



An Update on Canine Parvovirus in Ecuador and Worldwide: Pathogenesis, Diagnosis, Epidemiology, Treatment, and Control

Evelin Pamela Rodríguez Huera^{1*}  and David Fabricio Martínez Matamoros² 

¹Veterinary Medicine Program, State Polytechnic University of Carchi, Carchi 040150, Ecuador

²Veterinary Medicine Program, National University of Loja, Loja 110101, Loja, Ecuador

*Corresponding author's Email: evelin.rodriguez@upec.edu.ec



ABSTRACT

Canine parvovirus (CPV) remains one of the most relevant viral pathogens affecting domestic dogs worldwide, particularly in young and unvaccinated populations, due to its high environmental resistance and rapid transmission. The present study aimed to provide an updated and integrative overview of epidemiology, pathogenesis, diagnosis, and control of CPV through a systematic qualitative review. A comprehensive literature search was carried out using major academic databases, including PubMed, SciELO, and Scopus, considering studies published between 2020 and 2026. A total of 312 articles were initially identified, of which 70 met the inclusion criteria and were selected for article review. The results confirmed the global circulation of multiple variants (CPV-2a, CPV-2b, and CPV-2c), including the presence of CPV-2b in Ecuador, as well as the persistence of the virus in diverse environments and its impact on susceptible populations such as puppies between 6 weeks and 6 months of age and susceptibility, and some breeds such as Doberman Pinschers, Rottweilers, and American Pit Bull Terriers. Molecular diagnostic techniques, particularly PCR and qPCR, remain the most reliable tools for early detection. Despite advances in supportive therapy and emerging antiviral approaches, vaccination continues to represent the most effective preventive strategy. Continuous surveillance and improvements in diagnostic and immunization strategies are essential to reduce the impact of CPV infections.

Keywords: Canine parvovirus, Genoma, Pathogenesis, Transmission

INTRODUCTION

The most common viral infections that may affect various body systems are Canine parvovirus (CPV), distemper canine, and canine infections respiratory disease (Decaro et al., 2020). Although CPV is not considered a notifiable disease, it remains a significant health concern due to its high morbidity in susceptible canine populations and its high mortality in susceptible canine populations and its high mortality in untreated puppies (90%), despite survival rates exceeding 80-90% with intensive supportive care (Zhou et al., 2024). Nevertheless, its impact can be substantially reduced through appropriate vaccination programs and biosecurity measures (López et al., 2023; WOA, 2025).

Canine parvovirus emerged in the late 1970s and has continued to evolve in response to host adaptation (De la Torre et al., 2018). Currently, CPV is recognized as one of the leading causes of severe viral gastroenteritis in dogs worldwide (Grecco et al., 2024). The virus belongs to the genus *Protoparvovirus* within the family *Parvoviridae* (Ozaeta et al., 2025). Canine parvovirus is characterized as a small, non-enveloped virus containing a single-stranded DNA genome, features that confer remarkable environmental stability and facilitate its persistence and transmission (López-Astacio et al., 2023). Furthermore, CPV exhibits a relatively high mutation rate, similar to RNA viruses, which has contributed to the emergence of new variants with enhanced host-cell tropism, replication efficiency, and pathogenicity (Loza-Vega, 2025).

Despite its relatively small genome, CPV contains essential genes involved in viral replication and infectivity (Ozaeta et al., 2025). The molecular structure of parvovirus is codified by non-structural proteins such as NS1, which plays a critical role in viral replication (López-Astacio et al., 2023). On the other hand, structural proteins are encoded by the *cap* gene and form capsid proteins VP1 and VP2. These proteins are involved in key virus-host interactions, contributing to cellular invasion, viral dissemination, and persistence of infection (López-Astacio et al., 2023).

REVIEW ARTICLE
Received: June 19, 2026
Revised: July 21, 2026
Accepted: August 18, 2026
Published: September 30, 2026

From an epidemiological perspective, canine parvovirus continues to represent a major challenge for veterinary medicine due to its rapid spread, environmental resilience, and potential for indirect transmission through fomites (López- Astacio et al., 2023). A recent study has identified CPV-2b as the predominant antigenic variant circulating in Ecuador (Loor-Giler et al., 2025). Moreover, puppies between three weeks and six months of age have been reported to be at a higher risk of developing severe clinical disease, while infection appears to occur more frequently in males than in females (Ozaeta et al., 2025). Although adult dogs can also become infected, many remain asymptomatic and shed the virus through their feces, thereby contributing to environmental contamination and viral maintenance within susceptible populations (De la Torre et al., 2018).

