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A comprehensive review of canine parvovirus infection

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ABSTRACT

Canine Parvovirus 2 (CPV-2) remains one of the most significant and lethal infectious diseases of domestic dogs worldwide, decades after its pandemic emergence in the late 1970s. This comprehensive review synthesizes the current, multifaceted understanding of the virus. We explore its etiology as a non-enveloped, single-stranded DNA virus, its origin from Feline Panleukopenia Virus, and its rapid evolution into globally distributed antigenic variants (CPV-2a, 2b, 2c). The pathogenesis is detailed, highlighting a fecal-oral transmission route and a profound tropism for rapidly dividing cells, which dictates the classic clinical presentation of severe hemorrhagic gastroenteritis and profound leukopenia. This review critically examines the cornerstones of treatment, which remains aggressive supportive care centered on fluid resuscitation, broad-spectrum antibiotics to combat secondary sepsis, and antiemetic therapy. The critical role of vaccination with modified-live virus (MLV) vaccines is emphasized as the primary tool for prevention. We also address the central challenge in immunization: navigating the "window of susceptibility" created by maternally derived antibody (MDA) interference, which persists as the primary cause of vaccine failure in puppies. Furthermore, this review addresses the formidable challenges of control, driven by the virus's exceptional environmental resilience, which necessitates specific disinfection protocols and strict biosecurity. Finally, the significant economic impact and public health importance are discussed, underscoring the disease's substantial (though indirect) societal burden through high treatment costs, the psychological distress of "economic euthanasia," and its status as a paradigm for emerging infectious diseases.

Introduction

Canine parvovirus (CPV) is a member of the *Parvoviridae* family and the *Parvovirinae* subfamily, both of which are known to infect vertebrate species. The *Parvovirinae* subfamily is categorized into three genera: Parvovirus, Erythrovirus, and Dependovirus. CPV is classified under the feline parvovirus subgroup of the Parvovirus genus (Ogbu *et al*., 2017). Canine parvovirus (CPV) represents one of the most important infectious agents affecting domestic dogs worldwide, comprising two types, CPV-1 and CPV-2, both associated with severe clinical disease in canines. CPV-1, also known as the Minute Virus of Canines, is genetically unrelated to CPV-2, whereas CPV-2 acts as a major enteropathogen causing gastroenteritis in dogs and puppies. CPV-2 is a non-enveloped virus with an icosahedral capsid containing single-stranded DNA (Tuteja *et al*., 2022).

Dogs infected with canine parvovirus (CPV) typically develop clinical illness within 3–7 days, characterized by severe gastroenteritis, lethargy, vomiting, and often bloody diarrhea (Terzungwe *et al*., 2018). Mortality rates vary depending on treatment and vaccination status, reaching up to 91% in untreated or unvaccinated dogs, whereas timely and appropriate supportive therapy markedly improves survival to 68–95%. In puppies, CPV infection may also result in fatal myocarditis due to cardiac inflammation. Despite the existence of effective vaccines, recurrent outbreaks of the disease continue to occur worldwide (Kelman *et al*., 2019).

The persistence of CPV outbreaks is primarily attributed to the virus's high environmental stability and transmissibility (Voorhees *et al*., 2019). Moreover, interference of maternally derived antibodies with early vaccination in young animals remains a major obstacle to effective disease control (Wiedermann *et al*., 2016). Genetic variation, viral recombination, and co-infection further exacerbate clinical severity and complicate prevention and control efforts (Castillo *et al*., 2020).

Canine parvovirus (CPV) remains a major contributor to illness and death among young dogs worldwide, causing substantial economic losses that extend beyond treatment costs. These losses encompass disruptions in breeding programs, financial strain on animal shelters, and negative impacts on pet ownership, making CPV an important issue in both veterinary and public health sectors (Kelman *et al*., 2019). Mortality in untreated cases can surpass 90%, whereas intensive supportive therapy and hospitalization can increase survival to over 90% (Mazzaferro, 2020). Although widespread vaccination campaigns have targeted CPV-2, the virus continues to be a primary cause of puppy mortality in many regions (Decaro, 2020). Molecular studies utilizing advanced diagnostic methods have identified ongoing genetic variations and novel viral strains that may compromise vaccine performance and influence disease severity. Therefore, continuous molecular surveillance and periodic vaccine updates are essential to address these evolving CPV genotypes (Ogunlaja and Oginni, 2022). Therefore, this comprehensive review aims to synthesize the most current and critical findings regarding canine parvovirus infection. Specifically, this review details the recent evolutionary dynamics of CPV-2 variants, evaluates corresponding advancements in molecular diagnostics, and assesses current strategies for treatment and prevention, providing an up-to-date resource for professionals in the field.

Etiology

Both canine parvovirus type 1 (CPV-1), also called the minute virus of canines (MVC), and canine parvovirus type 2 (CPV-2) are members of the *Parvoviridae* family but differ markedly in genetic and antigenic composition, belonging to separate genera (Tuteja *et al*., 2022). Based on the classification by the International Committee on Taxonomy of Viruses, CPV-2 is designated as Carnivore Protoparvovirus 1 under the genus Protoparvovirus, while CPV-1 is grouped within Bocaparvovirus together with bovine and human bocaviruses (Cotmore *et al*., 2019). Both viruses are

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non-enveloped, small, single-stranded DNA viruses approximately 25 nm in size (Tuteja *et al*., 2022). CPV-1 shows closer genetic affinity to bovine parvovirus than to other mammalian parvoviruses, sharing only 25–26% nucleotide and 12–13% amino acid identity with CPV-2, indicating clear genetic divergence (Jager *et al*., 2021). The genome of CPV-1 encodes three major open reading frames, one of which produces a 570-residue protein, whereas CPV-2, belonging to Protoparvovirus, exhibits a distinct genomic organization (Cotmore *et al*., 2019).

Genomically, CPV-2 possesses two open reading frames that encode non-structural (NS1, NS2) and structural (VP1, VP2) proteins, with VP2 acting as the dominant capsid protein responsible for antigenic specificity (Tuteja *et al*., 2022). CPV-2 is considered to have evolved from the feline panleukopenia virus (FPV) through host adaptation involving six to seven amino acid substitutions in the VP2 domain interacting with the host transferrin receptor (Li *et al*., 2022). This ancestral strain later diversified into three main antigenic variants CPV-2a, CPV-2b, and CPV-2c defined by amino acid substitutions at positions 297, 305, and 426, altering antigenicity and host preference. Due to its non-enveloped structure, CPV demonstrates exceptional resilience in the environment, retaining infectivity for extended periods under unfavorable conditions (Dunowska *et al*., 2025).

