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CANINE DISTEMPER IN WILDLIFE: A SILENT CROSS-SPECIES THREAT

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Abstract

Canine distemper virus (CDV) is a highly contagious morbillivirus belonging to the family *Paramyxoviridae*, infecting a wide range of carnivorous and some non-carnivorous species. Originally recognized in domestic dogs, it has evolved into an important multi-host pathogen with global distribution in wildlife. The virus produces severe systemic disease marked by immunosuppression and involvement of the respiratory, gastrointestinal, and nervous systems, frequently leading to high mortality in immunologically susceptible populations. Its capacity for cross-species transmission and maintenance through domestic dog reservoirs presents a serious threat to wildlife conservation. Diagnosis is based on molecular and immunological methods, while control primarily relies on vaccination and effective surveillance at the wildlife–domestic animal interface.

Keywords: Canine distemper virus, morbillivirus, wildlife disease, cross-species transmission, conservation impact

Introduction

India harbours rich wildlife diversity and has implemented significant conservation efforts to protect endangered species, particularly wild felids. However, infectious diseases increasingly threaten these initiatives, largely due to growing interactions between wildlife and domestic animals. CDV is a highly contagious, pantropic morbillivirus that infects a wide range of carnivores and some non-carnivorous species. Initially identified in domestic dogs (*Canis familiaris*), CDV has emerged as a major multi-host pathogen affecting wildlife globally. Its ability to cross species barriers and cause high mortality in immunologically susceptible populations has resulted in repeated outbreaks and population declines, posing a serious conservation concern.

Etiology and Viral Features

CDV belongs to the genus *Morbillivirus* within the family *Paramyxoviridae*. It is an enveloped, single-stranded negative-sense RNA virus with an approximately 15.7 kb genome. The virus encodes six structural proteins: nucleocapsid (N), phosphoprotein (P), matrix (M), fusion (F), haemagglutinin (H) and RNA-dependent RNA polymerase (L). Among these, the H protein is primarily responsible for host cell attachment and serves as the key determinant of host range and genetic variability. Although CDV is antigenically classified as a single serotype, it exhibits significant genetic diversity with multiple globally distributed genotypes, including America, Europe, Asia, Africa, Arctic and wildlife-associated lineages, largely driven by variation in the H gene. The virus is environmentally fragile and is rapidly inactivated by heat, ultraviolet radiation, desiccation, detergents and lipid solvents, reflecting its enveloped structure and limited environmental stability.

Host Range

CDV has one of the broadest host ranges among morbilliviruses, primarily affecting members of the order Carnivora. Within Canidae, domestic dogs act as the principal reservoir, while

infections are also reported in African wild dog (*Lycaon pictus*), grey wolf (*Canis lupus*), coyote (*Canis latrans*), jackals (*Canis aureus, Canis mesomelas*), foxes including red, Arctic and bat-eared fox and dingo (*Canis dingo*). In Felidae, CDV has been documented in lion (*Panthera leo*), tiger (*Panthera tigris*, including Amur tiger), leopard (*Panthera pardus*), snow leopard (*Panthera uncia*), clouded leopard (*Neofelis nebulosa*), leopard cat, jungle cat, fishing cat and domestic cat, often with subclinical infection. Other susceptible families include Mustelidae (ferret, mink, otters, weasels, badgers), Procyonidae (raccoon, coati, kinkajou), Ursidae (black bear, brown bear, giant panda), Viverridae (civets, genets, linsangs, palm civet in India), Hyaenidae (spotted and brown hyena) and Ailuridae (red panda). Beyond carnivores, occasional infections or exposure have been reported in non-human primates such as *Macaca mulatta* and *Macaca fascicularis*, seals including Baikal and Caspian seals, porpoises associated with related morbilliviruses and rare cases in peccaries

Transmission and Epidemiology

Domestic dogs act as the principal reservoir of CDV and transmission occurs mainly through aerosolized respiratory droplets and direct contact with ocular, nasal and oral secretions. Viral shedding may also occur via urine, faeces and skin exudates during the acute phase of infection. Shedding can persist for up to 90 days, even in subclinical cases, thereby contributing to silent transmission within susceptible populations. Due to its environmental fragility, CDV survival outside the host is limited and spread depends largely on close contact between infected and susceptible animals

Pathogenesis and Host Specificity

CDV enters the host via the respiratory or oral route, initially replicating in macrophages and tonsillar tissue before spreading to regional lymph nodes within 24 hours. This is followed by a lymphoid phase marked by dissemination to lymphoid organs, causing severe lymphoid depletion, leukopenia and immunosuppression. Subsequent viremia results in systemic spread to the gastrointestinal tract, liver (Kupffer cells) and respiratory epithelium, leading to fever and multisystem involvement. In later stages, epithelial infection via nectin-4 causes respiratory, gastrointestinal, urinary and endocrine involvement, while CNS invasion leads to demyelination, encephalitis, seizures, paralysis and ataxia. Host specificity and tissue tropism are governed by interactions between the viral H protein and cellular receptors, primarily SLAM (CD150) on immune cells during early infection and nectin-4 on epithelial cells during later stages. Variations in key amino acid residues of the H protein, particularly at position 549, influence receptor binding affinity, host adaptation and cross-species transmission.

