

Influenza in Dogs and Cats

Emily Beeler, DVM^{a,b,*}

KEYWORDS

• Influenza • Dog • Cat • Emerging disease • H5N1 • H3N8

For the first time in history, influenza viruses have been documented as the cause of natural outbreaks of illness in dogs and cats. The years 2003 to 2004 brought the discovery of both canine influenza (H3N8) in racing greyhounds in Florida and avian influenza (H5N1) in zoo cats in Thailand. Dogs and cats may come to play a role in the evolution of new strains of human influenza. As a result, small animal veterinarians face new challenges in caring for their patients and a heightened public health responsibility. Canine influenza (H3N8), which is not known to be zoonotic, is the strain small animal practitioners are most likely to encounter. However, veterinarians must also be aware of less common, but perhaps more consequential, strains.

INFLUENZA BASICS

Influenza viruses belong to the family Orthomyxoviridae. There are three major types of influenza: A, B, and C.¹ Influenza A, which is the focus of this article, is of the greatest significance: it is the most mutable, causes human influenza pandemics, and has the widest host species range.²

Influenza A is an enveloped virus. The envelope, derived from the plasma membrane of the cell in which the virus was assembled, makes influenza A easier to inactivate than nonenveloped viruses.³ Cold temperatures prolong survival of influenza virus in water and on surfaces. It is inactivated by most properly performed cleaning and disinfection protocols. Heat, common disinfectants, hand washing, and laundering are all useful in reducing contamination (**Table 1**).

The influenza A genome contains eight separate strands of RNA that code for 11 proteins. Two of the 11 are called hemagglutinin (H) and neuraminidase (N).⁴

^a Los Angeles County Department of Public Health, Veterinary Public Health and Rabies Control Program, 7601 East Imperial Highway, Building 700, Room 94A, Downey, CA 90242, USA

^b College of Veterinary Medicine, Western University of Health Sciences, 309 E Second Street, Pomona, CA 91766, USA

* Los Angeles County Department of Public Health, Veterinary Public Health and Rabies Control Program, 7601 E Imperial Highway, Building 700, Room 94A, Downey, CA 90242, USA. E-mail address: ebeeler@ph.lacounty.gov

Table 1 Influenza A survival times, inactivation periods, and inactivation procedures for various environmental conditions	
Environment	Survival Time for Influenza A Viruses
Water	
Frozen	Indefinitely
32°F	Over 30 days
71°F	4 days
132°F–140°F	0.5–3 hours ⁴⁵
Feces (generally from infected birds)	
Frozen	Indefinitely
39°F	30–35 days
68°F	7 days ⁴⁵
Cloth, paper, tissues (no visible organic matter)	8–12 hours ⁴⁶
Nonporous plastic, stainless steel (with no visible organic matter)	1–2 days ⁴⁶
Sanitation Need	Procedure
Hand cleaning	Warm soap and water for ≥ 20 seconds. If no organic matter is adhered to hands, alcohol-based hand sanitizer may be used; enough should be applied to keep hands moist for ≥ 20 seconds. ⁴⁷
Surface disinfection (after visible organic matter removed)	1% bleach, 70% ethanol, quaternary ammonium salts (such as Roccal or Triple 2), and many other disinfectants. Contact time ≥ 10 minutes. ⁴⁸
Towels, bedding, cloth	Normal laundering with detergent ⁴⁷
Instrument sterilization	Moist heat autoclaving at 250°F for 15 minutes, at 140°F for 30 minutes, ⁴⁸ or cold sterilization with glutaraldehyde solutions. ⁴³
Cooking	Heat to minimum of 165°F ⁴⁹

Numerous copies of these two proteins project through the envelope and appear as “spikes” coating the viral surface (**Fig. 1**).

Hemagglutinins allow the virus to attach to sialic acid receptors on cell surfaces and thereby infect cells. Hemagglutinin plays the predominant role in the species specificity and tissue tropism of the virus. Sixteen different serotypes of hemagglutinin have been identified: H1 to H16.⁵

Neuraminidase orchestrates the exit of newly formed virions from an infected cell by cleaving sialic acid receptors that would otherwise bind to the hemagglutinin. Nine neuraminidase serotypes have been discovered.¹ Influenza A viruses are subcategorized by these two surface proteins. Examples include H1N1, H5N1, and H9N2.