Understanding the pathogenesis of CPV is essential for the development of effective prevention and control strategies, as genetic variations among viral strains may influence both virulence and host immune responses (López- Astacio et al., 2023). In parallel, advances in diagnostic technologies, including polymerase chain reaction (PCR), immunofluorescence assays, and serological tests, have enabled more sensitive and specific detection of infection, facilitating early and accurate diagnosis (Li et al., 2025). In this context, there is a growing need to integrate current knowledge on CPV in a critical and structured manner, not only to summarize existing evidence about specific aspects of the disease but also to identify gaps and emerging challenges. The present review aimed to address knowledge gaps by integrating current evidence on CPV pathogenesis, global distribution and epidemiology, and the molecular characterization of the CPV variants circulating in Ecuador. Furthermore, it systematically synthesized the limited available evidence regarding the effectiveness of the diagnostic, vaccination, and control strategies implemented in the country through a systematic qualitative review of recent literature.

MATERIALS AND METHODS

The present study was designed as a qualitative systematic review aimed at integrating current scientific evidence on CPV, with particular emphasis on its epidemiology, pathogenesis, diagnostic approaches, treatment and control strategies. The methodological approach followed established principles for systematic reviews according to PRISMA 2020, with the aim of ensuring transparency, reproducibility and rigor in the process of identifying, selecting and synthesizing available literature.

A comprehensive search was conducted in recognized academic databases, including PubMed, SciELO, and Scopus, to identify relevant studies published between 2020 and 2026. The mentioned databases were selected due to their broad coverage of veterinary literature. PubMed was prioritized as a primary source of high-quality scientific evidence, while SciELO facilitated access to regional studies, particularly from Latin America. Google Scholar was used as a complementary tool to identify additional literature not indexed in conventional databases (Page et al., 2021). Table 1 summarizes the distribution of articles retrieved from each database included in the literature search.

Table 1. Databases used for the literature search

Database	Results
PubMed	66
Scopus	231
SciELO	15
Total	312

The search strategy combined descriptors in both English and Spanish, using Boolean operators such as “AND”, “OR”, and “NOT”. The general search equation applied was (“canine parvovirus” OR “CPV” OR “canine parvoviral enteritis”) AND (epidemiology OR pathogenesis OR diagnosis OR treatment OR therapy). Filters were applied to include articles published within the selected timeframe. While open-access publications were prioritized to facilitate accessibility, this may have introduced a selection bias by excluding relevant high-quality studies available in subscription-based journals (Page et al., 2021).

Applying the filters corresponding to the year of publication, open access articles, language, and accessibility criteria, the search yielded a total of 312 records. After applying the inclusion and exclusion criteria, as well as removing duplicates, theses, and irrelevant articles, 70 studies were selected for a detailed analysis. The selection process was conducted according to the PRISMA 2020 framework, ensuring transparency in the identification, screening, eligibility, and inclusion stages, as shown in Figure 1 (Page et al., 2021).

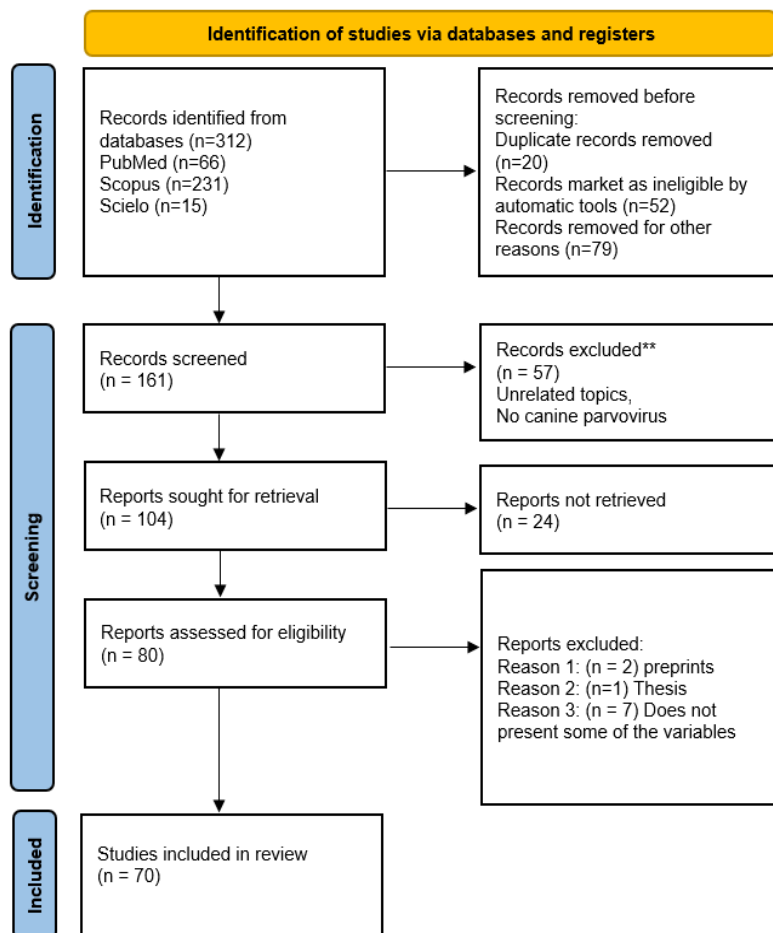


Figure 1. PRISMA flowchart for identification, screening, eligibility, and inclusion of studies about epidemiology, pathogenesis, diagnosis, treatment, and control of canine parvovirus