History

Canine parvovirus type 2 (CPV-2) first appeared in the late 1970s as a novel pathogen of domestic dogs and rapidly disseminated worldwide within two years. Initially identified in 1978, it caused severe outbreaks of hemorrhagic gastroenteritis and subacute myocarditis in kennels and shelters (Miranda and Thompson, 2016a). CPV-2 originated from feline panleukopenia virus (FPV) or a closely related carnivore parvovirus through several mutations in the VP2 gene that modified host receptor binding specificity (Voorhees *et al*., 2019). The designation "CPV-2" distinguished it from the pre-existing canine parvovirus type 1 (CPV-1), also known as the minute virus of canines (MVC) (Tuteja *et al*., 2022). CPV- 1, first isolated from healthy dogs in 1967, was once considered nonpathogenic but is now recognized to cause mild to severe respiratory and enteric diseases in neonatal puppies (Jager *et al*., 2021).

Shortly after its emergence, the original CPV-2 strain was replaced by a new variant, CPV-2a, in 1979. Subsequent substitutions at the VP2 residue 426 produced variants CPV- 2b and CPV-2c, identified around 1984 and 2000, respectively. These antigenic and genetic changes were associated with altered host ranges, including enhanced replication capacity in cats and variable infectivity in canine and other cell cultures (Miranda and Thompson, 2016b). CPV-2c, characterized by glutamic acid at position 426 of the VP2 capsid, was first detected in Italy in 2000 and later spread globally (Battilani *et al*., 2019).

Evolutionary studies revealed that CPV exhibits an exceptionally high mutation rate, comparable to RNA viruses, influencing disease diagnosis and epidemiology (Balboni *et al*., 2019). Since its initial detection, the virus has continued to diversify into numerous antigenic variants worldwide (Umar *et al*., 2024). A newly identified CPV-2a mutant (VP2 324Leu) highlights the need for ongoing molecular surveillance to monitor the spread and evolution of emerging strains (Balboni *et al*., 2019). The virus's global persistence and genetic diversification are largely driven by its high mutation frequency and strong selective pressure from host immunity and vaccination (Zhou *et al*., 2017).

Epidemiology

Canine parvovirus (CPV) is globally distributed and affects dogs of all breeds, ages, and geographic regions, with both CPV-1 and CPV-2 infections reported in Asia, Australia, New Zealand, the Americas, Europe, and Africa (Duque-García *et al*., 2017; de Oliveira *et al*., 2018; O'Neill *et al*., 2025). Since its emergence in 1978, CPV-2 has rapidly spread worldwide within two years, becoming one of the most important viral pathogens of domestic and wild canids (Miranda and Thompson, 2016a). Puppies aged approximately six weeks to six months are the most susceptible to CPV-2 infection, while CPV-1 primarily affects neonatal puppies less than two weeks old (Leopardi *et al*., 2022). In a study from India, mongrels were the most affected breed with a prevalence rate of 51.16%, followed by the Labrador Retriever at 9.68%, and males were more frequently infected than females, at 63.57% and 34.43%, respectively (Zhou *et al*., 2024).

The prevalence of CPV infection varies widely among geographical regions, ranging from 16.5% to 70%, depending on the population examined, diagnostic methods, and vaccination coverage (Umar *et al*., 2024). In India, an overall prevalence of 41.15% was reported in dogs with gastroenteritis (Zhou *et al*., 2024). In Egypt, CPV infection was detected in 59.7% of dogs showing clinical signs of parvovirus (Ammar *et al*., 2024). In Ghana, a cross-sectional study recorded a prevalence rate of 61.14% among dogs presented to a veterinary teaching hospital between August 2023 and July 2024 (Agbota *et al*., 2025).

An epidemiological investigation in Baghdad revealed infection rates of 58.3% in dogs up to six months of age and 11.11% in those older than 18 months (Oleiwi *et al*., 2025). In Nigeria, CPV prevalence in the southeastern region showed significant variation depending on vaccination status and age (Ukwueze *et al*., 2018).

Molecular studies reveal notable genetic diversity among CPV variants. In Ecuador, CPV-2b was the predominant genotype (84.54%) during 2022–2023 (Loor-Giler *et al*., 2025). In China, CPV-2c has shown a continuous rise since its first detection in 2009, surpassing CPV-2a in prevalence (Zhou *et al*., 2017). Global genomic analyses identified CPV-2a (37.55%) and CPV-2c (36.02%) as the dominant variants, exceeding CPV-2b (14.17%) and CPV-2/2-like (12.26%) (Zhang *et al*., 2010). CPV-2a predominates in Asia, whereas CPV-2c is more common in South America and has gradually replaced CPV-2a in several regions (Li *et al*., 2025). In Nigeria, CPV-2c (91.5%) was the major strain, genetically linked to Asian variants (Ndiana *et al*., 2021). Studies from Uruguay reported replacement of former CPV-2c strains by CPV-2a of Asian origin (Grecco *et al*., 2018), while in Europe, CPV-2c, first reported in Italy in 2000, is now widespread in Italy, Portugal, and Germany (Dei Giudici *et al*., 2017). CPV has also been detected in various wild and domestic species, including raccoons, mink, otters, coyotes, yaks, cats, and raccoon dogs (Ikeda *et al*., 2002). In Canada, CPV-2 cases peak between July and September, whereas in tropical and subtropical regions infections occur year-round, with higher rates in spring and winter in India (Zhou *et al*., 2024).

Pathogenesis

After oronasal exposure, CPV-1 and CPV-2 replicate initially in lymphoid tissues but differ in cell and organ tropism. Within one to two days post-infection, CPV-2 multiplies in the oropharyngeal and mesenteric lymph nodes before spreading through viremia (Fagbohun *et al*., 2020). Transmission occurs mainly by the fecal–oral route or contact with contaminated objects. The virus preferentially infects rapidly dividing tissues, such as intestinal epithelial cells and lymphocytes. CPV-2 exhibits a specific affinity for the intestinal crypt epithelium, lymphoid tissues, and bone marrow hematopoietic cells. Peak viremia occurs at three to four days post- infection, when viral dissemination causes destruction of mitotically active cells (Parrish and Kawaoka, 2005). Table 1 summarizes the sequential pathogenesis of canine parvovirus infection, from initial oronasal exposure and lymphoid replication to systemic dissemination, molecular mechanisms of apoptosis, intestinal and bone marrow damage, host immune response, and factors associated with disease severity and prognosis.

