Rapid risk assessment: Schmallenberg virus in imported live animals and germplasm

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Approved for general release

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CONTENTS

1. Executive Summary .................................................. 1
2. Introduction .......................................................... 2
3. Schmallenberg virus risk assessment .......................... 3
4. References ............................................................ 7
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1. Executive summary

A new disease causing fever, reduced milk production, diarrhoea, and abortion emerged in Germany in August 2011. A virus was demonstrated by a newly developed real-time RT-PCR and named Schmallenberg virus (SBV), according to the location where the disease was first described. Since then, disease caused by SBV has been reported throughout Europe.

Horizontal transmission of SBV requires the presence of a competent vector. The only known vectors are Culicoides spp. A Culicoides surveillance programme has been operating in New Zealand since 1991. Sentinel cattle are monitored for seroconversion to viruses transmitted by Culicoides spp. To date, no seroconversion has been detected in sentinel cattle and no Culicoides have been trapped. As there is no evidence of a competent vector in New Zealand, it can be concluded that if SBV were introduced into New Zealand with imported viraemic live animals, the disease would be unable to establish.

Although recent evidence indicates SBV may be found in the semen of infected bulls, foetal malformations are only likely to occur if a foetus is infected at a vulnerable stage of pregnancy, estimated to be between day 28 and day 50 of pregnancy in sheep, in cattle between day 62 and day 110, and in goats around day 40. It is therefore very unlikely that foetal malformations would be seen in progeny derived from infected germplasm.

This assessment concludes that there is no justification for any additional risk management measures against SBV in the import health standards for live animals or their germplasm from any country.
2. Introduction

Schmallenberg virus (SBV) was first detected in blood samples taken from clinically affected dairy cows during October 2011 on a farm near the German city of Schmallenberg. Since then this virus has spread throughout much of Europe and has been associated with foetal malformations in a number of species. Recent studies have also shown that SBV may be present in the semen of infected bulls.

A number of countries have imposed sanitary measures against SBV on products from the European Union. To date, New Zealand has not considered such measures to be necessary, principally because of its established freedom from the only recognised vector of SBV, *Culicoides* spp.

The Animal Imports team of MPI has requested an assessment of the risks posed by SBV in imports of live animals and their germplasm. This assessment considers the risks posed by the introduction of SBV associated with any live animal species or germplasm from countries where this virus has been reported.
3. Schmallenberg virus risk assessment

3.1. HAZARD IDENTIFICATION

3.1.1. Aetiological agent

Schmallenberg virus (SBV) is an emerging orthobunyavirus of ruminants (64). Viruses belonging to the Orthobunyavirus genus are mostly mosquito-transmitted although some are tick transmitted. Arthropods such as culicoid flies and phlebotomines are also recognised to transmit orthobunyaviruses. Other members of this genus recognised as having animal health importance include Akabane virus and Simbu virus (2).

3.1.2. OIE list

SBV is not listed by the OIE.

In February 2012, the OIE convened a meeting of experts to review existing knowledge of this new virus and provide information to its Members and to stakeholders. Based on the available information at that time, the expert group concluded that the risk for human health was negligible. The experts also determined that the viraemic period of SBV was short and that virus transmission most likely occurs by vectors such as mosquitoes or biting midges, with apparent similarity to the transmission of the bluetongue virus. The experts also assessed the risk of the possible spread of the disease through trade and concluded that the risk of disease spread from trade in meat and milk was negligible whereas for semen, embryos, and live animals the experts made recommendations for safe trade.

The OIE has produced a technical factsheet with information on this emerging disease (61).

3.1.3. New Zealand status

SBV has not been detected in New Zealand and is considered to be exotic (63).

3.1.4. Epidemiology

SBV was first detected from blood samples taken from clinically affected dairy cows with diarrhoea and pyrexia during October 2011 on a farm near the German city of Schmallenberg. Metagenomic analysis of these samples detected a novel orthobunyavirus of cattle (10). The first acute infections that became associated with SBV were noted in August 2011, while the first malformations in stillborn animals caused by this virus were detected in the Netherlands in December 2011 (3).

