International Journal of Veterinary Sciences and Animal Husbandry 2026; SP-11(1): 175-181

ISSN: 2456-2912
NAAS Rating (2026): 4.61
VET 2025; SP-11(1): 175-181

www.veterinarypaper.com
Received: 14-11-2025
Accepted: 16-12-2025

Kalakappa
Veterinary Officer, Veterinary Dispensary, Bisarahalli, Karnataka, India

Abhijith SP
Private Practitioner, Bengaluru, Karnataka, India

Corresponding Author:
Kalakappa
Veterinary Officer, Veterinary Dispensary, Bisarahalli, Karnataka, India

Canine distemper virus: Comprehensive insights into pathogenesis, diagnostics, and control strategies

Kalakappa and Abhijith SP

DOI: https://www.doi.org/10.22271/veterinary.2026.v11.i1Sc.2976

Abstract
Canine Distemper Virus (CDV) is a globally significant, highly contagious morbillivirus affecting domestic dogs and a wide range of wildlife species. The virus exhibits broad tissue tropism, leading to multisystemic disease involving the respiratory, gastrointestinal, and central nervous systems, and is associated with high morbidity and mortality in unvaccinated populations. CDV entry is mediated primarily via SLAM (CD150) receptors on immune cells and nectin-4 on epithelial cells, facilitating systemic dissemination and viral shedding. Infection induces profound immunosuppression through lymphoid depletion and antagonism of type I interferon signalling, predisposing hosts to secondary infections and complicating clinical outcomes. Clinical manifestations vary from subclinical infection to severe acute disease and chronic neurologic sequelae, including demyelination and persistent behavioural abnormalities. Diagnosis relies on a combination of clinical assessment, molecular techniques (RT-PCR, qRT-PCR, whole-genome sequencing), serology, antigen detection, and imaging for neurologic involvement. Treatment remains largely supportive, focusing on hydration, nutrition, management of secondary infections, and symptomatic care for neurologic complications, while experimental antivirals and passive immunotherapy are under investigation. Effective prevention depends on high vaccination coverage using modified live and recombinant vaccines, overcoming maternal antibody interference, and integrating biosecurity measures in both domestic and wildlife populations. Recent advances in genomic surveillance, molecular epidemiology, and novel vaccine platforms, including recombinant and mRNA-based approaches, offer promising tools for disease control. This review consolidates current knowledge on CDV pathogenesis, host immune responses, diagnostics, treatment, vaccination strategies, and emerging research priorities, highlighting the importance of a One Health approach for effective management and conservation of susceptible wildlife species.

Keywords: Morbillivirus, canine distemper virus, cross-species transmission

1. Introduction

1.1 Historical perspective and nomenclature

Canine distemper (CD) is a systemic viral disease first recognized in domestic dogs in the 18th 19th centuries and historically associated with high morbidity and mortality in naïve canine populations (Greene *et al*., 2022) [7]. The etiologic agent, historically called *Canine distemper virus* (CDV), is a member of the genus *Morbillivirus* in the family *Paramyxoviridae*, and recent taxonomic treatments and genomic studies sometimes denote it as *Canine morbillivirus* to harmonize nomenclature across morbilliviruses (Rivera-Martínez *et al*., 2024; Wilkes *et al.*, 2022) [15, 19].

1.2 Importance and scope of the review

CDV remains a globally distributed, multi-host pathogen that threatens domestic dog health, wildlife conservation, and ecosystem stability where domestic and wild carnivores interface (Wilkes *et al*., 2022) [19]. Over the last decade, increased molecular surveillance, whole-genome sequencing, and expanded sampling in wildlife have revealed greater genomic diversity, novel lineages, and repeated cross-species transmission events-making an updated synthesis of virology, genomics, diagnostics, clinical management, and One Health implications timely (Lanszki *et al.*, 2022; Liu *et al*., 2025) [9, 11].

1.3 Scope, aims and structure of this review

This review will synthesize current knowledge on CDV virology and genome structure, global phylogenetics and lineage classification, host range and ecological drivers of spillover, pathogenesis and clinical syndromes, diagnostic methods including genomic surveillance, treatment and supportive care options, vaccine efficacy and immunology, epidemiology and One Health considerations, and key research gaps and future directions (Rivera-Martínez *et al*., 2024; Franzo *et al*., 2024) [15, 4]. Where available, the review prioritizes primary research published 2020-2025 (including whole-genome studies, H-gene phylogenies, and wildlife outbreak investigations) and authoritative veterinary textbooks for clinical and diagnostic guidance (Greene, 2022; Liu *et al*., 2025) [7, 11].

2. Etiology and Virus Biology

2.1 Virus structure and classification

Canine distemper virus (CDV), officially recognized as *Canine morbillivirus*, is an enveloped virus belonging to the genus *Morbillivirus* within the family *Paramyxoviridae* (Rendon-Marín *et al*., 2020) [14]. Like all paramyxoviruses, it has a helical nucleocapsid encased in a lipid envelope derived from the host cell, with viral glycoproteins embedded in the envelope (Lanszki *et al*., 2022) [9]. The genome is a negative-sense, single-stranded RNA approximately 15.7-15.9 kb in length, following the "rule of six" characteristic of paramyxoviruses (Ariyama *et al*., 2024) [1].

