

**Import Risk Analysis:
Hatching eggs of domestic
ducks (*Anas platyrhynchos*
domesticus and *Cairina*
moschata) from the European
Union, Canada, the United
States of America, and
Australia**

May 2012

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1. Executive Summary

This import risk analysis supplements MAF's *Import Risk Analysis: Hatching eggs from chickens (Gallus gallus) from the European Union, Canada, the United States of America, and Australia*, dated 28 January 2009¹. The diseases considered to be a risk in chicken hatching eggs are assumed to be a risk in duck hatching eggs and the risk mitigation steps previously outlined for chicken hatching eggs are considered appropriate for the importation of duck hatching eggs.

This analysis outlines the additional biosecurity risks associated with the importation of duck hatching eggs from the European Union, Canada, the United States of America, and Australia.

As a result of further risk assessments of diseases found in ducks, it is concluded that the following organisms should be considered a risk in duck hatching eggs and options are presented for their effective management:

- Duck virus enteritis virus (anatid herpesvirus 1)
- Goose and Muscovy duck parvoviruses (Muscovy ducks and their hybrid breeds only)
- Muscovy duck reovirus (Muscovy ducks and their hybrid breeds only)
- *Chlamydia psittaci*

¹See: <http://www.biosecurity.govt.nz/files/regs/imports/risk/hatching-eggs-eu-ra.pdf>

2. Introduction

The diseases previously determined to be a risk in chicken hatching eggs are assumed to be a risk in duck hatching eggs. This risk analysis is therefore limited to a consideration of the additional biosecurity risks that may be posed by the importation of hatching eggs of Pekin ducks (*Anas platyrhynchos domesticus*) and their hybrids, or Muscovy ducks (*Cairina moschata*) and their hybrids.

3. Background

A New Zealand poultry company wishes to import duck hatching eggs from the European Union. MAF has previously examined the risks associated with chicken hatching eggs from the European Union, Canada, the United States of America, and Australia². It is assumed that the diseases previously determined to be a risk in chicken hatching eggs are also a risk in duck hatching eggs. To support the development of an import health standard for duck hatching eggs, this supplementary risk analysis specifically examines the additional risks that may be associated with duck hatching eggs.

4. Commodity Definition

The commodity considered by this risk analysis is hatching eggs of Pekin ducks, Muscovy ducks, and their hybrids, from the European Union, Canada, the United States of America, and Australia. The eggs will be sourced from duck breeding flocks compliant with the standards described in Chapter 6.4 of the OIE *Code* (or equivalent) and be clean (free of faeces) when collected, unwashed, and have intact shells (uncracked). Following collection, the eggs will be disinfected in accordance with Chapter 6.4 of the OIE *Code* (or equivalent).

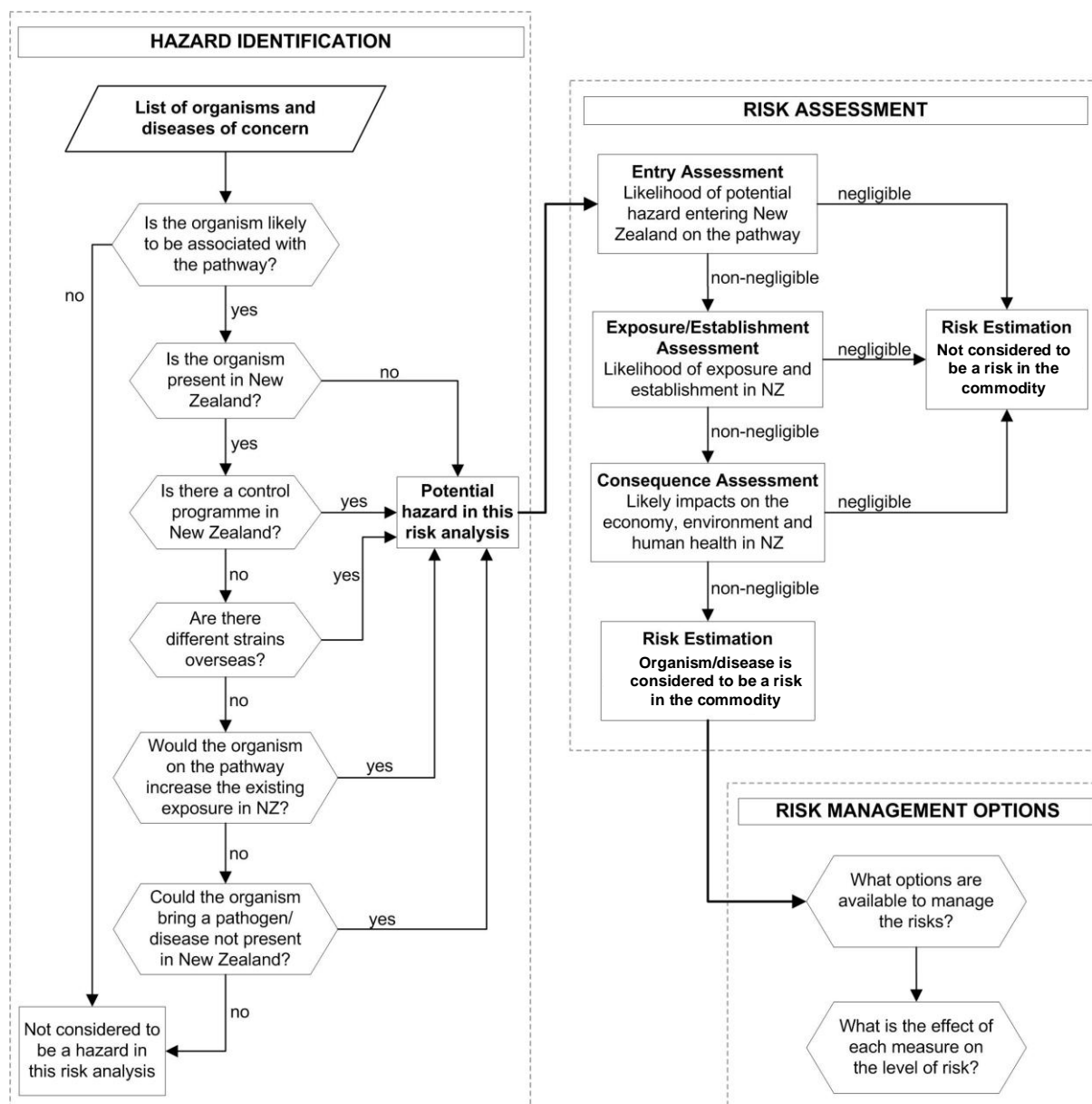
5. Risk Analysis Methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1*³ and in Section 2 of the OIE *Code*. The risk analysis process used by MPI is summarised in Figure 1.