At the molecular level, CPV-2 activates caspase-8 and caspase-12, inducing the accumulation of reactive oxygen species and reducing mitochondrial membrane potential, which releases cytochrome c. The virus binds to transferrin receptor 1 (TfR1), facilitating endocytic entry into ly-

Table 1. Pathogenesis and molecular mechanisms of canine parvovirus infection.

Pathogenesis stage	Pathological process	Clinical impact / Consequence	Reference
Initial exposure and primary replication	Following oronasal exposure, CPV-1 and CPV-2 initially replicate in lymphoid tissues	Early infection before systemic dissemination	(Fagbohun *et al*., 2020)
Dissemination through viremia	Within 1–2 days post-infection, CPV-2 proliferates in the oropharyngeal and mesenteric lymph nodes, then spreads through the bloodstream	Viral dissemination to multiple target organs	(Parrish and Kawaoka, 2005)
Tissue tropism	The virus infects rapidly dividing tissues such as intestinal epithelium, lymphocytes, and bone marrow	Damage to actively proliferating tissues	(Parrish and Kawaoka, 2005)
Intestinal mucosal damage	CPV-2 targets intestinal crypt epithelial cells, leading to villous atrophy and disruption of mucosal integrity	Hemorrhagic enteritis, severe diarrhea, and bacterial translocation	(Mazzaferro, 2020)
Hematopoietic suppression	Replication in the bone marrow and lymphoid organs causes destruction of hematopoietic cells	Leukopenia, neutropenia, lymphopenia, and immunodeficiency	(Mylonakis *et al*., 2016)
Molecular mechanisms of apoptosis	Activation of caspase-8 and caspase-12, increased ROS production, decreased mitochondrial membrane potential, and cytochrome c release	Cellular apoptosis and tissue damage	(Zhou *et al*., 2017)
Cell entry pathway and cell cycle disruption	Binding to TfR1 facilitates endocytosis into lysosomes and activates EGFR/p27 and STAT3/cyclin D1 pathways	Cell cycle arrest and enhanced viral replication	(Zhou *et al*., 2017)
Association between viral load and prognosis	High viral load in blood and feces correlates with disease severity	Poor prognosis and increased mortality	(Mylonakis *et al*., 2016)
Host immune response	Early interferon release, NK cell activation, and formation of neutralizing antibodies	Viral clearance and tissue recovery	(Mylonakis *et al*., 2016)
Predisposition to secondary infection	Leukopenia reduces immune defense against opportunistic bacteria	Sepsis and increased mortality	(Mazzaferro, 2020)
Lesions in puppies	Young puppies have a higher rate of cell turnover	More severe lesions and faster disease progression	(Mazzaferro, 2020)

sosomes and activating EGFR (Y1086)/p27 and STAT3 (Y705)/cyclin D1 signaling, leading to cell cycle arrest (Zhou *et al*., 2017). Destruction of intestinal crypts results in villous atrophy, impaired mucosal integrity, and hemorrhagic enteritis, allowing bacterial translocation into the bloodstream (Mazzaferro, 2020). Replication in bone marrow and lymphoid organs causes profound leukopenia particularly neutropenia and lymphopenia leading to immunosuppression (Mylonakis *et al*., 2016).

Higher viral loads in blood and feces correlate with increased disease severity and poor prognosis. Host defense relies on early interferon release, natural killer cell activation, and production of virus-neutralizing antibodies crucial for viral clearance (Mylonakis *et al*., 2016). Recovery depends on adequate antibody formation and regeneration of lymphoid and intestinal cells. Immunosuppression due to leukopenia predisposes infected animals to secondary bacterial infections, significantly increasing mortality (Mazzaferro, 2020). Younger puppies exhibit more severe lesions because of higher cell turnover in developing tissues.

Clinical manifestations

Clinical manifestations of canine parvovirus infections differ notably between CPV-1 and CPV-2, with CPV-2 producing severe hemorrhagic gastroenteritis and myocarditis, whereas CPV-1 mainly affects the respiratory tract of neonatal puppies (Tuteja *et al*., 2022). CPV-2 infection typically presents after an incubation period of three to seven days with nonspecific enteric signs such as anorexia, lethargy, vomiting, and diarrhea ranging from mucoid to hemorrhagic, often accompanied by dehydration and fever (Mylonakis *et al*., 2016). The diarrhea is frequently watery with a metallic odor and may contain blood, leading to rapid fluid loss and severe dehydration (Oliveira *et al*., 2018). Marked leukopenia, commonly below 2000–3000 cells/μL, arises from neutropenia and lymphopenia due to bone marrow and lymphoid tissue destruction (Mylonakis *et al*., 2016). Clinically, affected dogs exhibit sunken eyes, dry mucous membranes, prolonged capillary refill time, weak pulse, and cold extremities, which, if untreated, can progress to hypovolemic shock.

Myocardial infection by CPV-2 may occur in puppies infected transplacentally or during the early postnatal period when cardiomyocytes remain mitotically active, resulting in acute myocarditis that can progress to sudden death or congestive heart failure (Ford *et al*., 2017). Infection acquired in utero or within the first two weeks of life can damage the developing myocardium, with clinical manifestations such as heart failure or sudden death appearing up to two months later (Kilian *et al*., 2018). Some CPV-2–infected dogs exhibit systemic inflammatory response syndrome (SIRS) upon presentation, which is linked to poor prognosis (Mylonakis *et al*., 2016).

In contrast, canine parvovirus type 1 (CPV-1), or minute virus of canines, produces a spectrum of respiratory disease in neonatal puppies ranging from subclinical to fatal (Dines *et al*., 2023). CPV-1 may cause pneumonia, myocarditis, and enteritis in young dogs and transplacental infection in pregnant females, leading to fetal death or embryo resorption (Ford *et al*., 2017). Pathologic findings in CPV-1 (also termed canine bocavirus-1 or MVC) infection include bronchitis, interstitial pneumonia, and enteritis, consistent with its respiratory and enteric tropism (Velez *et al*., 2023). Canine bocavirus type 2 (CBoV-2) DNA has been identified in the lungs, liver, lymph nodes, and intestines of infected dogs, though histopathological changes were minimal, suggesting uncertain pathogenic significance (Doulidis *et al*., 2024). Disease caused by CPV-1 is generally less severe than that of CPV- 2, with experimental infections producing only mild to moderate clinical signs compared to the highly virulent CPV-2 variants (Tuteja *et al*., 2022).