MUTATION

The mutability of influenza A leads to annual outbreaks of influenza and occasional pandemics. Mutation occurs two ways.

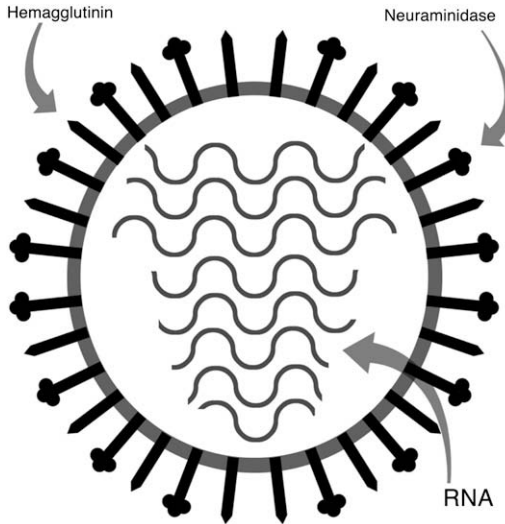


Fig. 1. Hemagglutinin and neuraminidase surface proteins, influenza A virus.

In antigenic “drift,” single nucleic acid point mutations occur when influenza RNA is replicated within an infected cell. There is no “proofreader” enzyme to correct these mutations. Drift causes the constant low-level change seen in human seasonal flu.

In antigenic “shift” the host range of an influenza virus abruptly changes. Typically two different influenza viruses enter the same cell and swap genes during replication. These new viruses may have a different host range than the original viruses.⁵

ECOLOGY OF INFLUENZA A VIRUSES

Wild waterfowl are the reservoir for all known influenza A subtypes.¹ Many influenza A viruses replicate in the birds’ intestinal mucosa and are shed in feces, without causing apparent illness. Periodically, new influenza A strains emerge from the wild, adapt to new species, and cause epizootics or panzootics.¹

Most of the time, however, influenza viruses exhibit species-specific infectivity. An individual strain of influenza is highly contagious, or highly adapted, to only one species (**Table 2**). The most common strains of influenza A infect humans, poultry, pigs, horses, and dogs.² Outbreaks have been documented in harbor seals.⁶ Ferrets are susceptible to human influenza viruses.² The H5N1 avian influenza virus is

Species	Well Adapted: Highly Transmissible Between Members of Same Species	Partly Adapted: Not Highly Transmissible Between Members of Same Species
Humans	H3N2, H1N1	H5N1, H9N2, H7N2, H7N3, H7N7 ⁵
Cats	None known.	H5N1 ^{19–30}
Dogs	H3N8 ^{39–43}	H5N1, H3N2 ^{11,34–38}

transmissible between cats on a small scale, but has not been shown to spread rapidly or efficiently in cats.

A large number of influenza A viruses infect poultry. Clinical signs are primarily seen in chickens and turkeys, and ducks are often asymptomatic. Virus is shed in feces and respiratory secretions. Most avian influenza viruses are considered low pathogenicity avian influenza (LPAI) viruses, causing drops in egg production and mild respiratory signs. A subset of LPAI viruses, those containing hemagglutinins H7 or H5, can mutate to become highly pathogenic avian influenza (HPAI) viruses. HPAI causes sudden death, dyspnea, diarrhea, cyanosis, neurologic signs, and hemorrhage and necrosis in multiple internal organs.⁷

PANDEMIC INFLUENZA

The influenza pandemic of 1918 to 1919 was one of the largest and most severe human disease outbreaks in history, taking up to 50 million lives.⁵ This virus was later labeled an H1N1 influenza A virus. Half of the deaths were in healthy adults. Many died from acute respiratory distress syndrome (ARDS).⁸

Genetic sequencing of the 1918 virus suggests it originated when a bird strain transferred to and adapted to humans; it was an entirely new virus in the human population, unrelated to the seasonal influenza of the time. The originating bird host remains a mystery. In contrast, milder human influenza pandemics in 1957 and 1968 followed mutation of the circulating seasonal influenza virus, which had acquired a few genes from avian viruses.⁹

CONCERNS FOR A NEW PANDEMIC

It is unknown if new pandemics can be predicted.⁸ In recent years, five influenza A subtypes of avian origin have been transmitted to humans, causing illness without becoming highly transmissible from human to human (see **Table 2**). All are candidates for becoming the next pandemic strain. One of them, HPAI H5N1, has spread over a vast geographic area and caused the greatest concern.