²Import risk analysis: *Hatching eggs from chickens (Gallus gallus) from the European Union, Canada, the United States of America, and Australia*. See: <http://www.biosecurity.govt.nz/files/regs/imports/risk/hatching-eggs-eu-ra.pdf>

³ See www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf.

Figure 1. The risk analysis process



5.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms possibly associated with the commodity that were not considered in MAF's import risk analysis for chicken hatching eggs. Table 1 shows these organisms, together with some of the key information considered in determining whether each organism should be considered further. This list was compiled from the text *Diseases of Poultry*, 11th edition, ed. Saif, Y.M., and from a review of the scientific literature of poultry diseases.

Table 1. Preliminary hazards identified in duck hatching eggs

Organism/Disease	New Zealand status [#]	Infection in duck eggs?	Infection on duck eggs?	Needs further consideration?
Picornaviridae				
Duck virus hepatitis type 1	Exotic, notifiable	No	Yes	Yes
Astroviridae				
Duck virus hepatitis type 2	Exotic, unwanted	No	Yes	Yes
Duck virus hepatitis type 3	Not recognised	No	Yes	Yes
Herpesviridae				
Duck viral enteritis	Exotic, notifiable	Yes	Yes	Yes
Goose herpesvirus	Not recognised	No	No	No
Experimental transmission of goose herpesvirus was shown to cause mortality in Muscovy ducks (Gough and Hansen 2000) although natural infection of either Muscovy ducks or Pekin ducks has not been described.				
Paramyxoviridae*				
Avian paramyxovirus type 4	Present (Stanislawek et al 2002)	N/A	N/A	No
Avian paramyxovirus type 6	Present (Stanislawek et al 2002)	N/A	N/A	No
Avian paramyxovirus types 8 and 9	Not recognised	Unknown	Unknown	No
APMV-8 and 9 have been reported from ducks but are usually identified as incidental findings during surveillance for avian influenza and reports suggesting pathogenicity have not been located. (Shortridge et al 1980; Alexander and Senne 2008).				
Parvoviridae				
Muscovy duck virus	Not recognised	Yes	Yes	Yes
Goose parvovirus	Exotic, unwanted	Yes	Yes	Yes
Reoviridae				
Reovirus infection of Muscovy ducks	Not recognised	Yes	Yes	Yes
Circoviridae				
Duck circovirus	Not recognised	No	Yes	Yes
Bacteria				
<i>Chlamydia psittaci</i>	Some serovars exotic	Yes	Yes	Yes
Available evidence suggests serovars A and B of <i>C. psittaci</i> are present in the New Zealand avian population, whereas exotic serovars C and E are more commonly associated with ducks (MAF 2010).				

[#]Based on the information from the register of unwanted organisms at <http://www.biosecurity.govt.nz/pests/registers/uor> and from the Biosecurity (Notifiable Organisms) Order 2010 http://www.legislation.govt.nz/regulation/public/2010/0265/latest/whole.html?search=ts_regulation_biosecurity_resel&p=1

N/A. = Not applicable because assessment is not relevant to this process when the organism is recognised as present in New Zealand.

*Avian paramyxoviruses 4, 6, 8, and 9 were not considered to be potential hazards in the previous chicken hatching eggs risk analysis as they are not associated with chickens

5.2. HAZARD IDENTIFICATION

For each organism identified as requiring further consideration in Table 1, the epidemiology is discussed, including a consideration of the following questions:

1. Could the imported commodity act as a vehicle for the introduction of the organism?
2. If the organism requires a vector, could competent vectors be present in New Zealand?
3. Is the organism exotic to New Zealand?
4. If it is present in New Zealand,
 - i. is it "under official control", which could be by government departments, by national or regional pest management strategies or by a small-scale programme, or
 - ii. are more virulent strains known to exist in other countries?

For any organism, if the answer to question one is “yes” (and the answer to question 2 is “yes” in the cases of organisms requiring a vector) and the answers to either questions 3 or 4 are “yes”, it is classified as a potential hazard requiring risk assessment.

Under this framework, organisms that are present in New Zealand cannot be considered as potential hazards unless there is evidence that strains with higher pathogenicity are likely to be present in the commodity to be imported. Therefore, although there may be potential for organisms to be present in the imported commodity, the risks to human or animal health are no different from risks resulting from the presence of the organism already in this country.

If importation of the commodity is considered likely to result in an increased exposure of people to a potentially zoonotic organism already present in New Zealand, then that organism is also considered to be a potential hazard. However, consistent with Article 2 of the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement), any sanitary measures applied to a commodity to manage risk associated with such organisms must be no more restrictive than our domestic requirements.

5.3. RISK ASSESSMENT

In line with the MPI and OIE risk analysis methodologies, for each potential hazard requiring risk assessment the following analysis is carried out:

- | | |
|-----------------------------|---|
| a) Entry assessment - | the likelihood of the organism being imported in the commodity. |
| b) Exposure assessment - | the likelihood of animals or humans in New Zealand being exposed to the potential hazard. |
| c) Consequence assessment - | the consequences of entry, establishment or spread of the organism. |
| d) Risk estimation - | a conclusion on the risk posed by the organism based on the release, exposure and consequence |

assessments. If the risk estimate is non-negligible, then the organism is classified as a risk.

It is important to note that all of the above steps may not be necessary in all risk assessments. The MPI and OIE risk analysis methodologies make it clear that if the likelihood of entry is negligible for a potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises where the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of exposure to susceptible species in the importing country is negligible, or where both entry and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

5.4. RISK MANAGEMENT

For each organism classified as a risk, a risk management step is carried out, which identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a risk, these are described alongside options of similar, lesser, or greater stringency where available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all risks. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted. As obliged under Article 3.1 of the SPS Agreement the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

5.5. RISK COMMUNICATION

After an import risk analysis has been written, the Imports Standards Team of MPI analyses the options available and proposes draft measures for the effective management of identified risks. These are then presented in a draft IHS which is released together with a risk management proposal that summarises the options analysis, the rationale for the proposed measures and a link to the draft risk analysis. The package of documents is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to these documents are reviewed before a final IHS is issued.