Inclusion and exclusion criteria

The inclusion criteria were established to ensure that the scientific relevance and methodological quality of the selected studies were assessed using the appropriate Joanna Briggs Institute (JBI) critical appraisal tools according to the study design. Each study was evaluated for methodological rigor, data collection procedures, outcome assessment, and potential sources of bias. The results of the quality assessment were considered during the synthesis and interpretation of the evidence. Original peer-reviewed articles addressing CPV in relation to its epidemiology, pathogenesis, diagnosis, treatment, disease control, and investigation conducted in Ecuador were included. Studies involving domestic dogs of any age, breed, or geographic region were considered, as well as investigations evaluating viral transmission, prevalence, risk factors, clinical manifestations, immune response, and preventive strategies such as vaccination and biosecurity measures. Additionally, studies about diagnostic methodologies, including enzyme-linked immunosorbent assays (ELISA) tests, PCR-based techniques, virus isolation, serological assays, or molecular characterization, were included.

Studies conducted exclusively on species other than canines, articles unrelated to CPV, or investigations focused solely on unrelated viral diseases were excluded. Likewise, narrative reviews, editorials, preprints, thesis, and conference abstracts without complete data, duplicate publications, and documents lacking primary scientific information or full-text access were not considered. This criterion was applied due to accessibility limitations and is recognized as a potential source of bias in the study selection process (Page et al., 2021).

Study selection process

The study selection process was carried out in different sequential stages according to the PRISMA flow guidelines by independent reviewers with conflict resolution. Initially, 312 identified records were screened in databases by title and abstract, and studies that did not meet the established thematic criteria related to CPV were excluded. Subsequently, potentially relevant articles were assessed through full-text review to determine their final eligibility.

The final selection of studies was conducted through a systematic evaluation process that identified 70 articles meeting the established inclusion criteria and considered suitable for qualitative synthesis. Although the review primarily included studies published in English within the predefined time frame, 7 articles published in Spanish were also

incorporated due to their scientific relevance and contribution to the topic. In addition, one study published in 2016 was retained despite falling outside the selected publication period because of its significant relevance and frequent citation in the literature, and one article published in 2018 because it has relevant information on Ecuador. The complete flow of the study selection process is presented in Figure 1.

RESULTS

The family *Parvoviridae* comprises a large group of viruses containing a single-stranded DNA genome ranging from 4 to 6 kilobases, enclosed within a small non-enveloped icosahedral capsid (Ozaeta et al., 2025). The virion measures approximately 18-26 nm in diameter (Yin et al., 2022). The replication occurs within the nucleus of actively dividing cells (Li et al., 2025). *Parvoviridae* comprises viruses that infect a wide range of vertebrate and invertebrate hosts and is classified into three subfamilies, including *Densovirinae*, which includes parvoviruses that infect invertebrate hosts; *Parvovirinae*, which comprises viruses infecting vertebrate hosts such as mammals, birds, and reptiles; and *Hamaparvovirinae*, is a subfamily that infects crustaceans and insects (López-Astacio et al., 2023). *Hamaparvoviridae* subfamily is classified based on nucleotide sequence homology within the helicase domain (Loza-Vega, 2025). The subfamily *Parvovirinae* has been further divided into eight genera according to the host species they infect (Table 2).

Table 2. Genera of the family *Parvoviridae*, their principal hosts, and susceptible species

Genus	Hosts	Susceptible species
<i>Amdoparvovirus</i>	Carnivores	Foxes (Aleutian mink disease virus), Minks (Aleutian disease virus)
<i>Aveparvovirus</i>	Galliform birds	Chickens (Chicken parvovirus), Turkeys (Turkey parvovirus)
<i>Bocaparvovirus</i>	Carnivores	Canines (Canine minute virus, Canine bocavirus 1), Felines (Feline bocavirus)
	Pinnipeds	California sea lion (Pinniped bocaparvovirus 1 and 2)
	Primates	Humans (Human bocavirus 1, 2a, 2b, 2c, 3, and 4), Gorillas (Gorilla bocavirus)
	Ungulates	Bovines (Bovine parvovirus), Swine (Porcine parvovirus 1, 2, 3, 4-1, 4-2)
	Equines	Horses (Equine parvovirus)
<i>Dependoparvovirus</i>	Anseriformes	Ducks (Duck parvovirus), Geese (Goose parvovirus)
	Chiroptera	Bats (Bat adeno-associated virus)
	Pinnipeds	California sea lion (Adeno-associated virus)
	Primates	Humans (Parvovirus B19), Non-human primates (Rhesus macaque, Pig-tailed macaque), Squirrels (Chipmunk parvovirus)
<i>Erythroparvovirus</i>	Ungulates	Bovines (Bovine parvovirus 3)
	Carnivores	Canines (Canine parvovirus), Felines (Feline parvovirus), Minks (Mink enteritis virus), Raccoons, Arctic wolves
<i>Protoparvovirus</i>	Primates	Humans (Bufavirus 1a, 1b, and 2)
	Rodents	Rats (Kilham rat virus, Rat parvovirus 1), Mice (Minute virus of mice, Mouse parvovirus 1-5), Hamsters
	Ungulates	Swine (Porcine parvovirus type 1, Kresse strain, NADL-2)
	Chiroptera	Fruit bats (<i>Eidolon helvum</i> parvovirus)