HIGHLY PATHOGENIC AVIAN INFLUENZA H5N1

HPAI H5N1 is causing one of the largest panzootics in poultry ever documented, and has crossed the species barrier several times, infecting humans, tigers, leopards, domestic cats, palm civets (a kind of exotic cat), macaques, a stone marten (a weasel-like animal), a mink, and a few dogs.^{10–12} It first caused poultry outbreaks in Hong Kong in 1997 and later spread throughout Southeast Asia, westward across Asia, and into Europe, the Middle East, and Africa. Wild waterfowl, the typically asymptomatic reservoirs of influenza A, died from HPAI H5N1 by the thousands in 2005 and 2006 at a large lake in China.¹⁰

Human infections with HPAI H5N1 are infrequent, occurring in places where poultry outbreaks occur, with a case fatality rate of 60%. Most people are infected by very close contact with infected poultry or their feces, or ingestion of raw poultry products.¹⁰ There are isolated cases of human-to-human transmission of the virus. Infected humans exhibit respiratory signs and fever, with many developing ARDS. Neurologic signs and diarrhea can also occur. Elevated aspartate aminotransferase (AST), alanine transaminase (ALT), and creatinine have been found in some patients. Lymphopenia upon presentation is statistically associated with a higher case fatality rate.¹³ The HPAI H5N1 virus attaches best to human bronchiolar epithelium and alveoli, and poorly to the tracheal epithelium. Preferential binding occurs to Type II

pneumocytes, which produce surfactant and are more metabolically active than Type I cells. Perhaps the most telling, H5N1 adheres very well to pulmonary macrophages, which plays a role in inflammation and ARDS.^{14,15}

There have been few autopsies performed in cases of human HPAI H5N1. Feline cases, although less closely reported and tracked, have been documented in much greater detail.

INFLUENZA IN CATS

Experiments in the 1970s and 1980s showed that cats could become infected with human influenza A (H3N2, H2N2). They shed the virus from the nose or pharynx, developed an antibody response, and transmitted it from cat-to-cat, but did not become ill.¹⁵⁻¹⁷ Before the appearance of HPAI H5N1, there were no known natural occurrences of influenza A illness in cats.

HIGHLY PATHOGENIC AVIAN INFLUENZA H5N1 IN CATS

In February 2004, the World Health Organization reported a die-off of 14 out of 15 cats in one household in Thailand. Two of three cats tested were positive for H5N1. At least one of these cats had been in contact with dead chickens.¹⁸

Subsequent reports identified H5N1 infection in tigers in China,¹⁹ tigers and leopards from zoos in Thailand,^{20,21} a domestic cat in Thailand,²² domestic cats in Iraq and Turkey,²³ three stray cats on a German island,²⁴ and cats in an Austrian animal shelter.²⁵ In all cases, except that in the Austrian shelter, cats were found dead or had severe illness. Clinical signs included high fever, dyspnea, panting, ataxia, and convulsions. In most of these cases, ingestion of raw infected birds or direct contact with poultry feces were likely sources. A serosurvey of stray cats near infected poultry markets showed that 20% were seropositive, indicating prior infection.²⁶ There have been no reports of humans contracting the virus from cats.

The 2004 outbreak of H5N1 in a tiger zoo in Thailand happened after tigers ate raw, infected chickens. The cats' diet was changed to cooked meat and moribund cats were euthanized to stop the outbreak. More cats became ill despite the intervention, and tiger-to-tiger transmission was suspected.²¹

The 2006 report of H5N1 infection in three cats in an Austrian animal shelter was not associated with clinical signs. Cats had been tested because some had climbed into a poultry pen where an H5N1-infected swan was kept. Only 3 of 40 cats tested were positive for viral RNA on pharyngeal swabs. A week later two of these cats were rechecked, and were negative. These two cats did seroconvert, as did another cat that had been negative for viral RNA on pharyngeal swab.²⁵ Some feared this meant cats could silently carry and shed the virus. However, given the very limited spread of the virus under shelter conditions, this is unlikely to be the case. It is not known if any cats had shed live virus.

