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A REVIEW: CANINE AND FELINE INFLUENZA

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ABSTRACT

Dogs and cats were initially not considered to be susceptible or being potential reservoirs of influenza virus, they are now known to be vulnerable to several Influenza A Virus (IAV) subtypes and may serve as the source of infection to other animals and human. In 2004, Influenza A virus H3N8 was the first flu outbreak reported in dog. In less than a decade, 7 and 4 IAV subtypes have been reported in dogs and cats respectively, with two novel reassortant subtypes, Canine influenza virus (CIV) H3N1 (originating from Human Pandemic H1N1 and avian-origin H3N2) and H5N2 (Avian-origin H5N2 and swine-origin H9N2) in dog. This review focuses on different IAV subtypes identified in canine and feline species, and serve as guide for pet owners and veterinarians on diagnosis of infected dog and cat. However, a continuous closeness between human and pet animals especially dogs and cats prompt great concern about anthropozoonosis and zooanthroposis of Influenza virus. Thus, development of active surveillance and control plans for influenza and other emerging viral diseases in pet animals under the framework of "One World, One Health" will be an invaluable tool for control and prevention of IAV infections.

1. INTRODUCTION

Influenza A virus (IAV) belongs to the family orthomyxoviridae with 8 segments of negative sense linear single stranded ribonucleotide genome. These genes code for structural and non- structural protein including Haemmagglutinin gene (HA), Neuraminidase gene (NA), Polymerase complexes genes (PB1, PB2 and PA), Matrix protein gene (M), Non structural protein gene (NS) and Non protein gene (NP). These gene segmentations afford them antigenic drift (point mutation) and antigenic shift (genetic reassortment). Genetic alterations might result in formation of virion that are virulent and or cause the generation of entirely new IAV subtypes classified based on Haemagglutinin and Neuraminidase surface proteins (MacLachlan *et al.*, 2011). Presently, 18 haemagglutinin (H) and 11 neuraminidase (N) proteins have been identified (CDC, 2014) and haemagglutinin is an important determinant of the host specificity (Suzuki *et al.*, 2000).

IAV has been reported at different public media causing acute respiratory disease in human, domestic animals (chicken, turkey, pigs and horses) and wild animals (bat, tiger, leopard, ferret) (Webster *et al.*, 1992; Lipatov *et al.*, 2004; Keawcharoen *et al.*, 2004; Crawford *et al.*, 2005). Dogs and cats which were initially not considered to be susceptible or being potential reservoirs of influenza virus (Beeler, 2009) are now known to be vulnerable to several IAV subtypes (Dubovi *et al.*, 2008) and may serve as the source of infection to other animals and human.

This review was aimed at given an overview of various IAV subtypes identified in dogs and cats. Also, history, clinical signs, diagnosis, control and prevention of IAV infections were highlighted.

2. MATERIALS AND METHODS

This review employed about 60 publications on canine and feline influenza from Google search engine, PubMed and CDC website. Both influenza outbreak and subsequent experimental studies were included in the review. These publications were compared and presented in a narrative style and the findings were described qualitatively.

3. SUSCEPTIBILITY OF DOG AND CAT BASED ON BREED, AGE AND SEX

Although the first case of CIV was reported in race greyhound, it has been reported in other breeds of dog including Beagle, English foxhound, Yorkshire terrier, Pekingese, Dalmatian etc (Crawford *et al.,* 2005; Daly *et al.,* 2008; Payungporn *et al.,* 2008). It is believed that all breeds of dogs are susceptible to CIV (Dubovi *et al.,* 2008). Also, infection is not limited by age or sex. There is no special breed preference reported in cats (Kuiken *et al.,* 2004; Songserm *et al.,* 2006a; Sponseller *et al.,* 2010; Song *et al.,* 2011).

