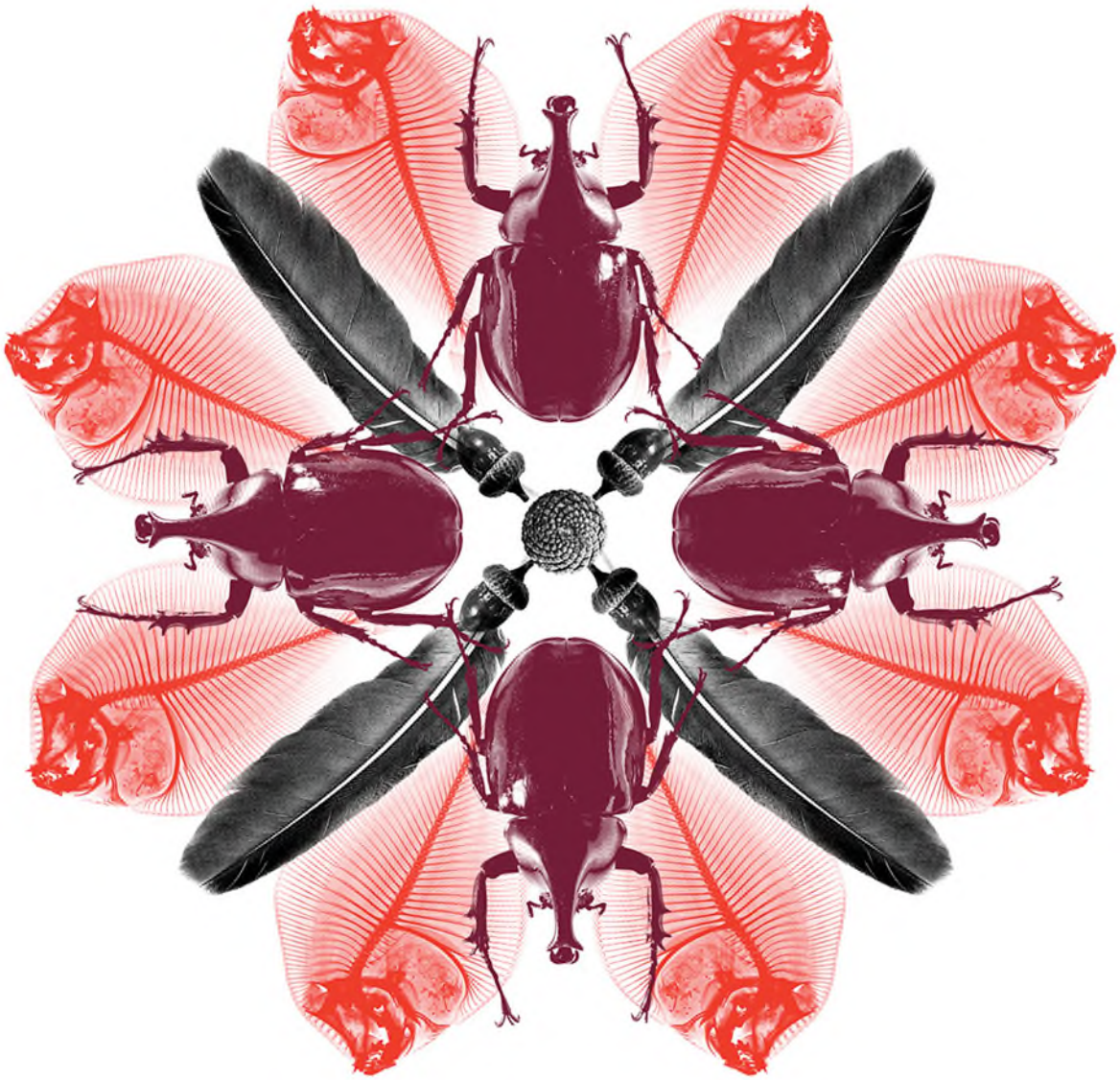
Australian Government
Department of Agriculture

Review of risks associated with canine influenza virus

August 2019



Cataloguing data

This publication (and any material sourced from it) should be attributed as: Department of Agriculture 2019, *Review of risks associated with canine influenza virus*, Canberra, August. CC BY 4.0.

This publication is available at agriculture.gov.au/biosecurity/risk-analysis/animal.

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Summary

The Department of Agriculture has reviewed whether the level of biosecurity risk in relation to canine influenza has changed since the completion of the *Importation of dogs and cats and their semen from approved countries: final policy review* (*Dog and cat review 2013*), released in November 2013. The *Dog and cat review 2013* considered the biosecurity risks for Australia associated with the importation of companion animals and their reproductive material from approved countries.

