Canine Infectious Respiratory Disease

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KEYWORDS

- CIRD - Kennel cough - *Bordetella* - Parainfluenza virus - Pneumonia

KEY POINTS

- Multiple bacterial and viral pathogens can result in clinical signs of canine infectious respiratory disease complex (CIRDC), with coinfection by multiple pathogens commonly identified.
- A clinical diagnosis of CIRDC can typically be made with a history and physical examination; however, an etiologic diagnosis should be pursued in dogs with severe or prolonged clinical signs or in disease outbreak situations.
- Clinical signs of CIRDC are typically mild and self-limiting, resolving after approximately 1 week.
- Antimicrobial treatment is warranted in dogs suspected to have bacterial pneumonia and optimally should be directed based on bacterial culture and susceptibility results. The development of pneumonia should raise concern for underlying canine distemper virus infection or other underlying immunosuppressive disease.
- Vaccinations do not induce sterilizing immunity; therefore, more comprehensive prevention strategies are necessary, especially in group-housing situations.

Canine infectious respiratory disease complex (CIRDC), commonly referred to as "kennel cough," refers to a syndrome characterized by acute onset of contagious respiratory disease in dogs that can be caused by a wide range of etiologic agents.[1] These infections are of particular concern when large numbers of dogs are housed together, such as in animal shelters, boarding facilities, or day-care facilities.[2] Outbreaks associated with CIRDC are reported worldwide,[2–4] and dogs are more likely to develop clinical signs of CIRDC the longer they are in a group-housing environment.[5]

Historically, the most common pathogens associated with CIRDC have been canine parainfluenza virus (CPIV), canine adenovirus type 2 (CAV-2), and *Bordetella bronchiseptica* (**Table 1**).[6] In the past 2 decades, outbreaks of novel pathogens, including

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Table 1
Summary of the primary pathogens associated with canine infectious respiratory disease complex

Organism	Incubation Period (d)	Clinical Presentation	Vaccination
B bronchiseptica	2–6	Variable ranging from commensal to mild upper-respiratory signs to severe bronchopneumonia	Parenteral inactivated; attenuated live intranasal, mucosal vaccine
Mycoplasma cynos	3–10	Clinical syndrome not completely described. Isolated as a single agent from dogs with pneumonia	None available
Streptococcus equi subsp *zooepidemicus*	Probably days	Although has been associated with severe, rapidly progressing hemorrhagic pneumonia in overcrowded environments, can also cause mild upper-respiratory signs or subclinical infections	None available
Canine adenovirus 2	3–6	Mild upper-respiratory signs and harsh cough of 2-wk duration	Attenuated live parenteral and mucosal vaccines; cross-protection for CAV-1
Canine distemper virus	3–6	Respiratory signs in combination with lethargy, ocular discharge, fever; rapidly progressive and can include GI and central nervous system signs	Parenteral attenuated live and recombinant vaccines; core vaccines
Canine herpesvirus-1	6–10	Subclinical or mild respiratory signs in adults; moderate to severe ocular changes; severe disease in neonates	None available
Canine influenza virus	2–4	Variable, ranging from subclinical to severe clinical disease with secondary bacterial infection	Parenteral inactivated vaccines for H3N2, H3N8, or both
Canine parainfluenza virus	3–10	Highly contagious; upper-respiratory signs lasting up to 10 d	Attenuated live parenteral and mucosal vaccines
Canine respiratory coronavirus	Probably days	Variable; subclinical to mild upper-respiratory signs	None available; no cross-protection afforded by the CCoV vaccine

Data from Sykes JE. Canine Viral Respiratory Infections. In: Sykes JE, ed. Canine and Feline Infectious Diseases. Saint Louis: W.B. Saunders; 2014:170-181.

canine herpesvirus-1 (CHV-1) and canine influenza virus (CIV), have been reported, and advances in molecular identification methods have identified other potential pathogens that may be playing a role in this disease complex.[3,7–13]

The agents associated with CIRDC are transmitted via the aerosol route and frequently cause subclinical or mild upper-respiratory signs that last on average 1 to

2 weeks. More severe clinical signs can be noted in dogs that have coinfections with multiple CIRDC pathogens or secondary bacterial pneumonia.[14–17] Here, potential pathogens involved in CIRDC are discussed, including both viral and bacterial pathogens (see **Table 1**), preventative measures to limit the spread of these infections, available diagnostics, and treatment options.

