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Rapid Risk Analysis

Canine influenza virus subtypes H3N8 and H3N2 in imported dogs and cats

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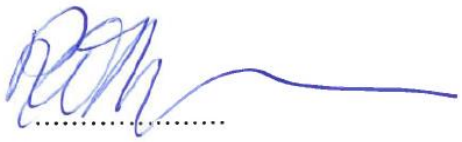
Approved for IHS development



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1.0	First peer-reviewed version	February 2019

New Zealand is a member of the World Trade Organisation and a signatory to the Agreement on the Application of Sanitary and Phytosanitary Measures ("The Agreement"). Under the Agreement, countries must base their measures on an International Standard or an assessment of the biological risks to plant, animal or human health.

This document provides a scientific analysis of the risks of canine influenza virus types H3N8 and H3N2. It assesses the likelihood of entry, exposure, establishment and spread of these agents in relation to imported cats and dogs and assesses the potential impacts of these organisms should they enter and establish in New Zealand. The document has been internally and externally peer reviewed.

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Contents

1 Executive summary

This document presents a qualitative analysis of the risk posed by canine influenza virus subtype H3N8 (CIV H3N8) and canine influenza virus subtype H3N2 (CIV H3N2) in dogs and cats imported into New Zealand.

The methodology for this risk analysis follows the guidelines as described in Biosecurity New Zealand Risk Analysis Procedures — Version 1 and in Chapter 2 of the Terrestrial Animal Health Code of the World Organisation for Animal Health (OIE), (2018).

The likelihood of CIV H3N8 and/or CIV H3N2 being present in imported dogs is assessed to be high. The likelihood of subsequent exposure and transmission of these pathogens to susceptible New Zealand domestic dogs is assessed to be moderate. The consequences of entry and establishment of CIV H3N8 and CIV H3N2 are assessed to be moderate. Both CIV H3N8 and CIV H3N2 are assessed to be presenting a moderate risk in imported dogs.

In imported cats, the likelihood of CIV H3N2 being present, the subsequent exposure and transmission of the pathogens, and the consequences of entry and establishment are assessed to be low.

Overall risk of CIV H3N2 in imported cats is assessed to be low.

Accordingly, risk management options have been presented which include one or a combination of the following measures:

1. No diagnosed cases of the disease in dogs or cats in the exporting country in the three months before export.
2. The dog or cat was not exposed to other dogs/cats with respiratory disease during the 21 days prior to shipment.
3. The dog was fully vaccinated against CIV H3N8 and H3N2 in the exporting country between 365 days and 14 days before export.
4. The cat or dog was tested negative for CIV H3N8 and CIV H3N2 with a polymerase chain reaction (PCR) test conducted on a sample collected within 5 days prior to export.

2 Introduction

Background

The Animal Import Team of the Ministry for Primary Industries, New Zealand (MPI) has requested a qualitative analysis of the biosecurity risks associated with the importation of cats and dogs infected with CIV H3N8 and CIV H3N2.

The "*Import Risk Analysis (IRA): Cats, Dogs and Canine Semen - November 2009"* (IRA 2009) assessed only the influenza A virus subtype H5N1 and estimated the risk of it entering New Zealand via imported dogs or cats to be negligible. Since the IRA 2009 was published, two other influenza virus subtypes (canine influenza virus subtype H3N8 and canine influenza virus subtype H3N2) have emerged in dogs in several countries, causing mild to severe respiratory disease (Crawford et al., 2005; Song et al., 2008). There have also been a number cases of influenza A infections reported in cats (Jeoung et al., 2013).

MPI complied the "*Rapid Risk assessment*: Canine Influenza H3N2" (RRA 2016) in 2016 following the first canine influenza virus subtype H3N2 outbreak in dogs in Chicago. This risk assessment concluded that the likelihood of canine influenza virus subtype H3N2 entering New Zealand through imported dogs was negligible. That document however, did not address the potential risk posed by imported cats infected with canine influenza virus subtype H3N2.

A significant proportion of dogs and cats imported into New Zealand originate from canine influenza endemic countries. In 2017, collectively 11.68% of dogs and 11.98% of cats that were imported into New Zealand came from the United States, Canada, Singapore and South Korea where canine influenza outbreaks have been reported (MPI, 2018).

Current Import Health Standard

Based on the IRA 2009 and RRA 2016 the Import Health Standard (IHS) for cats and dogs managed the risk associated with canine influenza viruses through pre-export clinical examination and certification on the freedom from clinical signs of contagious and/or infectious diseases prior to export to New Zealand.

In February 2018, a dog that was imported from the United States displayed clinical signs of a respiratory disease on arrival in quarantine in New Zealand. Diagnosis of canine influenza subtype H3N2 was confirmed by PCR testing of nasal secretions on 2 March 2018. All dogs arriving in New Zealand from approved countries, except from Australia must spend 10 days in quarantine.

Following this incident in March 2018 MPI made an urgent amendment to the IHS, which states that

(1) For at least 21 days prior to shipment:
   a) The cat or dog was not kept in a place where there were cats or dogs showing clinical signs of infectious respiratory disease; and
   b) The cat or dog showed no clinical signs of infectious respiratory disease.

Justification for reviewing the current Import Health Standard

The above measure may have contributed to the prevention of CIV infected cats and dogs entering New Zealand as since this measure was put in place no CIV cases in NZ quarantine occurred. However, this measure relies on importer/owner declarations and does not mitigate the risk of a dog or cat incubating the disease during shipment after exposure to an infected asymptomatic animal(s) prior to leaving the country of origin for New Zealand.

Given the wider distribution of canine influenza virus subtypes H3N8 and H3N2 in some countries and the potential consequences of these viruses spreading and establishing in New Zealand, there is a need for further risk assessment and to evaluate appropriate risk mitigation options.

This rapid risk analysis examines the risks posed to New Zealand domestic dogs (*Canis familiaris*) and cats (*Felis catus*) by importing dogs/cats infected with canine influenza virus subtype H3N8 and canine influenza virus subtype H3N2 and evaluates the possible risk management measures.

3 Methodology

3.1. GENERAL PROCEDURES

The methodology used in this risk analysis is consistent with the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1* (Biosecurity New Zealand, 2006), and the OIE Handbook on Import Risk Analysis for animals and animal products (OIE, 2010). The risk analysis process comprises three main steps: Hazard identification, risk assessment and risk management options.

3.1.1. Hazard identification

Hazard identification includes formal identification of the organism (potential hazard associated with the commodity), whether it is the cause of an OIE listed disease, its New Zealand status, and a discussion on the epidemiology and characteristics of the organism and the disease. The hazard identification section is concluded by a determination of whether the organism is identified as a hazard or not. If the organism is identified as a hazard, it is subjected to risk assessment.

3.1.2. Risk assessment

Risk assessment consists of:

a) Entry assessment: The likelihood of a hazard (pathogenic organism) being imported with the commodity.

b) Exposure assessment: Describes the biological pathway(s) necessary for exposure of susceptible animals or humans in New Zealand to the hazard and the ability for the organism/disease to establish and spread in the country.

c) Consequence assessment: Describes the likely potential consequences of entry, exposure and establishment or spread of an imported hazard.

d) Risk estimation: An estimation of the risk posed by the hazard associated with importing products. This is based on the entry, exposure and consequence assessments. If the risk estimate is assessed to be higher than negligible (i.e. High, Moderate, or Low) then the hazard is assessed to be a risk and risk management measures may be justified to reduce the level of risk to an acceptable level.