In early 2018, Australia identified a case of canine influenza virus (CIV) in an imported dog in the Melbourne post-entry quarantine facility (PEQ). Due to the application of strict biosecurity measures the incident was isolated and resolved in the PEQ facility. This incident was similar to a case that occurred in a New Zealand quarantine facility approximately1 month earlier in 2018.

The department then commenced a review of the biosecurity risk associated with CIV in dogs to determine if the current biosecurity measures in place continue to meet Australia's appropriate level of protection (ALOP) in light of the changing global distribution of the disease and identification of the disease in an imported dog in the Australian PEQ facility. The policy review of CIV was completed after considering new and relevant previewed scientific information, advice from international scientific experts and operational practicalities.

The policy review recommends biosecurity measures for the importation of dogs and cats and their semen from approved countries be enhanced for CIV. This policy review replaces the former CIV chapter in the *Dog and cat review 2013.*

Canine influenza

In 2013 canine influenza virus was identified by Australia as a disease of biosecurity concern for dogs through a process of risk review. That review was published as *Importation of dogs and cats and their semen from approved countries: final policy review*, November 2013 (Department of Agriculture 2013). Since the publication of the risk review the distribution of CIV has increased and more is known about the subtypes involved. Consequently a review of the policy for this emerging disease is necessary.

Background

CIV is a highly contagious viral pathogen. It causes respiratory illness (canine influenza) of varying severity in the majority of infected dogs. CIV can form part of the canine infectious respiratory disease complex (CIRD) in conjunction with other infectious agents such as canine parainfluenza virus, canine adenovirus type-2 and canine herpesvirus 1. Canine influenza is not an OIE-listed disease and is not nationally notifiable in Australia (Department of Agriculture 2017; OIE 2018). There is no evidence to suggest that CIV can infect humans (Abente et al. 2016; Li et al. 2018; Voorhees et al. 2017).

CIV is an influenza A virus of the family Orthomyxoviridae (genus *Influenzavirus A*) (Hilling & Hanel 2010; Sun et al. 2017). Influenza A viruses can spill over into new host species. The emergence of CIV in the late 1990s has been attributed to a host jump by influenza A from horses and birds directly to dogs (Rodriguez et al. 2017; Voorhees et al. 2017). Two subtypes of CIV have been identified in dogs – H3N8 and H3N2 (Abente et al. 2016; Jirjis et al. 2010; Kim et al. 2013; Lee et al. 2009; Voorhees et al. 2017). A number of strains have been isolated from each subtype. Cats have been shown to become infected with H3N2 but not H3N8 (Dalziel et al. 2014; Kim et al. 2013). Infection of cats was believed to be due to close proximity to infected dogs in a shelter environment (Dalziel et al. 2014) and is regarded as a 'spill over' event.

H3N8 is endemic in the United States, whereas H3N2 is endemic in the Republic of Korea, China, Thailand and the United States (Abente et al. 2016; Chanvatik et al. 2016; Lee et al. 2009; Li et al. 2010; Voorhees et al. 2017). Outbreaks of H3N2 have also been detected in Canada and Singapore (Low 2018; Weese 2018b).

Other influenza A subtypes have been isolated from dogs, such as pandemic H1N1/2009, avian H5N1, H1N1, H1N2 and human H3N2 (Chen et al. 2018; Lin et al. 2012; Songserm et al. 2006). Seroprevalence studies suggest that such infections are uncommon. A serological survey of pet dogs in several Chinese provinces found only 1.5% dogs were seropositive for pandemic H1N1/2009 (Sun et al. 2014), whereas a similar study in Italy found 0.7% of dogs showed evidence of exposure to H1N1/2009 (Dundon et al. 2010). Transmissibility of pandemic H1N1/2009 was shown to be poor between dogs (Lin et al. 2012), and it is likely these infections were passed on from their owners or caused by ingestion of infected poultry meat (Lin et al. 2012; Songserm et al. 2006).

There is no evidence that CIV has been involved in outbreaks of CIRD within Australia. However, CIV was detected and contained without spread in imported dogs at post entry quarantine (PEQ) facilities in New Zealand in March 2018 (Bingham 2018) and in Australia in May 2018.

Technical information

Agent properties

Influenza A viruses, such as CIV, have a lipid envelope, are 80–120 nm in diameter and contain a genome of 8 segments of single-stranded, negative-sense RNA (Hilling & Hanel 2010).

Influenza viruses can persist and remain infective on skin, fabrics and contaminated equipment, making fomites important in transmission (Bean et al. 1982). At 35–40% relative humidity and a temperature of 28°C, influenza A viruses persist on hard, nonporous surfaces such as stainless steel and plastic for 24–48 hours, but for less than 8–12 hours on porous surfaces such as cloth and paper (Bean et al. 1982). Influenza A virus was transferred from stainless steel surfaces to hands for up to 24 hours and from paper tissues to hands for up to 15 minutes (Bean et al. 1982). The virus persists on hands for up to 5 minutes after transfer from environmental surfaces (Bean et al. 1982). Sakaguchi and colleagues (2010) found that influenza A viruses can survive on personal protective equipment such as gloves, gowns and surgical masks for 24 hours. There is no known difference in environmental persistence between the 2 subtypes of CIV.

