoological collections in the Hanover Zoo and four zoos in the USA.

Lack of virological confirmation means that the outbreak in the Hanover Zoo cannot be attributed with certainty to a pestivirus infection (35). Although extensive virological and serological investigations were carried out in the American outbreaks, interpretation of the results was complicated by the diagnosis of malignant catarrhal fever (MCF) in some of the animals and by administration of BVDV vaccines to in-contact animals. Nevertheless, pestiviruses were isolated from two wildebeest (Connochaetes spp.), a nilgai (Boselaphus tragocamelus), an axis deer (Cervus axis) a barasingha deer (Cervus duvauceli), muntjac (Muntiacus reevesi), serow (Capricornis sumatraensis), pigmy goats (Capra hirus) and a waterbuck (Kobus ellipsiprymnus). There was also serological evidence of pestivirus infection in 49 species of ruminant but many of these had been vaccinated (20). The species with antibodies apparently due to natural infection are included in Table II.

Veterinarians in charge of valuable zoological collections should be aware of the dangers of pestivirus infection in ruminants, and the possibility of enhanced pathogenicity of isolates crossing species barriers. A policy of quarantine and testing to prevent the introduction of PI animals should be followed.
<table>
<thead>
<tr>
<th>Species</th>
<th>Continent [Captive (C) or free-living (FL)]</th>
<th>Evidence for infection</th>
<th>Reference</th>
</tr>
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<td>Camelidae</td>
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<tr>
<td>Axis deer (Cervus axis)</td>
<td>America (C)</td>
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<td>20</td>
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<td>Barasingha deer (Cervus duvauceli)</td>
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<td>20</td>
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<td>Chinese water deer (Hydropotes inermis)</td>
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<td>Europe (FL)</td>
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<td>3</td>
</tr>
<tr>
<td>Pygmy goat (Capra hircus)</td>
<td>America (C)</td>
<td>VI</td>
<td>20</td>
</tr>
<tr>
<td>Chamois (Rupicapra rupicapra)</td>
<td>Europe (FL)</td>
<td>AB</td>
<td>3</td>
</tr>
<tr>
<td>Serow (Capricornis sumatraensis)</td>
<td>America (C)</td>
<td>VI</td>
<td>20</td>
</tr>
<tr>
<td>Urial (Ovis vignei)</td>
<td>America (C)</td>
<td>AB</td>
<td>20</td>
</tr>
<tr>
<td>American bighorn (Ovis canadensis)</td>
<td>America (C)</td>
<td>AB</td>
<td>54</td>
</tr>
<tr>
<td>Barbary sheep/Aoudad (Ammotragus lervia)</td>
<td>America (C)</td>
<td>AB</td>
<td>20</td>
</tr>
<tr>
<td>Mountain goat (Oreamnos americanus)</td>
<td>America (C)</td>
<td>AB</td>
<td>20</td>
</tr>
<tr>
<td>African buffalo (Syncerus caffer)</td>
<td>Africa (FL)</td>
<td>VI/AB</td>
<td>29, 59</td>
</tr>
<tr>
<td>Gaur (Bos gaurus)</td>
<td>America (C)</td>
<td>AB</td>
<td>20</td>
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</tbody>
</table>
PESTIVIRUS INFECTION OF FREE-LIVING RUMINANTS

Pestiviruses have been recovered from dead roe deer (Capreolus capreolus) (62), fallow deer (Dama dama) (77), African buffalo (Syncerus caffer), giraffe (Giraffa camelopardalis) (29, citing W. Plowright) and wildebeest (Connochaetes spp.) (55). The role of the viruses in causing diseases in these free-living ruminants is uncertain. Similar uncertainty relates to outbreaks of disease resembling mucosal disease in white-tailed deer (Odocoileus virginianus) and mule deer (Odocoileus hemionus) in the USA (35).

Serological surveys have revealed the presence of antibodies to pestiviruses in the sera of several species of free-living ruminants (Table II). In Europe, low seroprevalence rates of 5 to 10% have been detected in red deer (Cervus elaphus), sika deer (C. nippon), roe deer (Capreolus capreolus), fallow deer (Dama dama), chamois (Rupicapra rupicapra) and ibex (Capra ibex) (3, 38, 40). In North America, antibody to BVD virus has been demonstrated in white-tailed and mule deer, pronghorn antelope (Antilocapra americana) and moose (Alces alces) (35, 71).

An outstanding study on 3,359 sera from ruminants and other species in nine African countries by Hamblin and Hedger (29) detected antibody to BVDV (NADL strain) in 17 of 29 species of ruminants. Seroprevalence rates of between 30 and 87% were detected in African buffalo, kudu (Tragelaphus strepsiceros), eland (Taurotragus oryx), waterbuck, defassa waterbuck (Kobus defassa), reedbuck (Redunca arundinum), sable antelope (Hippotragus niger) and oryx (Oryx gazella). It is reasonable to believe that disease due to pestiviruses is occurring in these 8 species at least, since seroprevalence rates of this order are associated with a widespread low incidence of disease in sheep and cattle. It is also reasonable to suppose that many other species of ruminants harbour pestiviruses.