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6. Duck Virus Hepatitis Types 1, 2 and 3

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Duck virus hepatitis (DVH) is caused by any one of three different viruses (Duck hepatitis virus (DHV) types 1, 2, and 3).

DHV-1 has been classified as an *Avihepatovirus*, a new genus in the *Picornaviridae* family (Tseng et al 2007). Three distinct genotypes have been identified, designated *Duck Hepatitis A virus* (DHAV) types 1, 2, and 3 (Wang et al 2008).

DHV-2 has been classified as an *Avastrovirus* species in the *Astroviridae* family (Gough et al 1985) and has been renamed *Duck Astrovirus* type 1 (DAstV-1).

DHV-3 has also been classified as an *Avastrovirus* species (Kim et al 2008; Todd et al 2009) and has been renamed *Duck Astrovirus* type 2 (DAstV-2). Sequence analysis has shown that DAstV-1 (DHV-2) and DAstV-2 (DHV-3) are distinct isolates and may represent different species (Todd et al 2009).

These duck hepatitis viruses are distinct from duck hepatitis B virus (DHBV), an *Avihepadnavirus*, which does not cause significant clinical disease in ducks (Yang et al 2008).

6.1.2. OIE list

Duck virus hepatitis is included in the OIE list of notifiable diseases.

6.1.3. New Zealand status

Duck hepatitis virus is listed as a notifiable exotic organism (MAF 2011).

6.1.4. Epidemiology

Duck virus hepatitis was first described in 1950, causing severe losses in ducklings in Long Island, New York (Levine and Fabricant 1950). DVH has subsequently been described in most duck-growing areas of the world although the viruses vary in their distributions. DHAV-1 is distributed widely and disease is usually endemic where it is found (Gough and McNulty 2008a). DHAV-2 and DHAV-3 have only been identified in Taiwan, South Korea and China (Kim et al 2009). DAstV-1 (DHV-2) has only been reported in the UK prior to 1985, and DAstV-2 (DHV-3) has only been reported in the USA (Woolcock 2008a).

Diagnosis of DVH is based on the characteristic disease pattern in the flock. Opisthotonus, ataxia, and mortality in ducklings are the most common presenting clinical signs. The main lesions are in the liver, which is enlarged with petechial and ecchymotic haemorrhages (Woolcock 2010).

It is not possible to distinguish between DHV-1, DHV-2, or DHV-3 on the basis of clinical findings and pathology alone. Distinctions can be made from the responses of ducklings, embryonated eggs, and cell cultures to the isolated viruses (Woolcock 2010).

Duck hepatitis viruses may be isolated in duck embryos, day-old ducklings, and duck embryo liver cell cultures, or less easily in chicken embryos. Immunological tests are not used extensively for the routine identification of DHV-1, DHV-2, or DHV-3. Serum neutralisation

tests can be used to assay immune responses to vaccination (Woolcock 2008b). DHV-1 and DHV-3 viruses remain viable for many weeks in faeces. Ducklings become infected as a result of contact between live birds, movement of people and/or fomites and insufficient cleaning of growing sheds (Woolcock 2008a).

Natural transmission occurs by the faecal-oral route (Tripathy and Hanson 1986; MingShu et al 1997) and recovered ducks may excrete virus in their faeces for up to 8 weeks post infection (Woolcock 2008a). Spread occurs horizontally by contact with infected ducklings or fomites (Gough and McNulty 2008a; 2008b). Newly hatched ducklings produced by breeders on infected premises remained uninfected when taken to an area where no ducks were being kept (Asplin 1958).

Vertical transmission is not thought to occur (Gough and McNulty 2008a), although there may be a possibility of transmission on egg surfaces so newly hatched ducks could become infected from this source. However, as noted in the commodity definition, the eggs will be disinfected in accordance with Chapter 6.4 of the OIE *Code* (or equivalent) following collection, which would be expected to remove any viral contamination on the egg surface.

6.1.5. Hazard identification conclusion

The aetiological agents of DVH are not transmitted in the egg and any surface contamination of imported eggs will be removed by disinfection of the commodity. It is concluded that DHV-1, DHV-2, and DHV-3 are not considered to be potential hazards in the commodity.

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7. Anatid Herpesvirus 1

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Duck virus enteritis (DVE) is also referred to as duck plague, anad herpes, eendenpest and peste du canard. The aetiological agent, a herpesvirus, is a member of the alphaherpesvirinae subfamily of the Herpesviridae (Woolcock 2008).

7.1.2. OIE list

Not listed.

7.1.3. New Zealand status

Duck virus enteritis has not been reported in New Zealand and is listed as an unwanted notifiable organism (MAF 2011).

7.1.4. Epidemiology

The disease was first described in the Netherlands in 1923 (Baudet 1923). The specificity for ducks was substantiated and the disease termed “duck plague”, a distinct viral disease of ducks (Bos 1942). DVE has been reported in the USA, Canada, Europe, Asia, and Brazil but not Australia (Hansen and Gough 2007).

Duck virus enteritis is reported in ducks, geese, and swans ranging in age from seven days to mature breeders. In susceptible flocks the first signs are often sudden high mortality with a significant drop in egg production. In flocks of chronically infected partially immune birds only occasional deaths occur (Sandhu and Metwally 2008). Birds that do recover may be immune to reinfection and become carriers, shedding virus in their faeces or on the surface of eggs for a prolonged period (Richter and Horzinek 1993; Shawky and Schat 2002).

Natural susceptibility has been limited to members of the family Anatidae (ducks, geese, coot, and swans) of the order Anseriformes. The infection has not been reported in other avian species, mammals, or humans (Jansen 1964; Sandhu and Metwally 2008). DVE is considered a threat to wild and migratory waterfowl populations (Shawky and Schat 2002).

In commercial ducks, introduction of the virus is usually associated with transmission from wild waterfowl. The spring migration and onset of breeding season for waterfowl is noted as driving seasonal occurrences. Some species are highly susceptible (such as domestic Muscovy ducks) while others (such as mallards) appear more resistant (Gough 2008).

Horizontal spread is the principal mechanism of transmission. In a susceptible host, the causative agent of DVE replicates primarily in the mucosa of the digestive tract and then spreads to the bursa of Fabricius, thymus, spleen, and liver. The virus is shed in faeces and in oral-respiratory secretions, and is transmitted by direct contact between infected and susceptible birds, or indirectly by contact with a contaminated environment (Sandhu and Shawky 2003).

