Review Article

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Canine Distemper Virus: A persistent threat to domestic and wild carnivores

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ABSTRACT

Canine distemper virus (CDV) is a major causative agent of the disease, causing a contagious infection with high morbidity and mortality in dogs and various wild carnivores. The virus, belonging to the genus Morbillivirus (family Paramyxoviridae), exhibits extensive interspecies adaptation, potentially threatening the health of both domestic animals and protected wildlife populations. Distemper infection causes multisystem disorders, including those affecting the respiratory, gastrointestinal, and central nervous systems, and induces severe immunosuppression, increasing susceptibility to additional infections. Although vaccination has successfully reduced the incidence in some regions, CDV remains prevalent worldwide, particularly in areas with low vaccination coverage and large feral dog populations. Advances in molecular analysis have enabled the identification of multiple CDV genotypes through variations in the hemagglutinin (H) gene, which plays a key role in virus binding to the host and influences virulence. This ongoing genetic variation poses challenges to the effectiveness of existing vaccines. Furthermore, the spread of CDV to various wildlife species, such as wolves, civets, and lions, further complicates disease control from a One Health perspective. This review comprehensively outlined the virology, epidemiology, pathogenesis, diagnostic methods, and prevention and control measures for CDV. A more comprehensive understanding of the biology and distribution patterns of CDV is expected to support the development of sustainable animal health policies and strengthen conservation efforts for vulnerable species globally.

Introduction

Canine distemper virus (CDV) is a major pathogen infecting domestic dogs and various wild carnivores worldwide (Franzo *et al*., 2024). The disease is highly contagious and characterized by high morbidity and mortality, particularly in young or unvaccinated animals (Wilkes, 2022). CDV infection affects multiple organ systems, including the respiratory tract, gastrointestinal tract, and central nervous system, and induces severe immunosuppression, making animals more susceptible to opportunistic infections (Beineke *et al*., 2015). Although vaccination programs have successfully reduced the incidence of the disease in many regions, cases of distemper still occur periodically, particularly in areas with large feral dog populations and low vaccination coverage (Viana *et al*., 2015).

Globally, distemper cases have been reported on nearly every continent, with prevalence rates varying depending on environmental conditions, animal population structure, and the effectiveness of local control programs (Uhl *et al*., 2019). In several regions of Africa and Asia, including Indonesia, CDV infections are still found in domestic dogs and wildlife such as foxes, civets, and wolves (Wipf *et al*., 2025). Serological surveys in Indonesia have shown the presence of CDV antibodies in stray dogs, indicating that the virus is circulating endemically in these populations (Dorji *et al*., 2020). These findings underscore the importance of enhanced epidemiological monitoring and the implementation of integrated One Health-based control strategies to prevent interspecies transmission (Manandhar *et al*., 2023).

Taxonomically, CDV is classified in the genus Morbillivirus and the family Paramyxoviridae (Libbey and Fujinami, 2023). This genus also includes Measles virus (the cause of measles in humans) and Rinderpest virus (the agent of rinderpest in cattle), which share strong genetic and antigenic similarities (Oleaga *et al*., 2022). CDV is a single-stranded, negative-polarity RNA virus with a genome size of approximately 15.7 kb, encoding six major structural proteins: nucleocapsid (N), phosphoprotein (P), matrix (M), fusion protein (F), hemagglutinin (H), and large polymerase (L) (Tan *et al*., 2011). The H and F proteins play a crucial role in the binding and fusion of the virus with target cells, while nucleotide variations in the H gene are frequently utilized for phylogenetic analysis between strains and to identify emerging variants that could potentially affect the performance of vaccines used (Duque-Valencia *et al*., 2019a).

The impact of distemper is widespread, affecting not only the health of domestic animals but also seriously impacting wildlife conservation efforts (Liang *et al*., 2024). CDV infections have been detected in various species, including African lions, wild dogs (Lycaon pictus), and even red pandas, reflecting the virus's ability to adapt and transmit between species (Candela *et al*., 2025). This cross-species transmission poses a threat to the sustainability of endangered animal populations and underscores the importance of ongoing molecular surveillance to understand the dynamics of viral evolution in the natural environment (Rios-Usuga *et al*., 2025).

This review article aims to provide a comprehensive scientific description of canine distemper virus, covering aspects of virology, epidemiology, pathogenesis, diagnostic methods, and prevention and control approaches. By deepening our understanding of the biological characteristics and transmission patterns of CDV, the information presented is expected to aid in the formulation of more targeted animal health policies, raise public awareness of the importance of vaccination, and strengthen conservation efforts for wildlife susceptible to CDV infection.

Etiology

CDV is a negative-stranded, single-stranded RNA virus belonging to the genus Morbillivirus in the family Paramyxoviridae (Rendon-Marin *et al*., 2019). This virus is the primary cause of distemper in dogs and various wild carnivores, with its genome components and constituent proteins playing key roles in the mechanism of infection and pathogenesis (Rive-

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ra-Martínez *et al*., 2024).

Description of the CDV genome

CDV is classified in the genus Morbillivirus and the family Paramyxoviridae (Karki *et al*., 2022). This virus has a genetic material in the form of single-stranded, negative-polarity RNA with a length of approximately 15,690 nucleotides (Siering *et al*., 2024). The CDV genome is linearly arranged and encodes six main structural proteins: the nucleocapsid (N), phosphoprotein (P), matrix protein (M), fusion glycoprotein (F), hemagglutinin glycoprotein (H), and the large polymerase enzyme (L) (Beaty and Lee, 2016).

The N protein functions to form the nucleocapsid, which protects the genomic RNA and is a core component of the viral replication complex (Bringolf *et al*., 2017). The P protein acts as a polymerase cofactor, associating with the L protein to support viral RNA synthesis (Röthlisberger *et al*., 2010). The M protein plays a role in virion assembly and aids in the release of infectious particles from host cells (Dietzel *et al*., 2011). Meanwhile, the F and H proteins play crucial roles in the binding and fusion of the virus with the target cell membrane, which influences tissue tropism and the virulence of a strain (Zhai *et al*., 2025). The L protein is an RNA-dependent RNA polymerase enzyme responsible for the transcription and replication of the viral genome (Silin *et al*., 2007).