Influenza A viruses are inactivated by a range of disinfectants and chemicals, including alcohol (e.g. ethanol, isopropanol), chloroxylenol, bleach, phenols (toxic to cats), quaternary ammonium compounds, oxidising agents (for example Virkon®) and detergents (AVMA 2009). Bleach and quaternary ammonium compounds are commonly used as disinfectant agents in shelters (CFSPH 2009). Cleaning prior to disinfection is essential as the effectiveness of these agents is reduced in the presence of organic matter (CFSPH 2009).

The surfactant action of soaps and detergents is also an effective decontaminant for CIV as it destroys the outer lipid envelope of the virus (Grayson et al. 2009). Soap and water or alcohol-based hand gels are suitable for personal disinfection against influenza viruses (Grayson et al. 2009).

Epidemiology

Host Range

H3N8

H3N8 infections have been reported in dogs and pigs (Parrish, Murcia & Holmes 2014). H3N8 circulating in dogs has not been reported to be transmitted back to horses due to selective adaption of the virus to dogs (Parrish, Murcia & Holmes 2014). There is no evidence that dogs or pigs can transmit H3N8 to other species of animals (Dalziel et al. 2014; Pulit-Penaloza et al. 2017). Cats and horses have only been infected experimentally with H3N8 (Parrish, Murcia & Holmes 2014).

H3N2

H3N2 infections have been reported in dogs and cats (Kim et al. 2013). During an outbreak of H3N2 at a dog shelter, cats housed in a separate building at the shelter site also became infected with H3N2 (Jeoung et al. 2013; Lei et al. 2012; Song et al. 2011). Fomites may have been the cause of cats developing the infection and transmission was not maintained amongst the cats (Jeoung et al. 2013; Lei et al. 2012; Song et al. 2011). Mice, ferrets and guinea pigs have been infected experimentally with H3N2, however transmission of H3N2 from experimentally infected animals to other animals was not deemed efficient (Kim et al. 2013).

Distribution

H3N8

Sequencing identified that the CIV caused by H3N8, which was isolated in the United States, originated from a host jump of equine influenza virus H3N8 to dogs resulting in a dog-specific influenza A virus that caused acute respiratory disease (Crawford et al. 2006).

This was not the first time equine influenza has been demonstrated to transfer from horses to dogs. A retrospective study conducted in the United Kingdom identified equine influenza virus H3N8 as the cause of an outbreak of severe respiratory disease in a single pack of 92 English foxhounds in 2002 (Daly et al. 2008). The dogs were all located on a single property and were housed in close proximity to horses with equine influenza (Daly et al. 2008). In addition, the dogs had been fed horse meat sourced externally with an unknown health status (Daly et al. 2008). This was an isolated case that did not involve further transmission to other animals (Daly et al. 2008).

During the equine influenza outbreak in Australia in 2007, 23 dogs in close proximity to infected horses were found to have seroconverted (Crispe et al. 2011; Kirkland et al. 2010). Ten of the 23 dogs also developed clinical signs consistent with an influenza infection (Crispe et al. 2011). There was no evidence of the equine influenza virus being transmitted from dogs to other animals (including other dogs) and becoming established in Australia (Crispe et al. 2011; Kirkland et al. 2010). Similarly a study conducted by Zhou and colleagues (2016) identified antibodies for equine influenza using haemagglutination inhibition in 5 out of 600 (0.83%) sera samples collected from pet dogs in China.

Outbreaks of H3N8 in the United States were different to incidents in other countries, in that virus was able to successfully adapt and propagate in dogs. Clinical respiratory disease caused by H3N8 was first identified in 22 racing greyhounds in Florida, United States in 2004 (Hilling & Hanel 2010; Payungporn et al. 2008). The greyhounds developed the disease after sharing racing facilities in Florida that had been used by horses infected with equine influenza (Crawford et al. 2006; Rivailler et al. 2010). In 2004, the disease spread to 14 greyhound racetracks across 6 US states, leading to the infection of approximately 10,000 racing greyhounds (Hilling & Hanel 2010; Payungporn et al. 2008). In 2005, outbreaks of H3N8 occurred in 11 US states and infected approximately 20,000 racing greyhounds before spreading to pet dogs (Hilling & Hanel 2010). A retrospective study of 520 sera collected from racing greyhounds between 1999 and 2004 found that 26% of dogs had antibodies for CIV and EIV H3 proteins, suggesting that H3N8 may have been circulating in the United States greyhound population in Florida since 1999 (Anderson et al. 2012).