Not all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of entry is negligible for a certain 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 when the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of susceptible species being exposed is negligible, or when both entry and exposure are non-negligible but the consequences of introduction are assessed to be negligible.

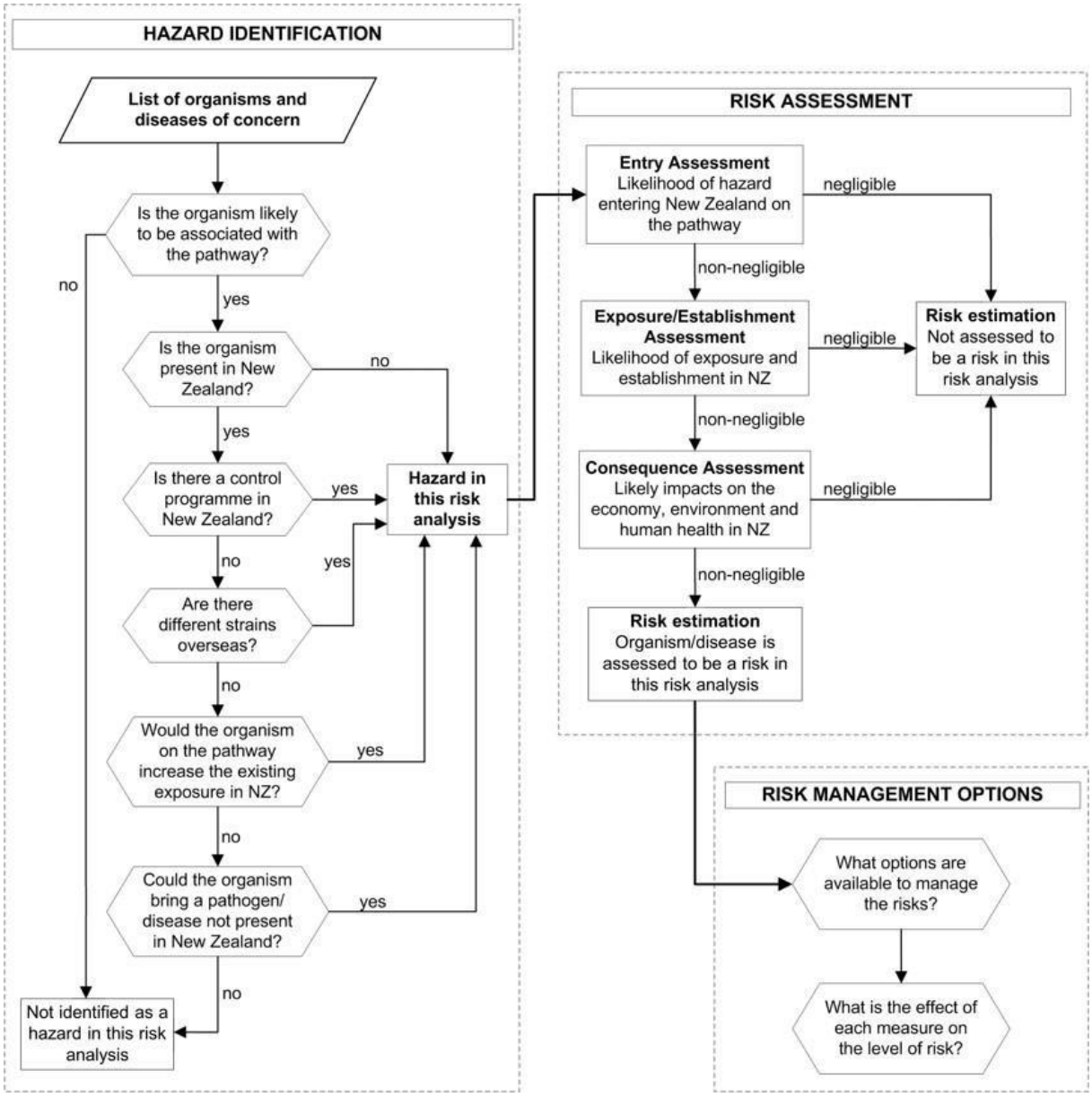

Figure 1. **The risk assessment process.**

3.1.3. Risk management

For each organism assessed to be a risk, options are identified for managing that risk. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when the IHS and risk management proposal documents are drafted.

As obliged under Article 3.1 of the World Trade Organization's Agreement on the application of Sanitary and Phytosanitary measures (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. That is, 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.

For each canine influenza virus subtype assessed to be a risk several risk management options are identified. A qualitative evaluation of the level of risk reduction for each identified risk

management option also is presented. This evaluation is intended as a guide for selection of appropriate risk management options.

3.1.4. Risk communication

After a draft import risk analysis has been written, MPI analyses the options available and proposes draft measures for the effective management of the identified risks. These are then presented in a draft IHS that is released for public comment, together with a risk management proposal (RMP) that summarises the options analysis, the rationale for the proposed measures and provides a link to the draft risk analysis.

4 Scope and commodity definition

This rapid risk analysis qualitatively assesses the risk due to canine influenza virus subtype H3N8 and canine influenza virus subtype H3N2, associated with the importation of dogs and cats from all countries.

5 Canine influenza A virus subtype H3N8

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family: Orthomyxoviridae
Genus*: Influenzavirus A*
Species (Type): CIV H3N8
This is a 'negative-sense', segmented ribonucleic acid (RNA) virus.

5.1.2. OIE list

Canine influenza infection caused by CIV H3N8 is not an OIE listed disease.

Infection with avian influenza viruses in poultry is an OIE listed disease. In addition, infection with avian influenza viruses of high pathogenicity in birds other than poultry (including wild birds) and equine influenza are OIE listed diseases (OIE, 2019).

5.1.3. New Zealand status

A serological survey conducted in 2009 in New Zealand did not detect antibodies for CIV in 251 serum samples obtained from a wide range of breeds of dogs of various ages. This survey used the blood samples submitted to the diagnostic laboratory as a part of diagnostic protocol for a range of clinical and non-clinical conditions and represented a wide geographic area of the country (Knesl, Allan & Shields, 2009).

CIV H3N8 is not a notifiable organism in New Zealand (Biosecurity (Notifiable Organisms) Order 2016).

5.1.4. Epidemiology

The Type A influenza viruses are further classified into 'subtypes' based on serologic reactivity of two surface glycoproteins, 'hemagglutinin' (HA) and 'neuraminidases' (NA) (Zhu et al., 2015; Daly et al., 2008). To date 16 HAs and 9 NAs have been reported (Tong et al., 2013). Each Influenza A subtype is named indicating its unique HA and NA protein combination (e. g. H3N8, H3N2).

Wild birds are known to be the main natural reservoir for influenza A viruses as most subtypes have been isolated from wild birds (Webster et al., 1992). Host specificity of various influenza A virus subtypes is not absolute. Most RNA viruses, including influenza A viruses tend to undergo mutation and assortment at a higher rate. Both these processes have led to influenza A virus subtypes crossing the species barriers and establishing in domestic birds, pigs, horses, dogs and humans (Sanjuan et al., 2010).

Until 2004 infection with Influenza A viruses has been known to cause respiratory diseases in humans, horses, pigs and domestic poultry. Dogs were not regarded as a reservoir for influenza A viruses because:

1. Dogs were not shown to be maintaining a canine-specific influenza A virus, and
2. Dog to dog transmission of influenza A virus had not been recorded (Gibbs & Anderson, 2010).