BACTERIAL ORGANISMS ASSOCIATED WITH CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX
Bordetella bronchiseptica

B bronchiseptica is a worldwide cause of respiratory disease in dogs and also causes disease in other species, including cats, pigs, rabbits, and people. Different strains of this gram-negative coccobacilli likely vary in their host range and ability to cause disease,[18–20] and isolation of *B bronchiseptica* from apparently healthy dogs can occur.[21–24]

Transmission of *B bronchiseptica* is via the airborne route, and it is highly contagious. Once inhaled, the organisms adhere to the respiratory cilia by way of adhesion molecules (fimbrial adhesions, filamentous hemagglutinin, pertactin, and lipopolysaccharides).[6] The organisms can evade the host defenses using virulence factors, such as the outer capsule, or the O antigen, which protects the bacteria from phagocytosis and complement mediated attacks.[25] Other virulence factors have also been described, including type III secretion systems that allow for bacterial colonization,[26–28] adenylate cyclase toxin with anti-inflammatory and immune evasion properties,[29,30] and exotoxins that cause necrosis of epithelial cells.[31] Once colonization has been established, altered respiratory epithelial cell function leads to excessive mucus secretion and further impairment of the local innate immune defenses, predisposing the host to infection by opportunistic secondary pathogens. The incubation period of *B bronchiseptica* ranges from 2 to 10 days. Clinical signs can vary dramatically. Mild upper-respiratory tract disease can lead to mucopurulent nasal discharge, sneezing, and a cough. In more severe disease cases that involve the lower-respiratory tract, signs of systemic illness can be present, including lethargy, decreased appetite, fever, and a productive cough. The organism can be shed for at least 1 month, and in some cases, for several months.[32,33]

Mycoplasma cynos

Many *Mycoplasma* spp are commensal organisms that colonize the mucous membranes of the respiratory tract, and their role in canine infectious respiratory disease is not clear. *Mycoplasma* spp are fastidious organisms that lack a cell wall, are the smallest known free-living organisms, and can be isolated from the lungs or trachea of about 25% of healthy adult dogs.[34] *M cynos* is the only *Mycoplasma* spp significantly associated with respiratory disease in dogs,[35] most commonly pneumonia.[36,37] It is still unclear if *M cynos* is a primary or secondary pathogen in dogs, because it can be cultured from the lungs of dogs both with[34,35] and without[35,36] other identifiable infectious respiratory disease. Recently it was associated with lethal bronchopneumonia in a litter of golden retriever puppies[38] and a colony of laboratory beagles.[39]

Experimental infection of dogs, by either endobronchial infection or exposure to infected dogs, results in development of clinical pneumonia, destruction and loss of respiratory cilia, and influx of neutrophils and macrophages into the alveolus.[37,40] Younger dogs are more likely to be infected with this organism as compared with older dogs, and dogs become infected within the first 2 to 3 weeks upon entering an animal shelter.[35] *M cynos* can persist in the lung for up to 3 weeks following infection and be

transmitted via aerosols.[40] It is unknown how long the organism can persist in the environment. However, other *Mycoplasma* spp can survive for several weeks outside of the host; therefore, it should be assumed that *M cynos* can also persist in the environment.

Streptococcus equi Subspecies zooepidemicus

Streptococcus equi subsp *zooepidemicus*, a β-hemolytic, Lancefield group C streptococcus, has emerged as a cause of acute, severe bronchopneumonia in dogs. It is a commensal organism of the upper-respiratory tract of horses, but also can cause opportunistic infections, such as abscesses, and endometritis and can result in abortion.[41] Outbreaks of severe hemorrhagic pneumonia have been described in several populations of group-housed dogs in which *S equi* subsp *zooepidemicus* was identified as the causative agent.[42–44] A history of contact with horses has been identified in some, but not all infected dogs.[45] Rarely, *S equi* subsp *zooepidemicus* can be isolated from dogs without clinical signs.[42–44]

Dogs initially have mild clinical signs, including a cough and nasal discharge; however, their clinical signs can rapidly progress within 24 to 48 hours of onset, resulting in development of severe acute fibrinosuppurative, necrotizing, and hemorrhagic bronchopneumonia.[46] The pathogenesis is not fully elucidated; however, bacterial exotoxin genes have been identified in *S equi* subsp *zooepidemicus*, and the course of clinical disease in dogs is similar to that of streptococcal exotoxin-induced toxic shock syndrome in people, in which an overzealous host immune response induces significant pathologic condition.[46,47] Coinfection with other pathogens has been noted in some, but not all cases. In experimental models, coinfection of *S equi* subsp *zooepidemicus* with CIV (H3N8) resulted in severe clinical signs, whereas infection with *S equi* subsp *zooepidemicus* alone did not induce disease.[16]