Diagnosis

Definitive diagnosis of canine parvovirus (CPV) infection is based on identifying viral antigen, DNA, or virus-specific antibodies alongside consistent clinical and hematologic findings, although diagnostic methods differ for CPV-1 and CPV-2 (Tuteja *et al*., 2022). In veterinary practice, the fecal antigen enzyme-linked immunosorbent assay (ELISA) remains the most common point-of-care method for CPV-2 detection, typically performed using rectal swabs. Lateral flow assays have also been evaluated, but their sensitivity varies according to viral load and sampling time (Marulappa and Kapil, 2009). Fecal testing is recommended for any puppy exhibiting vomiting and diarrhea (Mazzaferro, 2020). Table 2 summarizes the principal diagnostic methods for canine parvovirus (CPV) infection, including antigen detection, molecular assays, and hematologic findings, along with their diagnostic features, advantages, limitations, and relevant references.

Polymerase chain reaction (PCR) assays targeting the VP2 gene offer greater sensitivity than ELISA and can identify low viral loads of both

Table 2. Diagnostic approaches for canine parvovirus infection

Diagnostic method	Principle / Target	Key findings / Features	Advantages	Limitations	References
Fecal Antigen ELISA	Detection of CPV antigen in feces (rectal swab)	Rapid identification of CPV-2 infection in clinically affected dogs	Widely used, rapid, point-of-care test	Lower sensitivity compared to PCR; false negatives in early or late infection; may detect vaccinal virus	(Mazzaferro, 2020; Tuteja *et al*., 2022)
Lateral Flow Assay	Immunochromatographic detection of CPV antigen	Quick field-based screening tool	Simple, rapid, portable	Variable sensitivity depends on viral load and sampling time	(Marulappa and Kapil, 2009)
Polymerase Chain Reaction (PCR)	Detection of viral DNA (VP2 gene)	Identifies CPV-1 and CPV-2, including low viral loads	High sensitivity and specificity	May detect vaccine strains or subclinical infections, complicating interpretation	(Tuteja *et al*., 2022)
Quantitative Real-Time PCR (qPCR)	Quantification of viral DNA	Determines viral load (10^9 to single gene copy); correlates with disease severity	अत्यंत sensitive; prognostic value	Same limitations as PCR; requires specialized equipment	(Tuteja *et al*., 2022)
Hematologic Analysis	Evaluation of blood cell counts	Leukopenia, neutropenia (<1,000 cells/μL), lymphopenia; rebound during recovery	Supports clinical diagnosis and prognosis	Non-specific; cannot confirm infection alone	(Mylonakis *et al*., 2016; Tuteja *et al*., 2022)
Clinical Assessment	Observation of clinical signs	Vomiting, diarrhea, dehydration; indication for fecal testing	Essential for early suspicion	Non-specific signs	(Mazzaferro, 2020)

CPV-1 and CPV-2 (Tuteja *et al*., 2022). Quantitative real-time PCR (qPCR) additionally measures viral load, which is correlated with disease severity and prognosis. A highly sensitive qPCR assay has been established for CPV- 2 detection, capable of identifying viral quantities from 10^9 to a single gene copy. Despite superior sensitivity, PCR may complicate interpretation because it can detect attenuated vaccine strains or asymptomatic infections. When ELISA results are negative but infection remains suspected particularly in breeding kennels or shelters PCR confirmation is advised (Mazzaferro, 2020).

Hematologic analysis in CPV-2 cases generally shows marked leukopenia, primarily due to neutropenia (often <1,000 cells/μL) and lymphopenia (Mylonakis *et al*., 2016). CPV-2 infection suppresses leukocyte production, leading to profound depletion during acute phases and rebound counts during recovery (Tuteja *et al*., 2022). Clinical improvement usually parallels the restoration of leukocyte numbers. False-negative ELISA results can occur early in infection, during declining viral shedding, or in animals with excessive viral burden. Vaccination history should be considered, as both assays may detect vaccinal virus 4–10 days post-immunization. Fecal samples require at least 10^6 DNA copies per milligram for a positive ELISA result (Mazzaferro, 2020).

Differential diagnosis

The clinical features of canine parvovirus (CPV) infection resemble those of various acute gastroenteric and respiratory diseases in dogs, depending on whether CPV-1 or CPV-2 is involved (Mazzaferro and Powell, 2013). In CPV-2 infection, the combination of vomiting, hemorrhagic diarrhea, and acute leukopenia is highly indicative; however, differential diagnoses should include canine distemper, infectious canine hepatitis, parasitic enteritis, and other gastrointestinal disorders (Tuteja *et al*., 2022). Distemper must be considered in unvaccinated puppies showing gastrointestinal signs with concurrent respiratory or neurological manifestations. Canine coronavirus (CCoV) generally induces non-hemorrhagic enteritis, but in certain cases, virulent strains can cause hemorrhagic diarrhea, systemic illness, and leukopenia (Mylonakis *et al*., 2016). In neonatal dogs, respiratory infection due to CPV-1 must be distinguished from pneumonia caused by canine herpesvirus, bacterial pathogens, or other viruses to ensure accurate diagnosis (Li *et al*., 2025).

Bacterial diseases such as Salmonellosis, Campylobacteriosis, and *Clostridium* perfringens infection can mimic CPV-2 with hemorrhagic diarrhea, though they rarely produce the marked leukopenia typical of parvoviral enteritis. Intestinal parasites, including Ancylostoma caninum and Trichuris vulpis, may lead to bloody diarrhea and anemia in puppies but progress more gradually. Notably, prior anthelmintic treatment reduces the risk of CPV-2 infection (odds ratio 0.45). Mixed infections with canine circovirus, Giardia, Cryptosporidium, or coronavirus further complicate disease outcomes (Mazzaferro, 2020).