Generally, it is considered that interspecies transmission of a whole unmodified (no reassortment) IAV to dog or cat does not result into a sustained intraspecies transmission (Crawford *et al.,* 2005). Thus, formation of new sustained ancestry for influenza virus is rare (Webster *et al.,* 1992).

4. EPIDEMIOLOGY

Canine and feline influenza have been reported in several parts of the world including China, Japan, South Korea, Iraq, Thailand, Germany, United States and United Kingdom. The detection involved virus isolation, serosurveillance and genomic studies (Crawford *et al.,* 2005; Kuiken *et al.,* 2006; Songserm *et al.,* 2006b; Yingst *et al.,* 2006; Klopfleisch *et al.,* 2007; Daly *et al.,* 2008; Song *et al.,* 2008; Sponseller *et al.,* 2010; Song *et al.,* 2011; Zhan *et al.,* 2012). In Nigeria, serosurveillance had shown the presence of CIV in dogs. Oluwayelu *et al* (2014) reported seroprevalence of 40.5% and 0% of H3N8 and H3N2 respectively in pet and hunting dogs in Nigeria.

Majority of interspecies and intraspecies transmissions of IAV in dogs and cats under natural conditions had history of exposure to risk factors such as direct contact with infected animals and humans, ingestion of infected animals and aggregation of susceptible animals (Yoon *et al.,* 2005; Songserm *et al.,* 2006a; Songserm *et al.,* 2006b; Lin *et al.,* 2012; Sponseller *et al.,* 2010). Reported dogs and cats with H1N1 infection had history of contact with human who had influenza-like illness prior to the pets becoming ill (Sponseller *et al.,* 2010; Lin *et al.,* 2012). The direct transmission of the entire Equine-origin H3N8 virus to dog (Crawford *et al.,* 2005) also indicated contact with infected animal as a risk factor. Avian-origin H3N2 and H5N1 infections in dogs and cats were reported to have been transmitted through ingestion of infected birds especially during influenza outbreak in bird (Songserm *et al.,* 2006a; Songserm *et al.,* 2006b; Song *et al.,* 2008; Song *et al.,* 2011).

In decreasing order, IAV subtypes H3N2, H3N8, H5N2, H3N1, H1N1 and H5N1 may be transmitted in dog population when infected dog is comingled with uninfected ones in animal shelter, dog shows etc (Song *et al.,* 2009; Lin *et al.,* 2012; Jirjis *et al.,* 2010; Qian-qian *et al.,* 2012). Under the same condition, IAV Subtype H3N2 and H9N2 rapidly spread in cat population (Giese *et al.,* 2008; Zhang *et al.,* 2013).

5. INFLUENZA A VIRUS SUBTYPES IN DOG AND CAT

Various IAV subtypes have been identified in dog and cat. In dogs, these include CIV H3N8, CIV H3N2, CIV H3N1, Pandemic H1N1, Highly Pathogenic Avian Influenza (HPAI) H5N1, H5N2 and Avian-like H9N2 (Keawcharoen *et al.,* 2004; Crawford *et al.,* 2005; Thanawongnuwech *et al.,* 2005; Songserm *et al.,* 2006b; Harder *et al., 2010*; Lin *et al.,* 2011; Song *et al.,* 2012; Zhan *et al.,* 2012; Sun *et al.,* 2013). In cat, these IAV subtypes include Avian influenza H9N2; CIV H3N2, HPAI H5N1 and Pandemic H1N1 (Songserm *et al.,* 2006a; Sponseller *et al.,* 2010; Song *et al.,* 2011; Zhou *et al.,* 2015a).