CONCLUSION

Among ruminants other than cattle, pestiviruses are known to be serious pathogens of sheep in which they cause BD. Studies on this important congenital disease have helped to demonstrate the pivotal role of fetal infection in pestivirus infections.

Of the other domestic ruminants, goats are very susceptible to experimental infections with pestiviruses but natural disease is rare and although pestiviruses have been isolated from red deer there is no evidence that they cause disease in this species.

Similarly, the role of pestiviruses in other ruminant populations is largely unknown although there is some evidence that they may be significant: infection has been demonstrated serologically in more than forty species. Outbreaks of disease similar to those caused by pestiviruses in domestic ruminants have been described in captive and free-living ruminants, with pestiviruses being isolated from some such cases.

The elucidation of the epidemiology of pestiviruses in free-living ruminants and the antigenic relationship of isolates from various species would make an important contribution to further understanding of the biology of these fascinating viruses.

* * *
LES INFECTIONS PROVOQUÉES PAR LES PESTIVIRUS CHEZ LES RUMINANTS AUTRES QUE LES BOVINS. - P.F. Nettleton.

Résumé: Les pestivirus peuvent infecter un grand nombre de ruminants, domestiques, vivant en captivité ou en liberté. Le virus de la maladie de la frontière (border disease) est responsable d’une maladie congénitale dont l’importance est bien connue. On l’observe pratiquement dans tous les pays d’élevage ovin. L’auteur expose en détail les signes cliniques, la pathogénèse, le diagnostic, l’épidémiologie et la prophylaxie de cette maladie. On a décrit un foyer de maladie de la frontière apparu dans les conditions naturelles chez des chèvres domestiques. On a également la preuve sérologique et virologique de la fréquence des pestivirus dans cette espèce. Un pestivirus a été isolé chez un cerf d’élevage (Cervus elaphus) et on a la preuve sérologique que l’infection se rencontre chez cette nouvelle espèce domestique, dans de nombreux pays, avec une faible prévalence. Les pestivirus ont aussi été responsables de foyers de maladie chez des ruminants en captivité dans des jardins zoologiques.

Parmi les ruminants vivant en liberté, des pestivirus ont été identifiés chez le chevreuil européen (Capreolus capreolus), le daim (Dama dama), le buffle (Syncerus caffer), la girafe (Giraffa camelopardalis) et le gnou (Connochaetes spp.), mais dans aucun de ces cas la responsabilité du virus dans le développement de la maladie n’a été établie avec certitude. Des enquêtes sérologiques ont montré que beaucoup d’espèces de ruminants, vivant en liberté en Amérique du Nord, en Europe et en Afrique, présentent des anticorps dirigés contre les pestivirus, avec une prévalence variable.


* * *

LAS INFECCIONES PROVOCADAS POR PESTIVIRUS EN RUMIANTES DISTINTOS DE LOS BÓVIDOS. - P.F. Nettleton.

Resumen: Los pestivirus pueden infectar gran cantidad de rumiantes, tanto domésticos como en cautividad o en libertad. El virus de la enfermedad de la frontera (border disease) es responsable de una enfermedad congénita cuya importancia es bien conocida. Se lo observa en prácticamente todos los países de explotación ganadera ovina. El autor expone detalladamente los síntomas clínicos, la patogénesis, el diagnóstico, la epidemiología y la profilaxis de esta enfermedad. Se ha descrito un foco de enfermedad de la frontera que apareció en cabras domésticas en condiciones naturales. Se cuenta asimismo con las pruebas serológica y virológica que confirman la frecuencia de pestivirus en esta especie. Un pestivirus fue aislado en un ciervo de cría (Cervus elaphus) y se tiene la prueba serológica de que la infección puede hallarse en esta nueva especie doméstica, en varios países, con baja prevalencia. Los pestivirus han sido también causa de focos de enfermedad en rumiantes cautivos en jardines zoológicos.

Entre los rumiantes en libertad, se identificaron pestivirus en el corzo europeo (Capreolus capreolus), el gamo (Dama dama), el bufalo de Africa (Syncerus caffer), la jirafa (Giraffa camelopardalis) y el hu (Connochaetes spp.), pero en ninguno de estos casos se pudo establecer con certeza la responsabilidad del
virus en el desarrollo de la enfermedad. Investigaciones serológicas mostraron que muchas especies de rumiantes que viven en libertad en América del Norte, Europa y África presentan anticuerpos contra los pestivirus, con una prevalencia variable.

PALABRAS CLAVE: Cabra - Ciervo - Enfermedad de la frontera - Epidemiología - Ovinos - Patogénesis - Pestivirus - Profilaxis - Rumiantes - Rumiantes en libertad - Virus de la diarrea viral bovina - Virus de la enfermedad de la frontera.

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