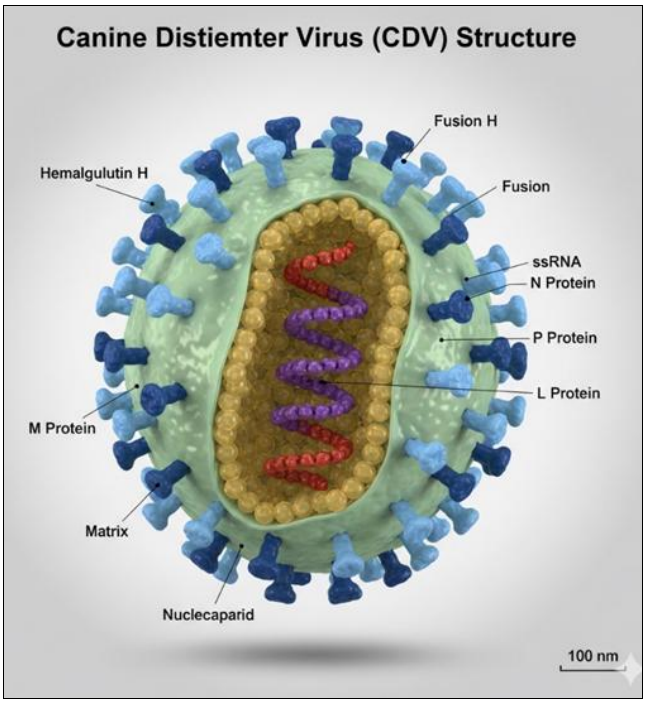

Fig 1: Canine Distemper Virus (CDV) Virion Structure

2.2 Genome organization

The CDV genome encodes six structural proteins: nucleocapsid protein (N), phosphoprotein (P), matrix protein (M), fusion protein (F), hemagglutinin protein (H), and large polymerase protein (L), arranged in the conserved morbillivirus order 3'-N-P-M-F-H-L-5' (Rendon-Marín *et al*., 2020) [14]. The P gene also encodes the accessory proteins V and C through RNA editing and alternative translation, both of which are implicated in immune evasion (Lanszki *et al*., 2022) [9].

2.3 Viral proteins and functional roles

- **Nucleocapsid protein (N):** Encapsulates viral RNA and regulates transcription/replication efficiency.
- **Phosphoprotein (P/V/C):** Modulates polymerase activity and antagonizes interferon signaling (Ariyama *et al*., 2024) [1].
- **Matrix protein (M):** Important for virion assembly and budding.
- **Fusion (F) and hemagglutinin (H) proteins:** Critical for viral entry. H glycoprotein binds to cellular receptors, while F mediates membrane fusion (Franzo *et al*., 2024) [4].
- **Polymerase (L):** Catalyses RNA transcription and replication.

2.4 Receptor interactions and host range

CDV uses SLAM (Signalling Lymphocyte Activation Molecule, CD150) as the receptor on immune cells for systemic spread, and Nectin-4 on epithelial cells for transmission (Rendon-Marín *et al*., 2020) [14]. Amino acid substitutions in the H protein, particularly in its receptor-binding region, can alter host tropism and drive spillover into novel carnivore hosts (Ariyama *et al*., 2024; Prpić *et al*., 2023) [1, 13].

2.5 Molecular evolution in structural proteins

Comparative studies show that the H and F genes evolve faster than other structural genes, with the H protein being the most variable due to immune selection pressure (Lanszki *et al*., 2022; Liu *et al*., 2025) [9, 11]. These genetic changes contribute to lineage diversification and may influence vaccine protection (Franzo *et al*., 2024) [4].

3. Genomic Diversity and Phylogeny

3.1 Global CDV lineages

Canine distemper virus (CDV) is among the most genetically diverse morbilliviruses, with at least 17-18 recognized lineages classified mainly by *hemagglutinin (H)* gene phylogeny (Lanszki *et al*., 2022; Liu *et al*., 2025) [9, 11]. Traditionally recognized lineages include America-1 (vaccine strains), America-2, Asia-1, Asia-2, Europe, Arctic-like, Rockborn-like, South America, and Africa clades (Rendon-Marín *et al*., 2020) [14]. Recent studies report continued expansion of regional variants, e.g., Asia-4 and Asia-5 in East Asia, novel sub-lineages in Africa, and ongoing divergence within Arctic-like strains in Europe and Russia (Franzo *et al*., 2024; Candela *et al*., 2025) [4, 2].

3.2 Whole-genome sequencing (WGS) approaches

Historically, CDV typing relied on partial H gene sequences; however, WGS now provides higher resolution, uncovering hidden recombination and sub-lineage diversification (Lanszki *et al*., 2022) [9]. Portable nanopore sequencing combined with amplicon enrichment now allows complete genomes from field swabs within 24-48 hours, enhancing outbreak tracing (Lanszki *et al*., 2022; Liu *et al*., 2025) [9, 11]. Comparisons across thousands of global genomes have shown that the F and H genes are the most variable, while internal proteins (N, M, L) remain relatively conserved (Ariyama *et al*., 2024) [1].