Vertical transmission of herpesviruses has been recognised, including DVE, although more recent reports suggest that DVE virus may be excreted only on the surface of the egg from infected carriers (Burgess and Yuill 1983; Sandhu and Leibovitz 1997). No DVE virus was recovered from eggs laid during a natural outbreak (Burgess and Yuill 1981a). However,

virus transmission through the egg has been reported in persistently infected ducks under experimental conditions (Burgess and Yuill 1981b; Gough 2008).

Diagnosis can be confirmed by demonstration of the virus in tissue samples, virus isolation, or PCR. Antibody develops in convalescent birds and may be detected by virus neutralisation, ELISA, or immunofluorescence (Sandhu and Metwally 2008). However, healthy birds may shed virus and show no detectable serum-neutralising antibody (Burgess and Yuill 1983).

7.1.5. Hazard identification conclusion

Duck virus enteritis occurs in many countries and can cause serious disease in ducks and other Anseriformes. Experimental studies have demonstrated transmission in the eggs of infected birds so DVE virus is considered to be a potential hazard in the commodity.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

Vertical transmission of DVE through both transovarial and cloacal contamination of eggs has been described in experimentally infected birds. On that basis, the likelihood of entry for DVE in the commodity is assessed to be non-negligible.

7.2.2. Exposure assessment

If hatching eggs with DVE were imported, the most likely outcome would be the introduction of the virus into the hatchery. From the hatchery, exposure would be likely to occur by infected ducklings and mechanical spread by humans and fomites. Exposure of wild and farmed ducks to the DVE-infected ducks would then disseminate the virus.

7.2.3. Consequence assessment

The consequences of introduction of DVE into New Zealand could vary greatly. Exposure of endemic and native duck populations would likely result in increased illness and mortality. DVE produces significant economic losses due to mortality, condemnations, and decreased egg production (Sandhu and Metwally 2008). If DVE were established in New Zealand, commercial duck producers would need to consider improvements in biosecurity and vaccination.

Because natural susceptibility to DVE is limited to members of the family Anatidae of the order Anseriformes, there would be no consequences for other avian species, mammals, or humans.

7.2.4. Risk estimation

Since the entry, exposure and consequence assessments are non-negligible, the risk estimation is non-negligible and DVE virus is classified as a risk in the commodity. Therefore, risk management measures are justified.

7.3. RISK MANAGEMENT

7.3.1. Options

Diagnosis of DVE is based on a combination of clinical signs, gross pathology, and histopathology together with identification of the agent either by virus isolation or by polymerase chain reaction (PCR). Virus isolation is best achieved from samples of liver,

spleen, or kidney whilst PCR tests have been developed that can be used on various tissues including oesophagus, liver, and spleen (Woolcock 2008).

The OIE *Manual* describes no specific test for DVE for international trade. As the humoral response to natural infection with DVE virus is often low and antibodies may be short-lived, it is assumed that cell-mediated immunity also plays a role in the infection. Immunological tests have limited value, although detection of neutralising antibodies to DVE virus in serum is possible and the *Manual* describes virus neutralisation assays performed in chicken or duck embryos using embryo-adapted virus, or in cell cultures (Woolcock 2008).

The Australian import conditions for duck hatching eggs note the use of a serum neutralisation test and require that there should be no evidence of illness in the source flock from 90 days prior to egg collection (AQIS 2009). A number of other diagnostic tests to detect seroconversion have been described, including an agar gel diffusion test, immunochromatographic strip tests, passive haemagglutination assay, dot-enzyme-linked immunosorbent assay and a commercial ELISA (Wu et al 2011).

The disease, if present, would be likely detected shortly after hatching so imported eggs could be hatched in post arrival quarantine (PAQ) and investigation of any mortality within PAQ could be required.

Vaccination of the source flock could be prohibited because the effects of vaccination on the reinfection of immunised birds, their carrier status, and vertical transmission of the virus are unknown.

One or more of the following options could therefore be considered in order to effectively manage the risk:

Option 1

Eggs from flocks in countries where DVE has not been recognised could be imported without sanitary restrictions.

Option 2

Vaccination against DVE could be prohibited in flocks supplying eggs for export.

Option 3

Eggs derived from flocks where virus isolation or PCR has demonstrated flock freedom from DVE could be considered eligible for import.

Option 4

Imported eggs could be hatched under secure quarantine conditions in New Zealand and material from embryos, dead-in-shell chicks, or hatchlings could be tested for DVE by virus isolation or PCR.

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8. Goose Parvovirus and Muscovy Duck Parvovirus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family: *Parvoviridae*, Genus: *Dependovirus*, Species: *Goose parvovirus* (GPV) (also called Derzsy's disease virus (DDV)) and *Duck parvovirus* (also called Muscovy duck parvovirus (MDPV) or Barbary duck parvovirus (BDPV)) (Tattersall et al 2005).

Other waterfowl parvoviruses can be categorised into GPV-related or MDPV-related groups based on their nucleotide sequences (Poonia et al 2006).

8.1.2. OIE list

Not listed.

8.1.3. New Zealand status

Waterfowl parvoviruses (GPV and MDPV) have never been recorded in New Zealand and Derzsy's disease is listed on the unwanted exotic organisms register (MAF 2011).

8.1.4. Epidemiology

Derzsy's disease is a highly contagious disease affecting young goslings and Muscovy ducklings, and has been reported from major goose and Muscovy duck farming countries in many parts of the world, including Europe, Asia, and the USA. Derzsy's disease is known by many different names, including goose influenza, goose plague, goose hepatitis, viral enteritis of goslings, infectious myocarditis, and hepatonephritis-ascites (Gough 2008).

Domestic geese, wild geese, and Muscovy ducks (including some hybrids) are the only species in which natural clinical disease has been observed. Other breeds of duck and domestic poultry appear refractory to infection and the disease has not been reported in other avian species or mammals (Gough 2008). The disease has no known public health significance (Irvine and Holmes 2010).

GPV and MDPV differ in host ranges, antigenicity, and nucleotide sequences but the clinical signs and pathological lesions caused by either virus are similar (Chang et al 2000). GPV can cause severe disease in both goslings and Muscovy ducklings, whereas MDPV is not pathogenic for geese (Glavits et al 2005; Gough 2008). Vaccinating Muscovy ducklings with GPV provides protection against both GPV and MDPV (Woolcock et al 2000).

Derzsy's disease is strictly age dependant, with birds building a progressive resistance to infection with age. 100% mortality may occur in goslings and Muscovy ducklings under 1 week old, with negligible losses in 4- to 5- week old birds (Gough 2008; Irvine and Holmes 2010).