Morphologically, CDV has a pleomorphic shape, measuring approximately 150–300 nm, and is surrounded by a lipid membrane derived from its host cell (Swati *et al*., 2015). Although the CDV genome is relatively conservative compared to other Morbilliviruses, phylogenetic analysis indicates genetic diversity forming several lineages, including America-1, Asia-1, Asia-2, Europe, and Arctic-like (Li *et al*., 2018). These genetic differences may influence the antigenic characteristics of the virus and impact variations in vaccine effectiveness across geographic regions (Van *et al*., 2023).

Important proteins

The CDV genome encodes six key proteins that play a critical role in the virus's replication, assembly, and infectious capacity: the nucleoprotein (N), phosphoprotein (P), matrix protein (M), fusion glycoprotein (F), hemagglutinin glycoprotein (H), and large polymerase (L) (Beaty and Lee, 2016).

The N protein functions to package and protect genomic RNA, forming a stable nucleocapsid, and also contributes to the regulation of transcription and replication (da Costa *et al*., 2021). Phosphoprotein P acts as a cofactor for the polymerase enzyme and associates with protein L to produce an active replication complex (Sugai *et al*., 2009). Furthermore, protein P plays a role in suppressing the host immune response, including inhibiting interferon activity (Tian *et al*., 2024).

The M protein (matrix protein) functions as a link between the nucleocapsid and the viral membrane, coordinating virion assembly and assisting in the release of new viral particles from the host cell surface (Bringolf *et al*., 2017). The F protein (fusion glycoprotein) mediates the fusion process between the viral envelope and the target cell membrane, allowing the nucleocapsid to enter the cytoplasm (Plattet *et al*., 2005). Activation of the F protein requires proteolytic cleavage by host enzymes, which also determines the infectivity and virulence of a strain (Dietzel *et al*., 2011).

The H protein (hemagglutinin glycoprotein) plays a role in recognizing and binding to host cell receptors, such as signaling lymphocyte activation molecule (SLAM/CD150) and nectin-4 (Otsuki *et al*., 2013). The specific binding between the H protein and these receptors contributes to tissue tropism and the virus's ability to infect various carnivorous species (Macías-González *et al*., 2025).

Meanwhile, the L protein (large polymerase protein) is a key enzymatic component of the RNA-dependent RNA polymerase (RdRp) complex, which carries out transcription and replication of the viral RNA genome (Benetka *et al*., 2011). This protein also contains methyltransferase and capping domains which are required to modify the viral mRNA so that it can be recognized and translated by the host cell ribosomes (Cheng *et al*., 2024).

Genetic variation between strains

Molecular analysis of the genes encoding the hemagglutinin (H) and nucleoprotein (N) glycoproteins shows that CDV exhibits high levels of genetic variation among isolates originating from various regions of the world (Ariyama *et al*., 2024). Phylogenetic studies have grouped CDV into several major lineages generally associated with their region of origin, such as America-1, America-2, Europe, Asia-1, Asia-2, Arctic-like, Africa, and South America-1 (Anis *et al*., 2018).

Differences in nucleotide and amino acid sequences in the H gene are crucial in determining these lineages, as this gene is the most variable and plays a direct role in the virus's interaction with host cell receptors and triggering a protective immune response (Tao *et al*., 2020). Mutations in the antigenic region or receptor-binding site of the H protein can affect the virus's ability to adapt to different host species and potentially reduce the effectiveness of vaccines developed based on conventional strains (Bi *et al*., 2023).

Several studies have shown that the Asia-1 and Asia-2 lineages are the most prevalent CDV variants circulating in dog populations in East and Southeast Asia, including Indonesia (Bhatt *et al*., 2019; Liu *et al*., 2025; Van *et al*., 2025). Meanwhile, the America-1 lineage is generally associated with vaccine strains widely used in various countries, and the Arctic-like lineage has been reported in cross-species infections involving dogs, foxes, and wildlife in Northern Europe (Alfano *et al*., 2025; Mira *et al*., 2018).

Genetic differences between CDV lineages not only impact antigenic variation but can also influence virulence and tissue tropism (Martella *et al*., 2006). Therefore, continuous molecular monitoring and H gene sequence analysis are crucial for identifying emerging new strains and assessing the suitability of available vaccines against field viruses in endemic areas (Riley and Wilkes, 2015).

Taxonomic relationship with Measles virus and Rinderpest virus

CDV belongs to the genus Morbillivirus in the family Paramyxoviridae and the order Mononegavirales (Budaszewski *et al*., 2016). This genus also includes several important mammalian pathogens, such as Measles virus (MV) in humans, Rinderpest virus (RPV) in cattle, Peste-des-petits-ruminants virus (PPRV) in sheep and goats, and Phocine distemper virus (PDV) in marine mammals (Libbey and Fujinami, 2023).

The taxonomic closeness between CDV, MV, and RPV is based on similarities in genome structure, gene arrangement, and nucleotide and amino acid sequence homology in several key structural proteins, particularly those encoding the hemagglutinin (H) protein, phosphoprotein (P), and large polymerase (L) (Oğuzoğlu and Koç, 2025). The sequence similarity between CDV and MV can even exceed 50%, indicating a very close evolutionary relationship between the two viruses (Duque-Valencia *et al*., 2019b).

Phylogenetic analysis indicates that the three viruses likely originated from a common Morbillivirus ancestor, then diversified as they adapted to different hosts (Wang *et al*., 2023). Host-switching events are thought to have contributed significantly to the evolution of this genus, as evidenced by the ability of CDV to infect a wide variety of carnivore species, including domestic dogs, ferrets, raccoons, and several large felid species (Wipf *et al*., 2025).