Results of experimental studies on HPAI H5N1 infection in cats are similar to what has been observed in the field. Cats inoculated oculo-nasopharyngeally, intratracheally, or by being fed infected 1-day old chicks, developed high fevers (over 104°F), dyspnea, conjunctivitis, and prominent nictitans. Infected cats transmitted the virus to their uninfected cage mates. These secondarily infected cats also became very ill. Clinical signs began 1 to 2 days postinfection for those experimentally infected and about 5 days after exposure to ill cats for those infected secondarily. Cats did *not* become infected when living next to or sharing bowls with infected dogs, nor did infected cats transmit virus to dogs. Swabs taken of the pharynx and rectum of ill cats revealed viral RNA, raising the possibility that virus might be shed in feces and

possibly be spread through the fecal-oral route. Feces from the cats were not reported to be evaluated directly for virus.²⁷⁻³⁰

Necropsies from both the naturally occurring and experimentally infected cases revealed systemic pathology, similar to that seen in chickens infected with HPAI H5N1. Commonly seen were hemorrhagic consolidated lungs, and bronchointerstitial and necrotic pneumonia with loss of bronchiolar and alveolar epithelium. Liver necrosis and encephalitis were also frequent. In some cases, enlarged tonsils and mandibular lymph nodes, pleural effusion, hemorrhagic pancreatitis, and hemorrhage in multiple organs was seen. Virus was found in the lungs, liver, and brain by immunohistochemistry and virus isolation.^{20-24,27-30} Virus was also extracted from pleural fluid, duodenum, kidney, spleen, and urine of the infected domestic cat from Thailand.²²

A dose-response in cat H5N1 infections has been demonstrated. When inoculated with high doses of virus, cats become severely ill as seen before. When inoculated with moderate doses, cats showed no clinical signs, but seroconverted and shed some virus from the pharynx. Cats inoculated with small amounts of virus do not become ill, seroconvert, or shed any virus.³⁰

Cats were protected from HPAI H5N1 by an experimental vaccine created from an LPAI H5N6 virus. The vaccine was administered to five cats subcutaneously, twice to each cat, 1 month apart. A month after the second dose, they were challenged with high doses of HPAI H5N1 virus. They developed only mild fevers, with the highest being 102.7°F, and no other clinical signs. In contrast, unvaccinated control cats became severely ill. Only two of the five vaccinated cats shed any viral RNA from the pharynx and rectum after H5N1 inoculation, and only one shed live virus. This last cat had produced the lowest levels of H5N1-specific antibodies in response to the vaccine. In contrast, all five cats used as unvaccinated controls shed large amounts of virus from the pharynx after infection, and two shed virus rectally.³⁰ The successful use of an imperfectly matched vaccine to protect cats and reduce viral shedding is encouraging for future efforts to protect both human and feline health.

The issue of cat-to-cat spread has triggered a great deal of concern. If the virus were to circulate more extensively in cats, it might adapt better to both humans and cats. H5N1 virus binds preferentially deep in the lungs in cats just as it does in humans¹⁴ and causes ARDS-like illness. The virus is shed rectally in both species.^{10,28} Because the behavior of the virus in cats and humans is so similar, it could theoretically be transmitted between these two species.

COMMUNICATING WITH THE PUBLIC, CALMING FEARS

Many pet owners became fearful following instances of cats infected with HPAI H5N1.³¹ Animal shelters in Germany reported a surge of phone calls and an increase in people seeking to relinquish their pets. Authorities feared a wave of cat-killings and clarified that it was illegal to shoot strays.³¹ The provision of clear advice from European authorities for regions that had had H5N1-positive birds helped to minimize public anxiety (**Box 1**). There have been no reports of cats infected with H5N1 in Europe since 2006, despite continued detection of the virus in European wild birds and poultry. The precautions recommended by the European Centers for Disease Control and Prevention appear to have been effective. There have been no reported human cases of H5N1 in Europe.