Older birds do not usually show clinical signs but can develop a subclinical infection, and latency may establish (Gough 2008; Irvine et al 2008; Irvine and Holmes 2010). However, some older birds may show nervous, locomotor, and enteric signs (ascites and profuse white diarrhoea), abnormal feather development and stunting (Poonia et al 2006; Gough 2008). Survivors may suffer growth retardation, loss of feathering, and ascites (Gough 2008) and

infection in older ducks results in degenerative skeletal muscle myopathy (Woolcock et al 2000; Glavits et al 2005; Poonia et al 2006).

Prominent pathological lesions in young birds include severe enteritis, hepatitis, myocarditis, and atrophy of the lymphoid organs (bursa of Fabricius, thymus, spleen). In less acute cases, perihepatitis, pericarditis, and ascites are frequent findings. In addition to the lesions observed in the geese, degenerative skeletal muscle myopathy, sciatic neuritis, and polio-encephalomyelitis are also frequently observed in Muscovy ducks infected with either GPV or MDPV (Kisary 1993; Glavits et al 2005; Poonia et al 2006).

Birds are infected either vertically via the ova or the egg-shell, or horizontally through the faecal-oral route (Schettler 1971). After entering the digestive tract, the virus replicates in the intestinal wall and then enters the blood stream leading to viraemia and dissemination (Kisary 1993; Gough 2008). In the case of egg transmission, embryos either die during incubation or hatch out in an infected state. The most serious GPV outbreaks occur following vertical transmission of the virus (Irvine and Holmes 2010).

The virus is distributed widely and has been detected in the small intestine, bursa of Fabricius, heart, liver, pancreas, spleen, bone marrow, thymus, blood, cardiac muscle, skeletal muscle, tongue, and brain of Muscovy ducklings from 2 days post-inoculation *per os* (Limn et al 1996; Takehara et al 1998; Yu et al 2002; Zhu et al 2010).

Infected birds excrete large amounts of virus in their faeces resulting in a rapid spread of infection by direct and indirect contact (Gough 2008). Infected birds younger than 1 month shed the virus continuously, even if they do not develop clinical signs (Kisary 1993). Recovered birds and those which contract the infection after the age of one month may become virus carriers. The carrier state lasts virtually lifelong (Kisary 1993) and affected birds can transmit the virus vertically in their eggs and horizontally in their faeces (Gough 2008; Irvine et al 2008; Irvine and Holmes 2010) although they do not shed the virus persistently (Gough 1987).

GPV and MDPV are extremely resistant to chemical and physical conditions and are not inactivated at temperatures of 65°C for 30 minutes or 56°C for 3 hours (Schettler 1971; Gough 2008). This means the potential for inanimate objects to spread infection is high (Kisary 1993; Poonia et al 2006), which has been demonstrated in several field outbreaks (Irvine and Holmes 2010).

Prophylaxis is based on vaccination of neonatal birds or breeding flocks, and elimination of carrier birds. Live and inactivated GPV, MDPV, and bivalent vaccines are available in most countries where the disease is endemic (Gough 2008; Irvine and Holmes 2010).

8.1.5. Hazard identification conclusion

GPV and MDPV may infect Muscovy ducks and their hybrids but not other species of duck. Since the viruses can be transmitted vertically by subclinically infected birds, they are considered to be potential hazards in the eggs of Muscovy or Muscovy hybrids but not in the eggs of other duck species.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

GPV and MDPV may be transmitted in the eggs of carrier Muscovy ducks. Therefore, the likelihood of introducing these viruses in Muscovy duck eggs from countries where the disease occurs is assessed to be non-negligible.

8.2.2. Exposure assessment

Introduction of the virus in hatching eggs would result in the hatching of infected ducklings that would be likely to transmit the disease when in contact with Muscovy ducks. Infected birds excrete large amounts of virus in their faeces resulting in rapid spread by direct and indirect contact. GPV and MDPV are extremely resistant to environmental conditions and inanimate objects can spread infection widely. Therefore, the likelihood of exposure is assessed to be non-negligible.

8.2.3. Consequence assessment

Derzsy's disease is an acute, highly fatal, rapidly spreading disease of young goslings and Muscovy ducklings. Infection of backyard Muscovy duck flocks, wild geese, or commercial poultry would be associated with non-negligible consequences.

Domestic geese, wild geese, and Muscovy ducks (including some hybrids) are the only species in which natural clinical disease has been observed (Gough 2008). There would be negligible consequences for other commercial poultry species, free-living avian species, or humans.

8.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimation is non-negligible and GPV and MDPV are classified as risks in the commodity. Therefore risk management measures can be justified.

8.3. RISK MANAGEMENT

8.3.1. Options

Serological tests do not distinguish between GPV and MDPV (Kisary 1977). Diagnostic tests for detection of antibodies include virus neutralisation, gel precipitation and ELISA tests. Virus can be detected by isolation of virus, electron microscopy on faeces or tissue sections, detection of viral antigen by immunofluorescence or by immunoperoxidase techniques, and PCR tests have been described to detect viral DNA (Gough 2008).

One or more of the following options could therefore be considered in order to effectively manage the risk:

Option 1

Eggs from duck species which are neither Muscovy ducks nor their hybrids could be imported without sanitary measures

Option 2

Muscovy duck eggs could be imported without restrictions from countries known to be free from GPV or MDPV.

Option 3

Eggs could be imported from duck flocks that are maintained as closed flocks and in which the disease has not occurred for several years (three to five years is suggested).

Option 4

Donor ducks could be kept isolated from other ducks for at least three weeks before eggs are collected, and the donors tested serologically before going into isolation and again three weeks after the end of the collection period.

Option 5

Imported eggs could be hatched under secure quarantine conditions in New Zealand and the hatchlings mixed with sentinel sero-negative New Zealand Muscovy ducklings. A representative sample of imported and sentinel birds could be tested serologically at the end of the quarantine period of at least three weeks.

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9. Reovirus of Muscovy Ducks

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Orthoreovirus*. Five orthoreovirus species are recognised, with avian reoviruses classified in group II (Fauquet et al 2005).

9.1.2. OIE list

Not listed.

9.1.3. New Zealand status

Muscovy duck reovirus (DRV) has not been recognised in New Zealand although there has been no active surveillance for this pathogen. DRV is not listed in the unwanted organisms register.