The transmission of IAV to dog and cat is shown in figures 1 and 2. Summary of influenza outbreaks and subsequent laboratory experiments are presented in table 1. In all, 7 and 4 IAV subtypes have been identified in dog and cat respectively, with two novel IAV reassortants in dog.

a. Influenza subtypes identified in dog

i. Pandemic H1N1

Influenza virus H1N1 cases have been reported in cats, pig and cheetahs after pandemic human influenza H1N1 outbreak of 2009 in Mexico and United States (http://vetmedicine.about.com/od/zoonotic/tp/H1N1news). Toward the end of 2009, two dogs were presented separately at a veterinary teaching hospital in China with history of respiratory abnormalities including severe cough with sputum. One of the owners reported that the family members had experienced influenza-like illness prior to the dog becoming ill. Reverse transcriptase polymerase chain reaction assay (rtPCR) carried out on the nasal swabs and virus isolates cultured from embryonated specific pathogen free chicken egg indicated the presence of H1N1/2009 influenza virus (Lin *et al.,* 2012). The genomic sequence analysis of all the 8 genes depicted that the viruses shared 98.9–100% nucleotide identity with human H1N1/2009 strain A/California/04/2009, suggesting a direct transmission from human (Lin *et al.,* 2012).

Also, it has been experimentally proven that interspecies transmission of H1N1/2009 among dogs is low (Lin *et al.*, 2012).

Table 1: Summary of Influenza outbreaks in dog and cats

Influenza virus type	Year	Location	Animal species/ breed	Source	Transmission	Reference
Pandemic H1N1	2009	Mexico, USA	Cat, Dog, Cheetah	Human	DOG: Interspecies, intraspecies (low in dog)	Lin *et al.*, 2012; Sponseller *et al.*, 2010
Avian-Origin CIV H3N2	2007(Dog); 2010(cat)	South Korea	Dog	Duck, chicken?	Interspecies, intraspecies	Song *et al.*, 2008; Song *et al.*, 2009; Song *et al.*, 2011; Jeoung *et al.*, 2013
CIV H3N1 (Reassortant)	2007-2009	South Korea	Dog	Avian (H3N2) and human or swine (H1N1)	Interspecies	Song *et al.*, 2012
Equine-origin CIV H3N8	2004	United State	Dog	Horse	Interspecies, intraspecies	Crawford *et al.*, 2005; Yamanaka *et al.*, 2009; Jirjis *et al.*, 2010
HPAI H5N1	2004	Thailand	Dog, cat, tiger, leopard	Duck, pigeon	Interspecies, intraspecies (unlikely)	Songserm *et al.*, 2006a ; Songserm *et al.*, 2006b; Giese *et al.*, 2008; Keawcharoen *et al.*, 2004
H5N2 (Reassortant)	2009	China	Dog	Turkey (H9N2) and Swine (H5N1)	Interspecies, intraspecies	Zhan *et al.*, 2012; Qian-qian *et al.*, 2012
Avian influenza virus H9N2	2011	China	Dog, cat	Avian ?	Interspecies(?), Intraspecies (inefficient in cat)	Amirsalehy *et al.*, 2012; Sun *et al.*, 2013; Zhang *et al.*, 2013; Zhou *et al.*, 2015a ; Zhou *et al.*, 2015b

ii. Avian-Origin Canine Influenza Virus H3N2

In South Korea, there was an outbreak of CIV H3N2 with severe respiratory diseases between May to September 2007 (Song *et al.*, 2008). The first case recovered after days of nasal discharges and sneezing. Other infected dogs further showed severe cough, fever, anorexia and some died. Increasing CIV antibody titres were also observed in 13 dogs housed in a clinic-owned shelter facility. It was suggested that this subtype might have come from untreated minced meats of ducks or chickens since it is common in South Korea to feed dogs with untreated duck and chicken meats including internal organs and head for fattening purpose (Song *et al.*, 2008). Interspecies transmission of avian-origin CIV H3N2 was later reported in a domestic farm dog at Southern China under natural condition (Su *et al.*, 2012). Phylogenetic analyses of the sequences of all eight viral RNA segments demonstrated that these are wholly avian influenza viruses of the Asia lineage.