The present data in the table are adapted from Ozaeta et al. (2025)

STRUCTURE AND GENOME OF PARVOVIRUSES

Parvoviruses are small, non-enveloped viruses, a characteristic that contributes to their remarkable environmental stability and resistance to physical and chemical inactivation (Tuteja et al., 2022). The virion consists of a highly structured icosahedral capsid approximately 18-26 nm in diameter, which protects the viral single-stranded DNA genome and plays a critical role in host-cell recognition, attachment, and entry (Flores et al., 2020). The capsid is assembled from 60 protein subunits, predominantly composed of VP2, the major structural protein that accounts for approximately 90% of the capsid and contains the principal antigenic determinants involved in host range specificity and immune recognition (Liu et al., 2025). In addition to VP2, the capsid contains VP1, a minor structural protein that shares

most of its sequence with VP2 but possesses a unique N-terminal region essential for viral infectivity and intracellular trafficking (López-Astacio et al., 2023). A third capsid protein, VP3, has also been identified in CPV and is generally considered a cleavage product derived from VP2 during virion maturation rather than a distinct gene product (Tuteja et al., 2022).

The *parvovirus* genome consists of a linear single-stranded DNA (ssDNA) molecule (Figure 2) ranging from approximately 4 to 6 kb in length and containing palindromic terminal sequences that fold into characteristic hairpin structures (López-Astacio et al., 2023). The terminal hairpins function as self-priming structures for DNA synthesis and are essential for viral replication. During replication, the hairpin termini are processed by viral and host cellular factors, generating primers that initiate DNA synthesis (Qi et al., 2020). Replication of parvovirus allows genome replication to proceed through a unidirectional process known as rolling-hairpin replication. The terminal palindromic sequences, together with adjacent nucleotides, contain critical cis-acting elements required for viral DNA replication, packaging, and genome encapsidation (Tuteja et al., 2022). In addition to serving as origins of replication, the terminal hairpins participate in the resolution of genomic termini through the action of viral proteins such as NS1 (Loza-Vega et al., 2025). The multifunctional protein introduces site-specific nicks at defined sequences, generating new 3'-OH ends that are subsequently extended by the host cell DNA replication machinery (López-Astacio et al., 2023). Consequently, productive viral replication is highly dependent on actively dividing cells, particularly those in the S phase of the cell cycle, where the cellular factors required for DNA synthesis are abundantly available (Pénzes et al., 2023).

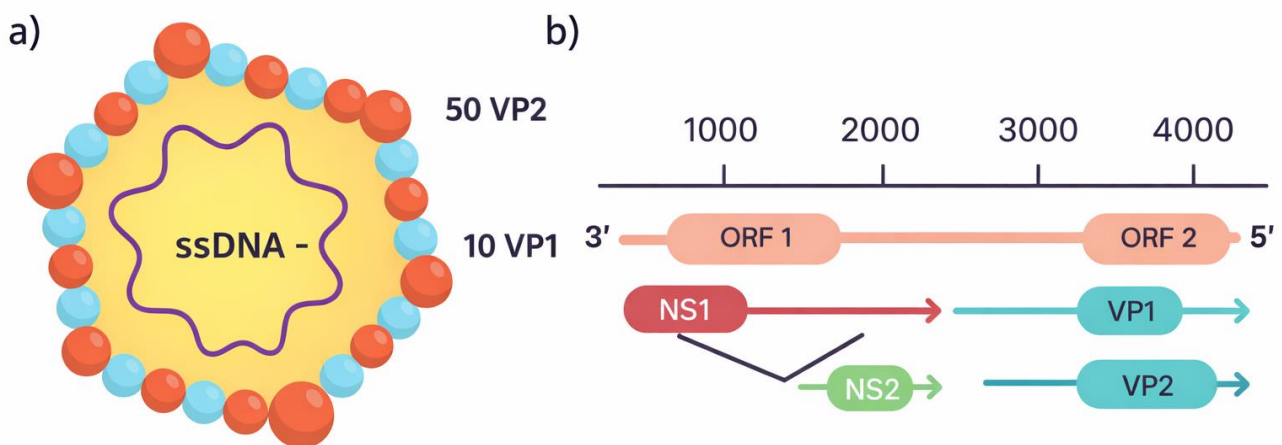


Figure 2. Representation of the structure of parvoviruses. **a:** Capsid of the virus. **b:** ORF 1 and ORF 2 with structural and non-structural proteins of the parvovirus. Source: Created by the authors based on Tuteja (2022).