9.1.4. Epidemiology

Muscovy duck reovirus has been identified as the cause of a disease of Muscovy ducks and Barbary ducks (Robertson et al 1986). Infected ducklings show characteristic necrotic foci in the liver and spleen (Malkinson et al 1981).

Reovirus infection of Muscovy ducks has been described in South Africa, France, Israel, and China (Malkinson et al 1981). Chinese isolates have also been shown to infect Muscovy-common duck hybrids (Huang et al 2004).

Muscovy ducks and geese are affected between ten days and six weeks and present with malaise and diarrhoea. Up to 20% mortality has been reported and individuals that recover are markedly stunted. The hock and metatarsal joints, as well as the gastrocnemius and tarsal flexor tendons, and sometimes the synovial bursae are markedly swollen (Malkinson et al 1981; Jones 2003).

Transmission studies have shown that goslings, Pekin ducks, and chickens are resistant to infection with DRV when inoculated subcutaneously (Malkinson et al 1981).

Reoviruses are recognised to be transmitted vertically through the egg. Virus may persist in breeding stock for long periods and Muscovy ducklings may be protected by maternal antibody (Malkinson et al 1981).

Sequence analysis suggests that DRV should be considered a distinct genogroup from other avian reoviruses (Kuntz-Simon et al 2002; Zhang et al 2006a) and recombinant vaccines have been developed which have been shown to protect against DRV (Kuntz-Simon et al 2002).

9.1.5. Hazard identification conclusion

It is concluded that DRV is a primary pathogen in Muscovy ducks and their hybrids and there is no evidence to support the position that this disease is present in New Zealand. As reoviruses are recognised to be transmitted in eggs, DRV is considered to be a potential hazard in the eggs of Muscovy ducks and their hybrids.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

DRV may be transmitted in the eggs of carrier Muscovy ducks or their hybrids. Therefore, the likelihood of introducing the virus in the hatching eggs of Muscovy ducks or their hybrids from countries where the disease occurs is assessed to be non-negligible.

9.2.2. Exposure assessment

Introduction of the virus in hatching eggs would result in the hatching of infected ducklings that would be highly contagious and transmit the disease in contact with Muscovy ducks.

Therefore, the likelihood of exposure is assessed to be non-negligible.

9.2.3. Consequence assessment

DRV causes malaise, diarrhoea, and mortality in ducklings. Infection of duck flocks would be associated with non-negligible consequences. If DRV were introduced, commercial duck producers of Muscovy ducks and their hybrids would need to consider improvements in biosecurity and vaccination.

Goslings, Pekin ducks, and chickens are resistant to infection with DRV. It is assumed that there would be negligible consequences for other commercial poultry species, free-living avian species, or humans.

9.2.4. Risk estimation

Because the entry, exposure and consequence assessments are non-negligible, the risk estimate is non-negligible and DRV is classified as a risk in the eggs of Muscovy ducks or their hybrids. Therefore, risk management measures can be justified.

9.3. RISK MANAGEMENT

9.3.1. Options

Pekin ducks are resistant to infection with DRV (Malkinson et al 1981) so no risk management measures are required for the eggs of this species.

DRV infection can be diagnosed serologically using either a serum neutralisation test (Giambrone 1980; Heffels-Redmann et al 1992), agar gel immunodiffusion test (Olson and Weiss 1972; Adair et al 1987), or an enzyme linked immunosorbent assay (Islam and Jones 1988; Zhang et al 2007). In addition to virus isolation, RT-PCR tests have been described to detect DRV in infected individuals (Zhang et al 2006b; Lin et al 2007).

Options for the importation of duck eggs could apply only to Muscovy ducks or their hybrids. One or more of the following options could therefore be considered in order to effectively manage the risk:

Option 1

Duck eggs could be imported without restrictions from countries known to be free from DRV.

Option 2

Eggs could be imported from flocks that are maintained as closed flocks and in which the disease has not occurred for several years (three to five years).

Option 3

Donor ducks could be kept isolated from other ducks for at least three weeks before eggs are collected, and the donors tested serologically before going into isolation and again three weeks after the end of the collection period, with negative results.

Option 4

Imported eggs could be hatched under secure quarantine conditions in New Zealand and the hatchlings mixed with sentinel sero-negative New Zealand Muscovy ducklings. A representative sample of imported and sentinel birds could be tested serologically at the end of the quarantine period of at least three weeks.

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10. Duck Circovirus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Circoviridae*; Genus: *Circovirus*. The *Circovirus* genus includes the species *Beak and Feather Disease Virus* (BFDV), *Canary Circovirus* (CaCV), *Goose Circovirus* (GoCV), *Pigeon Circovirus* (PiCV), *Porcine Circovirus – 1* (PCV-1), and *Porcine Circovirus – 2* (PCV-2).

Sequencing and phylogenetic analyses have confirmed *Duck Circovirus* (DuCV) as a novel and distinct circovirus (Fringuelli et al 2005) and a tentative species in this genus (Fauquet et al 2005), along with *Finch Circovirus* (FiCV) and *Gull Circovirus* (GuCV). DuCV is closely related to GoCV (Hattermann et al 2003; Soike et al 2004).

10.1.2. OIE list

Not listed.

10.1.3. New Zealand status

BFDV (Ortiz-Catedral et al 2009), PiCV (MAF 2009), GuCV (Smyth et al 2006), PCV-1 and PCV-2 (Garkavenko et al 2005) are known to be present in New Zealand.

DuCV has not been recorded in the New Zealand duck population. However, it is recognised that many duck diseases may occur in New Zealand despite them not having been detected (Hemsley 1996). Surveillance programmes of asymptomatic waterfowl and commercial duck farms in New Zealand have been limited and no specific testing for DuCV has been performed. DuCV is prevalent in many parts of the world (Banda et al 2007) and, in the absence of any surveillance to demonstrate freedom, it is reasonable to conclude that DuCV may be present in New Zealand.

10.1.4. Epidemiology

The worldwide distribution of DuCV is unknown (Banda et al 2007). It is highly prevalent and widespread in Hungary (Fringuelli et al 2005), Taiwan and China (ShaoHua et al 2010a, 2010b; Liu et al 2009, 2010), and has also been described in Germany (Hattermann et al 2003; Soike et al 2004) and the United States (Banda et al 2007).

DuCV has been recovered from Muscovy, mulard and Pekin ducks (Hattermann et al 2003; Soike et al 2004; Banda et al 2007) and cross-infection of other avian and mammalian species is considered unlikely (McNulty and Todd 2008). This host-specificity is supported by experimental work (Chen et al 2006; Woods and Latimer 2008) and there are no reports of natural infections in atypical hosts (Woods and Latimer 2008).