The genetic closeness between CDV and MV also contributes to antigenic cross-reactivity, which has been exploited in heterologous vaccine development research (Budaszewski *et al*., 2016). Furthermore, RPV—now declared extinct in the wild—has antigenic and molecular characteristics very similar to CDV, making it frequently used as a reference virus in

studies of Morbillivirus evolution and pathogenesis (Karki *et al*., 2022). As shown in Figure 1, this illustration depicts the simplified structural organization of the Canine Distemper Virus (CDV), highlighting its major components along with a concise linear representation of the viral genome.

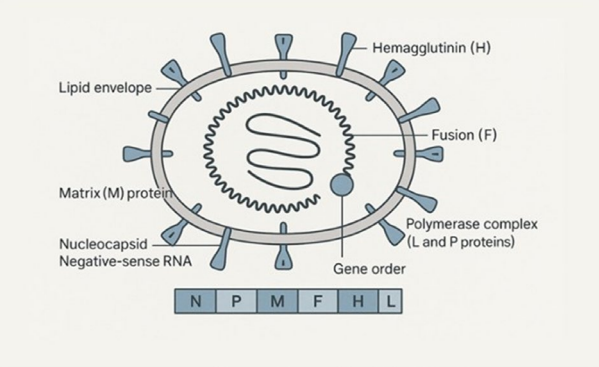

Figure 1. Simplified Structure of the CDV.

Host range

CDV has a very broad host spectrum and can infect various species within the order Carnivora (Nikolin *et al*., 2012). In addition to the domestic dog (Canis lupus familiaris) as its primary host, this virus is also found in several other families, such as Mustelidae (e.g., ferrets, minks, and otters), Procyonidae (raccoons), Felidae (lions, leopards, and other big cats), and Hyaenidae (hyenas) (Beineke *et al*., 2015). Infections in marine mammals, including seals from the family Phocidae, further demonstrate the virus's high cross-species adaptability (Jo *et al*., 2018).

The ability of CDV to cause cross-species infection is closely related to genetic variation in the gene encoding the hemagglutinin (H) glycoprotein, which determines the virus's affinity for host cell receptors, particularly Signaling Lymphocyte Activation Molecule/Cluster of Differentiation 150 (SLAM/CD150) and nectin-4 (Khosravi *et al*., 2015). Changes in these receptor-binding regions allow the virus to adapt to a wide range of carnivorous species, including wildlife not previously considered natural hosts (Huang *et al*., 2024).

CDV infections in wild animals such as raccoons and ferrets play a crucial role in maintaining virus circulation in the ecosystem, despite high vaccination rates among domestic dog populations (Lednicky *et al*., 2004). From a disease ecology perspective, these wildlife groups can act as potential reservoirs, contributing to the re-emergence of distemper in dogs and other susceptible species (Kapil and Yeary, 2011).

Evolutionarily, CDV's ability to adapt to multiple hosts reflects the virus's biological flexibility and underscores the need for a One Health approach to control efforts (Nikolin *et al*., 2017). Epidemiological monitoring of domestic and wildlife animals is crucial for understanding natural transmission patterns and preventing cross-species outbreaks that could threaten the sustainability of endemic carnivore populations (Reja *et al*., 2022).

Epidemiology

CDV has a near-global distribution and has been reported to infect both domestic dogs and wild carnivores on multiple continents (Beineke *et al*., 2015). Historically, distemper first appeared in Europe in the 18th century before rapidly spreading to other regions through trade and the movement of domestic animals (Stokholm *et al*., 2021). Cases of distemper in dogs were recorded as early as 1760, and a more detailed scientific description was provided by Edward Jenner in 1809 (Quintero-Gil *et al*., 2019). The causative agent, Canine morbillivirus, was isolated in 1905 in France. To date, CDV remains a major pathogen in dogs in many countries, particularly in areas with low vaccination coverage and suboptimal animal health surveillance systems (Uhl *et al*., 2019). Table 1 presents a summary of CDV epidemiology by region, endemic status, susceptible species, and dominant genotype.

In North America and Europe, widespread vaccination has drastically reduced the incidence of distemper, although infections continue to occur occasionally in wild animals such as foxes, raccoons, and wolves (Bergmann *et al*., 2021; Kapil and Yeary, 2011). Meanwhile, in Asia, Africa, and South America, CDV remains endemic, with incidence rates varying widely across countries (Fischer *et al*., 2016; Mousafarkhani *et al*., 2023; van de Bildt *et al*., 2002).

Several studies in Asia have shown that the Asia-1 and Asia-2 lineages are the most frequently detected CDV types, with distinct genetic characteristics from the standard America-1-based vaccine strain (Giacinti *et al*., 2022; Riley and Wilkes, 2015). In Japan and China, CDV outbreaks have been reported to cause high mortality in domestic dogs and wildlife, including endangered species such as Panthera pardus japonensis (Feng *et al*., 2016; Suzuki *et al*., 2015). In Indonesia, distemper cases continue to occur sporadically in dogs and wildlife in conservation areas, indicating that the virus continues to circulate in tropical environments (Mulia *et al*., 2021).

Geographically, CDV movement is influenced by climatic conditions, animal migration, and host population density. Urban areas, where pet dogs, wildlife, and humans interact more intensely, can become hotspots for virus transmission (Wilson *et al*., 2025). Therefore, mapping the spatial and temporal spread of CDV through molecular surveillance is a crucial step in understanding transmission patterns and developing appropriate control strategies for each endemic area (Candela *et al*., 2025).

Pathogenesis

CDV infection typically begins in the upper respiratory tract when an animal inhales virus-containing aerosol droplets from an infected individual (Rendon-Marin *et al*., 2019). Viral particles then bind to SLAM/CD150

Table 1. Geographic distribution and epidemiological characteristics of CDV in various regions of the world.