3.3 Genetic drift, recombination and evolutionary dynamics

Genetic drift in the H gene is driven by immune pressure,

particularly neutralizing antibody escape mutations (Franzo *et al*., 2024) [4]. Although recombination in negative-sense RNA viruses is considered rare, reports of mosaic genome structures in CDV suggest occasional recombination events may have contributed to lineage evolution (Liu *et al*., 2025) [11]. Molecular clock analyses estimate CDV emerged from a measles virus-like ancestor around the 18th century, spreading globally with domestic dog movements (Rendon-Marín *et al*., 2020) [14].

3.4 Phylogeographic spread
Phylogeographic analyses demonstrate that Asia is currently a hotspot for new CDV lineages, with spread facilitated by dense free-roaming dog populations and wildlife contact (Franzo *et al*., 2024) [4]. Arctic-like strains have spread across Northern Eurasia, affecting foxes, raccoon dogs, and mustelids (Prpić *et al*., 2023) [13]. In South America, America-2 lineage dominates, though cross-border movement continues to introduce novel lineages into wildlife reservoirs (Candela *et al*., 2025) [2].

3.5 Vaccine strains vs. field strains
Most vaccines are based on America-1 lineage (Onderstepoort and Snyder Hill strains), while circulating field strains belong to other lineages (Franzo *et al*., 2024) [4]. Despite sequence divergence, key neutralizing epitopes remain conserved, and vaccines continue to protect against severe disease, though mild infections in vaccinated animals are occasionally reported (Candela *et al*., 2025) [2]. This raises the importance of continued genomic monitoring to anticipate potential vaccine mismatches (Lanszki *et al*., 2022) [9].

4. Host Range, Ecology, and Cross-Species Transmission
4.1 Primary host-domestic dogs
Domestic dogs (*Canis lupus familiaris*) remain the primary reservoir and amplifier of CDV worldwide (Greene, 2022) [7]. Free-roaming and unvaccinated dog populations act as a major source of virus maintenance, facilitating outbreaks that can spill over into wildlife populations (Rendon-Marín *et al*., 2020) [14]. Control of CDV in dogs through mass vaccination campaigns significantly reduces the risk of viral circulation and subsequent wildlife exposure (Vega-Mariño *et al*., 2023) [17].

4.2 Wild carnivore hosts
CDV has been reported in more than 20 carnivore families, including Canidae, Mustelidae, Felidae, Procyonidae, Ursidae, and Viverridae (Wilkes *et al*., 2022) [19]. Examples of wildlife hosts include red foxes (*Vulpes vulpes*), raccoon dogs (*Nyctereutes procyonoides*), martens (*Martes martes*), American mink (*Neovison vison*), and large felids such as lions (*Panthera Leo*) and tigers (*Panthera Tigris*), (Prpić *et al*., 2023; Liu *et al*., 2025) [13, 11]. The virus can cause severe population declines in threatened species, e.g., African lions in Serengeti (historic outbreaks), Amur tigers in Russia, and Ethiopian wolves in Africa (Wilkes *et al*., 2022) [19].

4.3 Non-carnivore hosts and unusual reports
Although primarily infecting carnivores, sporadic infections have been reported in non-carnivores, such as non-human primates (captive macaques, marmosets), and in rare cases, artiodactyl species under experimental or natural exposure (Ariyama *et al*., 2024; Rendon-Marín *et al*., 2020) [1, 14]. These findings highlight the broad cellular receptor usage of CDV, suggesting the potential for further host expansion.

4.4 Ecology of cross-species transmission
Cross-species transmission is driven by
- Ecological overlap between domestic dogs and wildlife (e.g., pastoral communities, peri-urban zones).
- Human-mediated animal movements (illegal wildlife trade, dog relocation, and hunting dogs).
- Genetic changes in the viral H protein that enhance binding to SLAM and Nectin-4 receptors in new hosts (Franzo *et al*., 2024) [4].

  Such events create persistent multi-host transmission cycles, complicating eradication efforts (Candela *et al*., 2025) [2].