From 2004 to date, H3N8 has been identified in dogs in 40 US states (Rivailler et al. 2010). It was the dominant serotype circulating in the United States at the time of the 2013 policy review but has since been supplanted by H3N2. Since 2017, H3N8 appears to be endemic and geographically restricted to the north-eastern United States including New York, Vermont, New Hampshire and Pennsylvania (Dalziel et al. 2014; Hayward et al. 2010; Rivailler et al. 2010; Voorhees et al. 2017). While H3N8 is still present in the United States, the incidence of disease it causes appears low by comparison with H3N2 (Voorhees et al. 2017; Weese 2018c).

H3N2

H3N2 is the primary subtype of CIV circulating globally at this time (Pulit-Penaloza et al. 2017).

H3N2 originated in 2005 from an avian-origin influenza A virus (Voorhees et al. 2017). It has been hypothesised that the jump from birds to dogs was facilitated via the feeding of H3N2-infected chicken and duck meat to dogs farmed for the meat trade in Asia (Song et al. 2008; Voorhees et al. 2017). Transmission may have also occurred when live birds infected with H3N2 and dogs for consumption were placed in close proximity at markets (Song et al. 2008).

CIV caused by H3N2 was first reported in 4 pet dogs showing respiratory signs in China's Guangdong province from 2006 to 2007 (Li et al. 2010). In 2012, the rate of H3N2 seropositivity in farmed and pet dogs in Guangdong province was 12.2% and 5.3% respectively (Su et al. 2013).

H3N2 was also found to be circulating in dogs in the Republic of Korea in 2007 (Lee et al. 2009). A subsequent retrospective study of sera samples collected between 2004–2009 suggested that H3N2 may have been present in the Republic of Korea as early as 2005 (Lee et al. 2012; Voorhees et al. 2017). Lee and colleagues (2009) conducted a serological survey of dogs and found H3N2 antibodies in 19% of dogs produced for meat production as opposed to 0.5% (n=419) in pet dogs in the Republic of Korea.

H3N2 has also been reported to be circulating in Thailand. The seroprevalence of H3N2 was examined in dogs in a veterinary hospital in Bangkok, Thailand in 2012 (Chanvatik et al. 2016). Chanvatik and colleagues (2013) identified that 12.2% (n=164) of dogs tested had been exposed to H3N2.

International movement of dogs has been identified as an important factor in the spread of H3N2 from Asia to the United States. In early 2015, H3N2 was isolated from dogs with respiratory disease in Chicago (Voorhees et al. 2017). The disease was linked to the import of dogs rescued from meat markets in the Republic of Korea (Voorhees et al. 2017). H3N2 spread rapidly throughout the United States, especially in areas of high dog density such as kennels, shelters and veterinary hospitals (Voorhees et al. 2017). To date, H3N2 has spread to more than 40 states in the United States, including states where H3N8 is endemic (AVMA 2018).

In January 2018, H3N2 was detected in clinically affected dogs in Canada (Weese 2018c). The outbreak was confined to Ontario, Canada (Weese 2018c). The outbreak was associated with the rescue of dogs from China that had been imported through the United States to Canada (Weese 2018b). The disease also spread to shelter and pet dogs (Weese 2018c). This outbreak was contained by mid-April 2018, however a new outbreak was detected in Ontario in late October 2018 (Weese 2018b, d).

In March 2018, a clinically affected dog was detected in a New Zealand post entry quarantine facility (Bingham 2018). This case was managed within the facility and no further transmission occurred (Bingham 2018).

In May 2018, the first case of CIV due to H3N2 was detected in shelter dogs in the Pasir Ris Farmway area in Singapore, (Low 2018). To date, the outbreak is known to involve 4 shelters in the area (Low 2018). Singapore's Agri-Food and Veterinary Authority advised that H3N2 had not been detected from dogs with respiratory signs outside of these facilities, although private veterinarians have reported a number of respiratory cases where CIV was a differential diagnosis (Low 2018). The source of the outbreak has not been determined.

H3N2 was detected in May 2018 in a dog imported from Singapore that had entered the Australian post entry quarantine facility before showing clinical signs of respiratory disease. This case was managed and contained without further spread in the facility.

Transmission

Transmission of CIV can occur via direct or indirect contact (Crawford 2009). Direct transmission can occur via oronasal contact with infected dogs, or inhalation of aerosolised droplets released by coughing and sneezing dogs (Crawford 2009; Hilling & Hanel 2010; Jirjis et al. 2010). Indirect transmission can occur via fomites (Hilling & Hanel 2010). It is not known whether either subtype can be transmitted to dogs or other species via meat. As with other influenza A viruses, there no reports that either CIV subtype can be transmitted through semen. Fever associated with influenza infections has been found to reduce semen quality (Lugar, Ragland & Stewart 2017).