Hemorrhagic gastroenteritis (HGE) or acute hemorrhagic diarrhea syndrome (AHDS) presents similarly, with acute bloody diarrhea and hemoconcentration, but primarily affects adult small-breed dogs. Additional differentials in puppies with vomiting and diarrhea include dietary indiscretion, foreign body obstruction, or intussusception. Canine enteric coronavirus often causes mild diarrhea in puppies under six weeks old and may occur alongside CPV-2 variants. Complications such as aspiration pneumonia, hypoglycemia, hypoalbuminemia with edema, or intussusception increase morbidity and hospitalization time in CPV-2 cases (Mazzaferro, 2020). Chronic conditions like inflammatory bowel disease or food hypersensitivity rarely present as acute hemorrhagic enteritis (Mylonakis *et al*., 2016). In neonatal respiratory disease, minute virus of canines (CPV-1) must be differentiated from fading puppy syndrome, congenital defects, or septicemia to ensure precise diagnosis (Decaro *et al*., 2020).

Transmission

Canine parvovirus exhibits multiple transmission pathways depending on its type. CPV- 2 is primarily transmitted via the fecal-oral route, whereas CPV-1 can spread through respiratory secretions, feces, and transplacental infection. CPV-2 rapidly disseminates among dogs through direct fecal-oral contact or indirectly via oro-nasal exposure to virus- contaminated fomites (Ammar *et al*., 2024). Both CPV-1 and CPV-2 are highly resistant and ubiquitous in contaminated environments, easily adhering to inanimate surfaces (Mazzaferro, 2020). Infection occurs predominantly through oral or nasal exposure to virus-contaminated feces or fomites, including equipment, personnel, and the surrounding environment, underscoring the need for strict hygiene and biosecurity practices (Behdenna *et al*., 2019).

Infected dogs begin shedding CPV-2 in feces within three to four days after infection, often before clinical signs appear (Mazzaferro, 2020). During acute infection and for up to two weeks post-recovery, viral shedding occurs in large quantities, contributing significantly to environmental contamination and transmission (Freisl *et al*., 2017). Most infected dogs shed the virus for about seven to ten days, though some may continue for three to four weeks, and CPV-2c–infected dogs have been reported to shed for up to 51 days (Mazzaferro, 2020). This prolonged shedding period emphasizes the persistent environmental risk even after apparent clinical recovery.

Mechanical transmission via fomites also plays a crucial role, as vi-

ral particles can easily adhere to shoes, clothing, equipment, and vehicle tires (Golke *et al*., 2025). The extremely contagious nature of CPV-2 and its very low infectious dose fewer than 1,000 viral particles make even minimal contamination sufficient for infection. CPV-1 can cause transplacental infections in pregnant bitches, leading to embryonic resorption or fetal death, a route not typically observed in CPV- 2 (Sykes, 2014). Additionally, insects and rodents may act as mechanical vectors for both viral types (Mazzaferro, 2020). Wildlife such as foxes, wolves, and coyotes can become infected and shed the virus, serving as potential reservoirs for CPV-1 and CPV-2 (Zhou *et al*., 2017). Evidence from Australia indicates interspecies transmission between wild and domestic dogs, highlighting the epidemiological significance of wildlife reservoirs (Kelman *et al*., 2020).

Risk factors

Susceptibility to Canine Parvovirus (CPV) infection is a multifactorial phenomenon, with host-intrinsic factors being the most significant predictors of risk (Le *et al*., 2020). Vaccination status serves as the primary determinant, as unvaccinated dogs show a markedly greater likelihood approximately 11.76 times higher of developing clinical disease compared to those vaccinated (Doan *et al*., 2020). This increased vulnerability is closely tied to age and the dynamics of maternally derived antibodies (MDA). Puppies between six weeks and six months of age are especially at risk, passing through a "window of susceptibility" in which diminishing maternal antibodies are insufficient for protection yet still capable of interfering with vaccine-induced immunity (Jumaa *et al*., 2021). Table 3 summarizes the principal host, pathogen, and environmental risk factors associated with susceptibility to canine parvovirus infection and disease severity in dogs, together with their clinical implications and supporting references.

Beyond immunity, other host factors include a distinct breed predilection, with breeds such as Rottweilers, Doberman Pinschers, and German Shepherds consistently overrepresented in severe clinical cases, suggesting a potential genetic component to susceptibility (Sykes, 2014). Furthermore, the integrity of the gastrointestinal system is crucial, as concurrent infections with other enteric pathogens (e.g., Giardia and Canine Coronavirus) can significantly exacerbate mucosal damage and worsen disease severity (Jumaa *et al*., 2021). Compounding these host risks is the nature of the pathogen itself; as a non- enveloped DNA virus, CPV exhibits exceptional environmental resilience, capable of remaining infectious on surfaces and in soil for months to years, posing a persistent contamination threat (Zhou *et al*., 2024).

This remarkable environmental stability dictates the primary transmission routes and identifies key environmental risk factors. The virus's resistance to many common detergents means that fomite transmission via contaminated kennels, food bowls, soil, and even human hands or clothing is a highly effective route of infection. Consequently, risk escalates dramatically in high-density canine populations, such as animal shelters, breeding kennels, and pet stores, where stress and high contamination loads facilitate rapid outbreaks (Nandi and Kumar, 2010). Recent epidemiological studies have also highlighted a strong socioeconomic and geographic component; CPV clusters are significantly associated with postcodes experiencing greater socioeconomic disadvantage, which often correlates with reduced access to veterinary care and lower vaccination compliance (Kelman *et al*., 2020; Kantere *et al*., 2021).

Public health importance

While Canine Parvovirus (CPV-2) is definitively not a zoonotic pathogen posing no direct infectious threat to humans, its public health importance is significant and multifaceted, best understood through the indirect societal consequences articulated by the One Health framework. The virus's primary impact is on the human component of the human-animal bond, a relationship recognized for its contributions to human psychological well-being (Walsh, 2009). The acute onset, high mortality rate, and affliction of young puppies inflict a profound emotional and psychological burden on pet owners. This distress is frequently compounded by the financial strain of treatment, often leading to "economic euthanasia" a term describing the devastating situation where owners are forced to euthanize a pet with a treatable, albeit expensive, condition (Bubeck, 2023). This scenario creates significant moral distress and contributes to the recognized mental health challenges, including compassion fatigue and burnout, faced by the veterinary professionals who must manage these high-stakes, emotionally charged cases (Sykes, 2014).