Region	Endemicity status	Infected species	Dominant lineage/ Genotype	Main epidemiological information	Reference
Europe	Low – Sporadic	Domestic dogs, foxes, and wolves	*Europe lineage*	The first cases were recorded in the 18th century and a significant decline was due to mass vaccination	(Bergmann *et al*., 2021)
North America	Low – Sporadic	Raccoons, coyotes and pet dogs	*America-1 lineage*	Prevalence is low, but wild cases still occur in wild carnivore populations	(Kapil and Yeary, 2011)
East Asia (Japan and China)	Medium – High	Dog, civet, dan *Panthera pardus japonensis*	*Asia-1* and *Asia-2*	An outbreak with high mortality and significant genetic differences with the vaccine strain	(Feng *et al*., 2016; Suzuki *et al*., 2015)
Southeast Asia (including Indonesia)	Moderate – Endemic	Pet dogs and wildlife conservation	*Asia-1 lineage*	Sporadic cases are still being reported, vaccination is not yet widespread, and the virus continues to circulate	(Mulia *et al*., 2021)
Africa	High – Endemic	Dogs, lions and hyenas	*Africa lineage*	High cross-species transmission and threat to wildlife populations	(van de Bildt *et al*., 2002)
South America	High – Endemic	Dogs, raccoons, and foxes	*South America lineage*	High prevalence in areas with limited vaccination and high animal mobility	(Fischer *et al*., 2016)

receptors on mucosal immune cells, primarily T lymphocytes, macrophages, and dendritic cells (Zhao and Ren, 2022). After entering the host, the virus initially replicates in local lymphoid tissues, such as the tonsils and cervical lymph nodes (Burrell *et al*., 2020).

During the first viremic phase, the virus spreads through lymphoid tissues to other organs, including the spleen, thymus, and bone marrow (Lempp *et al*., 2014). This phase is accompanied by immunosuppression, indicated by a decrease in lymphocyte counts, weakened phagocytic activity, and impaired T and B cell function (Carvalho *et al*., 2012). This CDV-induced immunosuppression is a major factor in the increased susceptibility of infected animals to secondary infections, both bacterial and opportunistic viruses (Chludzinski *et al*., 2023).

During the secondary viremia phase, the virus begins to spread more widely to epithelial tissues and the central nervous system (CNS) (Rudd *et al*., 2006). CDV binds to the nectin-4 receptor on epithelial cells, allowing the virus to replicate in the respiratory tract, gastrointestinal tract, and skin (Sawatsky *et al*., 2012). In parallel, the virus enters the nervous system through the migration of infected immune cells across the blood-brain barrier or through neuronal spread (Geiselhardt *et al*., 2022). Infection of neurons and glial cells results in myelin damage, gliosis, and the formation of intranuclear and cytoplasmic inclusions—characteristic histopathological lesions of distemper involving the brain (Klemens *et al*., 2019).

Two major surface proteins, hemagglutinin (H) and fusion (F), play a critical role in determining the virulence and tissue tropism of CDV (von Messling *et al*., 2001). The H protein regulates the virus's recognition of specific host receptors, thus influencing the range of species it can infect (Chen *et al*., 2023). The F protein, on the other hand, facilitates the fusion of the viral envelope with the target cell membrane, enabling nucleocapsid entry into the cell and supporting cell-to-cell spread through syncytia formation (Rendon-Marin *et al*., 2019). Genetic changes in these two proteins are often associated with increased malignancy, shifts in target tissues, and the virus's ability to penetrate the nervous system (Vandevelde and Zurbriggen, 2005).

Immune response

CDV infection activates both the innate and adaptive immune responses, which interact to determine the severity of the disease and the chances of recovery in infected animals (Kapil and Yeary, 2011). Early on, the innate immune system detects the presence of the virus through pattern recognition receptors such as Toll-like receptors (TLRs) and RIG-I-like receptors (RLRs), which stimulate the production of type I interferon (IFN-α/β) (Klotz and Gerhauser, 2019). However, CDV can attenuate this interferon activation through the function of the V and C proteins derived from the phosphoprotein (P) gene, thereby diminishing the initial antiviral response and allowing the virus to replicate more efficiently in lymphoid tissues (Siering *et al*., 2021).

The adaptive immune response then plays a key role in clearing the virus and establishing long-term immunity (Carvalho *et al*., 2012). Neutralizing antibodies—particularly those directed against the hemagglutinin (H) and fusion (F) glycoproteins—are key components in protecting against subsequent infections (Xiao *et al*., 2025). These antibodies inhibit the binding and fusion of the virus to the target cell membrane, thus preventing the spread of infection throughout the body (Kalbermatter *et al*., 2019). Clinically, high levels of neutralizing antibodies are associated with increased resistance to disease and are an important parameter in assessing vaccination success (Bergmann *et al*., 2021).

In addition to the antibody response, cellular immune mechanisms involving T lymphocytes play a crucial role in combating CDV infection (Song *et al*., 2019). Cytotoxic T cells ($CD8^+$) are responsible for killing infected body cells, while helper T cells ($CD4^+$) assist in B cell maturation and enhance antibody production (Karabulut and Oğuzoğlu, 2025). Activation of both types of T cells is crucial for eliminating ongoing infections and preventing the virus from persisting in nerve tissue (Qeska *et al*., 2014).

A well-functioning immune response is usually able to clear the virus from epithelial and systemic tissues within 2–3 weeks of infection (Muñoz-Hernández *et al*., 2023). Conversely, if the adaptive immune response is not optimal, the virus can persist in the central nervous system and trigger chronic conditions such as progressive encephalitis or old-dog encephalitis (Espinoza *et al*., 2023).

Pathology

CDV infection causes pathological changes in various organ systems, reflecting the virus's ability to invade lymphoid, epithelial, and nervous tissues (Beineke *et al*., 2015). The type and severity of lesions are strongly influenced by the stage of infection, the host's immunological status, and the virulence of the strain (Alfano *et al*., 2022). In the early phase, the virus has a strong tendency to infect lymphoid cells, particularly T and B lymphocytes in the tonsils, thymus, spleen, and lymph nodes (Carvalho *et al*., 2012). Intense viral replication in these tissues leads to a generalized decrease in lymphoid cell numbers and follicular necrosis, which are major factors in the progressive immunosuppression of infected animals (Schobesberger *et al*., 2005).