As with other influenza A viruses, CIV has tropism for the respiratory tract (Reperant et al. 2012). More severe infections are often associated with infections of the lower respiratory tract, whilst highly transmissible influenza infections are often associated with infection targeting the upper respiratory tract (Reperant et al. 2012). CIV-infected dogs routinely shed live virus from their nasal, oral and/or oropharyngeal cavities (Reperant et al. 2012).

The persistence of shedding varies between CIV subtypes. Dogs infected with H3N8 typically shed virus for approximately1 week, whilst dogs infected with H3N2 can shed virus for up to 4 weeks (AVMA 2018). Peak shedding for both subtypes often occurs during the incubation period (AVMA 2018). Approximately 20% of infected dogs will remain asymptomatic but still spread the virus (Crawford et al. 2006; Hilling & Hanel 2010).

Dogs housed in communal facilities such as kennels, shelters, pet stores, veterinary clinics or attending dog shows are at highest risk of exposure to circulating CIV via contact with an infected dog or fomites (Pecoraro et al. 2014; Song et al. 2008). In these types of facilities there may be a significant proportion of unvaccinated and/or immune-suppressed dogs, including due to disease, stress and/or malnutrition on arrival. Such animals are at increased risk of acting as virus multipliers within that population.

Fomites include the clothing, footwear and hands of human handlers, food and water bowls, leads, bedding and surfaces (Hilling & Hanel 2010). The persistence of CIV on fomites depends on factors such as virus concentration in infective material, surface type, temperature and humidity (Killingley & Nguyen-Van-Tam 2013). CIV is more likely to persist for longer on a hard, non-porous surface than a soft, porous one (Killingley & Nguyen-Van-Tam 2013).

Pathogenesis

Influenza A viruses, such as CIV, replicate in the respiratory tract such as the mucosal epithelium cells lining the airways from the nose to the terminal airways, in bronchiolalveolar epithelium. Transfer of the CIV subtypes to dogs from birds and horses is thought to have been facilitated by the presence of haemagglutinin avian influenza-binding receptors on the bronchial and bronchioalveolar epithelial cells (Song et al. 2008), and equine-influenza binding receptors on tracheal and bronchial epithelial cells in dogs (Daly et al. 2008).

CIV replication leads to epithelial cell necrosis and destruction of the respiratory epithelial barrier, predisposing the dog to secondary infection by a variety of commensal bacteria. Secondary infections with *Bordetella bronchiseptica, E. coli*, *Klebsiella* spp., *Mycoplasma* spp.,

Pasteurella multocida, *Staphylococcus* spp. and *Streptococcus* spp. are common in CIV infected dogs and can be serious (Hilling & Hanel 2010; Larson et al. 2011; Watson, Bell & Toohey-Kurth 2016). In the lower respiratory tract, alveolar septa can become thickened by infiltration of inflammatory cells (Jung et al. 2010). Most clinically affected dogs recover without complications. However in less than 20% infection may lead to bronchopneumonia associated with virus-induced cell damage in the lower airway epithelium and complicated by secondary bacterial infection (Crawford 2009).

Influenza A viruses are able to suppress host antiviral and immune response by suppressing general protein synthesis by the host. The process responsible for this host protein 'shutoff' are unclear. However, a recent *in vitro* study suggested a differential effect on general host protein synthesis by the CIV subtypes (Nogales et al. 2017). H3N2 suppressed both host protein synthesis and interferon gene expression response *in vitro*, whereas H3N8 only had an effect on interferon gene expression (Nogales et al. 2017).

Diagnosis

The clinical signs of CIV are not specific enough for diagnosis and overlap with many of the agents associated with CIRD. Suitable diagnostic tests available for CIV include reverse transcription polymerase chain reaction (RT-PCR) and serological testing, such as haemaglutination inhibition (HI) (Anderson et al. 2012).

RT-PCR is a reliable and sensitive (95% sensitivity) test for detecting both H3N2 and H3N8 (Pecoraro et al. 2014). Successful diagnosis depends on sample collection during peak virus shedding early in the course of clinical disease (Anderson et al. 2012). Nasal swabs are the sample of choice for live dogs as it is difficult to obtain a good quality nasopharyngeal or oropharyngeal swab (Hanson, Tripp & Harvey 2016).