The socioeconomic ramifications extend beyond individual households to impact community resources. The intensive care required for CPV treatment, which includes aggressive fluid resuscitation, antiemetics, nutritional support, and prolonged hospitalization, represents a substantial economic burden (Mazzaferro, 2020). This financial barrier can limit treatment access, particularly in socioeconomically disadvantaged communities where vaccination compliance may already be low. This chal-

Table 3. Major risk factors associated with canine parvovirus infection

Risk factor category	Specific risk factor	Description / Mechanism	Clinical impact	References
Host-related factors	Vaccination status	Unvaccinated dogs are approximately 11.76 times more likely to develop clinical CPV disease than vaccinated dogs	Highest predictor of infection risk and disease occurrence	(Le *et al*., 2020; Doan *et al*., 2020)
	Age and maternally derived antibodies (MDA)	Puppies aged 6 weeks–6 months experience a "window of susceptibility" due to waning maternal antibodies that no longer protect but may still interfere with vaccination	Increased susceptibility in young puppies and vaccine failure risk	(Jumaa *et al*., 2021)
	Breed predisposition	Certain breeds such as Rottweilers, Doberman Pinschers, and German Shepherds show higher susceptibility, possibly due to genetic factors	Greater risk of severe clinical manifestations	(Sykes, 2014)
	Concurrent enteric infections	Co-infections with pathogens such as *Giardia* and canine coronavirus intensify intestinal mucosal damage	Increased disease severity and poorer prognosis	(Jumaa *et al*., 2021)
Pathogen-related factors	Environmental resilience of CPV	As a non-enveloped DNA virus, CPV remains infectious on surfaces and soil for months to years	Persistent environmental contamination and recurrent exposure risk	(Zhou *et al*., 2024)
Environmental factors	Fomite transmission	Transmission through contaminated kennels, food bowls, soil, clothing, and human hands	Rapid spread between susceptible dogs	(Nandi and Kumar, 2010)
	High-density dog populations	Shelters, breeding kennels, and pet stores facilitate rapid viral spread due to crowding, stress, and contamination load	Increased outbreak frequency and transmission rate	(Nandi and Kumar, 2010)
Socioeconomic / geographic factors	Socioeconomic disadvantage	Areas with reduced access to veterinary care and lower vaccination compliance show higher CPV clustering	Increased regional incidence and outbreak risk	(Kelman *et al*., 2020; Kantere *et al*., 2021)

lenge is critically amplified in the animal shelter environment. Shelters, which function as vital public service organizations, are uniquely vulnerable to CPV due to their high-density, high-turnover populations (Quimby *et al*., 2021). The extreme environmental hardiness of the virus means a single case can necessitate catastrophic operational responses, including facility-wide quarantine, costly and labor-intensive decontamination protocols, and the suspension of intake and adoption services, thereby diverting scarce resources from other essential animal welfare and public health programs, such as sterilization and rabies control (Mousazadeh *et al*., 2021).

From a broader scientific and epidemiological perspective, the emergence of CPV-2 represents a pivotal model in public health, serving as one of the most well-documented examples of a pandemic emerging infectious disease (EID). The so-called "Big Bang" emergence of CPV during the late 1970s, following a host-switch event likely originating from Feline Panleukopenia Virus, provides an exceptional real-world framework for examining the molecular processes underlying viral adaptation to novel hosts (Miranda and Thompson, 2016a). The virus's rapid worldwide spread and subsequent evolution into antigenic variants (CPV-2a, -2b, and -2c) highlight essential mechanisms of viral pathogenesis, host-range expansion, and the speed at which a new pathogen can permeate an unexposed population. This evolutionary history continues to shape surveillance systems and predictive modeling for other pathogens with pandemic potential (Jager *et al*., 2021).

Economic impact

The economic consequences of Canine Parvovirus (CPV) infection are profound, permeating multiple sectors from individual households to national animal welfare infrastructures. The most immediate and visible financial burden falls upon individual pet owners. The standard of care for CPV enteritis is notoriously expensive, demanding aggressive and prolonged hospitalization. This is not a disease amenable to simple outpatient management in its severe form. The costs are cumulative, arising from the necessity of continuous intravenous fluid therapy to manage dehydration and shock, parenteral broad- spectrum antibiotics to combat secondary sepsis (a frequent complication following bacterial translocation across the denuded intestinal barrier), potent antiemetics (e.g., maropitant and ondansetron) to control debilitating vomiting, and crucial nutritional support (Sykes, 2014). Furthermore, intensive monitoring through serial diagnostics, such as complete blood counts (to monitor for the characteristic neutropenia) and serum biochemistry, adds to the expense. In critical cases requiring plasma transfusions (for hypoproteinemia) or blood transfusions (for anemia), the cost can escalate, often reaching thousands of dollars per patient (González-Domínguez *et al*., 2024).

This substantial financial barrier to treatment directly precipitates a significant, and often tragic, secondary economic phenomenon: "economic euthanasia." Owners are frequently forced to decide between incurring catastrophic financial debt or euthanizing a pet that has a treatable condition with a fair prognosis, simply due to an inability to afford the high cost of intensive care (Horecka *et al*., 2020). This outcome highlights a severe economic disparity in animal healthcare access. The irony of this economic burden is its stark contrast with the affordability of prevention. The cost of a complete primary vaccination series is negligible compared to the financial and emotional expenditure of managing a single clinical CPV case. The economic impact also includes often-overlooked ancillary costs, such as the expense of environmental decontamination. The virus's extreme resilience requires the use of specific, potent virucides (e.g., accelerated hydrogen peroxide, sodium hypochlorite, and potassium peroxymonosulfate), as many common quaternary ammonium disinfectants are ineffective, adding a layer of cost and labor for clinics, shelters, and homes (Peng *et al*., 2023).

On a macroeconomic level, CPV poses a severe financial threat to animal welfare organizations and shelters, which operate on notoriously thin margins. An outbreak within a shelter environment is an economic catastrophe. It necessitates the immediate implementation of costly and labor-intensive quarantine protocols, the suspension of adoptions and intake (halting revenue streams and community service), and a massive diversion of staff and resources to outbreak management (Decaro and Buonavoglia, 2012). The costs of diagnostic testing for all exposed animals, coupled with providing intensive care for multiple affected individuals simultaneously, can deplete an organization's medical budget for months. In many shelter systems, the financial calculus of an outbreak, combined with the risk to the rest of the population, often leads to the mass euthanasia of exposed cohorts, representing a total and devastating economic loss (Kantere *et al*., 2021).