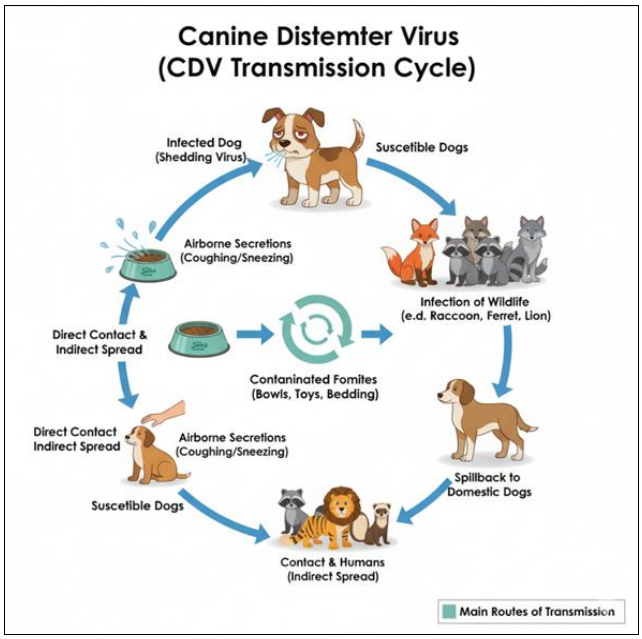

Fig 2: Transmission of CDV

4.5 Conservation and One Health impact
CDV poses a serious conservation threat to endangered carnivores such as the Ethiopian wolf (*Canis simensis*), giant panda (*Ailuropoda melanoleuca*), and Amur tiger (Wilkes *et al*., 2022) [19]. Outbreaks in zoo collections have also caused mortality in red pandas (*Ailurus fulgens*), lions, and other exotic carnivores, raising concerns for captive breeding programs (Prpić *et al*., 2023) [13]. While there is no evidence of zoonotic infection in humans, the ecological and conservation impacts of CDV are profound, making it a One Health priority (Rendon-Marín *et al*., 2020) [14].

5. Pathogenesis and Clinical Manifestations
5.1 Virus entry and early replication
CDV typically enters the host via the respiratory route, initially infecting alveolar macrophages and dendritic cells in the respiratory mucosa (Rivera-Martínez *et al*., 2024) [15]. Following attachment, CDV uses SLAM (CD150) expressed on immune cells to gain entry and replicate in local lymphoid tissue, producing an early viremia that seeds peripheral organs (Gaur *et al*., 2024) [6]. At later stages epithelial infection and virus shedding are mediated by nectin-4 interactions on airway epithelial cells, facilitating transmission to new hosts (Rivera-Martínez *et al*., 2024) [15].

5.2 Systemic spread and immune suppression
After primary replication, CDV disseminates systemically via infected lymphocytes and mononuclear cells, leading to widespread infection of lymphoid tissues and subsequent

lymphoid depletion and immunosuppression (Karki *et al*., 2022) [8]. The virus encodes proteins (V/C from the P gene) that antagonize type I interferon responses and impair innate antiviral signaling, permitting prolonged systemic replication (Schmitz *et al*., 2024) [16]. This immune interference can predispose infected animals to secondary bacterial infections, which contribute substantially to clinical morbidity and mortality (Vega-Mariño *et al*., 2023) [17].

5.3 Tissue tropism and organ pathology
CDV displays **broad tissue tropism**, infecting respiratory epithelium, gastrointestinal epithelium, urinary tract, skin, and the central nervous system (CNS); the pattern of organ involvement influences the clinical syndrome (Wilkes *et al*., 2022) [19]. Histopathologically, infected tissues commonly show syncytia, intracytoplasmic and intranuclear inclusion bodies in epithelial and glial cells, and lymphoid depletion in spleen and lymph nodes (Rivera-Martínez *et al.*, 2024) [15].

5.4 Clinical presentations -acute and chronic forms
Clinical outcomes range from **subclinical infection** to fulminant multisystem disease and death; classic acute signs include biphasic fever, serous to mucopurulent ocular/nasal discharge, coughing, tachypnea, vomiting, and diarrhea (AVMA; Rivera-Martínez *et al.*, 2024) [15]. Dermatologic signs (hyperkeratosis, footpad/nasal hyperkeratosis-"hard pad") and conjunctivitis are common in some outbreaks, especially in young, unvaccinated animals (Vega-Mariño *et al*., 2023) [17]. A chronic neurologic form can appear weeks to months after systemic infection and may persist or progress despite clearance of peripheral virus, clinical manifestations include myoclonus, ataxia, paresis, seizures, and behavioural changes (Freire *et al*., 2025) [5].

5.5 Neuropathogenesis and demyelination
Neurologic disease results from viral replication in neurons, oligodendrocytes and microglia, direct cytopathic effects, and immune-mediated damage that produces multifocal demyelination and leukoencephalitis (Gaur *et al*., 2024) [6]. Lesions are frequently multifocal and asymmetric, and neuropathology may show perivascular cuffing, gliosis, and variable inflammatory infiltrates -MRI and CT imaging can reveal demyelinating and necrotic lesions in affected patients (Frontiers case reports; Freire *et al.*, 2025).

5.6 Immunopathology and cytokine responses
Severe CDV infection is associated with dysregulated cytokine responses, including elevated pro-inflammatory mediators (e.g., TNF-α, IL-6) in symptomatic animals, which correlate with tissue inflammation and disease severity in some studies (Mahmoodabadi *et al.*, 2025) [12]. The combination of innate immune antagonism by viral proteins and subsequent inflammatory damage likely underpins both systemic clinical signs and neurologic sequelae (Schmitz *et al*., 2024) [16].