The parvovirus genome contains two major open reading frames (ORFs; Chen et al., 2023). The left ORF encodes the *rep* gene, which gives rise to the non-structural proteins, including NS1 and NS2 (López-Astacio et al., 2023). NS1 is a multifunctional protein essential for viral replication, possessing sequence-specific endonuclease (nickase), helicase, and ATPase activities (Magouz et al., 2023). In addition to initiating viral DNA replication, NS1 regulates viral transcription, interacts with host cellular replication machinery, and contributes to virus-induced cytopathic effects, including cell-cycle arrest, apoptosis, and oxidative stress-mediated cell damage (Pénzes et al., 2023). NS2, although less extensively characterized, plays important roles in viral replication, intracellular trafficking, nuclear export, and efficient virion production (Magouz et al., 2023).

The second ORF encodes the *cap* gene, which contains the genetic information required for the synthesis of the structural capsid proteins VP1 and VP2 (López-Astacio et al., 2023). VP1 is a minor capsid component that contains a unique N-terminal region (VP1u) enriched in basic amino acids, including arginine and lysine residues, which contribute to nuclear localization and infectivity (Mia and Hasan, 2021). Importantly, VP1u harbors a phospholipase A2 (PLA2) domain that facilitates viral escape from the endosomal compartment following cell entry (López-Astacio et al., 2023). In contrast, VP2 constitutes approximately 90% of the capsid structure and contains the major antigenic determinants responsible for host-cell receptor recognition, tissue tropism, and host-range specificity (Tuteja et al., 2022). Mutations within VP2 have been strongly associated with the emergence of novel antigenic variants and alterations in host susceptibility (Chen et al., 2024).

Expression of the *cap* gene is regulated through alternative splicing and the use of distinct translational initiation sites, allowing precise control of the VP1-to-VP2 ratio within mature virions (López-Astacio et al., 2023). Consequently, the sequence variability and genomic organization of ORF2 are of particular importance for understanding viral

evolution, molecular surveillance, and vaccine development (Mia et al., 2021). Due to their compact genomes and limited coding capacity, parvoviruses rely on multifunctional structural and non-structural proteins to complete their replication cycle (Magouz et al., 2023). The structural proteins coordinate multiple interactions with host cells and tissues, facilitating viral replication, dissemination, persistence, and adaptation within susceptible host populations (López-Astacio et al., 2023).

CELLULAR TROPISM

Parvoviruses exhibit a highly restricted cellular tropism that depends on both the presence of specific cell-surface receptors and the proliferative status of the host cell (Chen et al., 2024). In the case of *Protoparvoviruses* such as CPV, viral attachment is primarily mediated by the transferrin receptor type 1 (TfR1), which is highly expressed on actively dividing cells, including intestinal crypt epithelial cells, hematopoietic progenitor cells, and lymphoid tissues (Uhl and Fasina, 2025). The TfR1 serves as the primary cellular receptor for CPV and is a receptor that determines host range, tissue tropism, and viral pathogenicity (Uhl and Fasina, 2025).

Viral entry is initiated by receptor binding followed by clathrin-mediated endocytosis (Afumba et al., 2022). Acidification triggers conformational changes in the capsid, leading to the exposure of the unique N-terminal region of VP1 (VP1u), which contains a PLA2 domain required for disruption of the endosomal membrane and enables the escape of the viral particle into the cytoplasm (Shi et al., 2023). Subsequently, the virion is transported to the nucleus, where partial uncoating allows the release of the single-stranded viral DNA. Genome replication proceeds through a rolling-hairpin mechanism that depends on the inverted terminal repeats (ITRs) located at both the 5' and 3' ends of the viral genome, as well as the multifunctional NS1 protein (Magouz et al., 2023). NS1 recognizes the terminal sequences and functions as a site-specific nickase, helicase, and recruiter of host replication factors, thereby exploiting the cellular DNA replication and repair machinery for viral genome amplification (Uhl and Fasina, 2025). Alternative transcription and RNA splicing generate multiple viral mRNAs encoding both non-structural (NS) and structural (VP) proteins, which regulate viral replication, capsid assembly, and interactions with host antiviral responses (López-Astacio et al., 2023). As viral replication progresses, CPV induces cell-cycle dysregulation, oxidative stress, apoptosis, and other cytopathic effects that contribute to tissue damage and facilitate the release and dissemination of newly formed virions (Afumba et al., 2022).