Finally, the virus has significant economic implications for the commercial breeding industry. For a breeder, an outbreak can mean the total loss of projected income from one or more litters, as puppy mortality rates can approach 100% in unvaccinated populations. Furthermore, the (now rare) myocarditis form of CPV historically decimated breeding programs by causing the sudden death of entire, seemingly healthy litters weeks or months after birth (Dines *et al*., 2023). Beyond the immediate loss of "product," a CPV outbreak can inflict permanent damage on a kennel's reputation, leading to long-term financial losses and diminished market trust. Collectively, CPV functions as a significant economic drain, threatening the financial viability of animal care operations, the livelihoods of breeders, and the financial stability of individual pet owners.

Treatment

The management of Canine Parvovirus (CPV) infection represents a fundamental aspect of small animal emergency and critical care; however, it continues to pose considerable clinical difficulties. As there is no specific, commercially available antiviral agent that directly targets the virus, treatment is fundamentally aggressive, symptomatic, and supportive. The primary therapeutic goal is to maintain the patient's systemic homeostasis, allowing them to survive the acute viremic and gastrointestinal phases until their own immune response bolstered by a regenerating supply of leukocytes can clear the infection (Zhou *et al*., 2024). The success of treatment hinges on meticulous supportive care aimed at counteracting the three main pillars of CPV pathophysiology: profound dehydration, secondary bacterial sepsis, and severe gastrointestinal distress. Table 4 summarizes the principal therapeutic approaches for canine parvovirus infection, focusing on supportive care strategies targeting fluid imbalance, secondary bacterial infection, and gastrointestinal recovery, along with their clinical purposes and supporting references.

The non-negotiable cornerstone of all CPV treatment protocols is aggressive fluid resuscitation and maintenance therapy. The sheer volume of fluid lost through intractable vomiting and profuse hemorrhagic diarrhea leads rapidly to hypovolemic shock, severe dehydration, and life-threatening electrolyte disturbances (Mazzaferro, 2020). Establishing intravenous (IV) access is essential, with balanced isotonic crystalloids such as Lactated Ringer's Solution or Plasmalyte-A administered to restore fluid volume and alleviate dehydration. Critical electrolyte abnormalities must be addressed; hypokalemia is extremely common due to gastrointestinal losses and requires careful supplementation in maintenance fluids. Furthermore, hypoglycemia, particularly in young, anorexic puppies with depleted glycogen stores, must be monitored for and corrected via dextrose supplementation. In cases of severe hypoalbuminemia (protein-losing enteropathy) or systemic inflammation leading to third-spacing, the use of synthetic colloids (e.g., hetastarch) or, ideally, plasma transfusions may be warranted to restore colloid oncotic pressure and provide clotting factors (Gauthier *et al*., 2015).

Controlling secondary bacterial infection is the second critical pillar of therapy. The virus targets rapidly dividing cells, causing severe neutropenia, which annihilates the patient's primary defense against bacteria.

Table 4. Therapeutic strategies for canine parvovirus infection

Therapeutic category	Intervention	Mechanism / Purpose	Clinical benefit	References
General management	Supportive therapy (no specific antiviral)	Maintains systemic homeostasis during viremic and gastrointestinal phases until immune recovery	Improves survival through immune-mediated viral clearance	(Zhou *et al*., 2024)
Fluid therapy	Intravenous isotonic crystalloids (e.g., Lactated Ringer's, Plasmalyte-A)	Restores intravascular volume and corrects dehydration	Prevents hypovolemic shock and stabilizes circulation	(Mazzaferro, 2020)
Electrolyte management	Potassium supplementation	Corrects hypokalemia due to gastrointestinal losses	Prevents cardiac and muscular complications	(Mazzaferro, 2020)
Metabolic support	Dextrose supplementation	Corrects hypoglycemia, especially in young puppies	Maintains energy balance and prevents collapse	(Mazzaferro, 2020)
Colloid support	Synthetic colloids (e.g., hetastarch) or plasma transfusion	Restores oncotic pressure and provides clotting factors	Improves vascular stability and protein balance	(Gauthier *et al*., 2015)
Antimicrobial therapy	Broad-spectrum parenteral antibiotics (e.g., ampicillin, cefazolin ± enrofloxacin)	Prevents/treats bacterial translocation and sepsis due to neutropenia and mucosal damage	Reduces mortality associated with sepsis and endotoxemia	(Mazzaferro, 2020; Al-Hasan *et al*., 2009)
Antiemetic therapy	Maropitant, ondansetron	Controls vomiting via NK-1 and 5-HT3 receptor antagonism	Improves comfort and enables nutritional support	(Yalcin and Keser, 2017)
Nutritional support	Early enteral micro-nutrition (NG/NE tube feeding)	Provides nutrients to intestinal mucosa, promoting regeneration and microbiota balance	Shortens recovery time and enhances gut healing	(Mazzaferro, 2020)
Adjunct therapy	Fecal Microbiota Transplantation (FMT)	Restores intestinal microbiota balance	Reduces diarrhea duration and hospitalization time	(Pereira *et al*., 2018)
Alternative management	Outpatient treatment (SC fluids, long-acting antibiotics, antiemetics)	Cost-effective alternative to intensive care	Increases accessibility but carries higher clinical risk	(Venn *et al*., 2017)

This immune collapse occurs simultaneously with the destruction of the intestinal crypt epithelium, which destroys the gut mucosal barrier (Mazzaferro, 2020). This "perfect storm" of immunosuppression and a compromised gut barrier facilitates the translocation of enteric bacteria (e.g., *Escherichia coli* and *Clostridium* spp.) into the bloodstream, leading to sepsis and endotoxemia, which are common causes of death. Therefore, the administration of broad-spectrum parenteral antibiotics is not elective; it is essential. A common first-line protocol involves a beta-lactam (e.g., ampicillin, cefazolin) to target gram-positive and some gram-negative organisms, often combined with a fluoroquinolone (e.g., enrofloxacin) in severe sepsis for its potent gram-negative activity, despite theoretical risks of arthropathy in growing pups (Al-Hasan *et al*., 2009).