Dr Albert Osterhaus, a veterinarian researching influenza in cats, pointed out that H5N1-infected cats excrete 0.1% or less of the amount of virus that chickens do.³² Should HPAI H5N1 mutate, adapt fully to humans, and cause the next human pandemic, then contagion from infected humans, not cats, would be the main concern.

Box 1

European Centers for Disease Control and Prevention summarized recommendations for pet owners in locations where HPAI H5N1–positive birds have been found

1. Report stray, sick, or dead cats, or significant bird mortality, to local veterinary authorities.
2. Keep cats indoors, away from birds and their feces.
3. Do not let stray cats into your house.
4. If your pet carries a dead bird into the house, put on ordinary gloves and dispose of it as recommended by your Agricultural Department.
5. Consult your veterinarian if your cat displays breathing problems or nasal discharge.
6. Do not feed or water wild birds.
7. Keep dogs on a leash when outdoors to prevent them from eating birds.
8. Disinfect cages and equipment in contact with ill animals. Launder animal bedding with detergent.

Data from Influenza Team–European Center for Disease Control and Prevention. H5N1 infections in cats—public health implications. *Euro Surveill* 2006;11(15):2942.

Small animal veterinarians need to be ready to give clear advice to clients in the event of an HPAI H5N1 influenza outbreak in the United States, and should be acquainted with their local veterinary and health authorities in advance. Validated methods for commercial testing of cats for H5N1 are not currently available in the United States but may be developed should the virus arrive here.

INFLUENZA IN DOGS

Research in India in the 1970s demonstrated that dogs could be experimentally infected with human H3N2, shed virus from the pharynx, and seroconvert, but that they did not become ill.^{16,33} There were no reports of natural influenza outbreaks in dogs until the discovery of canine influenza H3N8 in Florida in 2004. Since then, there have also been isolated reports of dogs ill with HPAI H5N1 and an avian version of H3N2.

HIGHLY PATHOGENIC AVIAN INFLUENZA H5N1 IN DOGS

Dogs may become ill and die from ingestion of large doses of HPAI H5N1, but the virus does not appear to spread from dog-to-dog. In March 2006, Azerbaijani authorities reported that a stray dead dog tested positive for H5N1.³⁴ In Thailand, one dog ate an infected duck carcass and 5 days later developed a high fever, panting, and lethargy; it died the next day. Necropsy revealed bloody nasal discharge, severe pulmonary congestion with interstitial pneumonia, focal necrosis of the liver, and mild nephritis with tubular degeneration. Virus was isolated from the lungs, liver, kidneys, and urine.¹¹ An unpublished study found that 25% of dogs in one Thai village were seropositive for H5N1.³⁵ Thai researchers feared overreaction to the reports, and urged people not to abandon their dogs, but rather to prevent them from eating raw carcasses.³⁶

Results of experimental studies indicate that dogs are unlikely to play a role in transmission.²⁹ Beagles inoculated in the nose and trachea with HPAI viruses have shown no clinical signs, not even fever. Dogs inoculated oculo-nasopharyngeally showed transient fever and conjunctivitis. A few dogs shed viral RNA (but not live virus) from

the nose, and there was no evidence of rectal shedding. Most dogs seroconverted. On necropsy, there were no gross lesions, and viral RNA was not found in organs. Moreover, infected dogs did not transmit the virus to an uninfected cage mate, or to a cat.^{29,37} H5N1 viruses do not appear to be well adapted to dogs and outbreaks in dogs resulting from respiratory spread of current strains is unlikely.

To prevent canine infection, dog owners should be advised to keep dogs leashed when outdoors and prevent them from eating raw birds in areas HPAI H5N1 has been identified in birds.