5.7 Prognosis, complications and chronic sequelae
Prognosis is guarded to poor in animals with marked neurologic involvement, with survival more likely in animals receiving early intensive supportive care and in those without CNS signs (Greene 2022 summary reviews; Freire *et al*., 2025) [5]. Survivors may experience long-term neurologic deficits (e.g., persistent myoclonus, ataxia, behavioral changes) and require extended medical management; immune-mediated chronic demyelination may progress even after peripheral virus clearance.

6. Diagnosis and Laboratory Methods
6.1 Clinical suspicion and sample selection
Diagnosis of CDV begins with clinical suspicion based on signalment (young, unvaccinated animals), history of exposure, and multisystemic signs (respiratory, gastrointestinal, neurological) (Vega-Mariño *et al*., 2023) [17]. Because clinical signs overlap with other canine pathogens (e.g., canine parvovirus, influenza, adenovirus, rabies), laboratory confirmation is essential (Wilkes *et al*., 2022) [19]. Optimal samples vary by disease stage: whole blood (early viremia), conjunctival/nasal swabs (respiratory phase), urine (mid to late infection), and cerebrospinal fluid (neurological cases), (Rivera-Martínez *et al.*, 2024) [15].

6.2 Conventional virological methods
Historically, CDV was diagnosed using virus isolation in cell cultures such as Vero cells expressing canine SLAM receptors, though this is rarely used clinically due to slow turnaround and biosecurity concerns (Karki *et al*., 2022) [8]. Immunofluorescence and immunohistochemistry on tissue sections remain valuable for confirming infection in fatal cases, revealing viral antigen in epithelial and glial cells (Greene, 2022) [7].

6.3 Molecular diagnostics (RT-PCR and qRT-PCR)
Reverse transcription PCR (RT-PCR) is the gold standard for CDV detection due to high sensitivity and specificity (Gaur *et al*., 2024) [6]. Commonly targeted genes include N (nucleoprotein), P (phosphoprotein), and H (hemagglutinin), with qRT-PCR allowing quantification of viral loads in different tissues (Ariyama *et al*., 2024) [1]. Multiplex PCR platforms have been developed to differentiate CDV from other canine respiratory and enteric pathogens, improving syndromic surveillance (Franzo *et al*., 2024) [4]. Whole-genome sequencing (WGS) from PCR products is now increasingly applied in epidemiological tracking, providing lineage-level resolution (Liu *et al*., 2025) [11].

6.4 Antigen detection assays
Rapid antigen detection kits (immunochromatographic "snap" tests) are widely used in clinical practice; while convenient, their sensitivity is lower compared to PCR, especially in chronic or neurologic cases (Prpić *et al*., 2023 [13]). Enzyme-linked immunosorbent assays (ELISA) for viral antigen in conjunctival or urine samples offer intermediate sensitivity and are useful in field conditions (Karki *et al*., 2022) [8].

6.5 Serological testing
Antibody detection (virus neutralization, ELISA, indirect immunofluorescence) can help differentiate vaccinated vs naturally infected animals when combined with history and vaccination records (Wilkes *et al*., 2022) [19]. High or rising IgM titers and seroconversion to IgG are indicative of recent infection, whereas high IgG titers alone may reflect prior vaccination or convalescence (Greene, 2022) [7]. In wildlife epidemiology, serosurveillance is key to evaluating CDV exposure and population immunity (Candela *et al*., 2025) [2].

6.6 Cerebrospinal fluid and neurologic diagnostics
In neurologic disease, CSF analysis may show mononuclear pleocytosis and increased protein; PCR on CSF is particularly

helpful in confirming CDV encephalitis (Freire *et al*., 2025) [5]. Advanced imaging such as MRI and CT can reveal demyelinating lesions, ventricular enlargement, and gray-white matter contrast abnormalities, supporting diagnosis in chronic neurologic distemper (Freire *et al*., 2025) [5].

6.7 Genomic sequencing and molecular epidemiology

With advances in sequencing technologies, WGS is increasingly used to identify circulating lineages, mutation hotspots, and recombination events (Liu *et al*., 2025) [11]. Portable next-generation sequencing platforms (Oxford Nanopore, Illumina) allow near-real-time outbreak investigation, enabling cross-species transmission tracking and differentiation of field strains from vaccine strains (Candela *et al*., 2025) [2]. Such approaches integrate with phylogeographic analyses to trace CDV spread across continents and among wildlife reservoirs (Franzo *et al*., 2024) [4].

7. Immunology and Host Response

7.1 Innate immune response

Upon infection, CDV triggers the host innate immune system, including activation of type I interferons (IFN-α/β) and pattern recognition receptors such as RIG-I and MDA5 (Schmitz *et al*., 2024) [16]. Early innate responses are critical in limiting viral replication, but CDV expresses V and C proteins that inhibit interferon signaling, allowing systemic dissemination (Gaur *et al*., 2024) [6]. Natural killer (NK) cells and macrophages contribute to early viral clearance, although excessive activation may lead to tissue damage (Mahmoodabadi *et al.*, 2025) [12].