The capacity of influenza A viruses to cross the species barrier from horses to infect dogs was first identified in 2004. Crawford et al. (2005) reported an outbreak of canine respiratory disease in Florida, U.S.A. with more than 20 racing greyhounds being affected. The phylogenetic analyses showed that this virus originated due to direct interspecies transmission of equine influenza A virus subtype H3N8 (Crawford et al., 2005). Although this virus strain contained all eight genes of equine influenza virus, its monophyletic divergence makes it a distinct strain different to ancestral equine strain, therefore can be identified as "canine influenza virus" (Gibbs & Anderson, 2010).

Since the first outbreak in Florida the respiratory disease caused by CIV H3N8 has been diagnosed in greyhounds and pet dogs in most states of the U.S.A. and the District of Columbia (American Veterinary Medical Association [AVMA], 2019; Gibbs & Anderson, 2010). Anderson et al. (2013) stated that *"…results of the surveillance activities have indicated a wide geographic distribution of the virus with infections in pet and shelter dogs occurring sporadically in 39 states".* As of 2019, 43 states of the U.S.A. have reported CIV H3N8 outbreaks (Merck Animal Health, 2018) and the virus continues to spread.

Prevalence of CIV H3N8 in the U.S.A. varies in different geographical regions. In a cross-sectional study, serum samples collected during the 2005–2009 period from pet and shelter dogs with respiratory signs were tested with hemagglutination inhibition (HI) test for CIV H3N8 antibodies. An overall seroprevalence of 49% (618/1268, CI 46%–51%) dogs in 42 states, for antibodies against CIV H3N8 was estimated. However, investigators cautioned against extrapolating the results of this study for all dogs in the U.S.A., acknowledging the limitations of the study; dogs were not randomly selected and not all states were equally represented (Anderson, Crawford, Dubovi, Gibbs & Hernandez, 2013). Daly et al. (2008) retrospectively demonstrated evidence that a canine influenza outbreak in the United Kingdom that occurred in 2002 may have been caused by CIV H3N8.

Canine influenza cases have been reported in Australia. These cases occurred during the equine influenza outbreak in 2007. Several dogs that were in contact with horses infected with equine influenza virus subtype H3N8 developed influenza-like illness, although the virus could not be isolated from the affected dogs. Moreover, there was no epidemiological evidence that the virus had spread from dog to dog (Kirkland et al., 2010). Since Australia regained its country freedom from equine influenza in June 2008, no further cases were reported in dogs (Gibbs et al., 2010).

Zhou et al. (2016) conducted a serological survey of 600 samples collected from dogs in four major cities in China. Five of 600 (0.83%) sera samples were positive for influenza A subtype H3N8, but this study did not differentiate the specific strain of the virus whether it was CIV H3N8 or equine influenza H3N8 or avian influenza H3N8.

Like all influenza viruses, CIV H3N8 is contagious. It spreads via aerosolized droplets, direct contact with respiratory secretions containing the virus and through indirect contact with contaminated fomites. When aerosolized droplets containing virus particles are inhaled, the virus deposits on the mucous film covering epithelium of the upper respiratory tract. Infective virions deposited on fomite surfaces may gain entry to the epithelium of the upper respiratory tract through nares, conjunctiva or oral cavity (Hilling & Hanel, 2010). Faecal shedding of CIV H3N8 has not been reported and the virus can be detected in respiratory secretion of both symptomatic and subclinically infected dogs (CFSPH, 2016).

Under natural conditions, CIV H3N8 appears only to be affecting dogs (Su et al., 2014) and it spreads efficiently in the general dog population, causing widespread infection (Crawford et al., 2005; Anderson et al., 2011; Deshpande et al., 2009).

Jirjis et al. (2010) demonstrated dog to dog transmission of CIV H3N8 infection by exposing eight healthy dogs to four dogs that were experimentally infected with CIV H3N8. Prior to the experiment, all dogs tested negative with HI for CIV H3N8 antibodies. All dogs developed a range of clinical signs including ocular and nasal discharge, sneezing coughing, dyspnea and depression. The clinical signs peaked between 6–8 days post infection (dpi) and diminished from 9 dpi onward. However, seven out of eight dogs in contact-exposed group had varying degrees of coughing from the 7 dpi that lasted until 21 dpi. All experimentally infected dogs excreted virus in nasal secretions from 1 dpi while contact-exposed commenced shedding virus in secretion on 3 dpi.

Dogs of all ages and breeds are susceptible to infection (AVMA, 2019; Hilling & Hanel, 2010). Infected dogs shed virus before they exhibit clinical signs and shedding continues during the clinical phase of the disease (Hilling & Hanel, 2010).

In a transmission study, Crawford et al. (2005) tested sera samples collected from 46 asymptomatic dogs. These dogs were housed with dogs that were clinically affected by CIV H3N8. Out of 46 samples, 43 (93%) were seropositive for CIV H3N8. Crawford et al. (2005) stated that "*high seroprevalence in dogs with no history of respiratory disease indicates that infection with canine influenza virus can be subclinical and suggests efficient spread of the virus among dogs*".

The main risk factor for transmission is the exposure to infected dogs that are shedding the virus in group-housing environments such as animal shelters, kennels, and veterinary hospitals (AVMA, 2015). A study indicated that the infection was established and maintained in kennels with large numbers of incoming susceptible dogs (Hayward et al, 2010).

Although it originated from an equine strain of H3N8, the ability of CIV H3N8 to infect and establish in horses seems to have been lost (Rivailler et al., 2010). Collins et al. (2014) attributed the inability of CIV H3N8 to re-infect horses to the substantial changes in amino acid composition in hemagglutinin protein, compared to that of equine H3N8.

Notwithstanding the above, a virus transmission experiment conducted in China demonstrated that equine influenza H3N8 can be successfully transmitted to domestic cats with virus shedding and overt clinical signs. Experimentally infected cats transmitted the virus to contact cohorts (Su et al., 2014). However, reports on natural transmission of CIV H3N8 to cats are not available.

Information is lacking on survival of CIV H3N8 in the environment. It is considered to behave in a similar manner to other mammalian influenza A viruses (CFSPH, 2016) where influenza A and B viruses were shown to have survived for 24-48 hours on hard, nonporous surfaces such as stainless steel and plastic, but survived for less than 8-12 hours on cloth, paper, and tissues (Bean et al., 1982).

Clinical signs in CIV H3N8 infected dogs

Incubation period for CIV H3N8 typically ranges from one to five days with the majority of cases appearing within 2–3 days.

Around 80% of the dogs infected with H3N8 show clinical signs of upper respiratory tract infection. The remaining 20% of infected dogs that do not exhibit clinical signs can still shed the virus and spread the infection (AVMA, 2019; Hilling & Hanel, 2010).

Two clinical syndromes have been described: a mild upper respiratory tract form and a severe lower respiratory tract form.

In the mild form, clinical signs are consistent with initial infection of the upper respiratory tract. The clinical signs include a moist, soft cough that may persist for 10–30 days, purulent nasal discharge, and low-grade fever. This form of the disease is usually self-limiting unless secondary bacterial or viral infections occur. Clinical signs of upper respiratory tract infection with CIV H3N8 may mimic the clinical signs of the respiratory disease commonly known as 'kennel cough' caused by *Bordetella bronchiseptica* and parainfluenza virus complex (Payungporn et al., 2008; Hilling & Hanel, 2010).