In the respiratory tract, CDV infection triggers interstitial bronchopneumonia characterized by thickening of the alveolar septa, mononuclear cell infiltration, and damage to the bronchiolar epithelium (Volkan *et al*., 2022). Histopathologically, eosinophilic intranuclear and intraplasmic inclusions are often found in respiratory epithelial cells, a typical indicator of CDV infection (Shin *et al*., 2022). Meanwhile, in the digestive system, lesions generally appear in the form of necrotic enteritis accompanied by villous atrophy and damage to the intestinal glandular epithelium, conditions that often worsen clinical symptoms by facilitating secondary bacterial infections (Rendon-Marin *et al*., 2019).

CNS involvement is a key feature of chronic CDV infection (Freire *et al*., 2025). The virus can cross the blood-brain barrier by infecting endothelial cells or through the migration of infected leukocytes, then spread to neurons and glial cells (Rudd *et al*., 2006). Histologically, multifocal demyelination, gliosis, perivascular cuffing, and neuronal degeneration are seen (Feijóo *et al*., 2021). These changes are associated with the appearance of neurological symptoms such as tremors, seizures, ataxia, and limb weakness (Suwanpakdee *et al*., 2025). In persistent cases, this condition can progress to non-suppurative encephalitis or old dog encephalitis (ODE), which is progressive and ultimately fatal (Lempp *et al*., 2014).

CDV can also cause epithelial damage in various non-neural organs, including the skin, conjunctiva, and genitourinary tract (Rendon-Marin *et al*., 2019). In the skin, a common change is hyperkeratosis of the nose and footpads (hard pad disease), which occurs due to increased epithelial proliferation and keratin accumulation (Engelhardt *et al*., 2005). In the liver and kidneys, damage can include vacuolar degeneration of hepatocytes and renal tubular necrosis, reflecting the systemic cytopathic effects of the virus (Silva *et al*., 2022). The progression of canine distemper virus infection, from initial respiratory entry to systemic dissemination and neural involvement, is illustrated in Figure 2.

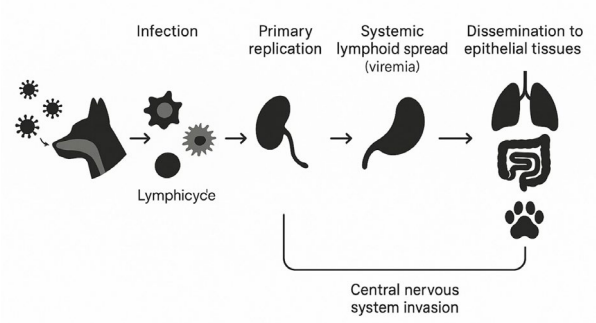

Figure 2. Schematic overview of CDV pathogenesis in dogs.

Clinical manifestations

The clinical manifestations of CDV infection vary widely and are influenced by factors such as age, immune status, and the virulence of the strain (Geetha and Selvaraju, 2019). Early in the disease, animals typically experience generalized fever (hyperthermia), which occurs as a systemic response to viral replication in the lymphoid tissue (Carvalho *et al*., 2012). This fever is often biphasic, with the first fever occurring during primary viremia and a second peak occurring as the virus spreads to the epithelial tissue and nervous system (Karki *et al*., 2022).

As the infection progresses, respiratory signs may develop, including serous to mucopurulent nasal discharge, conjunctivitis, and cough, indicating damage to the respiratory epithelium due to degeneration and necrosis of bronchiolar cells (Headley *et al*., 2018). If the gastrointestinal tract is involved, animals may experience vomiting and diarrhea, accompanied by severe dehydration resulting from intestinal epithelial damage and opportunistic bacterial infections (Lanszki *et al*., 2021).

Central nervous system involvement is a key characteristic of severe or long-lasting cases of distemper (Nägler *et al*., 2025). Common neurological manifestations include myoclonus (involuntary rhythmic muscle movements), tonic-clonic seizures, ataxia, paresis, and even paralysis (Freire *et al*., 2025). Myoclonus is often considered a hallmark of CDV infection and can persist even after the virus is no longer detectable in other tissues (Sarchahi *et al*., 2025). These neurological abnormalities result from demyelination and non-suppurative encephalitis that occur when the virus infects neurons and glial cells (Carvalho *et al*., 2012).

In general, the disease can range from acute to chronic (Liu *et al*., 2025). The acute form is usually fatal within a few weeks due to respiratory complications or severe gastroenteritis, while the chronic form can develop into progressive encephalitis with persistent neurological symptoms (Alfieri *et al*., 2006).

Diagnosis

Establishing a diagnosis of CDV requires a comprehensive approach that includes clinical assessment, pathological examination, and confirmation through laboratory tests based on immunological and molecular methods (Peserico) (Peserico *et al*., 2019). This integrated approach is essential because CDV infection often causes diverse symptoms and can mimic other infectious diseases in dogs (McDermott *et al*., 2023). Table 2 summarizes the various diagnostic techniques used to detect CDV, ranging from clinical methods and histopathology to immunological and molecular examinations.

Clinically, the initial diagnosis is generally based on the observation of typical symptoms such as biphasic fever, mucopurulent discharge from the eyes and nose, cough, and neurological manifestations such as myoclonus and seizures (Freire *et al*., 2025). However, these signs are nonspecific, so laboratory testing remains an essential step in confirming the cause of the disease (da Costa *et al*., 2019).

Histopathologically, CDV infection is characterized by the presence of intranuclear and intraplasmic eosinophilic inclusions in epithelial cells of the respiratory, digestive, and central nervous systems (Stancu *et al*., 2023). Predominant tissue changes include demyelination, perivascular cuffing, gliosis, and generalized lymphodepletion in lymphoid organs (Lempp *et al*., 2014). Immunohistochemical examination can be used to specifically identify viral antigens in tissues, providing confirmation of diagnosis with a high degree of sensitivity (Viana *et al*., 2020).

The direct immunofluorescence test (DFAT) is a conventional technique still widely used to detect CDV antigens in conjunctival, nasal epithelial, and lung tissue (Athanasiou *et al*., 2018). This approach is capable of rapid and accurate detection, especially during the acute phase of infection when antigen levels are still high (Kapil and Neel, 2015).