Miscellaneous Bacteria

Other bacterial species have been isolated from dogs with CIRDC, including *Streptococcus canis*, *Pasteurella* spp, *Pseudomonas* spp, *Staphylococcus* spp, and coliforms, such as *Escherichia coli* and *Klebsiella pneumoniae*[48,49]; however, they are likely to represent secondary opportunistic infections as opposed to primary pathogens.

VIRUSES ASSOCIATED WITH CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX

Canine Adenovirus 2

Canine adenovirus-2, genus *Mastadenovirus* of the family Adenoviridae, is a nonenveloped double-stranded DNA virus that is a worldwide cause of infectious respiratory disease in dogs.[50] CAV-2 infects the nonciliated bronchiolar epithelial cells; epithelial cells of the nasal mucosa, pharynx, and tonsillar crypts; mucous cells in the trachea and bronchi; and type 2 alveolar epithelial cells.[51] Clinical signs are most often mild and consist of sneezing, nasal discharge, and a dry cough; however, more severe clinical signs are observed when coinfections with other CIRDC pathogens are present.[52] Viral shedding typically wanes 1 to 2 weeks after infection; however, the virus can survive in the environment for weeks to months.[53]

Canine Distemper Virus

Canine distemper virus (CDV) is in the genus *Morbillivirus* and family Paramyxoviridae. CDV is an enveloped RNA virus that can cause a myriad of clinical signs, primarily respiratory, with variable gastrointestinal (GI) and neurologic signs.[54] CDV is highly contagious and spread through aerosol secretions. Viral particles initially infect

monocytes within the lymphoid and tonsillar tissues of the upper-respiratory tract and then disseminate throughout the body via the lymphatics.[55] CDV also infects lymphocytes, in particular, CD4$^+$ T lymphocytes, and causes widespread lymphocyte destruction, leading to lymphopenia in the first few days after infection; then widespread dissemination to multiple organ systems occurs.[56]

Clinical signs associated with CDV infection can vary from subclinical infection to death. Dogs that present with respiratory signs consistent with CIRD that also have GI signs, ocular discharge, neurologic signs, and/or an unknown vaccination history should be considered at high risk of having distemper. CDV is shed from all bodily secretions starting at 5 days after infection, and shedding can continue for up to 4 months.[54] Because of the viral envelope, environmental survival is only several hours, and routine disinfectants will inactivate the virus.

Canine Herpesvirus

Canine herpesvirus-1 (Canid alphaherpesvirus-1) is an enveloped doubled-stranded DNA virus belonging to the family Herpesviridae. The major clinical syndrome associated with CHV-1 is reproductive failure in bitches and severe illness in neonates.[52,57] CHV-1 also has been isolated from lung and tracheal specimens from dogs with rhinitis and pharyngitis, and a high seroprevalence has been noted in dogs housed in kennel situations, implicating it as a CIRDC pathogen.[58–62] Experimental infections of dogs with CHV-1 can lead to rhinitis, tracheobronchitis, and ocular signs, including keratitis and conjunctivitis.[63]

CHV-1 infects the epithelial cells of the upper-respiratory mucosa, and the incubation period is 6 to 10 days.[53] CHV-1, like other herpesviruses, becomes latent in neurologic tissue, and reactivation of latent infections can occur after considerable stress or pharmacologic immunosuppression, with intermittent shedding in respiratory secretions throughout the life of the patient.[52]

Canine Influenza Virus

Influenza viruses have segmented, negative-sense RNA genomes and are enveloped. CIV belongs to the family Orthomyxoviridae and genus Alphainfluenzavirus (influenza A) and is further subtyped based on its hemagglutinin (H) and neuraminidase (N) genes. Influenza viruses infect a wide variety of animals, including birds and mammals, and significant genetic reassortment can occur when multiple subtypes infect a single host.