In the severe form the lower respiratory tract is involved. Approximately 1–5% of the infected dogs develop the severe form of the disease (Parry, 2016). Clinical signs include high grade fever, tachypnea, purulent nasal discharge, depression and anorexia. These clinical signs are consistent with bronchopneumonia, including consolidation of the lungs and pleural effusion. A small percentage of infected dogs die peracutely from hemorrhagic pneumonia. Necroscopic examination of these dogs showed extensive hemorrhages in lungs, mediastinum and pleural cavity. Tracheitis, bronchitis, bronchiolitis, and suppurative bronchopneumonia were evident in histological examination (Crawford et al., 2005).

5.1.5. Hazard identification conclusion

The canine respiratory disease outbreaks caused by CIV H3N8 reported only in the U.S.A. Since it's detection in 2004, the virus has spread to over 43 states of the U.S.A. It is evident that movement of infected dogs is an effective pathway for the introduction of CIV H3N8 from one location to another.

Canine influenza infection caused by CIV H3N8 is now endemic in the U.S.A., from where New Zealand regularly imports dogs and this represents a pathway for the introduction of CIV H3N8 into New Zealand.

New Zealand is considered to be free from CIV H3N8. A serological survey, which used broadly reactive antibodies for antigens common across all influenza A viruses, demonstrated the absence of antibodies for influenza A viruses in dogs in New Zealand (Knesl et al., 2009).

CIV H3N8 meets the criteria for a hazard, therefore, it is concluded that CIV H3N8 is hazard in dogs imported from the U.S.A into New Zealand.

No cases of CIV H3N8 in cats have been reported therefore, it is not a hazard in cats imported into New Zealand.

6. Canine influenza virus subtype H3N2

6.1 HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: Orthomyxoviridae +
Genus*: Influenzavirus A*
Species (Type): CIV H3N2

This is a 'negative-sense', segmented RNA virus.

6.1.2. OIE list

Canine influenza infection caused by CIV H3N2 is not an OIE listed disease.

All avian influenza virus infections in poultry, highly pathogenic avian influenza virus infections in all bird species and equine influenza are OIE listed diseases (OIE, 2019).

6.1.3. New Zealand status

CIV H3N2 has not been detected in New Zealand (Knesl et al., 2009) and no reported cases of CIV H3N2 in New Zealand.

In February 2018, a dog that was imported from the United States displayed clinical signs of respiratory disease during post-arrival quarantine in New Zealand. Samples of nasal secretions tested positive for CIV H3N2 with a PCR test. Another dog that was quarantined in the premises at the same time with the infected dog was later diagnosed with CIV H3N2 after being given biosecurity clearance and it was later returned to quarantine (MPI, 2018). However, there is no evidence of the infection spreading outside of the quarantine and the organism did not establish in New Zealand (MPI, 2018).

6.1.4. Epidemiology

The canine influenza virus sub-type H3N2 contains hemagglutinin protein H3 and neuraminidase N2. There is a human strain of influenza virus sub-type H3N2 which has been shown to occasionally infect dogs (Chang et al., 1976; Voorhees et al., 2017; McCullers et al., 2011).

Host specificity of various influenza A virus subtypes is not stable and genomic changes have led to the emergence of new virus strains, which have crossed the species barriers and established in domestic birds, pigs, horses, dogs and humans (Lyoo et al., 2015).

An influenza virus A strain traversing a species barrier has been reported in South Korea in 2007. Song et al. (2008) reported a severe respiratory disease in dogs that were housed in geographically separated veterinary clinics in South Korea. Four outbreaks occurred with all affected dogs showing signs of severe respiratory disease. In one outbreak, all infected animals died two days after visiting the same animal hospital. Nasal swabs collected from all three dogs were tested positive for influenza A virus subtype H3N2 with reverse-transcription Polymerase Chain Reaction (RT-PCR). No other pathogens known to cause canine respiratory disease were detected in the samples. Song et al., (2008) hypothesized that this strain of the

virus has been originated through the assortment of genetic components of several influenza subtypes. Although CIV H3N2 was first detected in South Korea in 2007, a study conducted by Li et al. (2010) provided evidence of CIV H3N2 being present in China in 2006, before the South Korean outbreak.

CIV H3N2 is endemic in South Korea with a wide range of seroprevalence rates. Lee et al. (2009) conducted a serological survey of dogs during June to December 2007 in South Korea. Eight hundred and twenty nine (829) sera samples were collected from dogs residing in dog-farms and in pet dogs. The recorded CIV H3N2 prevalence ranged from 0.5% in pet dogs to 19% in farmed dogs.

At the time of above disease occurrences, avian influenza A subtypes H3N2, H5N1, H6N1 and H9N2 were circulating in domestic ducks and chicken in South Korea (Choi et al., 2005; Song et al., 2008). Researchers postulated that CIV H3N2 has been transmitted to dogs through feeding untreated meat of ducks or chicken. Given the close contacts among domestic chicken, ducks and dogs in South Korea, aerosol transmission of the virus from infected birds to dogs is also possible (Song et al., 2008). Li et al. (2010) hypothesized that a dog-adapted strain of avian influenza A subtype H3N2 proven to have existed in China during 2006–2007 period crossed the international barriers to South Korea through the pet dog trade.

CIV H3N2 is circulating also in China. A survey carried out in China using 882 canine serum samples collected between January 2012 and June 2013 recorded a seropositivity rate of 3.5% (Li et al., 2010). According to Lyu et al. (2019) the seropositivity rate has reached 6.3% by 2017. Moreover, the currently circulating clade is different to the clade that existed in 2012–2013 period (Lyu et al., 2019). It is believed CIV H3N2 originated in China and then reached South Korea (Li et al., 2010; Song et al., 2008).

Thailand has reported CIV H3N2 outbreaks in dogs. Bunpapong et al. (2014) reported the first outbreak in pet dogs in Thailand in January 2012. They concluded that a CIV H3N2 strain, closely related to Chinese and South Korean strains, is emerging in Thailand and causing an infectious respiratory disease in pet dogs.

In April 2015, the first CIV H3N2 outbreak in the U.S.A. occurred in Chicago. Sequence analyses of the virus suggested a single introduction of the virus, which then spread to the wider canine population (AVMA 2015; Voorhees et al., 2017). One news source indicated that more than 1000 dogs were affected in this outbreak with six deaths (AVMA, 2015).

Another outbreak of CIV H3N2 in dogs occurred in Connecticut in August 2018 (ProMed, 2018). Towards the end of 2018, over 30 states had reported canine influenza cases caused by CIV H3N2 (IDEXX Laboratories, 2018). CIV H3N2 is endemic in the U.S.A.

The most recent CIV H3N2 outbreak in dogs occurred in Oakland, California in June 2019 (ProMed, 2019).

Canada reported its first CIV H3N2 outbreak in January 2018 in dogs (International Society for Infectious Diseases [ProMed], 2018; Arsevska et al., 2018).

Canine respiratory disease outbreaks caused by CIV H3N2 have also occurred in Singapore. ProMed (2018) posted an article quoting a newspaper report of CIV H3N2 outbreak in Singapore. Over 500 dogs at several animal shelters were affected and a dog exported from Singapore to Australia tested positive for canine influenza in Australian quarantine (Department of Agriculture and Water Resources [DAWR], 2018). Following prompt application of biosecurity measures, CIV did not establish in Australia.

CIV H3N2 appears to have a broad host range infecting dogs, cats, ferrets and guinea pigs (Lee et al., 2016; Lyoo et al., 2015).