Following the recognition of SBV, infection spread over a large area of Europe including the Netherlands (11, 12), Belgium (13, 16), the United Kingdom (England (17, 19), the Channel Islands (24, 26), Wales (36), Scotland (40), and Northern Ireland (44)), France (18), Italy (20, 21) (including Sardinia (45)), Luxembourg (21), Spain (23), Denmark (29, 30), Switzerland (31-35), Austria (36), Poland (37, 54), Sweden (38, 53), Finland (40, 42, 52), Norway (41), Ireland (43), the Czech Republic (49, 53), Hungary (50), Estonia (51, 53), and Slovenia (56).
Infection of adults is followed by a short period of viraemia lasting five to six days and may be accompanied by clinical signs including fever, decreased milk production, and diarrhoea (10).

In December 2011, reports emerged (initially from the Netherlands (11) and then from Belgium (13, 14)) of SBV infection being associated with lambs born with defects such as crooked neck, hydrocephalus and stiff joints. Most deformed lambs were born dead, and those born alive were not viable. Similar signs were described in calves around the same time. It is now established that transplacental SBV infection of the foetus in utero causes malformations, with the main pathological findings being arthrogryposis, torticollis, scoliosis and kyphosis, and various brain malformations (8).

It is reasonable to assume that SBV infections act on the ruminant host in a manner similar to other members of the genus Orthobunyavirus, especially Akabane virus, and that foetal malformations are observed when infection occurs at a vulnerable stage of pregnancy. Therefore, the vulnerable stage in sheep is likely to be between day 28 and day 50 of pregnancy, in cattle between day 62 and day 110, and in goats around day 40 (7). Beer et al (2013) assumed that, in sheep, the risk period for transplacental SBV infection is days 25-38 of pregnancy, with the greatest risk on day 32 (3).

A recent study of SBV pathogenesis had identified neurons in the brainstem as the major target for viral replication in the developing foetus. The period of foetal vulnerability to SBV infection has been suggested to reflect the period between placentome development, which occurs at 28 days gestation in sheep, and the development of the blood brain barrier (between days 50 and 60 in sheep). This hypothesis explains why SBV infection of adult animals (that have an intact blood brain barrier) results in mild clinical signs with no apparent lesions in the central nervous system (64).

Very high seroprevalences have been described in dairy herds at the centre of the SBV epidemic (6). A cross-sectional survey of Belgian cattle between January and March 2012 concluded that almost all Belgian cattle had been exposed to SBV and the vast majority of these animals had developed post infection protective immunity against this virus (60). However, SBV re-emergence has subsequently been described in regions with a high seroprevalence (4 cited in 5). A high level of genetic variability has been described in SBV and it has been suggested that the accumulation of mutations in the natural course of infection may support immune evasion (5).

In addition to cattle and sheep, infection of goats with SBV and associated foetal malformation has been described (15, 37). Seropositive alpacas have been documented with no associated clinical signs (59). Wildlife surveys in affected regions have also shown a high number of SBV-seropositive red deer, roe deer, and mouflon (3, 28). It is unknown if non-ruminant species such as pigs and horses are susceptible to SBV infection (3).

Culicoides spp. are thought to be the main vector for SBV spread (3). In Belgium, pools of Culicoides spp. have been shown to be positive for SBV using RT-PCR (22, 39), with similar results reported from Italy (25), and Denmark (27, 62). The peak of SBV transplacental transmission in sheep in Germany in 2011 was shown to coincide with the peak periods of bluetongue virus serotype 8 (BTV-8) in Western Europe in 2006 and 2007, suggesting a very similar mode of transmission for SBV and BTV-8 (3).

Preliminary studies have indicated that direct horizontal transmission from infected sheep and cattle does not occur (3).
Two studies reported in December 2012 that analysis of semen from SBV-infected bulls had resulted in the detection of SBV RNA up to 3 months post-infection. Furthermore, in one bull a pattern of PCR-positive and PCR-negative consecutive semen batches was observed within 43 days, suggesting intermittent virus excretion in semen (47, 48). In January 2013 it was shown that subcutaneous inoculation of calves with 5 straws of SBV positive semen may result in SBV infection, confirmed by both RT-PCR and subsequent SBV-seroconversion (55).