PARVOVIRUS IN WILDLIFE SPECIES

Canine parvovirus emerged as a canine pathogen in the late 1970s following a host-switch event involving a feline panleukopenia virus (FPV)-like ancestor circulating among wild carnivores (Qi et al., 2020). Although the precise evolutionary pathway remains unresolved, molecular and phylogenetic evidence supports the hypothesis that CPV originated through the adaptation of an FPV-related virus capable of infecting carnivore species (Zhou et al., 2024). This hypothesis is further strengthened by the presence of intermediate viral variants circulating in wildlife populations that share genetic features of both FPV and CPV, together with the limited ability of classical FPV strains to effectively infect dogs (Franzo et al., 2023).

Wild carnivores play an important role in the ecology and maintenance of canine parvoviruses (CPV; Sarchese et al., 2025). Studies conducted in Türkiye and Italy have shown that wild canids, particularly urban red foxes (*Vulpes vulpes*), Eurasian badgers, and wolves, act as reservoirs of CPV infection and contribute to viral transmission between wildlife and domestic dog populations (Kurucay et al., 2023; Sarchese et al., 2025). The evidence supports the importance of wildlife-domestic animal interfaces in the epidemiology of canine parvoviral disease.

Its genomic evolution has been characterized by an unusually high substitution rate for a DNA virus, estimated at approximately 10^{-4} substitutions per nucleotide site per year, a value comparable to those reported for certain RNA viruses (Jantafong et al., 2022). The accelerated mutation rate has facilitated the emergence of novel antigenic variants with substantial genetic and phenotypic differences (Zhou et al., 2024).

Serological surveys have demonstrated a high prevalence of antibodies against FPV among wild felid populations, suggesting frequent exposure to the virus, likely through interactions with domestic cats or shared environmental sources of infection (Ozaeta et al., 2025). This finding supports the existence of complex transmission networks involving both domestic and wild carnivore hosts (Ozaeta et al., 2025).

CANINE PARVOVIRUS IN ECUADOR

Molecular and epidemiological studies conducted in Ecuador have demonstrated the circulation of the three classical antigenic variants of canine parvovirus type 2 (CPV-2), namely CPV-2a, CPV-2b, and CPV-2c (De la Torre et al., 2018). Recent evidence indicated that CPV-2b is currently the predominant variant detected in clinical samples reported in a multicenter study conducted between 2022 and 2023, which analyzed samples collected from six Ecuadorian provinces,

including Carchi, Chimborazo, Guayas, Imbabura, Pichincha, and Santo Domingo de los Tsáchilas (Loor-Giler et al., 2025). The study employed quantitative PCR (qPCR) and molecular characterization of the VP2 gene, enabling both viral load quantification and the identification of mixed infections involving multiple CPV variants (De la Torre et al., 2018; Loor-Giler et al., 2025).

Prior to the study by Loor-Giler et al. (2025), investigations with smaller sample sizes and more restricted geographic coverage reported different patterns of variant distribution, with CPV-2a identified as the predominant circulating strain, followed by CPV-2b (De la Torre et al., 2018). The changes in variants may be attributed to methodological differences, including limited sampling areas, lower sample numbers, and the use of conventional sequencing approaches without large-scale qPCR-based viral quantification.

Age-related analyses of CPV prevalence in Ecuador revealed that dogs between 3 and 12 months of age represented the largest proportion of positive cases, with the probability of infection decreasing progressively as age increased (Basurto et al., 2024; Loor-Giler et al., 2024). This age-associated susceptibility is likely related to the combined effects of immunological immaturity and the progressive decline of maternally derived antibodies, which create a period of increased vulnerability to infection (Basurto et al., 2024).

The majority of cases reported in Ecuador involve domestic dogs presenting with hemorrhagic gastroenteritis, with puppies younger than six months of age exhibiting the highest risk of developing severe clinical disease (Basurto et al., 2024). The findings are consistent with the established pathogenesis of CPV infection, which primarily affects rapidly dividing cells within the intestinal crypts, lymphoid tissues, and bone marrow (Basurto et al., 2024; Loor-Giler et al., 2024).

The detection of co-circulating CPV variants and mixed infections highlights the importance of continuous molecular surveillance in Ecuador (De la Torre et al., 2018). In a recent study, 78.47% of dogs with CPV-2 showed a high prevalence of infection, elevated viral loads, and ongoing viral evolution; regular monitoring of circulating strains is essential to evaluate potential impacts on vaccine effectiveness and to support the implementation of evidence-based prevention and control strategies (Loor-Giler et al., 2025).