To demonstrate dog-to-dog transmission of CIV H3N2, Song et al. (2008) used the virus isolated from the dogs involved in the South Korean outbreak to experimentally (intranasally) infect nine beagle puppies. Sneezing, nasal discharge and fever developed in the inoculated group after 2–7 days post–inoculation, while the control group did not show any of the clinical signs. Virus shedding in nasal discharge began at 1 dpi and continued to 6 dpi. The highest viral titer, $10^{6.1}$ (EID50/0.1mL) was reached by 4 dpi. Virologic, serologic, pathologic and phylogenic analyses were conducted and the results showed cross-species infection of an avian influenza A subtype H3N2 to dogs (Song et al., 2008).

CIV H3N2 has also been shown to infect domestic cats causing respiratory disease. Influenza A subtype H3N2 infection in cats has been reported since 1970. Paniker and Nair (1970) experimentally inoculated kittens with human influenza virus A2/Hong Kong/68. The kittens showed no clinical signs but shed the virus for 1 week, developing antibodies and transmitting the virus to a susceptible kitten. This influenza virus A2/Hong Kong/68 strain was later identified as influenza A H3N2 (Su et al., 2014).

Zhu et al. (2015) analyzed 24 canine and one feline isolates of CIV H3N2 and found that all genetic segments except for one, originated from Eurasian avian influenza viruses. Jeoung et al. (2013) compared genomes of isolates of CIV H3N2 from of dog and cats involved in an outbreak with the genome of the first CIV H3N2 strain (South Korean) and found no more than 2% divergence among HA gens of all the strains. The investigators concluded that the same CIV H3N2 strain infects both dogs and cats (Zhu et al., 2015; Jeoung et al., 2013).

Song et al. (2011) reported a CIV H3N2 outbreak in cats in South Korea. This outbreak occurred from December 2009 to January 2010 in cats that resided in an animal shelter in Seoul which housed both dogs and cats. Dogs developed the disease first. Investigations revealed that both species were infected with CIV H3N2. In this outbreak, dogs showed 25% mortality while cats showed 100% morbidity and 40% mortality (Song et al., 2011). Another transmission study reported 46.6% morbidity and 21.7% mortality in cats (Jeoung et al., 2013).

Experimental inoculation of susceptible cats with CIV H3N2 isolated from clinical cases reproduced the disease (Song et al., 2011; McCullers et al., 2011). The cats with the experimental CIV H3N2 infection had nasal shedding of viral titres ranging from $10^{2.8}$ to $10^{3.8}$ EID_{50}/mL^{-1}, which was lower than viral titers detected in experimentally infected dogs (Song et al., (2011).

McCullers et al. (2011) investigated the distribution of CIV H3N2 antibodies in cats in the U.S.A. by analyzing 117 sera samples collected from cats in diverse geographic locations and reported 25.6% seroprevalence. The virus appears to be endemic in the domestic cat population in the U.S.A. with a high seroprevalence rate.

CIV H3N2 can be a potential public health risk. Human strains of influenza A subtype H3N2 have not caused clinical disease in dogs. However, CIV H3N2 and the human strains of H3N2 share common biologic and physiologic features. Re-assortment of these strains in dogs to produce a strain with zoonotic potential cannot be ruled out (Voorhees et al., 2017; McCullers et al., 2011).

Zhu et al. (2015) forewarned that dogs have the potential to act as a 'mixing vessel' species in which avian, human and canine influenza viruses can reassort leading to the emergence of a new influenza virus strains with pandemic potential.

Several other authors have noted a public health concern in the possible emergence of new recombinant feline or canine influenza viruses in companion animals, with zoonotic potential (Song et al., 2011; Jeoung et al., 2013).

Inhalation of CIV H3N2 particles and contact with infected dogs or contaminated fomites transmits the disease to susceptible dogs and cats. Droplets and aerosols created by coughing and sneezing by infected animals, direct contact with nasal discharge (nose to nose contact) and contact with fomites contaminated with the virus provide sources of infection for uninfected dogs or cats. Segregation of susceptible dogs and cats in closed environments with infected animals favours transmission of the virus (CFSPH, 2016).

An oral route of infection also has been suggested. According to Song et al (2008), feeding dogs with untreated chicken and duck meat has transmitted the entire genome of the avian influenza A subtype H3N2 virus from infected birds to dogs. These researchers experimentally demonstrated dog-to-dog transmission of CIV H3N2 by inoculating the virus (isolated from naturally infected dogs) to healthy dogs, thus reproducing the disease (Song et al., 2008). Dog- to-dog transmission of this virus has also been experimentally demonstrated by Lee et al., 2009. Faecal shedding of CIV H3N2 by infected dogs does not occur, even in immunocompromised dogs (Hong et al 2013).

An important feature of exposure to CIV H3N2 is that asymptomatic infections occur. Dogs without clinical signs of respiratory disease and history of respiratory disease have been serologically tested positive (CFSPH, 2016; Bunpapong et al., 2014; Hilling et al., 2010; Song et al., 2008; Voorhees et al., 2017).

Dogs may continue to shed the virus up to 20–30 days after they become seropositive, requiring longer quarantine period for infected dogs in order to control the disease spread (AVMA, 2015).

Although dog-to-cat and cat-to-cat transmissions of CIV H3N2 has been demonstrated information about transmission from infected cats to dogs is lacking.

Natural and experimental infection of cats with CIV H3N2 have been reported (Song et al., 2008; McCullers et al., 2011; CFSPH, 2016; Kim et al., 2012).

In cats viral shedding has been reported on 2, 4 and 7 dpi (Song et al., 2011). These authors stated that cats can horizontally acquire the H3N2 infection in animal shelters shared with infected dogs.

Clinical signs in CIV H3N2 infected dogs and cats

CIV H3N2 infects dogs of all breeds and ages, leading to a respiratory disease syndrome (Hilling & Hanel, 2010).

Clinical signs are typical of respiratory tract infections. Fever, anorexia coughing, sneezing and nasal discharge are common clinical signs shown by CIV H3N2 infected dogs. These signs may be mild or severe and tend to subside within 7–10 days in most cases. However, during the outbreaks in South Korea and Thailand significant numbers of deaths were recorded.

Dogs experimentally infected with CIV H3N2 developed fever within 1–3 days, with clinical signs of respiratory disease appearing 2–8 days after inoculation. In cats respiratory signs developed 2–7 days after experimental inoculation (CFSPH, 2016).

Cats infected with CIV H3N2 show coughing, dyspnea and tachypnea and lethargy (CFSPH, 2016). Experimentally infected cats developed fever, nasal discharge, depression, coughing, sneezing and abdominal breathing 3 dpi (Song et al., 2011). Some cats developed conjunctivitis 2 dpi (Lei et al., 2012).

6.1.5. Hazard identification conclusion

The dog and cat importations have been shown to be an effective pathway for the introduction of canine influenza virus from one country to another. Through the international movement of dogs and cats, CIV H3N2 transmitted from China to South Korea (Li et al., 2010), from South Korea to Thailand (Bunpapong et al., 2014), from the U.S.A. to Canada (Arsevska et al, 2018), from Asia to the U.S.A (Voorhees et al., 2018) from Singapore to Australia (DAWR, 2018) and from the U.S.A to New Zealand (MPI, 2018).

Based on the absence of serological evidence (Knesl et al., 2009) and lack of any reports of CIV H3N2 infections, New Zealand is currently free from CIV H3N2.

CIV H3N2 is endemic in the U.S.A., South Korea and Canada (Hayward et al., 2010; Lee et al., 2009; Song et al., 2008; Arsevska et al., 2018) from which New Zealand allows dog and cat imports. The current endemic status is not clear in Singapore which has reported canine influenza outbreaks in 2018 (ProMed, 2018; DAWR, 2018). Canine influenza disease status is not known for other countries from which New Zealand permits dog and cat imports.