Recent therapeutic strategies now emphasize comprehensive gastrointestinal support rather than mere symptom control (Wilson *et al*., 2014). The use of potent antiemetics such as maropitant (an NK- 1 receptor antagonist) and ondansetron (a 5-HT3 antagonist) remains essential to manage vomiting and enhance comfort; however, current nutritional management has shifted significantly (Yalcin and Keser, 2017). The previous approach of withholding food (NPO) to rest the intestines has been replaced by early enteral "micro-nutrition," which is vital for intestinal recovery. Once emesis is stabilized, gradual administration of a liquid diet through a nasogastric or nasoesophageal tube delivers nutrients directly to the intestinal mucosa, promoting villous regeneration, maintaining microbial balance, and shortening recovery time (Mazzaferro, 2020). Additional treatments such as Fecal Microbiota Transplantation (FMT) have also demonstrated potential in restoring gut microbiota health, reducing diarrhea duration, and minimizing hospitalization (Pereira *et al*., 2018). Considering the high expense of intensive care, modified outpatient regimens using subcutaneous fluids, long-acting antibiotics, and injectable antiemetics have been proposed as cost-effective alternatives, though with increased clinical risk (Venn *et al*., 2017).

Vaccination

Vaccination remains the single most effective and critical tool in preventing Canine Parvovirus (CPV) infection. The foundation of modern immunization protocols lies in the use of modified-live virus (MLV) vaccines, which contain attenuated, non-pathogenic CPV strains capable of replicating in vivo to stimulate strong and lasting immune protection involving both humoral and cell-mediated responses (Day *et al*., 2010). The proven antigenicity and efficacy of MLV CPV vaccines enable immunization even in the presence of low to moderate residual maternal antibodies. However, vaccination success is closely tied to navigating the "window of susceptibility," a crucial developmental phase when maternally derived antibodies (MDAs) decrease below protective levels but remain high enough to neutralize vaccine antigens, hindering active immune response development (Pardo *et al*., 2007).

The primary goal of the puppy vaccination series is to close this window of susceptibility. Contrary to common belief, the series does not simply "boost" immunity; instead, the repeated doses typically starting at 6–8 weeks and repeated every 2–4 weeks are intended to deliver a vaccine dose precisely when MDA levels are no longer interfering with immune activation. This timing varies greatly among individual puppies, even within the same litter (Decaro and Buonavoglia, 2012). Therefore, global vaccination guidelines, including those from the World Small Animal Veterinary Association (WSAVA), recommend that the final vaccine dose be administered at or beyond 16 weeks of age to ensure effective seroconversion, even in puppies with persistently high MDA levels (Day *et al*., 2010).

This standard protocol may require modification in high-risk environments, such as animal shelters, where dense populations, stress, and high pathogen exposure necessitate accelerated vaccination schedules. In such contexts, shelter medicine guidelines endorse high-titer, low-passage MLV vaccines for their greater ability to overcome moderate MDA interference (Decaro and Buonavoglia, 2012). Vaccination in these populations often begins earlier, around 4–6 weeks of age and continues every 2–3 weeks until 18–20 weeks to mitigate outbreak risks (Decaro *et al*., 2020). True vaccine failure in properly immunized animals is extremely rare and primarily results from MDA interference rather than vaccine deficiency. Upon completion of the primary series at or beyond 16 weeks, a booster at one year is recommended to establish a strong immunological memory. Subsequent studies have shown that immunity from core MLV CPV vaccines lasts at least three years, often lifelong, leading to the current triennial revaccination recommendation, which balances long-term protection with reduced vaccine exposure (Day *et al*., 2010).

Control

The control of Canine Parvovirus (CPV) represents a major biosecurity challenge in veterinary medicine due to the virus's exceptional environmental resilience. As a non- enveloped DNA virus, CPV remains highly infectious for months or even years, particularly in dark, moist, and organic-rich environments (Zhou *et al*., 2024). It resists freezing and is unaffected by most detergents or alcohol-based disinfectants, allowing contaminated areas such as kennels, parks, veterinary clinics, and soil to act as persistent infection sources. Therefore, effective management must extend beyond vaccination to include strict environmental disinfection and control of fomite transmission (Nandi and Kumar, 2010).

Because routine cleaning alone is ineffective, chemical disinfection forms the foundation of environmental control. Many common veterinary disinfectants, such as quaternary ammonium compounds (QATs), are inactive against CPV (Vargas *et al*., 2025). The standard virucidal agent remains sodium hypochlorite (household bleach) at a 1:30 dilution (≈5.25% solution) applied to pre-cleaned surfaces with a minimum 10-minute contact time to achieve inactivation (Cavalli *et al*., 2018). Newer disinfectants, including potassium peroxymonosulfate (e.g., Virkon S) and accelerated hydrogen peroxide (AHP), have gained favor in clinical and shelter settings for their comparable efficacy, improved safety, and material compatibility (Goyal *et al*., 2014). In high-risk environments such as shelters or hospitals, decontamination must be paired with strict outbreak protocols, including immediate isolation of symptomatic animals, dedicated staff, and barrier nursing procedures with proper PPE and hygiene (Weese, 2004). Diagnostic tests like ELISA or PCR are essential to confirm infection and identify asymptomatic carriers (Faz *et al*., 2017).

Long-term CPV control depends on achieving and maintaining herd immunity through widespread vaccination. While disinfection and isolation provide reactive control, vaccination proactively reduces the population of susceptible animals, lowering the effective transmission rate of the virus (Nandi and Kumar, 2010). When most dogs are immunized, the chance of contact between infected and susceptible hosts decreases, reducing viral persistence in the environment and indirectly protecting unvaccinated puppies in the susceptibility window. Comprehensive CPV prevention therefore requires an integrated approach combining vaccination, biosecurity, environmental hygiene, and rapid outbreak response.

Conclusion

Canine Parvovirus 2 (CPV-2), since its explosive emergence in the late 1970s, has established itself as one of the most significant and persistent canine pathogens globally, driven by its high virulence, rapid antigenic evolution (giving rise to new variants like CPV-2c), and exceptional environmental resilience. Although modified-live virus (MLV) vaccination is the cornerstone of prevention, its primary challenge is interference from maternally derived antibodies (MDA), which creates a "window of susceptibility" that remains the principal cause of vaccine failure and disease persistence in endemic areas. When prevention fails, clinical management which is solely intensive supportive care (fluid therapy, antibiotics, antiemetics) creates a substantial financial and emotional burden, often leading to "economic euthanasia" and threatening the operational viability of animal shelters. Moving forward, continued molecular surveillance, research into novel vaccine strategies to overcome MDA, and the development of affordable antivirals are essential. However, until such innovations are realized, CPV control depends on a multi-proNGED APPROACH: STRICT ADHERENCE to vaccination protocols (final dose at ≥16 weeks of age), meticulous biosecurity with proven virucides, and sustained public education.

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Conflict of interest

The authors have declared no conflict of interest.

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