EPIDEMIOLOGY IN SUSCEPTIBLE HOSTS

Canine parvovirus is a widely contagious agent that affects domestic and wild canid populations globally and is recognized as a major cause of viral gastroenteritis in dogs (Qi et al., 2020). Although dogs of all ages are susceptible to infection, the highest incidence of clinical disease is observed in puppies between 6 weeks and 6 months of age, particularly those with incomplete vaccination schedules, inadequate maternal antibody protection, or no previous immunization (Squires et al., 2024). Different studies conducted in the United States have identified breed-associated susceptibility, with Doberman Pinschers, Rottweilers, American Pit Bull Terriers, Labrador Retrievers, German Shepherd Dogs, and their crossbreeds reported to be at increased risk of developing severe disease (Flores et al., 2020; Jyothi et al., 2024).

Host susceptibility may also be influenced by factors that compromise immune function, including malnutrition, stress, parasitism, and concurrent infectious diseases (Jyothi et al., 2024). Co-infections with pathogens such as canine distemper virus, canine enteric coronavirus, rotavirus, astrovirus, adenovirus, calicivirus, and emerging viruses including norovirus, kobuvirus, sapovirus, and canine circovirus have been associated with increased disease severity and poorer clinical outcomes (Jyothi et al., 2024; Li et al., 2025). These findings suggest that viral and bacterial co-infections may enhance susceptibility to CPV infection and contribute to the progression of clinical disease (Jyothi et al., 2024; Li et al., 2025).

Although adult dogs can become infected, many develop subclinical or mild infections due to pre-existing immunity acquired through vaccination or previous exposure (Muñoz et al., 2021). Even after infection, animals may persistently shed viral particles in their feces, promoting environmental persistence and further transmission of the pathogen (Serena et al., 2021). The virus is predominantly spread via the fecal–oral pathway through direct contact with infected feces or indirectly through contaminated fomites (Ozaeta et al., 2025). The epidemiological success of CPV is largely attributable to its exceptional environmental stability, as the virus can remain infectious for extended periods and exhibits considerable resistance to many commonly used disinfectants and adverse environmental conditions (Miranda and Thompson, 2016).

PATHOGENESIS

The transmission of CPV-2 is by the fecal-oral route, through the ingestion of contaminated fecal material or contact with contaminated fomites (Flores et al., 2020). In high-density environments such as kennels, shelters, and breeding facilities, infected animals may also contribute to environmental contamination through body secretions, facilitating transmission to susceptible hosts (López-Astacio et al., 2023).

Canine parvovirus type 2 exhibits a specific tropism for rapidly dividing cells (López-Astacio et al., 2023). Following oral exposure, the virus initially replicates within lymphoid tissues of the oropharynx, including the tonsils and regional lymph nodes (Tuteja et al., 2022). Viral replication in the mentioned tissues is followed by dissemination through the bloodstream, resulting in a systemic viremia that typically develops within 3-5 days post-infection, and the first symptoms appear between day 3 and day 5 (Tuteja et al., 2022). During viremia, the virus targets hematopoietic progenitor cells in the bone marrow and lymphoid tissues, causing depletion of leukocyte precursors and leading to severe leukopenia, lymphopenia, and neutropenia (Maganga et al., 2023).

In puppies born to non-immune dams, infection during the first weeks of life may result in viral myocarditis due to active replication within cardiomyocytes (Tuteja et al., 2022). However, the clinical manifestation has become relatively uncommon globally because most neonatal puppies acquire maternally derived antibodies (MDA) through colostrum, which provide protection during the early postnatal period (Zhou et al., 2024).

As the infection develops, infected leukocytes facilitate the spread of CPV-2 to the epithelial cells undergoing active proliferation within the intestinal crypts of Lieberkühn (Tuteja et al., 2022). Viral replication destroys the crypt stem cells responsible for continuous epithelial renewal, thereby disrupting the regeneration of intestinal villi (Tuteja et al., 2022). Consequently, villous atrophy, crypt necrosis, and collapse of the intestinal mucosal architecture occur, leading to malabsorption, fluid loss, and the development of acute enteritis, often accompanied by hemorrhagic diarrhea (Grecco et al., 2024).

The destruction of lymphoid and hematopoietic cells results in immunosuppression, causing sepsis characterized by lymphopenia and neutropenia (Maganga et al., 2023). Simultaneously, disruption of the intestinal epithelial barrier, together with inflammation and crypt necrosis, facilitates bacterial translocation from the intestinal lumen into the bloodstream. Canine parvovirus type 2 frequently predisposes affected animals to secondary bacterial infections, endotoxemia, and septicemia, which are major contributors to disease severity and mortality (Maganga et al., 2023).

Infected dogs begin shedding large quantities of virus in their feces approximately 4-7 days after exposure, often before the onset of clinical signs (Maganga et al., 2023). The extensive viral shedding, combined with the remarkable environmental stability of CPV-2, contributes significantly to the rapid dissemination of the virus within susceptible populations and communal environments (Magouz et al., 2023).