Dog and cat imports represents an effective pathway for the introduction of CIV H3N2 into New Zealand.

CIV H3N2 meets the criteria for a hazard, therefore, it is concluded that CIV H3N2 is a hazard in dogs and cats imported into New Zealand.

6.2 RISK ASSESSMENT

6.2.1 Entry assessment

While CIV H3N8 is endemic in the U.S.A, CIV H3N2 is endemic in several countries including the U.S.A., Canada, and South Korea. New Zealand imports a significant proportion of dogs and cats from these countries.

Nine (CIV H3N2) canine influenza outbreaks have occurred in the U.S.A. from 2015 –2017 (Voorhees et al., 2018) while Canada reported one outbreak in 2018 (ProMed, 2018; Arsevska et al., 2018). South Korea continues to report canine influenza outbreaks in dogs and cats (Media report, The Korean Times, 2019). The current endemic status is not known for some countries with a history of canine influenza outbreaks (e. g. Singapore, Thailand). These countries have not declared freedom from canine influenza.

Both CIV subtypes H3N8 and H3N2 have no breed predilection or age limitations (Hilling & Hanel, 2010). New Zealand does not have import restrictions for dogs other than dogs from five prohibited breeds. Dogs less than eight weeks old cannot be imported into New Zealand. (MPI, CATDOG.GEN, 2018).

Canine influenza is a community acquired infection (Macejko, 2009) and both viruses spread from infected dogs to susceptible dogs via inhalation, direct contact with secretions of infected dogs and indirect contact with contaminated objects (AVMA, 2017; Hilling & Hanel, 2010). Infection commonly occurs in premises with high –frequency animal movements, such as shelters, boarding facilities, veterinary clinics (Lyoo et al., 2015).

A high percentage of dogs exposed to CIV H3N8 have been shown to be seropositive with no overt clinical signs, indicating subclinical infection in exposed animals (Crawford et al., 2005). Similarly, CIV H3N2 antibodies have been detected in asymptomatic dogs suggesting subclinical infection (CFSPH, 2016).

Infected dogs shed virus before clinical signs appear (Hilling & Hanel, 2010). The likelihood of dogs or cats destined for New Zealand being infected with these pathogens, while in premises with high –frequency animal movements during the immediate pre-export period cannot be ruled out. In 2018, a dog subclinically infected with CIV H3N2 arrived in New Zealand from the U.S.A. It was revealed later that this dog had been exposed to the virus in an animal shelter in the U.S.A. before export to New Zealand (MPI, 2018). Similarly, an infected asymptomatic dog arrived in Australia from Singapore and developed the clinical signs while in quarantine in Australia (DAWR, 2018).

As per the New Zealand import requirements only clinically healthy animals that are fit to travel are likely to be certified (MPI, CATDOG.GEN, 2018). However, the detection of subclinically infected or exposed dogs or cats during the pre-export clinical examination is highly unlikely. If a dog were to be infected with either pathogen just before leaving the originating country for New Zealand, it is likely that it would arrive in New Zealand incubating canine influenza virus infection.

Given that:

- both pathogens have successfully sustained infectivity and transmissibility in dog populations (Lee et al, 2009; Payungporn et al, 2008),
- disease caused by these pathogens is endemic in some countries,
- New Zealand permits dog imports from countries that are canine influenza endemic and from countries with histories of canine influenza outbreaks but endemic status is unclear,
- dogs of all breeds and all ages are susceptible for the infection,
- the likelihood of aggregation of dogs that are being prepared for export to New Zealand with canine influenza virus infected dogs,
- the detection of sub-clinically infected dogs prior to export is unlikely and
- dog imports presents a clear pathway for canine influenza virus to enter into New Zealand, as evidenced by the canine influenza case in New Zealand quarantine in 2018, the likelihood of entry of CIV H3N8 and/or CIV H3N2 into New Zealand through imported dogs is assessed to be high.

Cats are susceptible to CIV H3N2 infections and dogs may play a role in the transmission of CIV H3N2 to cats (Song et al., 2011; Su et al., 2014; Paniker & Nair., 1970). Su et al. (2014) experimentally demonstrated the susceptibility of cats to an equine strain of H3N8, however natural infection of cats with CIV H3N8 has not been reported. A seroprevalence survey conducted on samples collected from cats in diverse geographic locations in the U.S.A in 2009-2010 did not detect antibodies against CIV H3N8 (McCullers et al., 2011).

The cat-to-cat transmission of CIV H3N2 occurs when infected and susceptible cats are in close contact (Jeoung et al., 2013). However cat–to–dog transmission is not reported. The viral antibodies have been detected in asymptomatic cats suggesting subclinical infection (McCullers et al., 2011; Jeoung et al., 2013).

The last reported CIV H3N2 infection in cats involved thirteen animals in an animal shelter in New York, USA (University of Wisconsin - Madison, 2016). There have been no further reports since Song et al. (2011) on the occurrence of canine influenza infections in cats in South Korea.

The frequency of canine influenza infection outbreaks in cats is lower when compared to the disease frequency of dogs (Su et al., 2014; Harder & Vahlenkamp, 2010).

The kittens experimentally inoculated with influenza H3N2 showed no clinical signs but shed the virus for 1 week, developing antibodies and transmitting the virus to a susceptible kitten (Paniker & Nair, 1970).

Considering that:

- evidence exists for subclinical CIV H3N2 infection in cats (Su et al., 2014; Paniker & Nair, 1970).
- the same CIV H3N2 strain infects both dogs and cats (Zhu et al., 2015; Jeoung et al., 2013),
- New Zealand permits cat imports from countries where CIV H3N2 outbreaks have occurred in cats,
- detection of subclinically infected asymptomatic cats prior to export is unlikely,
- in cats, influenza A viruses have lower transmission efficiency (Su et al., 2014),
- cats shed less number of viral particles compared to dogs (Song et al., 2011; Su et al., 2014), and shedding lasts only for 7- 10 days (Hilling & Hanel., 2010),
- the feline behavior (less social contacts than dogs) (Su et al., 2014) and
- the less number of cats imported into New Zealand per year compared to the number of dogs (MPI, 2018),

  the likelihood of entry CIV H3N2 into New Zealand through imported cats is assessed to be low.

The likelihood of entry of CIV H3N8 into New Zealand through imported cats is assessed to be negligible.

6.2.2 Exposure assessment

Dogs and cats imported to New Zealand from all countries, except from Australia, must complete a 10-day quarantine in one of four commercial quarantine facilities (MPI, 2018).

The frequency of animal movements is high in quarantine facilities because the ‘all in–all out’ policy cannot be adopted. New arrivals and animals that have partially completed quarantine, are housed together. In this context, dog and cat quarantine facilities provide an ideal environment for the transmission of CIV H3N8 and CIV H3N2 to susceptible animals due to the shared air space and aggregation of dogs/cats from various origins.

Transmission of canine influenza infection from an infected dog to a susceptible dog has already occurred within a quarantine facility in New Zealand. A dog in the quarantine facility

acquired the virus from the infected dog but did not show clinical signs, except for a mild cough. It was given biosecurity clearance and later tested positive for CIV H3N2. The likelihood of this scenario repeating in the future cannot be excluded.