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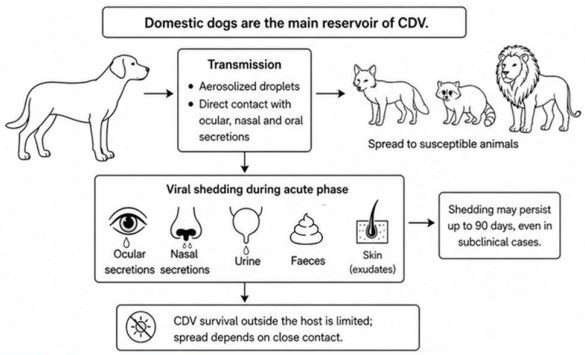

Figure: Transmission and Epidemiology of CDV

Pathology

Gross lesions: Pneumonia, enteritis, lymph node enlargement or depletion and neurological degeneration.

Microscopic lesions: Bronchointerstitial pneumonia, lymphoid depletion, syncytial cells and eosinophilic intracytoplasmic/intranuclear inclusion bodies in lungs, kidney, intestine, urinary bladder and brain. In felids, demyelinating lesions may be less pronounced than in canids.

Clinical Disease in Wildlife

Disease ranges from subclinical infection to acute fatal disease. Common signs include fever, ocular/nasal discharge, respiratory distress, gastrointestinal signs, neurological dysfunction and dermatological changes such as hyperkeratosis and enamel defects. Chronic neurological disease often leads to fatal encephalitis.

Diagnosis

Diagnosis of CDV infection involves a combination of histopathological, immunological, molecular, serological and virological methods.

Histopathological examination typically reveals characteristic inclusion bodies along with tissue necrosis in affected organs. Immunohistochemistry enables detection of viral antigens across multiple tissues, including occasional unusual sites such as the pancreas in tigers. Molecular techniques such as RT-PCR targeting N, H and P genes, real-time PCR and nucleotide sequencing followed by phylogenetic analysis are widely used for sensitive and specific detection as well as strain characterization. Serological diagnosis includes virus neutralization tests, considered the gold standard, along with ELISA and indirect fluorescent antibody tests (IFAT). Virus isolation can be performed using Vero-SLAM cell lines for confirmation and further characterization of the virus.

Molecular Epidemiology

CDV has a single serotype but multiple evolving genotypes. The H gene shows highest variability, leading to geographic clustering: Asia-1, Asia-2, Africa, Europe/South America and wildlife-specific strains. Spillover from domestic dogs to wildlife is the dominant transmission pattern.

Conservation Impact

CDV has been associated with major wildlife population declines across multiple regions and species. Notable impacts include a severe outbreak in African lions in the Serengeti, resulting in approximately one-third population loss, repeated pack collapse events in African wild dogs and critical endangerment episodes in Ethiopian wolves. In addition, infections have been documented in Amur tigers, while mortality has been reported in captive giant pandas and black-footed ferrets have experienced near-extinction level impacts during outbreaks. In India, CDV has been confirmed in multiple wild felids, with evidence of ongoing circulation in both captive and free-ranging populations, highlighting its continued threat to wildlife conservation.

Treatment and Control

There is no specific antiviral treatment for CDV and disease management is primarily supportive and symptomatic. Experimental antiviral approaches, including polymerase inhibitors, fucoidan, flavonoids, phenolic acids, mesenchymal stem cells and silver nanoparticles, have demonstrated partial inhibitory effects against viral replication in laboratory studies. Vaccination remains the cornerstone of prevention, with modified live virus vaccines such as Onderstepoort and Rockborn strains being widely used, although their application in wildlife is limited by safety concerns. Recombinant canarypox-vectored vaccines have shown promising protective efficacy in several species. However, overall control of CDV is constrained by logistical challenges in wildlife vaccination, limited accessibility of target populations and continuous spillover from domestic dog reservoirs.

Conclusion

CDV is one of the most important infectious threats to global carnivore conservation. Its wide host range, strong immunosuppressive ability and persistent spillover from domestic dogs make it difficult to control. Effective management requires integrated surveillance, molecular epidemiology, improved vaccination strategies and strict reduction of contact between domestic dogs and wildlife populations.

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