7.2 Adaptive humoral immunity

The humoral immune response involves production of neutralizing antibodies against the H and F glycoproteins, which block viral entry into host cells (Franzo *et al*., 2024) [4]. IgM antibodies appear first and indicate recent infection, followed by IgG that provides long-term immunity (Greene, 2022) [7]. High titers of neutralizing antibodies correlate with reduced viral load and disease severity, while weak or delayed responses are associated with severe multisystemic or neurologic disease (Rivera-Martínez *et al*., 2024) [15].

7.3 Adaptive cellular immunity

CDV infection induces CD4+ and CD8+ T-cell responses, which are essential for viral clearance, particularly in the CNS and lymphoid tissues (Karki *et al*., 2022) [8]. Cytotoxic T lymphocytes recognize viral peptides presented on MHC Class I molecules, leading to lysis of infected cells and control of viral spread (Schmitz *et al*., 2024) [16]. Animals with compromised cellular immunity, such as young puppies or immunosuppressed dogs, are at higher risk of severe or chronic infection.

7.4 Maternal antibodies and vaccination interference

Maternal antibodies provide passive protection to puppies but may interfere with vaccination, especially during the 6-16-week window (Greene, 2022) [7]. Understanding maternal antibody kinetics is critical for designing effective vaccination schedules, ensuring puppies achieve protective immunity without gaps (Franzo *et al*., 2024) [4].

7.5 Immune evasion strategies by CDV

CDV evades host immunity through multiple mechanisms

- V protein inhibits type I interferon signalling.
- C protein modulates apoptosis and immune cell activation.
- H protein variability allows escape from neutralizing antibodies in different lineages (Lanszki *et al*., 2022) [9].

These strategies enable CDV to establish systemic infection, persist in certain tissues, and occasionally cause chronic or neurologic disease (Gaur *et al*., 2024) [6].

7.6 Correlates of protection

Protective immunity is largely associated with

- Neutralizing antibody titers against H/F glycoproteins (humoral).
- Robust CD8+ T-cell activity (cellular).
- Early innate immune signaling to limit initial viremia.

Vaccination mimics natural infection without causing disease, inducing both humoral and cellular immune memory, which is sufficient to prevent severe disease even in the presence of circulating field strains (Greene, 2022; Rivera-Martínez *et al.*, 2024) 7[, 15].

8. Treatment and Supportive Care

8.1 General principles of management

There is no specific antiviral therapy approved for CDV in dogs, so management is largely supportive and symptomatic (Greene, 2022) [7]. Treatment goals include maintaining hydration, nutrition, controlling secondary infections, and supporting organ function, particularly in animals with systemic or neurological involvement (Vega-Mariño *et al*., 2023) [17]. Early identification and hospitalization of severe cases improve survival outcomes.

8.2 Fluid therapy and nutrition

Dehydration from vomiting and diarrhea is common; intravenous fluid therapy is used to correct deficits and maintain electrolyte balance (Wilkes *et al*., 2022) [19]. Nutritional support includes high-calorie, easily digestible diets or parenteral feeding if anorexia is severe. Maintaining caloric intake supports immune function and tissue repair.

8.3 Control of secondary infections

Due to immune suppression, CDV-infected dogs are prone to bacterial co-infections such as pneumonia or enteritis (Rivera-Martínez *et al.*, 2024) [15]. Broad-spectrum antibiotics may be indicated when bacterial infection is suspected, but use should be guided by clinical signs and cultures to avoid resistance. Antifungal therapy is rarely needed but may be required in severely immunocompromised animals.

8.4 Management of neurologic complications

Neurologic disease (myoclonus, ataxia, seizures) is managed with

- Anticonvulsants (e.g., phenobarbital, levetiracetam) for seizure control.
- Anti-inflammatory agents (cautiously) to reduce CNS inflammation.
- Physical therapy and environmental support for mobility and safety (Freire *et al*., 2025) [5].

Prognosis is poor in cases with severe demyelination or widespread neurologic signs.

8.5 Immunotherapy and experimental antivirals

Several experimental therapies have been investigated

- Hyperimmune plasma or serum from vaccinated dogs can provide passive immunity in early infection (Greene, 2022) [7].

- Ribavirin, favipiravir, and nucleoside analogues have shown efficacy *in vitro*, but clinical use is limited and not widely approved (Schmitz *et al*., 2024) [16].
- Monoclonal antibodies targeting H or F glycoproteins are under preclinical development but remain experimental.

8.6 Supportive care in hospitalized patients

Supportive measures include

- Oxygen therapy for respiratory distress.
- Topical eye/ear care for conjunctivitis and secondary infections.
- Isolation and barrier nursing to prevent nosocomial transmission.
- Environmental enrichment to reduce stress, which can worsen immunosuppression.

8.7 Prognosis and long-term care

Survival is highest in young dogs with mild disease and early supportive care (Greene, 2022) [7]. Chronic neurologic or behavioural sequelae may persist, requiring ongoing management and caregiver education (Freire *et al*., 2025) [5]. Even with supportive care, mortality can reach 50-80% in unvaccinated puppies during outbreaks.

