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Rapid risk assessment:

Vesicular stomatitis virus in live animals and their germplasm

Prepared for the Ministry for Primary Industries
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Contents

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1 Executive summary

Import Health Standards (IHSs) for cattle, deer, sheep, goats, alpacas, llamas and horses, and their germplasm have requirements for vesicular stomatitis (VS). Until 2015, VS had been listed as a disease of importance for international trade by the World Organisation for Animal Health (OIE). Accordingly, many of the measures in IHSs reflect the previous OIE *Terrestrial Animal Health Code* (hereafter referred to as the *Code*) recommendations.

During the OIE process for delisting VS, New Zealand provided a supportive submission (appended). Accordingly, this rapid risk assessment examines the risk of VS being introduced and establishing as a result of importing animals and germplasm. This assessment not only takes into account the OIE's rationale for delisting VS, but also, an exotic disease response context. This is because VS is clinically indistinguishable from foot and mouth disease (FMD).

New Zealand's import risk analyses for importing animals and germplasm include the possibility that an initial diagnosis of vesicular disease, before laboratory confirmation of the viral aetiology, might trigger the initial response usually reserved for FMD.

The likelihood of entry of VS in animal germplasm is assessed to be negligible and the risk of transmission through germplasm imports is assessed to be negligible. The likelihood of entry of VS for live animals is assessed to be very low, the exposure assessment is assessed to be negligible, and the risk of transmission through live animal imports is assessed to be negligible.

In the very unlikely event an imported animal ever showed clinical signs suggestive of VS, a laboratory confirmed diagnosis of VS can be attained within one day (Spence, 2016). Further, laboratory confirmation would be required before a full FMD response was initiated (Bingham, 2016; Pleydell, 2016). The testing methodologies for VS are outlined in the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* (hereafter referred to as the *Manual*).

Since there are reliable and rapid diagnostic tests available, the concerns around VS triggering an FMD response are no longer valid. There is no viraemia with VS infection and no carrier status in mammals. Further, infection is primarily insect-borne and there are no known vectors present in New Zealand.

2 Background

The OIE aims to provide transparency in the global animal disease and zoonosis situation and safeguards world trade by publishing health standards for international trade in the *Code*.

Harmonisation ensures a consistent approach to addressing risks and means that countries should base their sanitary and phytosanitary measures on relevant international standards where they exist.

In 2013, the OIE Terrestrial Animal Health Standards Commission (*Code Commission*) invited submissions on the proposed delisting of VS. New Zealand's submission supported delisting VS, concluding that:

"Natural transmission of vesicular stomatitis to humans is recognised although subsequent disease is inconsequential. Infection is not associated with significant morbidity or mortality in domestic animals, or significant morbidity or mortality in wildlife. Using the criteria in Article 1.2.2. for determining if a disease should be listed, VS should not be included in the OIE list".

New Zealand's full submission to the OIE supporting the delisting of VS has been appended (see Appendix 1).

The *Code Commission* met in February 2014 and considered Member countries' submissions on the proposed delisting of VS and reported their rationale for delisting as follows: "The *Code Commission* rejected Member Countries' requests to retain VS as a listed disease, as none provided an adequate rationale based on the listing criteria of Article 1.2.2."

The listing criteria can be found in the *Code* chapter, *Criteria for Listing Diseases* (OIE, 2015). A referenced explanation for the delisting of VS can be found in the *Code Commission's* meeting report which considered the listing criteria (international spread and effects on human and animal health etc.) (OIE, 2014). The report concluded that natural transmission of vesicular stomatitis to humans is recognised although subsequent disease is inconsequential. Infection is not associated with significant morbidity or mortality in domestic animals, or significant morbidity or mortality in wildlife.

The *Code Commission* determined that by using the criteria in Article 1.2.2., VS should not be included in the OIE list.

At the May 2015 OIE General Session, the *Code Commission's* recommendation to delist VS was adopted.

Accordingly, the disease is no longer listed by the OIE and there no international trade recommendations for risk management

3 Introduction

This rapid risk assessment examines the risks associated with importing VS susceptible species and germplasm. The re-examination of VS has been as a result of the OIE delisting the disease and removing risk management recommendations when importing animals and embryos.

The purpose of this document is to provide a risk assessment to inform the Ministry for Primary Industries (MPI) Animal Imports team in making risk management decisions with respect to VS. Currently, IHSs contain measures for VS that are based on previous risk analyses and past *Code* recommendations.