CIV is caused by 2 subtypes of influenza that have adapted to spread throughout the dog population. The first was documented in a population of racing greyhounds in Florida in 2004. The virus is closely related to equine influenza virus subtype H3N8.[64,65] Serosurveys of greyhounds indicate that the virus first emerged in the canine population between 1999 and 2000.[3] The virus then spread throughout the country, mostly being reported in kennels and shelters, and sporadically within the pet population.[10] The overall prevalence of this infection has been declining, and extinction in the US dog population has been suspected. In 2015, an outbreak of CIV was noted in Chicago, Illinois caused by an H3N2 influenza subtype that is genetically similar to a strain previously reported in South East Asia, suspected to be the result of a mutated avian influenza virus that has now adapted to the dog.[66] Since 2015, this strain of CIV has spread throughout the United States,[66] and reintroductions from Asia have resulted in the appearance of additional outbreaks.

CIV typically causes mild clinical respiratory signs, including lethargy, cough, nasal and ocular discharge, and occasionally more severe clinical signs associated with pneumonia. Clinical signs may be more severe with H3N2 than H3N8 infections. After experimental infection, H3N8 CIV induced necrotizing and hyperplastic tracheitis and

bronchitis in all dogs, and mild bronchiolitis and pneumonia in some dogs.[8] Many dogs also developed secondary bacterial pneumonia.[8] Viral shedding decreases dramatically 1 week after infection; however, H3N2 has been isolated from dogs up to 3 weeks after infection.[8,67]

Canine Parainfluenza Virus

CPIV is an enveloped single-stranded negative-sense RNA virus belonging to the family Paramyxoviridae and genus *Rubulavirus* and is closely related to simian virus 5. CPIV is a highly contagious cause of respiratory disease in dogs worldwide. Before introduction of vaccines, CPIV could be isolated from up to 50% of dogs with respiratory disease in a kennel situation.[68]

CPIV is spread via respiratory droplets, and infection occurs within the respiratory epithelial cells. Dogs can exhibit no clinical signs or mild clinical signs of a dry, harsh cough for 2 to 6 days with or without pyrexia and nasal discharge. Clinical signs appear more severe when coinfections occur. In experimental infections, clinical signs of respiratory disease are absent or very mild.[69] Histologic examination shows rhinitis with mixed inflammatory cell infiltration, tracheobronchitis, and bronchiolitis with loss of ciliated respiratory cells, and epithelial hyperplasia.[69] Viral shedding decreases 1 to 2 weeks after infection. The envelope of CPIV renders it susceptible to inactivation by most commercial disinfectants.

Canine Respiratory Coronavirus

Canine respiratory coronavirus (CRCoV) is a group 2a coronavirus in the family Coronaviridae and is an enveloped RNA virus. This virus was first described in a group of shelter dogs with respiratory disease in 2003 in the United Kingdom and has now been identified in dogs worldwide.[12] It can be found in dogs with and without clinical signs of respiratory disease.

Infection with CRCoV is associated with mild clinical signs, including nasal discharge, cough, and sneezing.[70] Experimental CRCoV infection of dogs resulted in mild respiratory disease, with virus infecting most respiratory tissue and respiratory associated lymphoid tissue, such as the tonsils and local lymph nodes. Infection of lymphoid tissue is associated with histopathologic changes that include damage to or loss of respiratory cilia.[53,70] Although respiratory tissue appears to be the primary site of viral replication, CRCoV has also been detected in the stool or intestines of dogs that presented with primary respiratory disease in the absence of GI signs and in 2 dogs with coinfections with other enteric viral pathogens in the absence of respiratory signs.[71] Viral shedding has been detected up to 10 days after infection.

Other Viruses Potentially Associated with Canine Infectious Respiratory Disease Complex

Molecular techniques have identified other viruses in dogs afflicted with CIRDC. Pantropic canine coronavirus, a group 1a coronavirus, is a strain of canine enteric coronavirus (CCoV) that was isolated from a group of dogs in Italy with severe respiratory disease. This strain of pantropic CCoV has been associated with severe clinical disease in puppies less than 3 months of age, as compared with those 6 months and older. Outbreaks have been reported throughout Europe, and coinfection with canine parvovirus has also been reported.[72]

Canine pneumovirus is a member of the family Paramyxoviridae. Infections have been reported in dogs from 2 animal shelters in the United States that had respiratory disease with no other causative agents discovered.[73] Other viruses that have been identified in dogs with respiratory disease include canine reovirus, canine bocavirus, and canine

hepacivirus.[13] The role of these viruses in the induction of respiratory disease is unclear, and further investigation is warranted to elucidate their individual roles in CIRDC.