Dog-to-dog transmission was experimentally demonstrated with the infected and the exposed dogs showing pyrexia, virus shedding, seroconversion, severe necrotizing tracheobronchitis and brochioalveolitis (Song *et al.*, 2009).

iii. Canine Influenza virus H3N1 (Reassortant)

A novel reassortant CIV H3N1 virus was identified in nasal swabs of sick dogs (showing clinical respiratory signs such as cough, nasal discharge and fever) during surveillance which spanned from 2007 to 2009 in South Korea (Song *et al.*, 2012). The genomic sequence analysis of the amplified genes revealed that its HA gene had 99% similarity with CIV H3N2 influenza strain (A/canine/Korea/GCVP01/2007) circulating in South Korea during that time. Furthermore, phylogenetic study revealed that the CIV H3N1 originated from avian-origin CIV H3N2 (which were circulating in South Korea and China that time) and pandemic H1N1 viruses based on HA and NA gene cluster closeness. Further studies indicated that HA gene originated from CIV H3N2 while the other 7 genes originated from pandemic H1N1. Experimental infection of dog showed low virus shedding with mild pathogenicity compared to CIV H3N2 (Song *et al.*, 2012).

iv. Equine-origin Canine influenza virus H3N8

Equine-origin CIV H3N8 was first identified in racing greyhounds in Florida in 2004 and was associated with respiratory disease outbreaks (Crawford *et al.*, 2005; Yoon *et al.*, 2005). This

influenza subtype shares more than 96% nucleotide sequence identity with contemporary Equine IAV subtype H3N8 suggesting direct transmission of the entire virus from horse to dog without reassorting with other strains (Crawford *et al.,* 2005). The haemagglutinin of this subtype was found to differ from contemporary equine viruses by 5 conserved amino acid substitutions (Payungporn *et al.,* 2008). However, the virus shed from dogs in close contact with an experimentally Equine Influenza H3N8 infected horse had no substitution of amino acids in their haemagglutinin (Yamanaka *et al.,* 2009). Also, these experimental dogs present no clinical signs though the haemagglutination inhibition (HI) test carried out showed that all the dogs exhibited seroconversion. It might be that amino acid substitutions in haemagglutinin of H3N8 influenza outbreak in Florida increase virulence.

Intraspecies transmission by experimental infection has been reported (Jirjis *et al.,* 2010). All the CIV H3N8 infected dogs showed respiratory abnormalities while 75% of uninfected dogs commingled with the infected dogs showed respiratory abnormalities. In addition, the serological evidences and isolation of closely related H3N8 subtype in dogs from different geographic locations in United States and other countries of the world strengthen the acclaimed dog-to-dog transmission (Crawford *et al.,* 2005; Yoon *et al.,* 2005).

v. Highly Pathogenic Avian Influenza H5N1

HPAI H5N1 was reported in a dog after ingesting H5N1-infected duck carcass during an outbreak in Thailand in 2004 (Songserm *et al.,* 2006b). Based on genomic sequence and phylogenetic analysis, the isolated virus possessed similar haemagglutinin and neuraminidase genes found in H5N1 viruses from chickens, ducks, tigers and humans infected in Thailand during the same time that the dog was infected. Also, seroprevalence study in Central Thailand revealed 25% prevalence of H5N1 antibodies among 629 village dogs (Butler, 2006).

The intraspecies transmission of HPAI H5N1 in dogs has been experimentally proved to be unlikely compared to other adapted CIV H3N2 (Giese *et al.,* 2008; Song *et al.,* 2009).

vi. H5N2 (Reassortant)

It was first reported in Shandong, China in 2009 from dog exhibiting respiratory signs (Zhan *et al.,* 2012). The genomic sequence and phylogenetic analysis revealed that its neuraminidase gene (NA) is closely related to NA gene of avian influenza virus (AIV) A/turkey/Wisconsin/66(H9N2) and A/turkey/WI/ 1966(H9N2) while the remaining 7 genes (including HA gene) are closely related to the corresponding genes of A/swine/Fujian/F1/ 2001(H5N1). Thus, it was declared to be a reassortant influenza virus. However, the prevalence of anti-H5N2 antibodies was found to be 3.21% (6/187).