Over the past twenty years, nucleic acid-based diagnostic techniques have emerged as the primary method for detecting CDV infection (Uhl *et al*., 2019). Reverse transcription polymerase chain reaction (RT-PCR) allows for highly sensitive identification of viral RNA, even in antigenically depleted samples (Elia *et al*., 2006). Genes frequently targeted for amplification include the nucleocapsid (N), phosphoprotein (P), and hemagglutinin (H) genes (Glišić *et al*., 2024). Quantitative RT-PCR (qRT-PCR) can also be used to determine viral load, helping to evaluate the severity of infection and the effectiveness of supportive treatment (Sehata *et al*., 2015).

For molecular epidemiology purposes, H gene sequence analysis is often performed to identify lineages and genetic diversity between strains

Table 2. CDV diagnostic approach based on method, detection target, and analysis objective.

Diagnostic methods	Type of examination	Target detection / Specific marker	Advantages and applications	Diagnostic purpose / Additional information
Clinical evaluation	Physical examination and observation of symptoms	Typical symptoms: biphasic fever, mucopurulent discharge, cough, myoclonus, and seizures	Quick and easy to do in the field	Initial diagnosis and laboratory confirmation is needed because the symptoms are not pathognomonic
Histopathology	Tissue examination (lungs, brain, and lymphoid)	Eosinophilic intranuclear and intraplasmic inclusions, demyelination, and gliosis	Describes specific lesions of CDV infection	Helps identify typical lesions and differential diagnosis with other infections
Immunohistochemistry (IHC)	Detection of viral antigens in tissue	CDV antigen protein in epithelial or nerve tissue	High sensitivity and immediate visual results	Confirmation of CDV infection through detection of specific antigens
Direct Fluorescent Antibody Test (DFAT)	Direct immunofluorescence	CDV antigen in the conjunctiva, nasal epithelium, or lungs	Fast and accurate results in the acute phase of infection	Rapid confirmatory diagnosis in acute cases
Reverse Transcription Polymerase Chain Reaction (RT-PCR)	Molecular detection of viral RNA	N, P, or H gene	High sensitivity and can detect viruses even when antigens decrease	The gold standard for molecular diagnosis of CDV
Quantitative RT-PCR (qRT-PCR)	Quantification of viral nucleic acids	Gene N or H	Measuring viral load and supporting therapy monitoring	Assessment of infection levels and effectiveness of treatment
H gene sequencing	Advanced molecular analysis	Hemagglutinin (H) gene	Identification of lineage and genetic variation	Molecular epidemiology and vaccine strain suitability
Enzyme-Linked Immunosorbent Assay (ELISA)	Serological examination	CDV antibodies or antigens	Efficient and suitable for population surveillance	Seroepidemiological studies and post-vaccination antibody monitoring
Virus Neutralization Test (VNT)	Seroneutralization test	Neutralizing antibodies against CDV	High specificity and gold standard for protective immunity	Evaluation of vaccine effectiveness and individual immune status

(Ariyama *et al*., 2024). Phylogenetic studies of this gene play a crucial role in monitoring the dynamics of viral evolution and assessing the suitability of circulating strains to vaccines (Li *et al*., 2018). Serological tests, such as enzyme-linked immunosorbent assay (ELISA) and virus neutralization test (VNT), are used to detect CDV-specific antibodies or antigens (Lysholm *et al*., 2025). VNT is considered the standard method for assessing post-vaccination protection, while ELISA is more practical for population surveillance and seroepidemiological studies (Kim *et al*., 2018). Figure 3 provides a concise visual summary of the major diagnostic methods used to detect Canine Distemper Virus, highlighting clinical signs, antigen detection assays, molecular techniques, and serological tests.

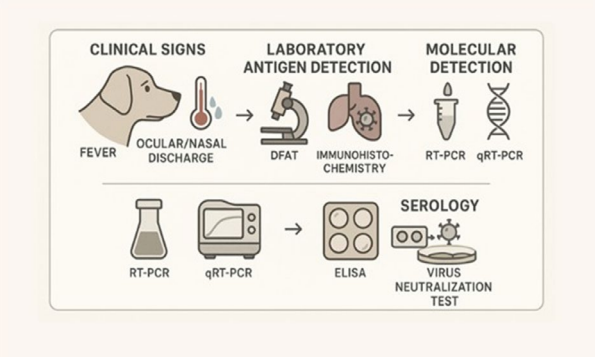

Figure 3. Diagnostic approaches for CDV.

Differential diagnosis

A careful differential diagnosis of CDV infection is essential because the clinical presentation often resembles various viral diseases and other neurological disorders in dogs (Rivera-Martínez *et al*., 2024). Accurate diagnosis is crucial to avoid misdiagnosis, especially in the early stages when symptoms are still general and nonspecific (Sarchahi *et al*., 2022).

In the respiratory phase, CDV can cause signs similar to those of canine adenovirus type 2 (CAV-2) or canine parainfluenza virus (CPIV), both of which cause tracheobronchitis and pneumonia (Day *et al*., 2020). However, CDV is generally accompanied by more pronounced systemic and neurological symptoms and a longer disease course (Freire *et al*., 2025).

In gastrointestinal disorders, canine parvovirus (CPV-2) is the pathogen most frequently causing similar symptoms, such as severe vomiting and diarrhea (Truyen *et al*., 2024). Important differences can be seen through hematological and histopathological examinations: CPV-2 typically presents with severe panleukopenia and intestinal crypt damage, while CDV more frequently causes generalized lymphodepletion and the formation of eosinophilic inclusions in various epithelial tissues (Carvalho *et al*., 2012).

The neurological manifestations of CDV can also mimic several other diseases, including rabies, toxoplasmosis, and Neospora encephalitis (Nägler *et al*., 2025). Rabies is usually distinguished by the bite history, aggressive behavioral changes, and characteristic ascending paralysis, while CDV is often characterized by rhythmic myoclonus, non-suppurative encephalitis, and multifocal demyelination (Suwanpakdee *et al*., 2025). Neurological symptoms may also occur in both Neospora caninum and Toxoplasma gondii infections, but definitive diagnosis can be made through serological or molecular detection of specific protozoa (Postma *et al*., 2019).