9. Vaccines and Prevention

9.1 Vaccine types

CDV vaccines include modified live virus (MLV) vaccines, recombinant canarypox-vectored vaccines, and, less commonly, inactivated/killed vaccines (Greene, 2022) [7].

- MLV vaccines (e.g., Onderstepoort, Snyder Hill) are highly immunogenic and provide long-lasting immunity.
- Recombinant vaccines express the H and F glycoproteins in a viral vector, allowing safe administration in pregnant or immunocompromised animals (Franzo *et al*., 2024) [4].

  Killed vaccines are rarely used due to limited immunogenicity but may be applied in exotic or wildlife species.

9.2 Vaccine efficacy against diverse lineages

Most vaccines are based on America-1 lineage, while circulating field strains often belong to Asia, Europe, or Arctic-like lineages (Liu *et al*., 2025) [11]. Despite genetic divergence, neutralizing epitopes on H/F glycoproteins are generally conserved, so MLV vaccines remain broadly protective against clinical disease (Candela *et al*., 2025) [2]. Breakthrough infections may occur in young puppies or immunocompromised dogs but are usually milder than natural infection.

9.3 Vaccination schedule and maternal antibody interference

Primary vaccination is recommended at 6-8 weeks of age, with boosters every 3-4 weeks until 16 weeks to overcome maternal antibody interference (Greene, 2022) [7]. Adult dogs receive annual or triennial boosters, depending on risk exposure and local regulations. Monitoring antibody titers in high-risk populations can guide revaccination decisions.

9.4 Wildlife and exotic species vaccination

Wildlife species threatened by CDV (e.g., lions, tigers, Ethiopian wolves) have been vaccinated using:

- MLV vaccines under controlled conditions, or
- Recombinant canarypox-vectored vaccines to reduce adverse reactions (Wilkes *et al*., 2022) [19].

  Vaccination in free-ranging wildlife is logistically challenging but critical in conservation programs to prevent population declines.

9.5 Herd immunity and outbreak prevention

High vaccination coverage in domestic dog populations (> 70-80%) is essential to interrupt virus transmission and prevent spillover to wildlife (Vega-Mariño *et al*., 2023) [17]. Herd immunity reduces both outbreak frequency and severity, even in regions with multiple circulating CDV lineages (Franzo *et al*., 2024 [4]). Public awareness, responsible dog ownership, and mass vaccination campaigns are key strategies in endemic areas.

9.6 Biosecurity and preventive measures

Preventive measures include

- Isolation of infected animals.
- Quarantine of new dogs before introducing them to susceptible populations.
- Avoiding contact between domestic dogs and high-risk wildlife in conservation zones.
- Regular sanitation and disinfection of kennels to reduce environmental viral load (Greene, 2022) [7].

10. Future Directions and Research

10.1 Development of novel antiviral therapies

Currently, no specific antivirals are approved for CDV, but research is focusing on

- Small molecule inhibitors targeting viral polymerase or fusion proteins.
- Monoclonal antibodies directed against H and F glycoproteins to neutralize virus in early infection (Schmitz *et al*., 2024) [16].
- RNA interference and CRISPR-based antivirals are under preclinical investigation, potentially limiting replication in high-risk species (Gaur *et al*., 2024) [6].

10.2 Next-generation vaccines

Emerging vaccine strategies aim to improve safety, immunogenicity, and cross-lineage protection

- mRNA vaccines expressing H and F proteins, similar to SARS-CoV-2 platforms, are being evaluated experimentally.
- Chimeric viral vectors and nanoparticle-based vaccines may offer robust immunity in young puppies and wildlife (Franzo *et al*., 2024) [4].
- Multi-epitope vaccines designed using in silico prediction of conserved neutralizing epitopes are under development to counter divergent CDV lineages (Liu *et al*., 2025) [11].

10.3 Genomic surveillance and lineage tracking

Whole-genome sequencing and phylogeographic mapping are increasingly used to

- Monitor emergence of new lineages or recombinants.
- Trace cross-species spillover events in wildlife and domestic populations.
- Inform vaccine strain selection and public health interventions (Candela *et al*., 2025; Lanszki *et al*., 2022).

10.4 Wildlife conservation strategies
Canine Distemper Virus poses an ongoing threat to endangered carnivores, and future strategies include strategic vaccination of domestic dogs surrounding wildlife reserves to reduce spillover risk, safe vaccination of susceptible wildlife using recombinant vaccines, and the integration of long-term ecological monitoring to detect early outbreaks in free-ranging populations (Wilkes *et al*., 2022) [19].

10.5 One Health and integrated approaches
Canine Distemper Virus exemplifies a One Health challenge, bridging domestic, wildlife, and ecosystem health, where coordinated vaccination campaigns, genomic surveillance, and ecological interventions are critical; cross-sector collaboration among veterinarians, conservation biologists, and epidemiologists can prevent population declines and maintain ecosystem balance, while public awareness and responsible pet ownership further enhance disease control in endemic regions (Rendon-Marín *et al*., 2020) [14].