MPI Animal Imports have already made the decision to remove the VS measures for horses and horse germplasm (from the United States of America [USA]).

4 Hazard identification

4.1 AETIOLOGICAL AGENT

Vesicular stomatitis virus (VSV) is a member of the genus *Vesiculovirus* in the family *Rhabdoviridae*. The two major serotypes are New Jersey and Indiana (The Center for Food Security and Public Health [CFSPH], 2016).

VS is important mainly because it is clinically indistinguishable from foot and mouth disease (FMD) (Sellers & Daggupaty, 1990; Rodriguez, 2002; Mare & Mead, 2004).

4.2 NEW ZEALAND STATUS

VS is exotic and listed as a notifiable organism.

4.3 EPIDEMIOLOGY

The epidemiology of VS has been reviewed in several MPI live animal and animal germplasm import risk analyses and most recently in the *Report of the Meeting of the OIE Terrestrial Animal Health Standards Commission* Paris, 11-20 February 2014 (OIE, 2014).

This section contains epidemiological information reiterated from the OIE report and MPI import risk analyses.

VS is restricted to the Americas, but in the past it has also been reported in France during the First World War and in South Africa (1886 and 1897) (European Food Safety Authority [EFSA], 2012). Despite large numbers of livestock movements from the Americas, the disease failed to establish in Europe or South Africa.

The disease is endemic in Central and South America and outbreaks occur each year from southern Mexico to northern South America (Rodriguez, 2002). In the USA, the disease occurs sporadically in some southern states.

The disease occurs in horses, cattle and pigs and, more rarely, in sheep and goats (Swenson & Allende, 2015; CFSPH, 2008). Naturally occurring infections in South American camelids are rare (Bridges et al., 1995; Fowler, 1992; Schmidtmann et al., 1999).

Natural transmission of VS to humans is recognised although subsequent disease is inconsequential. Infection in humans is acute and self-limiting with signs similar to influenza. Vesicles are rare and deaths have not been reported (EFSA, 2012).

The most commonly held view is that the virus is transmitted by an insect vector. Virus has been isolated from the sand fly *Lutzomyia shannoni*, which is the most likely vector (Braverman, 1994; Comer et al., 1994; Rodriguez et al., 1996; Schmidtmann et al., 1999; Stallknecht, 2000).

The maintenance hosts of the virus have not yet been conclusively established, but deer, raccoon (Stallknecht, 2000) and the cotton rat, *Sigmodon hispidus* (Jimenez et al., 1996), have been found to have antibody to the virus.

Outbreaks of disease occur sporadically in the USA and are always associated with insect transmission (Lubroth et al., 2006; Rodriguez, 2002; Rodriguez et al., 1996). The virus is found in epithelial tissues of the mouth, nose, and coronary region of the hooves, teats and lymph nodes while it is not found in blood (Lubroth et al., 2006). There are no references to it being excreted in semen. There is no evidence of a carrier state in cattle, horses, or swine (EFSA, 2012), indicating that international spread through trade in animals and their germplasm is highly unlikely.
Lesions on teats and feet are primary lesions caused by entry of the virus directly at these sites (Wilks, 1994). Similarly, in experimental infection of pigs, lesions occurred at the injection sites but there was

no viraemia (Howerth et al., 1997). In a description of the pathogenesis of the disease it is stated that virus replicates in the lower layers of the epidermis and there is no description of viraemia (Mare & Mead, 2004). Mead (2000) states that viraemia does not occur in mammalian hosts.

Infection in animals is typified by a short febrile period and full recovery. The incubation period is short, ranging from 2 to 8 days after infection with an average of 3 to 5 days. The most common early signs are excessive salivation and drooling. The disease is characterised by vesicles, papules, erosions and ulcers. Vesicles are caused by the action of the virus on the tongue, lips, buccal mucosa, teats, and in the coronary band epithelium of cattle, horses, pigs and many other species of domestic and wild animals.

Vesicular lesions in horses generally occur on the upper surface of the tongue, lips, around nostrils, corners of the mouth, and gums. Lesions in horses may also be expressed as crusting scabs on the muzzle, lips or ventral abdomen. Affected pigs usually first show signs of lameness caused by foot lesions (EFSA, 2012). Observational studies on outbreaks indicated several subclinical infections with limited observed clinical signs, both in equidae and cattle.