DIAGNOSIS

Diagnosis of disease associated with CIRDC starts with collection of a history and thorough physical examination. Most causative agents of CIRDC have short incubation periods ranging from a few days up to 2 weeks. A history of exposure to other dogs is often present because most agents are transmitted by inhalation of respiratory droplets, although fomite transmission can take place with some pathogens. Most dogs will exhibit mild clinical signs of a paroxysmal, harsh cough; serous ocular discharge; nasal discharge; and/or sneezing. Typically, energy and appetite will remain normal. Dogs that are exhibiting pyrexia, lethargy, decreased appetite, or other more severe clinical signs likely have secondary bacterial infections.

Complete blood count, serum biochemistry, and urinalysis are usually normal or show evidence of inflammation, including mild to moderate neutrophilia, presence of band neutrophils, and lymphopenia. Thoracic radiographs are often normal or have mild abnormalities ranging from an interstitial to a bronchointerstitial pulmonary pattern. Dogs with more severe clinical signs or secondary bacterial infection can have an alveolar pulmonary pattern.

Findings on the history, physical examination, blood work, and radiographs can raise suspicion for disease caused by a pathogen within the CIRDC; however, an etiologic diagnosis cannot be elucidated without pathogen-specific diagnostic assays. For dogs that (1) have severe or rapidly progressive clinical signs, (2) have clinical signs that last for more than 7 to 10 days, or (3) exist in an outbreak setting, an attempt to obtain an etiologic diagnosis is recommended.

Bacterial cultures can be performed from specimens obtained from nasal swab, oropharyngeal swab, tracheal wash, or bronchoalveolar lavage. Cultures of the upper-respiratory tract should be interpreted with caution because they can yield growth of normal flora, and many CIRDC pathogens, such as *B bronchiseptica*, can be isolated from healthy animals. Isolation of the same pathogen from multiple animals within an outbreak setting would likely be more meaningful. Collection of a tracheal wash or bronchoalveolar lavage specimen is indicated in dogs with more severe clinical signs or evidence of pneumonia. Growth of a CIRDC pathogen such a *B bronchiseptica* or *M cynos* in a dog with consistent clinical signs can provide some support for their involvement; however, coinfection with other pathogens should still be considered, and negative test results do not rule out the presence of other pathogens (such as CDV). Growth of multiple bacterial species may represent opportunistic secondary infection or contamination. False negatives can occur with low bacterial burden or if antimicrobials have been administered.

Molecular diagnostic assays, such as those based on the polymerase chain reaction (PCR), have become widely available from commercial laboratories. Respiratory panels have been developed using real-time PCR that detect the nucleic acid from pathogens, including CPIV, CAV-2, CDV, CRCoV, CHV, CIV, *B bronchiseptica*, and *Mycoplasma* spp.[15,74–76] Swabs of the nasal cavity, oropharyngeal cavity, or specimens collected from the lower-respiratory tract can be submitted for PCR. False negative results can be common because of transient or low-level shedding or sample degradation during transit to the laboratory. Vaccination within the previous few weeks with live-attenuated vaccines can lead to false positive results.[77]

Virus isolation is increasingly being replaced with PCR assays, but is still offered by specialized virology laboratories (eg, the Animal Health Diagnostic Laboratory at

Cornell University, Ithaca, NY, USA). Swabs from the upper-respiratory tract or specimens collected from the lower-respiratory tract can be submitted. Virus isolation suffers from some of the same pitfalls as PCR with false negatives because of low or intermittent viral shedding or degradation of virus particles during transit. If possible, the laboratory should be contacted in advance of specimen collection to provide instruction on storage and transport conditions for collected specimens.

Serologic assays for measurement of antibodies to CIRDC viral pathogens are available; however, their clinical use is limited because antibodies occurring in response to vaccination cannot be distinguished from those produced owing to infection or to subclinical infection.

TREATMENT

Treatment of dogs with uncomplicated signs of CIRDC typically involves supportive care. Clinical signs in most dogs will resolve without treatment, so if clinical signs have been present for less than 1 week and the dog is bright with a good appetite, no specific therapy is recommended under current guidelines.[78] Expectorant medications, such as guaifenesin, have not been shown to be beneficial in reducing clinical signs of CIRDC and therefore are not recommended.[6] Use of a cough suppressant can be considered in order to provide relief for affected dogs and their owners, especially as the cough can persist for weeks and can occur throughout the night. Over-the-counter antitussive medications might be somewhat effective in dogs with mild clinical signs. Narcotic antitussives, such as hydrocodone, are more effective at reducing clinical signs; however, administration is contraindicated in animals with productive cough because there can be diminished clearance of bacteria when they are administered, predisposing to secondary infections.[79] There are currently no labeled antiviral therapies for dogs with CIRDC and no published recommendations for administration of commercially available influenza antivirals used in human medicine; therefore, antiviral therapy is not recommended.