In like manner, dog-to-dog transmission of the virus was experimented. Majority of the infected and the exposed dogs exhibited mild respiratory signs including transient pyrexia, conjunctivitis, sneezing, nasal discharge and mild coughing, virus shedding and seroconversion, but there was no fatal disease (Qian-qian *et al.,* 2012). The interspecies transmission of H5N2 from infected dog to cat and chicken has been proven in a contact exposure experiment (Hai-xia *et al,* 2014).

vii. Avian-like H9N2

Avian–like H9N2 was first isolated in 2011from nasal mucus samples of dog during a 2-year influenza surveillance in Guangxi, China. Nucleotide studies revealed that the HA and NA genes of this subtype has 99.7% and 99.5% similarities with those of H9N2 viruses isolated from chicken in 2011. Phylogenetic analysis revealed the novel genotype of this isolate and denoted as U. Serosurveillance between 2010-2011 detected a prevalence range of 20.21 -28.98%, while in 2012, the rate was higher (44.85%) (Sun *et al.,* 2013). Experimental infection also confirms the susceptibility of dogs to avian origin H9N2 (Amirsalehy *et al.,* 2012).However, susceptibility may vary between breeds and H9N2 subtype origin (Amirsalehy e*t al.,* 2012; Zhou *et al.,* 2015b).

Infected dog present mild clinical signs including weak, coughing, sneezing, nasal discharge, mild pyrexia, appetite and vomiting. However, infection may be subclinical (Sun *et al.,* 2013; Zhang *et al.,* 2013).

b. **Influenza subtype identified in cat**

i. Pandemic H1N1

In 2009, an old castrate domestic cat that was kept indoor in a single-cat household was diagnosed of Pandemic HINI (2009) (Sponseller *et al.,* 2010). This cat was reported to develop respiratory difficulties some days after its close human family members had influenza-like illness. Real-time rtPCR assay identified this virus as pandemic (H1N1) 2009 (Eurasian matrix) which differs from endemic swine H1N1 (North America matrix). Genomic sequences of HA, NA and M genes from the virus isolate further confirmed the similarities with the same genes of pandemic H1N1 (2009) virus.

ii. Canine influenza virus H3N2

In 2010, interspecies transmission of CIV H3N2 virus was reported in cats (and dogs) at an animal shelter in South Korea with 100% morbidity and 40% mortality (Song *et al.,* 2011). The nucleotide sequences of the feline isolate displayed 98.0–99.8% similarities with all eight gene segments of CIV H3N2. Also, serological evidence of canine H3N2 in stray cats found near live poultry market in China suggested that interspecies transmission can originate from birds (Zhou *et al.,* 2015a).

Also, the intraspecies transmission of CIV H3N2 was found to be rapid in cats with endemic or epidemic respiratory diseases characteristics (Jeoung *et al.,* 2013).