New Zealand domestic dogs and cats presents a naïve population in which immunity for canine influenza is non-existent. If an infected dog were to be released from quarantine, there is a moderate likelihood that it would spread the disease to other dogs in the same household or in the community, thus, establishing the infection in New Zealand.

Dog shows are popular events in New Zealand with multiple shows being held in every month in various locations in both North and South islands (Dogs New Zealand, 2019 – on line). The likelihood of participation of recently imported dog subclinically infected with canine influenza viruses in these events is assessed to be moderate. In this scenario, a large number of dogs would likely to be exposed to the virus thus facilitating the spread and establishment of the viruses.

Given the epidemiology and existing pathogen exposure pathways, the likelihood of CIV H3N8 and H3N2 transmission, spread and establishment in New Zealand domestic dog population is assessed to be moderate.

Compared to dogs, cats are less social animals and are normally confined to their own household. Therefore, any canine influenza outbreak in cats is likely to be confined to individual premises.

There are just under 40 affiliated cat clubs throughout in New Zealand and there is a cat-show season where people are able to show their cats and compete (New Zealand Cat Fancy, 2019). Despite the likelihood of a recently imported cat being participating in a cat-show, the shorter incubation period and the narrow virus shedding period in cats are likely to limit the exposure of susceptible cats to infected cats.

Given that:

- cats shed less virus within a short period (Song et al, 2008) and
- because of the shorter incubation period (Lei et al., 2012), a cat recently exposed to CIV H3N2 is likely to exhibit clinical signs, thus, allowing early diagnosis

The likelihood of spreading and establishment of canine influenza infection in New Zealand domestic cat population is assessed to be low.

6.2.3 Consequence assessment

Variable morbidity and mortality rates have been reported in several outbreaks of canine influenza infection. Crawford et al. (2005) reported a 36% case-fatality rate (8 out of 22 infected) during the first outbreak of CIV H3N8 infection in the U.S.A. All 3 clinically affected dogs involved in the third outbreak of CIV H3N2 in South Korea in 2007 died (Song et al., 2008).

In general the morbidity rate of 80% with a mortality rate less than 10% can be expected in dogs exposed to these viruses (AVMA, 2019; Hilling & Hanel, 2010). Given the highly infectious nature, if the viruses were to be established in New Zealand, respiratory disease outbreaks involving large numbers of dogs can be expected. Infected dogs will show low to severe respiratory disease symptoms with some fatalities occurring.

Being an exotic pathogen to New Zealand, canine influenza virus entering and establishing in New Zealand, may lead to the implementation of disease control and/or eradication programs. Movement restrictions of dogs and additional biosecurity measures may require to be imposed on premises where aggregation of dogs from various origins take place, such as boarding kennels, and veterinary hospitals.

Animal welfare, cancellation/postponement of popular events such as pet shows and competitions would be additional impacts in the case of canine influenza infection entering and establishing in New Zealand. If canine influenza outbreaks were to occur in New Zealand, participation in popular events such as pet shows and competitions would be restricted only to dogs that were vaccinated against CIV.

Countries importing dogs/cats from New Zealand may insist on evidence of freedom from canine influenza infection in imported animals, which may lead to additional regulatory requirements.

Canine influenza viruses have become adapted to canine and feline species but are not known to cause disease in other species or in humans. However, the potential for new strains emerging with cross-species infectivity exists (Jeoung et al., 2013; McCullers et al., 2011; Song et al., 2011; Voorhees et al., 2017; Zhu et al., 2015).

Zhu et al. (2015) forewarned that dogs have the potential to act as a 'mixing vessel' species in which avian, human and canine influenza viruses can reassort leading to the emergence of a new influenza virus strains with pandemic potential.

Several authors have noted the public health concern of the possible emergence of new recombinant feline or canine influenza viruses in companion animals with zoonotic potential (Song et al., 2011, Jeoung et al., 2013). Because of the tendency of influenza viruses for frequent re-assortments, the likelihood of a new virus strain evolving in dogs with zoonotic potential cannot be ignored.

The overall consequences of canine influenza infection spreading and establishing in New Zealand dog population has been assessed to be moderate.

The New Zealand cat population is composed of distinct segments; domestic cats, and feral cats. In 2011, the number of the domestic cats was recorded as 1.419 million and the feral cat population has been estimated to be over 2.5 million. However, physical contacts among these populations is thought to be minimal (The New Zealand Companion Animal Council New Zealand, 2016). Therefore, a canine influenza outbreak in domestic cats is unlikely to be spilled over to feral cats.

Varying degrees of morbidity and mortality in cats infected with CIV H3N2 has been reported. A morbidity rate of 100% and a mortality rate of 40% have been reported in cats in South Korea (Song et al., 2011). However, there has been no fatalities in cats naturally infected with canine influenza reported in the U.S.A. (AVMA, 2019). The underline causes for the variations in morbidity and mortality rates in different geographical locations yet to be investigated.

The consequences of canine influenza infection outbreak would be limited to low to moderate respiratory disease in infected cats with a fewer number of cats deaths compared to infected dogs.

Animal welfare, owner distress and financial costs associated with treatment and control of the disease are additional consequences of canine influenza infection occurrence in cats in New Zealand.

Accordingly, the overall consequences of canine influenza infection spreading and establishing in New Zealand cat population has been assessed to be low.

6.2.4 Risk estimation

CIV H3N8 and CIV H3N2 are assessed to be risks in dogs imported into New Zealand.

For dogs the likelihood of CIV H3N8 and CIV H3N2 entry is assessed to be 'High' with 'Moderate' exposure likelihood and 'Moderate' consequences.

The risk posed by CIV H3N8 and CIV H3N2 in dogs imported into New Zealand is estimated to be 'Moderate'.

CIV H3N2 is a risk in cats imported into New Zealand and the entry, exposure and consequence assessments of CIV H3N2 are assessed to be 'Low'.

The risk posed by CIV H3N2 in cats imported into New Zealand is estimated to be 'Low'.

6.3 RISK MANAGEMENT

The following information has been considered in evaluating the risk management options.

- Canine influenza is not an OIE listed disease.
- The world distribution of the disease vary with disease endemic in some countries (U.S.A. (CIV H3N8 and CIV H3N2), South Korea (CIV H3N2)) while some countries have reported occasional outbreaks of H3N2 (Singapore, Canada, Thailand).
- Both CIV H3N8 and CIV H3N2 can cause subclinical infections.
- Subclinically infected animals shed the virus in nasal and ocular secretions and are capable of transmitting the disease to susceptible animals in close contact.
- Antibodies for the virus first appear approximately 10 days after infection.
- The virus can be detected in respiratory secretions in infected animals.
- Monovalent and bi-valent canine vaccines are available in some countries (AVMA, 2017; Rodriguez et al., 2017).
- Monovalent vaccines are not cross-protective (Rodriguez et al., 2017).

The following options are evaluated:

1. Country freedom from canine influenza infection in dogs and cats
2. Certified non-exposure
3. Vaccination (dogs only)
4. Serological testing for antibodies
5. Virus isolation
6. PCR testing for viral antigens
7. Monitoring for new and emerging strains of influenza virus in dogs and cats

6.3.1 Country freedom

Provided that effective surveillance programs for the detection of canine influenza virus infections are in place in the country, this option is appropriate for dogs and cats imported from countries with no history on canine influenza infection.

This option is likely to be highly effective in managing the risk of canine influenza viruses entering New Zealand through the importation of dogs and cats from these countries.

6.3.2 Certified non-exposure

The current cat and dog IHS contains the following conditions:

"For at least 21 days prior to shipment:

- The cat or dog was not kept in a place where there were cats or dogs showing clinical signs of infectious respiratory disease; and
- The cat or dog showed no clinical signs of infectious respiratory disease".