Serological tests such as HI, which has a sensitivity of 99.6% and a specificity of 94.6%, can also be used to confirm CIV infection, especially in cases where PCR results are negative but the index of suspicion is high (Anderson et al. 2012). A serological response usually develops 7–10 days post infection and will continue to increase until 14 days (CFSPH 2015; Jirjis et al. 2010). Paired acute (sick for < 7 days) and convalescent (10–14 days later) serum samples are ideal for diagnosis of recent active infection (Anderson et al. 2012). Seroconversion is defined as at least a four-fold increase in antibody titres between acute and convalescent sera (Crawford et al. 2006). No cross reactions were reported when using a HI assay developed for CIV H3N2 in dogs experimentally infected with human influenzas (seasonal H3N2 and pandemic H1N1) (Shim et al. 2015).

Clinical signs

The incubation period for both CIV subtypes is 2 to 5 days (Hilling & Hanel 2010). Dogs of any age, breed and health status are susceptible. Immunocompromised dogs, puppies and elderly dogs are at higher risk of severe infection (Merck 2016).

Clinical disease is usually mild, consisting of an acute onset of coughing, sneezing, nasal discharge, some ocular discharge, lethargy and anorexia (Lee et al. 2018; Li et al. 2018; Li et al. 2010; Payungporn et al. 2008; Voorhees et al. 2017; Watson, Bell & Toohey-Kurth 2016). Non-productive and dry coughing is the predominant sign and typically persists for up to 3 weeks (Payungporn et al. 2008). A low grade fever may also precede the onset of coughing (Hilling & Hanel 2010; Song et al. 2011).

The disease may progress to a lower respiratory tract infection and even occasionally death due to complications from pneumonia (Watson, Bell & Toohey-Kurth 2016). Severely infected dogs with pneumonia show typical signs such as high fever, inappetence, productive cough, increased respiratory rate and effort (Crawford et al. 2006; Watson, Bell & Toohey-Kurth 2016). Haemorrhagic pneumonia and subsequent death has been reported in H3N8 infections (Crawford et al. 2006). More severe lower respiratory tract disease is also associated with secondary bacterial infections (Payungporn et al. 2008; Watson, Bell & Toohey-Kurth 2016). Cats have a lower morbidity rate than dogs and commonly present with mild respiratory signs when infected with CIV (Jeoung et al. 2013).

The mortality rate associated with H3N8 is less than 10% in most cases (Hilling & Hanel 2010). However some H3N2 strains isolated from China and the Republic of Korea have been associated with mortality rates reported as high as 22–55% in some shelters (Jeoung et al. 2013; Lee et al. 2018; Song et al. 2011).

Treatment

Treatment consists of supportive care based on clinical signs and laboratory tests. There is no specific antiviral treatment for CIV. Antitussives do not reduce the frequency and duration of coughing (Crawford 2009). Antibiotic therapy is not recommended for dogs with mild respiratory signs associated with CIV as the disease is self-limiting and relatively short lived (Hilling & Hanel 2010). However in the case of secondary bacterial infections, antibiotics are indicated for immunosuppressed dogs or those with fever, purulent nasal discharge, productive cough and pneumonia (Crawford 2009).

Vaccination

Vaccines are available for both subtypes of CIV. Vaccination against CIV decreases both the likelihood of dogs becoming infected with CIV and the duration and intensity of viral shedding in infected animals (Cole & McNally 2009; Hilling & Hanel 2010). Furthermore, vaccination reduces the severity of clinical signs (Larson et al. 2011).

Monovalent inactivated antigen vaccines with an aluminium based adjuvant are available for both H3N8 and H3N2 (Cobey et al. 2018; Merck 2016). These vaccines do not provide cross-protection against the non-target subtype. Both monovalent vaccines are registered for use in the United States, Canada and the Republic of Korea (Merck 2016).

A bivalent inactivated vaccine that covers H3N8 and H3N2 is also available for use in the United States, Canada, Singapore and the Republic of Korea (Merck 2016). An intra-nasal bivalent live-attenuated vaccine is in development (Rodriguez et al. 2017). Mice given this intra-nasal live vaccine produced a stronger humoral immune response than to the inactivated vaccines available at the time this work was performed (Rodriguez et al. 2017).

Two vaccinations given 2 to 4 weeks apart followed by a single annual booster is the standard vaccination protocol for monovalent vaccines developed against H3N2 and H3N8 (Zoetis 2018). The bivalent vaccine requires 2 vaccinations to be administered 3 weeks apart (Zoetis 2018). The need for an annual booster with the bivalent vaccine is variable with manufacturers differing in their advice to clients (Zoetis 2018).

There is no vaccine available that is suitable for use in cats (Merck 2016).