The pathogenesis of DuCV, and avian circoviruses in general, is not completely understood and current knowledge is based on field experience, since experimental inoculation in ducks has not been performed (Hattermann et al 2003; McNulty and Todd 2008). However avian circovirus infections share many characteristics (Soike et al 2004) and it is reasonable to extrapolate from studies of other avian circovirus species.

Circoviral infection is typically reported in juvenile birds but has been reported in adults without previous clinical signs of disease (Banda et al 2007; Woods and Latimer 2008). In one study, ducks aged 40-60 days demonstrated greater susceptibility to DuCV infection than those at other ages (ShaoHua et al 2010a). Avian circoviruses are frequently detected in cloacal swabs several months following infection and it is known that adults may be carriers of the virus (McNulty and Todd 2008).

Horizontal transmission, via the respiratory or faecal-oral route, is thought to be the most common route of infection but vertical transmission may also occur, as with BFDV and PiCV (Fauquet et al 2005; Todd et al 2006; McNulty and Todd 2008).

There is no evidence to support a pathogenic role for DuCV in ducks (Hattermann et al 2003). DuCV has a low prevalence in the United States and the infection may not represent a significant problem for the duck industry (Banda et al 2007). However, in Hungary, Taiwan and China DuCV is associated with significant losses up to 70% (Soike et al 2004; Chen et al 2006; Woods and Latimer 2008). The factors that dictate the course and outcome of the infection are unknown but may include virus strain, route of exposure, virus dose, levels of maternal antibody and presence of other pathogens (Soike et al 2004; Fringuelli et al 2005; McNulty and Todd 2008; Woods and Latimer 2008). DuCV replicates in healthy birds, with little or no apparent signs of infection, and no pathological significance has been proven (Hattermann et al 2003).

Circoviruses are highly dependent on cellular enzymes for replication, which is typically intranuclear and occurs in targeted rapidly dividing cells such as the basal feather follicular epithelium, lymphoreticular tissues and intestinal crypt epithelium (Schmidt et al 2008; Woods and Latimer 2008). An *in situ* hybridisation (ISH) study of geese showed that GoCV could be found in all tissue types tested (bursa of Fabricius, spleen, thymus, bone marrow, liver, kidney, lung and heart) with the exception of brain, and was most abundant in the bursa, liver and small intestine (Smyth et al 2005). PiCV has additionally been found in the brain and semen of pigeons (Duchatel et al 2009). DuCV DNA has been isolated from extracts of liver, spleen, kidney, bursa, thymus, Harderian gland, heart, blood, lung and pancreas of ducks (Li et al 2009), but the target cells and major sites of virus replication remain to be identified (McNulty and Todd 2008). Intracytoplasmic globular or botryoid inclusions in these tissues are characteristic of other circovirus infection (McNulty and Todd 2008) but are not a common feature of DuCV infection (Fringuelli et al 2005).

Damage to lymphoreticular tissue impairs both humoral and cellular immune functions. This predisposes affected birds to secondary pathogens (Soike et al 2004; McNulty and Todd 2008) including *Escherichia coli*, *Riemerella anatipestifer*, *Pasteurella multocida*, duck hepatitis virus type 1 and reovirus (Liu et al 2010; ShaoHua et al 2010a). DuCV infection is commonly associated with growth retardation, feathering disorders and increased rearing losses (Soike et al 2004; Chen et al 2006; Woods and Latimer 2008) as well as signs of secondary infection, including mild arthritis and fibrinous pericarditis (Banda et al 2007). Many birds experience mild, subclinical infections (McNulty and Todd 2008; Liu et al 2010) and DuCV has been isolated in a healthy duck (ShaoHua et al 2010b). No particular clinical syndrome can be specifically associated with DuCV infection (Smyth et al 2005) and most mortalities result from secondary infections (Woods and Latimer 2000).

10.1.5. Hazard identification conclusion

Avian circoviruses closely related to DuCV are known to be present in New Zealand and DuCV is widespread in many parts of the world. DuCV can be present in healthy ducks

(ShaoHua et al 2010b) and there is no evidence to support a pathogenic role for DuCV in ducks (Hattermann et al 2003). In the absence of surveillance for DuCV in New Zealand there is no evidence to suggest that it should be considered exotic. DuCV is therefore assessed not to be a potential hazard.

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11. Avian Chlamydiosis

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Obligate intracellular gram-negative bacteria, *Chlamydia psittaci* (Andersen and Vanrompay 2008). Eight serovars, distinguished using monoclonal antibodies and with differences in their predominant host ranges, are recognised (Gilardi et al 1995).

Six serovars (A to E) of *Chlamydia psittaci* are recognised in birds (Everett et al 1999) :

- Serovar A is endemic in psittacines,
- Serovar B in Columbiformes, with some presence in turkeys,
- Serovar C has been isolated most frequently from Anseriformes with reports also from turkey and partridge,
- Serovar D is most common in turkeys (Phasianiformes) with single isolates being identified from a seagull and a budgerigar,
- Serovar E has been reported from ducks, pigeons, and ratites,
- Serovar F has been reported only from a single psittacine (parakeet).

11.1.2. OIE list

Listed.

11.1.3. New Zealand status

Psittacosis was first described in 66 imported Australian parrots in 1954, with diarrhoea, listlessness, and death affecting at least 31 birds (Cairney 1954). Laboratory investigations between 1984 and 1985 identified *C. psittaci* isolates from budgerigars, parakeets, pigeons, rosellas, and cockatiels (Bell and Schroeder 1986). Psittacosis is considered to be prevalent in New Zealand wild pigeons, with a prevalence rate of between 9.5% and 25% (Motha et al 1995).

An unpublished survey of faecal samples from captive and wild endangered and threatened avian species was reported by Motha et al (1995) to have detected *C. psittaci* in a number of species, including kakapo, takahe, and kiwi. However, a subsequent survey found no evidence of psittacosis in native psittacines with perhaps the exception of kakas and wekas on Kapiti Island and it was suggested that the earlier unpublished findings were likely to have been false positive results due to the choice of test (Motha et al 1995).

Given the patterns of host preference of *C. psittaci* serotypes, the above evidence is consistent with the presence of serotypes B (Columbiformes) and possibly A (Psittaciformes) of *C. psittaci* in the New Zealand avian population (MAF 2010).