Mortality is extremely low. The data on production losses are limited, but they seem to be variable (EFSA, 2012).

Although the OIE prescribes no VS-related measures for international trade for any animal since the disease has been delisted, the *Manual* provides laboratory standards for identifying VS and serological tests (ELISA, virus neutralisation and complement fixation) and antigen tests for diagnosis (Swenson & Allende, 2015).

Vesicular stomatitis virus can be readily isolated by the inoculation of several tissue culture systems, unweaned mice or embryonated chicken eggs. Viral ribonucleic acid (RNA) can be detected from epithelial tissue and vesicular fluid by conventional and real-time reverse transcriptase polymerase chain reaction (PCR). Viral antigen can be identified by an indirect sandwich enzyme-linked immunosorbent assay (ISELISA) which is the least expensive and most rapid test. The complement fixation (CF) test is also a good alternative. The virus neutralisation (VN) test may be used, but it is elaborate and time consuming (Swenson & Allende, 2015).

Convalescent animals develop serotype-specific antibodies within 4 to 8 days of infection that are demonstrated by a liquid-phase blocking ELISA (LP-ELISA), a competitive ELISA (C-ELISA) and VN. Other tests are CF, agar gel immunodiffusion and counter immunoelectrophoresis (Swenson & Allende, 2015).

4.4 HAZARD IDENTIFICATION CONCLUSION

VSV is an exotic pathogen of cattle, horses and pigs and more rarely of other animals including sheep and goats, llamas and alpacas. Therefore, it is identified as a hazard in these animal species and their germplasm.

4.5 OTHER CONSIDERATIONS

In previous MPI risk analyses, it has been stated that initial diagnosis of the disease before laboratory confirmation of the viral aetiology may trigger the massive initial response usually reserved for FMD. Alternatively, if an outbreak of FMD is incorrectly assumed to be VS, the response to the FMD outbreak might be delayed (Sellers & Daggupaty, 1990).

These concerns are no longer considered valid. In New Zealand, a full FMD response would only be initiated after laboratory confirmation (Bingham, 2016; Pleydell, 2016). As part of the testing procedure for vesicular lesion investigation of any FMD susceptible animal, laboratory testing for VS would be carried out. MPI's diagnostic tests for VS include PCR and antigen ELISA. These test results would be available within one day. Additionally, virus isolation may also be carried out if considered appropriate (Spence, 2016).

5 Risk assessment

5.1 ENTRY ASSESSMENT

There is a considerable body of evidence showing that viraemia does not occur in VS thus, introducing the virus through importing clinically healthy animals or their germplasm would not be possible. Viraemia has not been demonstrated in infected animals and there is no evidence for a carrier state in recovered animals (Howerth et al., 1997; Lubroth et al., 2006; Ministry of Agriculture and Forestry [MAF], 1999; Mare & Mead, 2004).

This may account for the failure of the disease to spread beyond the Americas.

However, imported animals in the incubatory period could introduce the virus with subsequent vesicular lesions emerging. The likelihood of this occurrence is very low since the incubatory period is short and the United States Department of Agriculture (USDA) quarantines premises where VS is suspected or confirmed in susceptible species. Quarantined properties are eligible for release 21 days after the onset of lesions in the last affected animal on the premises (Animal and Plant Health Inspection Services [APHIS], 2012).

Outbreaks are sporadic in the USA, with varying numbers of premises quarantined at any given time. There is a seasonal factor to outbreaks but currently there are no VSV-affected premises under quarantine in the USA (USDA, 2016).

Considering that the USDA quarantine VS suspect or confirmed premises, the likelihood of introducing the virus with clinically healthy imported animals is assessed to be very low in the case of animals sourced from the USA.

Germplasm is assessed to have a negligible likelihood of entry since viraemia in mammals is not known to occur (Howerth et al., 1997; Lubroth et al., 2006; MAF, 1999; Mare & Mead, 2004).

5.2 EXPOSURE ASSESSMENT

The disease is transmitted primarily by insect vectors. Outbreaks of disease occur sporadically in the USA and are always associated with insect transmission (Lubroth et al., 2006; Rodriguez, 2002; Rodriguez et al., 1996). The disease has never spread outside of the Americas, indicating that the presence of a competent vector and also other factors unique to endemic regions (e.g. suitable climatic conditions and the presence of an as yet unidentified reservoir host) are necessary for the establishment of the disease.