iii. Highly Pathogenic Avian Influenza (HPAI) H5N1

HPAI H5N1 has the ability to infect and kill avian species, mammals including man. A Zoo at Suphanburi in Thailand reported death of 2 leopard and 2 tigers after being fed with chicken carcasses during H5N1 virus outbreak in 2003 - 2004. Clinical signs and necropsy findings showed that these zoo animals had respiratory abnormalities. Reverse transcriptase-PCR identified H5N1 from RNA extracted from tissue and isolated virus. Nucleotide sequencing and phylogenetic analysis confirmed further that this influenza subtype was identical to the circulating H5N1 in the poultry during that time (Keawcharoen *et al.,* 2004). During the same influenza outbreak period, high mortalities were also recorded in domestic cats (Kuiken *et al,* 2006) but the susceptibility of cat was not reported immediately until experimental confirmation using human isolate of HPAI H5N1 (Kuiken *et al.,* 2004). However, within the same period of outbreak, a cat was reported to have eaten HPAI H5N1 infected pigeon and later died 7 days post-ingestion after showing pyrexia, convulsion and ataxia 2 days before death (Songserm *et al.,* 2006a). Sequencing and phylogenetic analysis of the HA and NA genes of HPAI virus isolated from the cat and the pigeon that died during the outbreak showed that the HA and NA genes of the viruses were similar to each other as well as to those of the viruses isolated from tigers, chickens, and humans in Thailand (Songserm *et al.,* 2006a). Seroprevalence studies conducted in China from 2010 to 2013 indicated low prevalence of H5N1 using haemagglutination inhibition test and microneutralization assay (Sun *et al.,* 2015; Zhou *et al.,* 2015a)

However, transmission of H5N1 from experimentally infected dog to cat has been experimentally proven to be unlikely (Giese *et al.,* 2008).

iv. Influenza virus H9N2

A Seroprevalence study conducted from 2010 to 2012 in China evidently showed that cat can seroconvert against H9N2. Sera samples of 0.034% and 0.023% were tested positive for H9N2 antigen using Haemagglutination inhibition test (1:20 titre) and microneutralization assay (1:80 titre) respectively (Zhou *et al.,* 2015a). This study further showed that cats near poultry market and those showing influenza-like signs have higher risk of possessing rise in antibody titer. Experimental evidence showed that influenza H9N2 virus can be recovered from intranasally inoculated cat. Also, intraspecies transmission of this subtype has been reported (Zhang *et al.,* 2013)

6. DIAGNOSIS

a. Clinical signs

Most of the infected dogs were presented with upper respiratory abnormalities such as cough with or without sputum, nasal discharges and panting, which can last up to 30 days (Yoon *et al.,* 2005; Kirkland et al., 2010; Payungporn *et al.,* 2008; Songserm *et al.,* 2006b; Lin *et al.,* 2012; Sponseller et al., 2010). Other clinical signs may include pyrexia, anorexia, mild depression, lethargy and lung crackle (sound). Some of the infected dogs and cats may recover while some die peracutely depending on the IAV subtype virulence, extent of respiratory system compromise and secondary infections. Radiograph may further show the condition of the lower respiratory tract (Kirkland *et al.,* 2010; Payungporn *et al.,* 2008; Sponseller *et al.,* 2010).

b. Pathology

Generally, lung lesions were seen and these include discolorations, foci necrosis, haemorrhage and consolidation. Lesion may extend to pleural cavity and adjacent tissues. Histopathology may display cilia destruction, trachietis, and bronchitis, mild to moderate interstitial pneumonia. Suppurative pneumonia in combination with neutrophil infiltration and extensive erosion have also been reported (Yoon, *et al.,* 2005; Songserm *et al.,* 2006a; Songserm *et al.,* 2006b; Payungporn *et al.,* 2008; Song *et al.,* 2011). It is worth noting that IAV H3N1 and Pandemic H1N1 subtypes causes mild symptoms in dogs and cats (Lin *et al.,* 2012; Sponseller *et al.,* 2010; Song *et al.,* 2012).

c. Laboratory diagnosis

Clinical samples that can be collected for virologic diagnosis may include pair sera and swabs from nasal cavity, rectum and oropharynx. At post-mortem, affected organs in addition to lungs are collected. Serology using Enzyme linked immunosorbent assay (ELISA) that detect antibodies against IAV nucleoprotein followed by Haemagglutination inhibition (HI) test or Fluorescent antibody test (FAT) using specific antigen have been reported (Benedictis *et al.*,2010). Virus antigens in the pathologic tissue(s) can be identified through immunohistochemistry technique using specific monoclonal antibody specific for IAV nucleoprotein (Songserm *et al.,* 2006b).