Control

Dog-to-dog transmission of CIV occurs via co-mingling of infected dogs with naïve dogs (Jirjis et al. 2010). Dogs kept in high density or transient populations such as kennels, dog day care, shelters, racing environments and dog farms have a greater risk of direct (via contact and aerosols) and indirect (via fomites) exposure than pet dogs kept at home (Buonavoglia & Martella 2007; Larson et al. 2011; Pecoraro et al. 2014). Higher seroprevalence has been found in dogs farmed for meat in China and the Republic of Korea than in pet dogs (Lee et al. 2009; Su et al. 2013). The seroprevalence of CIV is also high in dog shelters in the United States with a study by Holt and colleagues (2010) identifying 42% of dogs in a Philadelphia shelter as seropositive for CIV.

Stringent biosecurity procedures for quarantine and isolation of infected dogs or dogs showing clinical signs of respiratory illness are necessary to prevent further spread in high density or transient populations. Given that shedding of H3N2 can continue for up to 4 weeks, the AVMA recommends isolation of infected dogs and in contact dogs for up to 4 weeks (AVMA 2018).

Human handling of infected dogs and then non-infected dogs in the absence of proper decontamination has contributed to the transmission of CIV in shelter environments (Miller & Hurley 2009). United States shelter staff have accidentally infected their own pet dogs through improper decontamination (Miller & Hurley 2009). Consequently, cleaning and disinfection of equipment and decontamination of personnel are important in reducing and or controlling the spread of CIV (Hilling & Hanel 2010). CIV is killed by readily available disinfectants such as alcohol, quaternary ammonium compounds and diluted bleach (1:30 dilution) solutions (Hilling & Hanel 2010). However the effectiveness of these agents is reduced in the presence of organic matter, so cleaning prior to decontamination is critical.

Vaccination is also useful in controlling ongoing transmission of CIV, especially in smaller shelters with lower dog throughput, as this environment has less dogs entering that are naïve to the infections circulating within the shelter environment (Dalziel et al. 2014). Vaccination reduces the duration of shedding in infected dogs, which reduces the rate of transmission of the disease (Dalziel et al. 2014; Hilling & Hanel 2010).

Current biosecurity measures

The following biosecurity measures were put in place to manage the biosecurity risk of CIV in imported dogs following the 2013 policy review.

For dogs imported from the United States only:

- Within 12 months and at least 14 days immediately before export, the dog must have a current vaccination status against canine influenza, in accordance with the vaccine manufacturer's recommendations.

For dogs from all countries:

- Within 5 days immediately before export, the dog must be subjected to a thorough physical examination by a registered veterinarian and found to be free from clinical signs of canine influenza.

Risk assessment

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by CIV:

- CIV is not an OIE listed disease.
- CIV is present in the United States, the Republic of Korea, Thailand, China, Singapore and Canada.
  - H3N8 is endemic to the United States only.
  - H3N2 is now endemic in the Republic of Korea, China, Thailand and the United States. H3N2 has emerged in Singapore.
  - China and Thailand are not approved countries to export companion animals to Australia.
- Outbreaks of H3N2 were detected in Canada in 2018 (Weese 2018a, d). The last outbreak was detected in late October 2018 and remains unresolved (Weese 2018d).
- Peak shedding of virus occurs in the preclinical incubation phase of infection (AVMA 2018). Shedding can last for1 week in H3N8 infections and up to 4 weeks in H3N2 infections (AVMA 2018).
- Sub-clinically infected dogs can also shed virus (AVMA 2009). A prolonged carrier and shedding status has not been reported following infection.
- Vaccination against CIV decreases both the likelihood of dogs becoming infected with CIV and reduces viral shedding in infected animals (Merck 2016).
- The United States, Canada, Singapore and the Republic of Korea have vaccines for CIV (Merck 2016).
- Dogs in high density communal facilities such as shelters and kennels have the highest risk of contracting CIV due to the increased likelihood of direct contact with an infected dog and exposure to aerosolised virus particles (Buonavoglia & Martella 2007; Larson et al. 2011; Pecoraro et al. 2014).
- Fomite transmission is also very important in the transmission of CIV, as the virus can survive for up to 48 hours on a surface given the correct conditions (Bean et al. 1982). Humans acting as fomites is a risk in the spread of CIV (Miller & Hurley 2009).
- Outbreaks of H3N2 in the United States and Canada have been linked to the importation of dogs rescued from shelters or dog meat farms in Asia (Weese 2018c).
- CIV is not notifiable in Australia.
- CIV was detected in Australia in an imported dog in post-entry quarantine in May 2018. Further spread of CIV from this case did not occur.
- A study conducted by Animal Medicines Australia in 2016 identified that 38% of households in Australia own at least1 dog and that there is an estimated population of 4.8 million dogs nationally.
- There is no CIV vaccine currently approved for use in Australia.
- The Australian dog population is naïve to either subtype of CIV.
- In most dogs, the health impact of CIV infection is typically mild; with most dogs recover without complications (Lee et al. 2018; Payungporn et al. 2008; Voorhees et al. 2017). However, pneumonia associated with secondary bacterial infection occurs in a low proportion of CIV infected dogs but can be fatal (Crawford et al. 2006; Watson, Bell & Toohey-Kurth 2016).