Virus has been isolated from the sand fly *Lutzomyia shannoni*, which is the most likely vector (Braverman, 1994; Comer et al., 1994; Rodriguez et al., 1996; Schmidtmann et al., 1999; Stallknecht, 2000). This vector is not present in New Zealand and there are no known competent vectors here.

Infected animals with vesicular lesions introduced into New Zealand could theoretically transmit the virus in a limited fashion to other animals through direct contact involving minor abrasions of the oral mucosa or skin.

In view of the above, the likelihood of exposure from importing live animals is assessed to be negligible.

5.3 RISK ESTIMATION

For animal germplasm, the likelihood of entry is assessed to be negligible. Accordingly, the risk estimate for animal germplasm is negligible.

For live animals, the likelihood of entry is assessed to be very low, and the exposure assessment is assessed to be negligible. Therefore, the risk estimate for VS in live animals is negligible.

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6 Appendix 1 – New Zealand's submission to the OIE

6.1 INTERNATIONAL SPREAD

VS is primarily an insect-borne virus but it can also be transmitted by contact (Lubroth et al., 2006). Outbreaks of disease occur sporadically in the USA and are always associated with insect transmission (Lubroth et al., 2006; Rodriguez, 2002; Rodriguez et al., 1996). The virus is found in epithelial tissues of the mouth, nose, and coronary region of the hooves, teats and lymph nodes (Lubroth et al., 2006). It is not found in blood (Lubroth et al., 2006). There are no references to it being excreted in semen.

6.2 COUNTRY FREEDOM

VS is currently limited to the Americas, but in the past it has also been reported in France (1915 and 1917) and in South Africa (1886 and 1897) (EFSA, 2012).

6.3 SIGNIFICANT MORTALITY

Infection in animals generally is typified by a short febrile period and full recovery. The incubation period is short, ranging from two to eight days after infection with an average of 3-5 days. The most common early signs are excessive salivation and drooling. The disease is characterised by vesicles, papules, erosions and ulcers. Vesicles are caused by the action of the virus on the tongue, lips, buccal mucosa, teats, and in the coronary band epithelium of cattle, horses, pigs and many other species of domestic and wild animals. Vesicular lesions in horses generally occur on the upper surface of the tongue, lips, around nostrils, corners of the mouth, and gums. Lesions in horses may also be expressed as crusting scabs on the muzzle, lips or ventral abdomen. Affected pigs usually first show signs of lameness caused by foot lesions (EFSA, 2012).

Observational studies on outbreaks indicated several subclinical infections with limited observed clinical signs, both in equidae and cattle. The mortality is negligible. The data on production losses are limited, but they seem to be variable (EFSA, 2012).

In humans, vesicular stomatitis is an acute, self-limiting infection with signs similar to influenza. The incubation period is usually three to four days, but it can be as short as 24 hours or as long as six days. The symptoms can include fever, muscle aches, headache and malaise. Vesicles are rare, but can occasionally be found on the mouth, lips or fingers. Deaths have not been reported, and most people recover without complications in four to seven days. (EFSA, 2012)

6.4 DIAGNOSIS

VSV can be readily isolated by the inoculation of several tissue culture systems, unweaned mice or embryonated chicken eggs. Viral RNA can be detected from epithelial tissue and vesicular fluid by conventional and real-time reverse transcriptase PCR. Viral antigen can be identified by an indirect sandwich enzyme-linked immunosorbent assay (IS- ELISA) – this is the least expensive and most rapid test. The complement fixation (CF) test is also a good alternative. The virus neutralisation (VN) test may be used, but it is elaborate and time- consuming (OIE, 2010)

Convalescent animals develop serotype-specific antibodies within 4–8 days of infection that are demonstrated by a liquid-phase blocking ELISA (LP-ELISA), a competitive ELISA (C-ELISA) and VN. Other described tests are CF, agar gel immunodiffusion and counter immunoelectrophoresis (OIE, 2010).

6.5 CONCLUSION

Natural transmission of vesicular stomatitis to humans is recognised although subsequent disease is inconsequential. Infection is not associated with significant morbidity or mortality in domestic animals, or significant morbidity or mortality in wildlife. Using the criteria in Article 1.2.2. For determining if a disease should be listed, vesicular stomatitis should not be included in the OIE list.

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