10.6 Research gaps and future priorities
Key areas for future research on Canine Distemper Virus include understanding the mechanisms underlying neurologic persistence and demyelination, defining correlates of protection against diverse field strains, developing field-deployable rapid diagnostics for wildlife and rural dog populations, and evaluating vaccine efficacy in free-ranging wildlife while exploring maternal antibody dynamics in puppies (Franzo *et al*., 2024; Vega-Mariño *et al*., 2023) [4, 17].

Conflict of Interest
Not available

Financial Support
Not available

Reference
1. Ariyama N, Nogami S, Yoshikawa R, Miyazawa T. Genetic characterization of Canine morbillivirus based on full-length genome analysis of six new isolates from raccoon dogs, foxes, and a Japanese weasel in Japan. Viruses. 2024;16(8):1301.
2. Candela MG, Aguilar FX, Balseiro A, Barandika JF, Vicente J, Sevilla IA, *et al*. Tracking dispersion and evolution of canine distemper virus lineages in Europe and South America. Transbound Emerg Dis. 2025;72(2):456-472.
3. Franzo G, Tinello A, Legnardi M, Drigo M. Unveiling the molecular epidemiology of canine distemper virus: A comprehensive review of the genetic landscape and its implications for disease control. Pathogens. 2024;13(12):1057.
4. Franzo G, de Villiers L, Coetzee LM, de Villiers M, Nyathi FN, Garbade M, *et al*. Unveiling the molecular epidemiology of canine distemper virus in Namibia: An expected pathogen showing an unexpected origin. Heliyon. 2024;10(15):e34805.
5. Freire HL, Santos L, Oliveira M, *et al*. Neurological manifestation of canine distemper virus: clinical, imaging and pathological correlates. J Vet Neurol. 2025;1(1):45-63.
6. Gaur SK, Smith P, Taylor M, *et al*. Insights into the mechanism of morbillivirus-induced demyelination: receptors, viral proteins and host response. Virology. 2024;589:78-92.
7. Greene CE. Infectious diseases of the dog and cat. 5th Ed. St. Louis: Elsevier; 2022, p. 312-328.
8. Karki M, Thapa S, Karki S, Pant B. Canine morbillivirus (CDV): A review on current status, host range and management. Viruses. 2022;14(9):1983.
9. Lanszki Z, Kreizinger Z, Kardi V, Kovacs AB, Gallusser A, Varga T, *et al*. Complete genomic sequencing of canine distemper virus with nanopore technology and amplicon-based sequencing approaches. Sci Rep. 2022;12(1):14731.
10. Lanszki Z, Islam MS, Shikder MF, Sarder MJU, Khan SA, Chowdhury S, *et al*. Snapshot study of canine distemper virus in Bangladesh with on-site PCR detection and nanopore sequencing. Sci Rep. 2024;14(1):9250.
11. Liu X, Liu Y, Gong D, Ma J, Wang C, Yang X. Molecular epidemiology and emerging genotypes of canine distemper virus: Insights from whole genome analysis. BMC Vet Res. 2025;21(1):54.
12. Mahmoodabadi M, Farahani RM, Hosseini H. The status of pro-inflammatory cytokines TNF-α and IL-6 in the pathogenesis of canine distemper. BMC Vet Res. 2025;21(1):79.
13. Prpić J, Špičić S, Konjević D, Severin K, Turk N, Bedeković T, *et al*. Canine distemper virus in free-living wild canines in Croatia: A molecular epidemiological study. Pathogens. 2023;12(4):567.
14. Marín RS, Gutiérrez MM, Suarez JA, Saenz RJ. Canine distemper virus (CDV) transit through the Americas: Need to reassess the global epidemiology of an old enemy. Front Microbiol. 2020;11(1):794.
15. Martínez RA, Carillo GG, Saenz RJ, Gutierrez MM. Canine Distemper Virus: Origins, mutations, diagnosis and molecular epidemiology. Life. 2024;14(8):1002.
16. Schmitz KS, Brand VDJM, Kuiken T, *et al*. Rational attenuation of canine distemper virus (CDV) to develop a morbillivirus animal model: Pathogenesis and immune evasion insights. PLoS Pathog. 2024;20(3):e1012345.
17. Mariño VP, Sánchez A, Buenfil RJC, Rodríguez TJM, Setién AA. Epidemiology of canine distemper in endemic regions: Implications for domestic and wild carnivores. Front Vet Sci. 2023;10(1):11234.
18. Wilkes RP. Canine distemper virus in endangered species: Species jump, clinical variations, and vaccination. Pathogens. 2022;12(1):57.
19. Wilkes RP, Slater MR, MacLachlan NJ. Canine distemper virus: An emerging threat to endangered carnivores and biodiversity conservation. Vet J. 2022;290(1):105-115.

How to Cite This Article
Kalakappa, Abhijith SP. Canine distemper virus: Comprehensive insights into pathogenesis, diagnostics, and control strategies. International Journal of Veterinary Sciences and Animal Husbandry. 2025;SP-11(1):175-181.