These measures provide a level of assurance that the cat or dog has not been exposed to infected animals, however, the measures do not exclude recent exposures, therefore, subclinically infected animals may enter the export chain. Accordingly 'certified non-exposure' has been assessed as a risk mitigation option with low effect.

6.3.3 Vaccination

Inactivated monovalent vaccine products against CIV H3N8 and CIV H3N2 are commercially available in the U.S.A. (Shields et al., 2010; AVMA, 2017; Rodriguez et al., 2017). However, each vaccine does not confer immunity against the other strain (Rodriguez et al., 2017).

A bivalent, inactivated vaccine product against both CIV H3N8 and CIV H3N2 also have been approved for the use in dogs in the U.S.A and it is commercially available (MERCK, 2019). Primary vaccination, which comprised two doses of the vaccine given 2–4 weeks apart, can be administered to dogs over 7 weeks of age by intramuscular injection. Annual revaccination with one dose is recommended.

Live attenuated monovalent and bivalent vaccines against CIV H3N8 and CIV H3N2 have also been produced in the U.S.A for use in dogs (Rodriguez et al., 2017; Nogales et al., 2017). Producers of these vaccines claim superiority of live vaccines compared with inactivated virus vaccines; they produce both humoral and cellular immunity, intra-nasal administration mimic the natural infection, thus, enabling local immunity at the infection site, and only a single dose of the vaccine need to be administered (Rodriguez et al., 2017; Nogales et al., 2017). However, information is not available on the potential for reversal to virulent state of the attenuated viruses in the vaccine products and their commercial availability.

Vaccine product for the use in cats are not available.

It is important to note that canine influenza infection cannot be ruled out in CIV H3N8 vaccinated dogs with acute respiratory disease (Anderson & Crawford, 2011).

Canine vaccine products are available in a limited number of countries and this option is not applicable to all countries exporting dogs to New Zealand. Although vaccination may not completely prevent infection but it reduces the severity of the disease and virus shedding, therefore the effectiveness of this option is likely to be 'High'.

6.3.4 Serological testing for antibodies

Antibodies against canine influenza viruses can be detected 7–10 days post-infection (Anderson et al, 2012; Deshpande et al, 2009). The presence of antibodies indicates exposure, but not necessarily an active infection. For this reason, paired serum samples, collected during the initial stage when the animal exhibits clinical signs of the disease and again during the convalescent period 2-3 weeks after, are used. A four-fold increase in the antibody titre in the second sample is indicative of an infection.

The HI assay has been shown to have a 99.6% and 94.6% sensitivity and specificity respectively for CIV H3N8 antibodies when the cut-off point is set to 32 (Anderson et al., 2012). Provided the dog has not been vaccinated for H3N8 prior to the sample collection, HI test provides a definitive diagnosis.

Because of the time taken to develop antibodies against the virus, serological testing may not be suitable for screening dogs for import purposes, as the test may not detect the dogs exposed to the viruses immediately prior to export. Serological testing does not detect subclinically infected animals.

Accordingly, serological testing has low effect as a risk mitigation option.

6.3.5 Virus isolation

Canine influenza viruses can be isolated from clinical samples. However, the timing of the sample collection is crucial for the isolation of the sufficient number of virus particles. Samples must be collected from dogs that are showing clinical signs (Anderson & Crawford, 2011).

Although virus isolation allows for identification of other influenza viruses that cause respiratory disease in dogs, precise timing requirements for sample collection and longer turnaround time make this method unsuitable for screening dogs for canine influenza viruses in dogs that are being prepared for export to New Zealand.

Although virus isolation is an effective diagnostic tool, as a risk mitigation option for imported dogs and cats it has low effect.

6.3.6 Polymerase chain reaction (PCR) Testing

The PCR test, using specific primers for the influenza matrix gene or the CIV hemagglutinin (H3) gene, detects viral components in the sample. The virus does not have to be infectious and the test is able to differentiate virus stains containing hemagglutinin H3 from virus strains with hemagglutinin H1 (Anderson & Crawford, 2011; Lu et al., 2010). According to Anderson and Crawford (2011) vaccination with H3N8 vaccine products does not interfere with PCR testing.

Lee et al. (2016) reported the development of a ‘Multiplex real-time PCR test for detecting CIV H3N2 viral segments in dogs.

PCR testing could be used for effective screening dogs that are being prepared for export to New Zealand because:

1. Dogs infected with canine influenza viruses shed the virus before clinical signs appear (Hilling & Hanel, 2010),
2. The PCR test using H3 gene primers can detect either CIV H3N8 or CIV H3N2 (Anderson & Crawford, 2011; Lu et al., 2010), and

3. The relatively short turnover time for PCR test reported in some countries may allow for testing within the five days immediately prior to the date of export.

No PCR test has been reported for testing canine influenza virus in cats however, it may be possible to apply the same PCR test used in dogs, after validation for feline, to detect exposed or infected cats.

Following the arrival of a CIV H3N2 infected dog into the country from Singapore, Australia implemented PCR testing of all dogs exported from Singapore to Australia. Under this condition, all dogs exported to Australia from Singapore must be tested negative. This condition has been removed since 15 January 2019 requiring all dogs must be fully vaccinated with attenuated vaccine product against CIV H3N2 between 365 days and 14 days before export (DAWR, 2018).

Because viral shedding commences before the clinical signs appear, PCR testing offers an effective tool for detecting incubating and subclinically infected animals. The effectiveness of this option is likely to be 'High'.

6.3.7 Monitoring emerging influenza virus A strains affecting dogs and cats

There are reports of A subtypes such as H5N2, H5N1, transferring into new hosts (canine, feline) from their natural hosts but these infections do not tend to spread among the new host species (Crawford et al 2005). It would be prudent monitor the evolution of these strains as they may gain inter and intra-species infectivity.

6.3.8 Options

Option 1

The export certification to the effect that "No case of canine influenza caused by CIV H3N8 and/or CIV H3N2 in cats or dogs has been diagnosed in the country in the three months before the date of export".

This option is particularly appropriate for dogs and cats originating in countries with no history of canine influenza infection. It is likely to be highly effective in managing the risk of canine influenza viruses entering New Zealand through the importation of dogs and cats from countries, which have not reported CIV cases.

Option 2

The dog was fully vaccinated against CIV H3N8 and CIV H3N2 with MPI approved vaccine product/s in the exporting country as per the vaccine manufacturer's instructions between 365 days (12 months) and 14 days before the date of export.

Canine vaccine products are available in some countries and this option is not applicable to all countries exporting dogs to New Zealand. Although vaccination may not completely prevent infection but it reduces the severity of the disease and virus shedding, therefore the effectiveness of this option is likely to be 'High'.

Option 3

The cat or dog was tested with a negative result for CIV H3N8 and CIV H3N2 viral antigens with MPI approved PCR test, conducted on a sample collected within the 5 days prior to export.

Because viral shedding commences before the clinical signs appear, incubating and subclinically infected animals can be detected. The effectiveness of this option is likely to be 'High'.

Option 4

For at least 21 days prior to shipment:

- The cat or dog was not kept in a place where there were cats or dogs showing clinical signs of respiratory disease; and
- The cat or dog showed no clinical signs of respiratory disease.

This option is currently in place for all countries. Because it does not prevent export of subclinically infected, animals or recently-exposed animals, effectiveness of the option is likely to be 'low'.

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