Canine herpesvirus (CHV-1) and canine influenza virus (CIV) infections should also be considered as possible differential diagnoses, especially in cases presenting with predominantly respiratory symptoms without nervous system involvement (Day *et al*., 2020).

To ensure an accurate diagnosis, a combination of methods such as RT-PCR, immunohistochemistry, and specific serologic testing for CDV is highly recommended (Zhang *et al*., 2020). This comprehensive approach helps differentiate CDV from other diseases with similar clinical signs and provides a sound scientific basis for determining therapy and disease control strategies (Iribarnegaray *et al*., 2024).

Transmission

CDV is spread primarily through the air and through direct contact with bodily fluids of infected animals (Tonchiangsai *et al*., 2025). The virus can be found in various biological secretions, including nasal mucus, saliva, urine, and conjunctival fluid, especially during the acute viremia phase when viral load in epithelial and lymphoid tissues is very high (Sarchahi *et al*., 2022). Respiratory droplets containing the virus can remain airborne for short periods and serve as a source of transmission between animals, particularly in densely populated areas such as shelters, farms, and animal markets (Lazarus *et al*., 2025).

In addition to airborne transmission, CDV can also be transmitted indirectly through contact with contaminated objects or surfaces (fomites), such as eating utensils, cages, or human hands that have come into contact with infected animal secretions (Wilkes, 2022). Although the virus is relatively easily degraded in the external environment due to its sensitivity to heat, ultraviolet light, and lipid detergents, transmission is still possible if contact occurs soon after contamination (Macías-González *et al*., 2025).

The virus enters the host primarily through the respiratory tract after inhaling droplets containing infectious particles (Rendon-Marin *et al*., 2019). It then infects macrophages and lymphocytes in the tonsils and regional lymph nodes (Lan *et al*., 2009). From there, the virus spreads throughout the body via the blood and lymphatic system, causing primary viremia, which marks the beginning of spread to various target organs (Oleaga *et al*., 2022).

Risk factors

The incidence of CDV in the dog population is influenced by various epidemiological factors, including immune status, environmental conditions, and animal population dynamics (Ulaş *et al*., 2025). One reason for the high prevalence of this disease is low vaccination coverage (Pekkarinen *et al*., 2024). Dogs that do not receive routine primary or booster immunizations are highly susceptible to infection, particularly in early life when the immune system is not fully developed (Gulliver *et al*., 2025). Uneven vaccine distribution, particularly in rural areas, and the use of poorly standardized vaccines contribute to continued virus transmission in the field (Candela *et al*., 2025).

Another factor influencing the spread of CDV is the presence of feral or semi-feral dog populations, which act as the primary reservoir in the virus transmission cycle (Viana *et al*., 2020). These dogs are generally unvaccinated and frequently come into contact with domestic dogs, increasing the risk of cross-population transmission (Woodroffe *et al*., 2012). Epidemiological studies have shown that areas with high feral dog densities are positively associated with higher distemper incidence, particularly in densely populated urban areas and rural areas with limited animal health management systems (McDermott *et al*., 2023).

Furthermore, environmental conditions and climatic factors influence the stability and spread of the virus (Chludzinski *et al*., 2023). Moderate temperatures and high humidity can prolong the viability of virus particles, increasing the likelihood of transmission through the air or contaminated surfaces (Sawatsky *et al*., 2018). In tropical regions, the rainy season is often associated with a surge in CDV cases due to decreased animal immunity and increased dog population movement (Rivera-Martínez *et al*., 2024).

Urbanization and the movement of domestic animals contribute to the geographic spread of CDV (Kapil and Yeary, 2011). The movement of dogs between regions without proper quarantine practices can facilitate the introduction of new virus strains into previously disease-free areas (van de Bildt *et al*., 2002). Furthermore, interactions between domestic dogs, urban wildlife such as raccoons and foxes, and protected species

expand the host spectrum and increase the risk of cross-species transmission (Macías-González *et al*., 2025).

Vaccination

Vaccination is the primary strategy for controlling and preventing CDV infection in dogs and wild carnivores (Huo *et al*., 2025). Effective immunization programs have been shown to significantly reduce morbidity and mortality rates, while also helping to limit the spread of the virus in the environment (Nova *et al*., 2018). Currently, three types of vaccines are commonly used: live attenuated vaccines, recombinant vaccines, and DNA-based vaccines, each with different characteristics and levels of protection (Jiang *et al*., 2019).

Live attenuated vaccines have long been the most widely used type of vaccine in CDV immunization programs (Sixt *et al*., 1998). These vaccines are derived from attenuated virus strains through serial adaptation in tissue culture, such as the Onderstepoort, Snyder Hill, and Lederle strains (Carmichael, 1999). The vaccine works by simultaneously stimulating humoral and cellular immune responses, resulting in long-lasting protective immunity (Zhao *et al*., 2023). However, caution is needed when administering this vaccine to very young animals or individuals with compromised immune systems due to the potential for residual viral replication, which can lead to post-vaccination reactions (Du *et al*., 2022).

Advances in molecular biotechnology have enabled the development of viral vector-based recombinant vaccines, such as the canarypox-CDV vaccine, which contains the hemagglutinin (H) and fusion (F) genes from CDV (D. Liu *et al*., 2025). This type of vaccine offers a higher level of safety because the vector does not replicate in mammals, eliminating the risk of viral reactivation (Huo *et al*., 2025). Furthermore, recombinant vaccines can stimulate a rapid and robust immune response, even in animals with residual maternal antibodies, making them ideal for early immunization of puppies (Griot *et al*., 2004).

DNA vaccines are a promising new innovation for distemper prevention (Jensen *et al*., 2015). They use DNA plasmids that encode immunogenic antigens, such as the H or F proteins of CDV, into host cells (X. Liu *et al*., 2025). Once the antigen is translated intracellularly, it stimulates the activation of cytotoxic T cells and the production of specific antibodies (Klotz and Gerhauser, 2019). The advantages of DNA vaccines include high stability, safety, and the ability to generate long-term immunity without the need for additional adjuvants (Fomsgaard and Liu, 2021). However, these vaccines are still in the experimental research stage and are not yet commercially available (Bresalier and Worboys, 2014).