AVIAN INFLUENZA H3N2 IN DOGS

The only report of H3N2 in dogs to date occurred in 2007, when it was diagnosed in several dogs at three veterinary hospitals and one kennel in South Korea.³⁸ Clinical signs in the dogs ranged from having only sneezing and nasal discharge to having fever, cough, nasal discharge, and death. The dogs were likely infected when fed raw pieces of infected poultry. All dogs at the kennel seroconverted, suggesting dog-to-dog transmission. Experimental nasal inoculation of this virus into dogs produced fever (average 104°F) and necrotizing tracheal, bronchial, and alveolar lesions, but no extrarespiratory lesions. All infected dogs shed virus nasally, but no virus was detected in the feces. It is uncertain whether this strain will cause significant future outbreaks.³⁸

CANINE INFLUENZA H3N8

Canine influenza H3N8 appears fully adapted to, and highly contagious among, dogs. It was first discovered in 2004, when it caused respiratory disease and occasional hemorrhagic pneumonia in racing greyhounds in Florida. The virus is genetically similar to the H3N8 equine influenza virus circulating in horses and is thought to have jumped from horses to dogs. This was the first time in history that an influenza virus was discovered circulating in dog populations.³⁹ It is the strain of influenza that a small animal veterinarian will most likely encounter in the United States.

Canine influenza is now found throughout the United States. The outbreak spread through racing dog facilities in 2004. By 2005 it was infecting dogs in the general population.⁴⁰ The Cornell Animal Health Diagnostic Center posts statistics by state on numbers of seropositive dogs identified through their laboratory, and also notes states where there have been virus isolations: <http://www.diaglab.vet.cornell.edu/issues/civ-stat.asp>.

Analysis of archived dog blood revealed the virus was in Florida greyhounds as early as 1999.⁴¹ An equine H3N8 virus also appears to have independently jumped from horses to foxhounds in the United Kingdom in 2002, an event not directly linked to the Florida outbreak. It is unknown in each case how the virus first jumped from horses to dogs, although the consumption of horse meat (likely raw or undercooked) by dogs has been proposed.⁴² There is evidence that the dog-adapted virus has retained its ability to infect horses. Horses experimentally inoculated with canine influenza showed clinical signs similar to, but much milder than, those seen in equine influenza. The horses also shed virus and had histopathological lesions typical of equine influenza (Dr Cynda Crawford, personal communication, 2008).⁴³

Clinical Signs

Clinical signs of canine influenza H3N8 can resemble those in other infectious tracheobronchitis infections.⁴⁴ Up to 20% of infected dogs are asymptomatic. The most common signs are a low-grade fever, a cough (either soft or harsh) that lasts 10 to

30 days, and nasal discharge that may be either serous or purulent. A subset of dogs develop high fever (104°F to 106°F) and pneumonia. The case fatality rate is between 1% and 5%.⁴⁴

Diagnosis

Canine influenza H3N8 should be suspected in outbreaks of respiratory disease among fully vaccinated dogs. Serology is one of the most useful diagnostic approaches. Infected dogs typically seroconvert by day 7 after the onset of clinical signs. In locations where canine influenza is not yet endemic, a single positive serum antibody titer combined with compatible symptoms is suggestive of the disease. A four-fold increase in titer 2 to 3 weeks later confirms it. Serum samples may be shipped on ice packs directly to the Cornell Animal Health Diagnostic Center for testing. For details, see: <http://diaglab.vet.cornell.edu/issues/civ.asp#samp>.

Polymerase chain reaction (PCR)-based testing for the RNA of the virus may be performed on nasal swabs. They are most accurate if samples are collected from infected dogs before clinical signs appear.⁴⁴ A negative result does not rule out infection. Viral shedding begins before the onset of clinical signs and tapers throughout the first 2 to 3 days of illness. Many dogs are likely to no longer be shedding by the time they present to a veterinarian. Lung and distal trachea can be tested by PCR in fatal cases. The Cornell Animal Health Diagnostic Center and the University of California at Davis, Lucy Whittier Molecular and Diagnostic Core Facility, offer PCR testing for the virus. For details, see the above link for Cornell or the following for UC Davis: <http://www.vetmed.ucdavis.edu/vme/taqmanservice/pdfs/DiagnosticPacket.pdf>.

Treatment and Control

Educating staff and dog owners about protocols for preventing contagion should be an integral part of treatment, whether the dog is hospitalized or treated at home.