Influenza virus can be isolated in 10-day-old specific pathogen free (SPF) embryonated chicken or turkey egg. Allantoic fluid is harvested for haemagglutination test, immunofluorescent test etc. Cell culture such as Madin Darby Canine Kidney cell (MDCK) can be used because its surface possesses both sialic acid (SA) α- 2,3-galactose and SA α- 2,6 -galactose receptors at high and low concentration respectively (Kovbasnjuk *et al.,* 2000, Harder *et al., 2010*). Genetically engineered MDCK cell are available for high sensitivity (Oh *et al.*, 2008).

Also, RNA extraction can be done using swab sample and virus culture, and amplified by rtPCR using commercial extraction kit and specific forward and reverse primers for IAV gene of interest (Payungporn *et al.,* 2008). Further investigations may involve genomic sequencing and phylogenetic analysis.

7. CONTROL AND PREVENTION

Presently, there is no drug of choice for control of IAV infection. However, the use of broad spectrum antibiotic and other supportive therapy have been reported to reduce the severity of the disease (Yoon *et al.,* 2005). The use of iodine preparations to reduce the spread of the virus has been experimented and it promises to be effective. Vaccination of human and animal against influenza may be considered especially in epidemic areas to increase and boost antibody titre against common influenza subtype. Proper disposal and destruction of IAV infected carcasses should be done by appropriate department to impede contact by other susceptible animals and scavenging wild birds (Song *et al.,* 2011). Also, the feeding of uncooked poultry meat to dogs and cats should be avoided. Isolation of infected dog or cat should be done in a suspected IAV infection. Human with flu symptoms should avoid exposure of pet animals (Lin *et al.,* 2012).

8. RISK TO HUMAN

The evidence of different IAV subtypes in dogs and cats indicate their susceptibility. However, the infection of these animals with human-origin IAV subtypes loud their probable position on epidemiology of human influenza. The fact that these species are closest to human, as pet, guard and or for hunting purpose, zooanthroposis cannot be taken lightly. More so, the pattern of disease spread is made difficult in subclinical or asymptomatic infection in dog and cat. Another danger that may arise from multiple subtype susceptibility in dogs and cats is the possible generation of a new novel influenza subtype originating from reassortment of mixed infection of IAV subtypes (Crawford *et al.,* 2005; Payungporn *et al.,* 2008).

9. CONCLUSION

In contrast to previous belief, dog and cat are now known to be susceptible to increasing numbers of IAV subtypes. Less than a decade, 7 and 4 IAV subtypes have been reported in dogs and cats respectively, with two novel reassortant subtypes, CIV H3N1 (originating from Human Pandemic H1N1 and avian-origin H3N2) and H5N2 (Avian-origin H5N2 and swine-origin H9N2) in dog. Although, these IAV subtypes have varying potential to establish and cause disease in dog and cat populations, the adaptive characteristic of influenza virus may eventually facilitate these subtypes becoming a common infection in these populations. The susceptibility of dog and cat to avian and human origin IAV subtypes suggest need to consider them as potential foes since they can be hosts within which genomic reassortment can take place. Also, the continuous closeness between human and pet animals especially dogs and cats prompt great concern about zoonosis. Thus, development of active surveillance and control plans on influenza in pet animals under the framework of “One World, One Health” will be an invaluable tool for control and prevention of IAV infections.