The chief veterinary officers of the 27 European Union member states met in January 2012 and noted that none of the infections and diseases caused by other viruses to which SBV appears to be genetically similar, are notifiable, and that the data available suggest that the SBV infection does not deserve a different approach from diseases like Akabane. It was also stated that, based on currently available data, restrictive measures against European Union exports of ruminants and their products are not justified (1, 9).

Despite this, Russia was the first country to impose restrictions on the import of live small ruminants, meat, offal, semen, and embryos of small ruminants from Germany, the Netherlands, and Belgium (17). Since then, an increasing number of countries have introduced sanitary measures against SBV on products imported from the European Union (46, 57, 58).

3.1.5. Hazard identification conclusion

SBV infection has been described in a number of species (cattle, sheep, goats, deer, and alpacas) and bovine semen has been shown to contain infectious SBV. SBV is identified as a potential hazard in imported live animals and their germplasm.

3.2. RISK ASSESSMENT

3.2.1. Entry assessment

Live animals

SBV infection has been recognised in cattle, sheep, goats, deer, and alpacas. SBV would be most likely to be introduced into New Zealand by animals that are in the incubation period or viraemic at the time of entry. The period of viraemia for SBV is thought to be five to six days. Therefore the likelihood of a viraemic animal entering New Zealand is assessed to be low.

Semen

Recent results have shown that semen from infected bulls may contain SBV for up to 3 months after infection. The likelihood of entry in bovine semen is assessed to be non-negligible. In the absence of studies in other species, it is assumed that SBV may also be present in the semen of infected sheep, goats, deer, or alpacas.

Embryos

No studies have looked for the presence of SBV in embryos. Simbu viruses have not been reported in embryos collected for transplantation. For SBV to be transmitted, it is assumed that embryos would have to be collected during the viraemic phase of the disease. The likelihood of collecting embryos during a period of viraemia is low. IETS has classified Akabane as a
category 4 disease i.e. one for which preliminary work has been conducted or is in progress. The likelihood of SBV being transmitted in embryos is therefore assessed to be low.

3.2.2. Exposure assessment

Live animals

SBV is not contagious and could only be horizontally transmitted to other animals in New Zealand by competent insect vectors. As with other Simbu serogroup viruses, Culicoides spp. are considered to be the main vectors of SBV. A Culicoides surveillance programme has been operating in New Zealand since 1991. Sentinel cattle are monitored for seroconversion to viruses transmitted by Culicoides spp. (bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses). To date, seroconversion to arboviruses has not been detected in sentinel cattle and no Culicoides have been trapped. MPI is currently commissioning an operational research project to use wind trajectory modelling and climate modelling to evaluate this programme. Depending on the results of these studies, future surveillance may be limited to vector surveillance with serosurveillance in sentinels discontinued. As there is no evidence of a competent vector in New Zealand, the disease would be unable to establish (63).

In the absence of a competent vector in New Zealand, the likelihood of exposure is assessed to be negligible.

Semen and embryos

Imported embryos and semen would be transplanted or inseminated into susceptible recipients. Therefore, the likelihood of exposure is assessed to be high.

3.2.3. Consequence assessment

Although SBV-positive semen has been shown to transmit infection when inoculated subcutaneously, it is unknown if natural venereal transmission of this virus occurs. However, if a germplasm recipient became infected with SBV, this might result in a short period of fever, decreased milk production, and diarrhoea in that individual. However, SBV could only be horizontally transmitted to other animals in New Zealand by competent insect vectors. As there is no evidence of a competent vector being present in New Zealand the disease would be unable to establish.

Foetal malformations are only likely to occur if a foetus is infected at a vulnerable stage of pregnancy, which starts when placentomes first develop and ends when the foetal blood brain barrier begins to develop. It is therefore very unlikely that foetal malformations would be seen in progeny derived from infected germplasm.

3.2.4. Risk estimation

For live animals, the likelihood of exposure is assessed to be negligible. For germplasm imports, the consequences are assessed to be negligible. Therefore, SBV is not assessed to be a risk in imported live animals or their germplasm and risk management measures are not justified.
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