In general, the recommended CDV vaccination protocol begins at 6–8 weeks of age, with booster doses every 3–4 weeks until 16 weeks of age to overcome the influence of maternal antibodies (Griot *et al*., 2004). Booster doses are then given annually or every three years to maintain population immunity (Hill, 2006). The success of a vaccination program depends heavily on adherence to the immunization schedule, broad vaccination coverage, and maintaining vaccine quality through proper cold chain management (Lugelo *et al*., 2022).

From a veterinary public health perspective, mass vaccination programs for domestic dogs, combined with control of stray dog populations and education of pet owners, are crucial elements of a distemper eradication strategy (Wilkes, 2022). The implementation of a safe vaccine capable of eliciting a broad immune response also has the potential to reduce the risk of cross-species transmission of the virus, including in vulnerable wildlife such as members of the Mustelidae and Felidae families (Beineke *et al*., 2015).

Control

CDV control relies not only on vaccination but also requires the implementation of biosecurity principles, good shelter management, monitoring of wildlife reservoirs, and public education (Ahmad *et al*., 2024). This approach is comprehensive and aligns with the One Health concept, given CDV's ability to transmit across species and its potential threat to wildlife conservation (Rendon-Marin *et al*., 2019).

Strict biosecurity measures in shelters, clinics, and breeding facilities are key to preventing the spread of CDV (Gregers-Jensen *et al*., 2015). These procedures include segregating new or sick animals, routinely disinfecting equipment and the environment, and limiting direct contact between animals (Suwanpakdee *et al*., 2025). Because CDV is sensitive to heat and lipid detergents, disinfectants based on alcohol, quaternary ammonium, or sodium hypochlorite have been shown to be effective in inactivating the virus on surfaces (Imhoff *et al*., 2007).

Effective shelter management includes regulating animal density, maintaining a proper ventilation system, and monitoring the vaccination status of each individual (Day *et al*., 2016). Animals showing clinical signs should be immediately isolated and subjected to molecular testing to prevent transmission to other populations (Yang *et al*., 2020).

Wildlife, such as raccoons (Procyon lotor), foxes (Vulpes vulpes), and several mustelid species, plays an important role as reservoirs that maintain the natural circulation of CDV (Trebbien *et al*., 2014). Interspecies transmission can occur through direct contact, contamination of secretions, or shared water sources (Beineke *et al*., 2015). Therefore, routine epidemiological monitoring of wildlife populations is crucial to detect the presence of the virus and assess the risk of its spread to domestic animals (Macías-González *et al*., 2025).

Control approaches include vaccinating pets around conservation areas, limiting contact between domestic dogs and wildlife, and quarantining captive or reintroduced animals (Jessup *et al*., 2009). In conservation areas with high distemper incidence, genetic monitoring of viral strains is crucial to understand transmission patterns and potential cross-species adaptation (Rivera-Martínez *et al*., 2024).

The role of the community is also crucial in CDV control. Educational programs should emphasize the importance of routine vaccination, environmental hygiene, and prompt reporting of disease cases (Candela *et al*., 2025). Collaboration between veterinarians, shelter managers, and animal health authorities is necessary to establish an integrated reporting system capable of rapidly detecting outbreaks (Decaro *et al*., 2004).

Routine monitoring through active and passive surveillance of domestic and wildlife animal populations provides the basis for assessing the success of vaccination programs and biosecurity implementation (Kapil and Yeary, 2011). The use of molecular-based surveillance systems, such as RT-PCR and genetic sequencing, allows for monitoring of the dynamics of viral evolution and improves the ability to detect new strains early, potentially affecting vaccine effectiveness (Halecker *et al*., 2021).

Challenges and future research directions

Despite the implementation of various control measures, including vaccination and animal health education, CDV remains a global threat due to the complexity of the virus's evolution (Rivera-Martínez *et al*., 2024). The emergence of new strains with significant genetic variation often renders cross-protection from conventional vaccines less effective (Pratelli, 2011). This situation emphasizes the importance of continuous molecular monitoring to study mutation patterns in the H and F genes, which play a key role in determining the virus's antigenic properties (Rendon-Marin *et al*., 2019).

Furthermore, the risk of interspecies transmission of CDV between domestic and wild animals presents new challenges for conservation and disease ecology studies (Duque-Valencia *et al*., 2019a). Reports of infections in a variety of wild carnivores, including civets, raccoons, and even endangered species like tigers, highlight the need for an integrated, interdisciplinary approach (Rentería-Solís *et al*., 2014). In this regard, the application of One Health principles is crucial for integrating animal, human, and environmental health aspects into disease surveillance systems and control strategies (Danasekaran, 2024).

Future research should focus on developing a multivalent vaccine capable of providing broad protection against various CDV genotypes, as well as innovative molecular-based rapid diagnostic methods for early detection in the field (da Costa *et al*., 2019). Utilizing the integration of epidemiological, genomic, and ecological data through bioinformatics approaches is expected to improve understanding of the dynamics of virus spread and the effectiveness of prevention strategies (Panzera *et al*., 2015). Close collaboration between research institutions, veterinary authorities, and conservation organizations will be key to reducing the impact of CDV on domestic and wildlife populations in the future (Wang *et al*., 2022).

Conclusion

CDV remains a serious threat to the health of domestic dogs and wildlife globally. Although vaccination has been shown to reduce disease incidence, sporadic infections still occur due to low immunization coverage and genetic differences between virus strains. Controlling CDV requires a One Health approach with cross-sector collaboration through integrated surveillance, improved early diagnosis capabilities, and strengthened ongoing vaccination programs. Furthermore, further research on virus evolution and vaccine effectiveness against local strains is needed to support adaptive and sustainable control strategies.

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Conflict of interest

The authors have declared no conflict of interest.

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