Dogs that have clinical signs persisting beyond 1 week or any signs of bacterial pneumonia, such as pyrexia, lethargy, decreased appetite, or an alveolar pulmonary pattern on thoracic radiographs, should be treated with antimicrobials. Ideally, treatment of bacterial pathogens, *B bronchiseptica*, or opportunistic secondary pathogens should be guided by culture and susceptibility testing, because antimicrobial resistance is increasingly being recognized, especially among *Bordetella* isolates.

Empiric antimicrobial therapy should be based on the most likely agent to be present. Doxycycline is recommended for dogs with suspected *B bronchiseptica* or *M cynos* infection. If a bacterial infection is suspected to be secondary to an underlying viral infection, broad-spectrum antimicrobials are more appropriate. In severe cases, a parenteral antimicrobial combination that includes a fluoroquinolone and penicillin or clindamycin is recommended.[78]

Additional supportive care is advised and should be tailored to the patient's need. This additional supportive care can include hydration and/or nutritional support, oxygen therapy, nebulization, and coupage. Care should be taken to prevent further irritation to the trachea by avoiding a neck lead and removing barking triggers.

PREVENTION OF CANINE INFECTIOUS RESPIRATORY DISEASE

Vaccines are available for many common CIRDC pathogens (see **Table 1**): CAV-2, CDV, CPIV, CIV H3N8, and H3N2, and *B bronchiseptica.* With the exception of CDV, these vaccines do not produce sterilizing immunity but rather decrease the severity of clinical signs and magnitude of pathogen shedding.[5] The CDV vaccine is

a core vaccine that should be administered to all dogs. The remaining vaccinations are recommended in dogs that have risk of exposure.

Both mucosally administered (intranasal or transoral) and parenteral vaccines are available for CPIV, CAV-2, and *B bronchiseptica.* The route of vaccine delivery for these pathogens and its impact on the immune response have been debated in the literature. Intranasal or intraoral vaccination has been recommended to improve mucosal immune responses and permit rapid onset of protection in overcrowded environments, such as shelters. However, mucosal vaccination can sometimes result in vaccine-induced disease, and it can be difficult to know whether disease in a shelter environment is secondary to the vaccine or natural infection. Concern has also been raised that intranasal vaccine strains of *B bronchiseptica* might be capable of causing human disease in the immunosuppressed, although molecular evidence of this is lacking. Vaccination with intranasal vaccines is followed by the development of low titers of serum immunoglobulin G (IgG), whereas serum IgG responses are higher after parenteral vaccination.[80] A recent study suggested that intranasal vaccination may provide greater clinical protection against challenge than oral vaccination.[81] Additional studies are warranted to further assess the optimal vaccine type for dogs. Parenteral vaccines are available for reduction of clinical signs owing to CIVs, including individual H3N8 or H3N2 vaccines and combination (bivalent) vaccines.[82,83] No commercially available vaccines are available for reduction of clinical signs caused by CRCoV and CHV.

Although vaccination is a major prevention strategy, other precautions must be taken because immunization does not protect against all infections. In group-housing situations, precautionary measures should include an isolation period for dogs entering the population, rigorous daily monitoring for development of clinical signs within the group, and quarantine protocols for dogs with clinical signs associated with CIRDC. Care should be taken to prevent overcrowding and stress within the population. If an outbreak does occur, facilities should have an infectious disease protocol in place to limit exposure to other dogs in the facility, isolating ill animals from the population at large and applying proper disinfection protocols. An attempt should be made to determine the etiologic agent so targeted prevention and treatment protocols can be instituted.

SUMMARY

Contagious respiratory disease is a pervasive problem in group-housed dogs and pets that comingle with other dogs. Molecular techniques have led to discoveries of CIRDC pathogens that were not previously associated with the disease complex and highlighted the importance of coinfections in disease severity. With increased travel of dogs around the world, it is likely that novel pathogens will continue to emerge as CIV variants have in the past 2 decades. Because there are no specific therapies available for viral CIRDC pathogens, and available vaccines do not convey sterilizing immunity, prevention of infection is vital in group-housed dogs.

DISCLOSURE

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