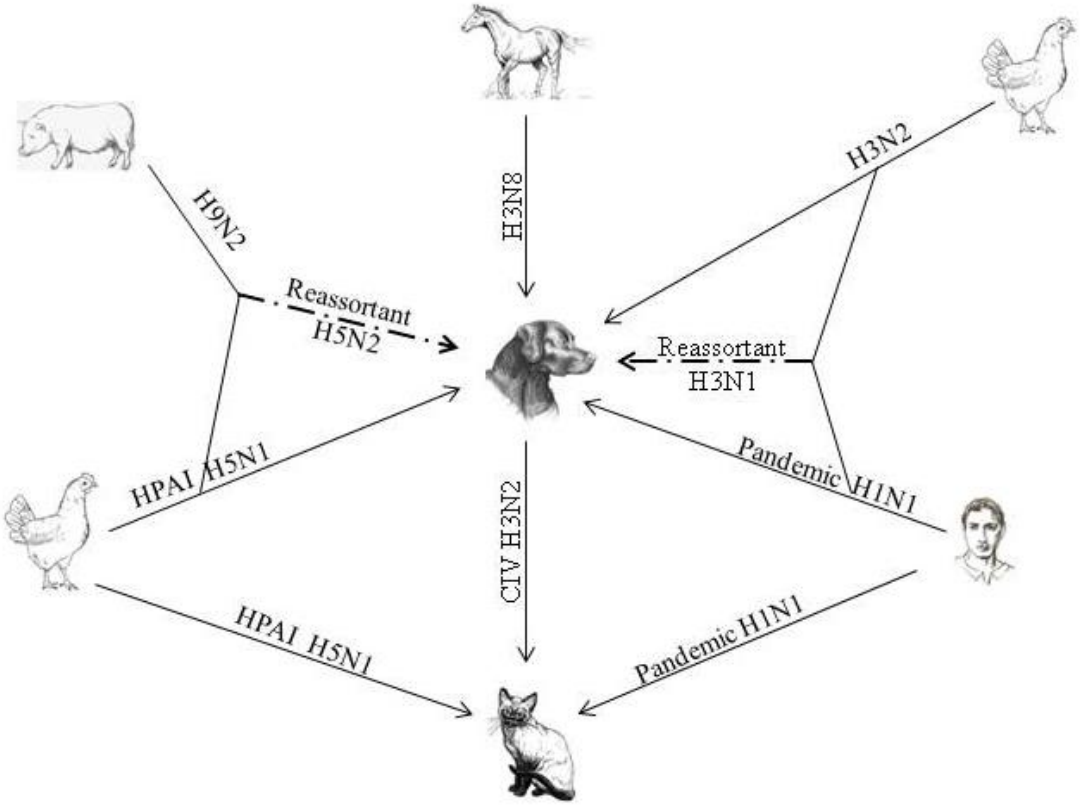

Figure showing interspecies transmission of Influenza A virus to dog and cat.
Legend: Direct transmission (Straight arrow)
Genomic reassortment occurred before transmission (broken arrow)

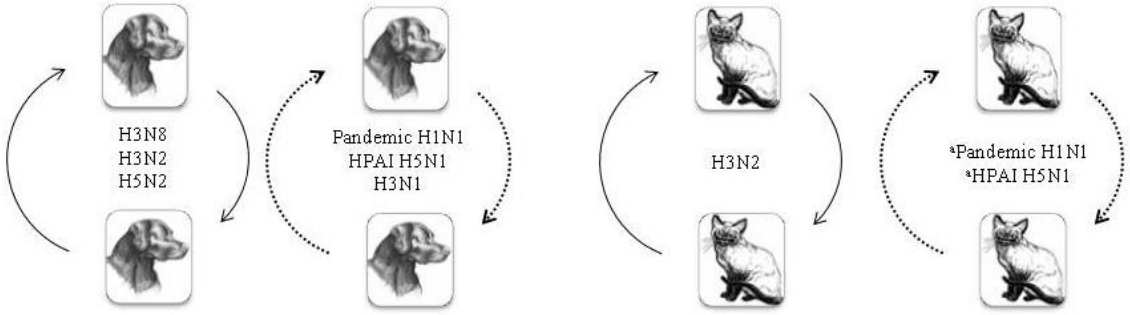

Figure showing intraspecies transmission of Influenza A virus to dog and cat.
Legend: [a] Intraspecies transmission is unknown
Intraspecies transmission is present (Straight line)
Intraspecies transmission is absent or low (Broken line)

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