The virus is easily transmitted through direct contact between dogs and through aerosolization (ie, coughing and sneezing). Humans may play a role in fomite transmission by touching an infected dog, then touching surfaces that other dog handlers come in contact with, such as doorknobs. Staff should change their clothes before going home to prevent bringing the virus home to their own dogs.⁴⁴

Suspect cases entering the clinic should be escorted to an examination room immediately. The floors, walls, and tables of the room should be thoroughly disinfected after use, with special attention to doorknobs and other objects that humans may touch. To prevent further transmission, dog owners should be counseled to isolate cases at home until recovery, to wash hands and food bowls and water bowls frequently with soap, and to change clothes before handling other dogs.

Hospitalized cases should be placed in isolation. At minimum, gloves and gown should be worn while handling the canine influenza patient. Hands should be washed with soap and water or disinfected with alcohol-based hand sanitizer after handling the dog. Shoes should be disinfected with an appropriately maintained disinfectant footbath upon exiting isolation. As with all influenza viruses, thorough cleaning and disinfection procedures inactivate the virus (see **Table 1**).⁴⁰

There is no recommended specific treatment for canine influenza,^{40,44} but broad-spectrum antibiotics are often used to control secondary bacterial infection and may reduce purulent nasal discharge. For pneumonia cases, transtracheal wash and culture may be needed to identify secondary bacterial infections.⁴⁴ Supportive treatment includes intravenous fluids, bronchodilators, coupage, and supplemental oxygen. Administering oseltamivir (Tamiflu, Roche Pharmaceuticals, Nutley, NJ) or other antivirals is not recommended for several reasons (**Box 2**).

Box 2**Reasons not to use oseltamivir for the treatment of Canine Influenza H3N8**

1. Timing of use. Oseltamivir acts by trapping viruses in infected cells. It must be administered very early, before the virus is widely spread in the body, to be of benefit. Most animals are presented to a veterinarian after this point.
2. Safety and efficacy. There have been no studies on the clinical use of oseltamivir in dogs.
3. Lack of need. Most dogs will recover from canine influenza with good supportive care.
4. Possible resistance. There is no system for detecting oseltamivir resistance in canine influenza should it appear.
5. Public health. Tamiflu overuse is being discouraged in both human and veterinary medicine. Should an influenza pandemic occur, antivirals will more likely be effective if they have not been overused.

Data from UC Davis Koret Shelter Medicine Program. Information—Canine influenza. May 2006. Available at: http://www.sheltermedicine.com/portal/is_canine_influenza_update.shtml#top3. Accessed July 30, 2008.

Prevention

As of yet there is no vaccine available to prevent canine influenza. The equine influenza vaccine should not be used on dogs.⁴⁴ It is not clear whether “antigenic drift” will be seen in the canine influenza virus as is seen in human seasonal influenza. This would necessitate regular updating of any vaccine to maintain its efficacy. Vaccinations for other canine respiratory diseases should be administered to maintain the health of the respiratory tract, and to help clarify when a canine influenza outbreak might be occurring.

Case Study: Canine Influenza Outbreak in Los Angeles County, 2007

A 5-month-old puppy of unknown origin presented at a veterinary clinic on July 10, 2007 with a fever of 104.5°F, nasal discharge, pneumonia, and vomiting. The puppy was placed in isolation and treated with intravenous fluids, antibiotics, a humidifier, and coupage. On July 13, the puppy was moved to an oxygen cage in the main treatment area, which was adjacent to the boarding section of the clinic. The puppy died soon after.

On July 17, clients began notifying the clinic of coughing in their dogs. The outbreak was reported by the owner of the practice to Los Angeles County Veterinary Public Health, which assisted with diagnostics. Approximately 43 dogs were identified in the outbreak, over a period of 17 days (**Fig. 2**). Most cases (72%) had been boarded at the clinic before onset, while others were outpatients or had contact with a boarded dog. Seven cases had thoracic radiographs performed, of which four had bronchopneumonia. The vast majority of cases were prescribed an oral antibiotic. Only the index case was hospitalized. Serology was performed on six coughing dogs and four healthy dogs that had been in the boarding area. Interestingly, all four healthy dogs were seronegative, despite likely exposure. Five of the six coughing dogs tested were seropositive. Two of these five had a convalescent serum sample drawn 3 weeks later, which revealed a fourfold rise in titer in both cases. Nasal and pharyngeal swabs of two other coughing dogs were negative by PCR.