11.1.4. Epidemiology

C. psittaci serovars can be distinguished in specialised laboratories by a panel of serovar-specific monoclonal antibodies (Andersen 1991; Andersen 1997). Restriction fragment length polymorphism analysis and genotyping techniques are also available to distinguish

serovars (Vanrompay et al 1997; Geens et al 2005). Of the 8 known serovars of *C. psittaci*, serotypes C and E are associated with ducks.

Chlamydiosis in ducks is usually a severe disease, with morbidity up to 80%, with up to 30% mortality (Andersen and Vanrompay 2008) although some outbreaks may be associated with few clinical signs (Arzey et al 1990; Newman et al 1992; Hinton et al 1993).

Transmission of *C. psittaci* occurs primarily through inhalation of contaminated material, with large numbers of chlamydiae found in the respiratory tract exudate and faeces of infected birds (Andersen 1996).

Vertical transmission of *C. psittaci* has been demonstrated in several avian species, including ducks, although the frequency of this appears to be fairly low (Wittenbrink et al 1993). It has been suggested that *C. psittaci* may either penetrate the shell of freshly laid eggs or be present in the egg contents due to either sperm contamination or transovarian haematogenous spread (Dickx and Vanrompay 2011). Vertical transmission is recognised as a possible route of introduction of *C. psittaci* into a poultry flock (Harkinezhad et al 2009).

Serologic evidence of *C. psittaci* infection of ducks was described in 1942 and within three years human infections due to contact with infected ducks had been reported (Harkinezhad et al 2009). Human cases of severe respiratory distress have been associated with zoonotic *C. psittaci* infections acquired from infected duck farms (Laroucau et al 2009). Transmission to humans typically involves inhalation of infectious aerosols during the handling of infected material. The symptoms of human infection range from severe systemic disease to a complete absence of clinical signs (Andersen and Vanrompay 2000).

11.1.5. Hazard identification conclusion

It is not known which serovars of *C. psittaci* are present in New Zealand although there is no evidence to indicate the presence of serovar C in the New Zealand duck population. Although transmission of *C. psittaci* primarily occurs horizontally from one infected bird to another susceptible bird in close proximity, vertical transmission is described and recognised as a possible route of introduction into a poultry flock.

Reflecting this, exotic strains (serovars C and E) of *C. psittaci* are considered to be a risk in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

C. psittaci may be transmitted in the eggs of infected ducks. Therefore, the likelihood of introducing the organism in duck hatching eggs imported from countries where serovars C and E of *C. psittaci* have been described in ducks is assessed to be non-negligible.

11.2.2. Exposure assessment

Although the occurrence of vertical transmission of *C. psittaci* appears to be fairly low, an outbreak of disease in a flock can be started from a single infected chick (Andersen and Vanrompay 2000).

Air-sampling of a hatchery for *C. psittaci* found high titres of live organisms following hatching of fumigated eggs whose shells were found to be negative for *C. psittaci* by both

PCR and culture (Dickx and Vanrompay 2011). The hatching of infected chicks is therefore highly likely to result in dissemination of infection to other chicks

Therefore, the likelihood of exposure is assessed to be non-negligible.

11.2.3. Consequence assessment

In ducks, serovar C of *C. psittaci* has a morbidity rate of 10-80% and a mortality rate of 0-30%, with some duck farms being infected with few or no clinical signs. Concurrent infections or stress increase the severity of the disease (OIE 2009).

Serovar E of *C. psittaci* has a diverse host range, and has been isolated from cases of fatal chlamydiosis in ratites, from outbreaks in ducks and turkeys, and occasionally from humans (Harkinezhad et al 2009).

Humans can become infected after inhaling contaminated dust, feathers or aerosolized secretions and excretions. Direct contact with infected birds can also spread disease. Human symptoms vary from mild, flu-like illness to severe atypical pneumonia with dyspnoea. Illness usually lasts for 7 to 10 days although complications including miscarriage, endocarditis, myocarditis, renal disease, hepatitis, anaemia and neurological signs are described (OIE 2009).

Considering the above, the consequences of introducing serovars C and E of *C. psittaci* are considered to be non-negligible.

11.2.4. Risk estimation

Because the entry, exposure and consequence assessments are non-negligible, the risk estimate is non-negligible and serovars C and E of *C. psittaci* are classified as a risk in imported duck eggs. Therefore, risk management measures can be justified.

11.3. RISK MANAGEMENT

11.3.1. Options

Because isolates of *C. psittaci* associated with ducks are limited to serovars C and E, there is no need for laboratory tests to distinguish between any serovars of *C. psittaci* isolated from breeding flocks or hatched ducklings.

The OIE *Code* contains no recommended measures to manage the risk of *C. psittaci* in imported hatching eggs.

Pharyngeal or nasal swabs are the preferred samples for isolating *C. psittaci* from live birds using cell culture techniques or embryonated eggs (Andersen 1996). Intestinal content, cloacal swabs, conjunctival scrapings and peritoneal exudate may also be used (OIE 2008).

Histochemical staining of impression smears of liver and spleen can be used to demonstrate the presence of *C. psittaci* using Giemsa, Gimenez, Ziehl-Neelsen and Macchiavello's stains. Immunohistochemical techniques are also described which are more sensitive than histochemistry although these require greater experience to ensure correct interpretation (OIE 2008).

A modified direct complement fixation test to detect serological evidence of exposure to *C. psittaci* is widely used (OIE 2008). Other serological tests available include the agar gel immunodiffusion test (Page 1974), the latex agglutination test, the elementary body

agglutination test (Grimes et al 1994; Grimes and Arizmendi 1996) and the micro-immunofluorescence test (OIE 2008).

One or more of the following options could therefore be considered in order to effectively manage the risk:

Option 1

Duck eggs could be imported without restrictions from countries where chlamydiosis has not been reported in commercial ducks.

Option 2

Eggs could be imported from flocks that are maintained as closed flocks and in which ongoing surveillance has demonstrated freedom from *C. psittaci*.

Option 3

Donor ducks could be kept isolated from other ducks for at least three weeks before eggs are collected, and the donors tested serologically for exposure to *C. psittaci* before going into isolation and again three weeks after the end of the collection period, with negative results.

Option 4

Imported eggs could be hatched in isolation in New Zealand and a representative sample of chicks could be tested serologically. Any dead hatchlings or dead-in-shell chicks could be examined to detect the presence of *C. psittaci* using histochemical staining of liver and spleen impression smears.

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