- The risk associated with CIV is the highly contagious nature of the disease which is often seen to result in waves of infection through populations of naïve dogs. Dogs can be infected with CIV multiple times (Hilling & Hanel 2010).
- Immunocompromised dogs, puppies and elderly dogs are at a higher risk of severe infection (Merck 2016).
- In the case of secondary bacterial infections, antibiotics are indicated for immunosuppressed dogs or those with fever, purulent nasal discharge, productive cough and pneumonia (Crawford 2009).
- The mortality rate associated with H3N8 is less than 10% in most cases (Hilling & Hanel 2010). However some H3N2 strains isolated from China and the Republic of Korea have been found to be more virulent with mortality rates reported as high as 22–55% in some shelters (Jeoung et al. 2013; Lee et al. 2018; Song et al. 2011).
- There is no specific antiviral treatment for CIV, and treatment is supportive. Antitussives do not reduce the frequency and duration of coughing (Crawford 2009).
- CIV infection can lead to significant financial cost to owners, animal shelters and animal rescue organisations. Stakeholders have previously identified CIV as a disease of concern.

Conclusion

Based on the preceding key points, it was concluded that risk management measures for CIV are warranted for dogs from all countries approved to export dogs to Australia.

The information available also indicated that risk management measures for CIV are not warranted for dog semen.

In countries where evidence of CIV of either subtype has not been detected:

- Within 5 days immediately before export, the dog must be subjected to a thorough physical examination by a registered veterinarian and found to be free from clinical signs of infectious or contagious disease (such as CIV).

In countries where evidence of CIV of either subtype has been detected (and a vaccine is available):

- Within 12 months and at least 14 days immediately before export, the dog must be fully vaccinated[1] against the circulating strain(s) of CIV in the exporting country as per the manufacturer's direction; and
- Within 5 days immediately before export, the dog must be subjected to a thorough physical examination by a registered veterinarian and found to be free from clinical signs of infectious or contagious disease (such as CIV).

[1] Fully vaccinated refers to dogs over 8 weeks of age that have undergone a primary CIV vaccination (inactivated vaccine) course, consisting of 2 vaccinations administered 2 to 4 weeks apart, within 12 months and at least 14 days immediately prior to export. If the dog was last vaccinated more than 12 months before export, it must receive a single booster vaccination within 12 months and at least 14 days immediately prior to export.

Glossary

Term or abbreviation	Definition
ALOP	Appropriate level of protection
appropriate level of protection (ALOP) for Australia	The *Biosecurity Act 2015* defines the appropriate level of protection (or ALOP) for Australia as a high level of sanitary and phytosanitary protection aimed at reducing biosecurity risks to very low, but not to zero.
Australian territory	Australian territory as referenced in the *Biosecurity Act 2015* refers to Australia, Christmas Island and Cocos (Keeling) Islands.
biosecurity	The prevention of the entry, establishment or spread of unwanted pests and infectious disease agents to protect human, animal or plant health or life, and the environment.
biosecurity measures	The *Biosecurity Act 2015* defines biosecurity measures as measures to manage any of the following: biosecurity risk, the risk of contagion of a listed human disease, the risk of listed human diseases entering, emerging, establishing themselves or spreading in Australian territory, and biosecurity emergencies and human biosecurity emergencies.
biosecurity risk	The *Biosecurity Act 2015* refers to biosecurity risk as the likelihood of a disease or pest entering, establishing or spreading in Australian territory, and the potential for the disease or pest causing harm to human, animal or plant health, the environment, economic or community activities.
endemic	Belonging to, native to, or prevalent in a particular geography, area or environment.
goods	The *Biosecurity Act 2015* defines goods as an animal, a plant (whether moveable or not), a sample or specimen of a disease agent, a pest, mail or any other article, substance or thing (including, but not limited to, any kind of moveable property).
host	An organism that harbours a parasite, mutual partner, or commensal partner, typically providing nourishment and shelter.
pathogen	A biological agent that can cause disease to its host.
PCR	Polymerase chain reaction
PEQ	Post-entry quarantine
quarantine	Official confinement of regulated articles for observation and research or for further inspection, testing or treatment.
risk analysis	Refers to the technical or scientific process for assessing the level of biosecurity risk associated with the goods, or the class of goods, and if necessary, the identification of conditions that must be met to manage the level of biosecurity risk associated with the goods, or class of goods to a level that achieves the ALOP for Australia.
stakeholders	Government agencies, individuals, community or industry groups or organisations, in Australia or overseas, including the proponent/applicant for a specific proposal that have an interest in the policy issues.

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