The clinic closed their boarding section to new admissions for 20 days beginning on August 1st. Surgeries and outpatient appointments were not cancelled. Staff were assigned to work in either the back of the clinic (where hospitalized patients were

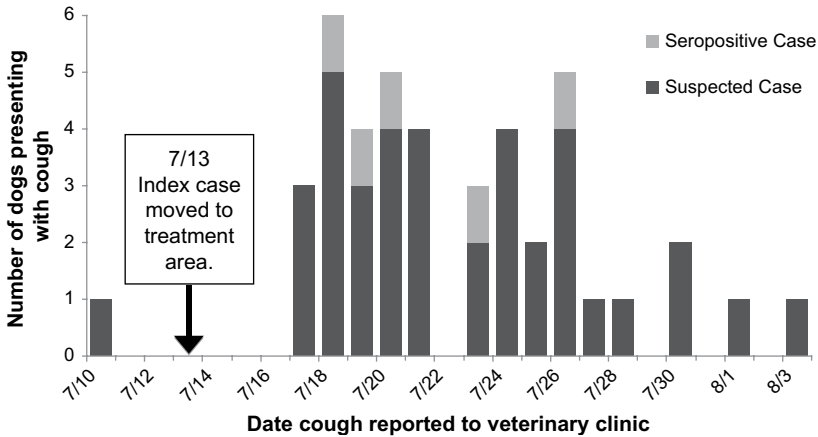


Fig. 2. Epidemic curve of canine influenza H3N8 at a veterinary clinic in Los Angeles County, 2007.

located), or the front, (where outpatients were seen), and were not allowed to cross between the two sections. The lobby was mopped with disinfectant six times daily. All fomites commonly touched by people (like phones, doorknobs, faucet handles, countertop pens, and door face plates) were disinfected four times daily during the outbreak and for 1 month after. The comprehensive approach this clinic took to infection control helped limit the outbreak to less than a month, despite the ongoing case-load in the practice. They reported almost no loss of clientele.

Canine Influenza H3N8 and Public Health

Canine influenza H3N8 is not considered to be zoonotic. However, because it has already exhibited a change in species specificity by jumping from horses to dogs, any human illness following contact with ill dogs should be noted and public health authorities alerted.

INFLUENZA IN CATS AND DOGS—GENERAL RECOMMENDATIONS

1. Know about the options for influenza testing. Serologic and PCR testing for canine influenza A H3N8 testing is commercially available. Tests for other influenza strains may possibly be performed by diagnostics labs through special arrangement.
2. Follow the news and the latest research. Two examples of free, online sources are:
 - a. Emerging Infectious Disease, a free journal published by the Centers for Disease Control and Prevention (CDC) can be found at: <http://www.cdc.gov/ncidod/eid>.
 - b. ProMed mail. Human, animal, and plant outbreak news from all over the world e-mailed daily, from the International Society for Infectious Diseases. <http://www.promedmail.org/>.
3. Know your local and state public health authorities.
4. Practice basic zoonosis control within your clinic. The National Association of State Public Health Veterinarians (NASPHV) has written a compendium of standards: <http://www.nasphv.org/Documents/VeterinaryPrecautions.pdf>.

5. Have a plan for handling outbreaks within your clinic. The Model Infection Control Plan for Veterinary Practices from the NASPHV can be found at: <http://www.nasphv.org/documentsCompendia.html>.
6. Educate staff and clients about influenza to minimize unreasonable fears. The most useful step you can take is to be knowledgeable and to have clear-cut recommendations ready.

SUMMARY

Since 2004, highly pathogenic avian influenza H5N1 has infected cats and dogs, primarily through ingestion of raw infected birds. The virus also spread from cat-to-cat in isolated cases. The virus apparently cannot be transmitted dog-to-dog. No cases of zoonotic transmission from a cat or dog have been reported. There is one report of illness in dogs due to avian H3N2 virus; these cases, reported from South Korea, were most likely acquired through consumption of raw infected poultry. Canine influenza H3N8 is the only influenza fully adapted to, and highly contagious among, dogs. It is the strain most veterinarians are likely to encounter in practice.

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