CLINICAL SIGNS

The incubation period of CPV infection typically ranges from 4 to 7 days (Purwitasari et al., 2022). Early clinical manifestations include lethargy, depression, anorexia, and fever, which are subsequently followed by vomiting, hemorrhagic diarrhea, dehydration, and progressive weakness (Gerlach et al., 2020; Suvethika and Kumar, 2021). Canine parvovirus infection results in loss of fluids, electrolytes, and plasma proteins through the gastrointestinal tract, leading to severe dehydration and, in advanced cases, hypovolemic shock (Purwitasari et al., 2022). The small intestinal epithelium serves as the primary site of viral replication, where infection of crypt epithelial stem cells disrupts normal epithelial renewal (Purwitasari et al., 2022). Consequently, villous atrophy, crypt necrosis, mucosal erosion, and intestinal hemorrhage develop throughout the duodenum, jejunum, and ileum, resulting in malabsorption and severe enteritis (Parrish and Sykes, 2021).

Clinically, CPV-2 infection is associated with two major disease manifestations, including enteritis and myocarditis (Sherasiya et al., 2024). The enteric form is by far the most common presentation and primarily affects puppies older than six weeks of age (Sherasiya et al., 2024). Clinical signs include acute hemorrhagic diarrhea, vomiting, pyrexia, anorexia, depression, abdominal discomfort, and rapid dehydration, which may progress to shock and death if left untreated (Zhou et al., 2024). The myocardial form of CPV-2 infection is currently less common and is generally observed in puppies infected during the neonatal period, particularly those born to non-immune dams lacking protective maternally derived antibodies (Dines et al., 2023). The viral replication within actively dividing cardiomyocytes leads to myocarditis characterized by myocardial inflammation, degeneration, and necrosis (Zhou et al., 2024). Clinically, affected puppies may present with acute heart failure, respiratory distress, arrhythmias, or sudden death (Gjurovski et al., 2023; Sherasiya et al., 2024).

From a pathological perspective, enteric CPV infection is characterized by segmental hemorrhagic enteritis, intestinal mucosal necrosis, crypt collapse, villous atrophy, and depletion of lymphoid tissues (Liu et al., 2025). Gross lesions may include gastric erosions, intestinal petechial or ecchymosis accumulation of serous to hemorrhagic intestinal contents, and enlargement of mesenteric lymph nodes (Zhou et al., 2024). In severe cases, extensive ulceration of the intestinal mucosa and marked gallbladder distension may also be observed (Ain-Fatin et al., 2020).

Recent studies investigating acute canine diarrhea have reported significant associations between CPV and canine coronavirus (CCoV) co-infections, although prevalence rates vary according to age group and geographic region (Li et al., 2025; Liu et al., 2025). Because the clinical manifestations of CPV are not pathognomonic, differential diagnoses

should include canine distemper, canine enteric coronavirus infection, hemorrhagic gastroenteritis of other etiologies, hookworm infection, coccidiosis, and other causes of acute enteritis (Chethan et al., 2023; Dines et al., 2023). In addition, intestinal parasitic infections caused by *Toxocara canis* and *Ancylostoma caninum* should be considered among the principal presumptive diagnoses, as these pathogens may produce clinical signs similar to CPV infection, including hemorrhagic diarrhea, vomiting, lethargy, and pale mucous membranes (Ain-Fatin et al., 2020).

DIAGNOSIS

Because the clinical manifestations of CPV infection may overlap with those of other causes of acute hemorrhagic gastroenteritis, early and accurate diagnosis is essential to facilitate appropriate case management and prevent viral dissemination, particularly in veterinary hospitals, shelters, breeding facilities, and other environments where multiple animals are housed together (Jantafong et al., 2022). Importantly, infected dogs may begin shedding virus in their feces before the onset of gastrointestinal signs, thereby increasing the risk of environmental contamination and transmission (Decaro et al., 2020).

Samples commonly used for CPV diagnosis include feces, rectal swabs, and whole blood (Tuteja et al., 2022). Confirmatory diagnosis can be achieved through different laboratory methods, including antigen-detection ELISA, PCR, qPCR, hemagglutination assays, electron microscopy, and virus isolation in cell culture (Decaro et al., 2020; Nandi et al., 2022). Among these approaches, PCR-based methods are considered the most sensitive and specific diagnostic tools currently available (Decaro et al., 2020).

In routine veterinary practice, rapid immunochromatographic tests and commercial ELISA kits are widely used because of their ease of application, rapid turnaround time, and high specificity, which frequently exceeds 90% (Decaro et al., 2020). However, their sensitivity is considerably more variable and has been reported to range from approximately 16% to 80% in ELISA tests when compared with molecular assays such as PCR and qPCR, which have high sensitivity and specificity, particularly during early infection or in samples containing low viral loads (Hyeon et al., 2023; Segev et al., 2022; Table 3). In addition, the rapid antigen test has 96.4% sensitivity and 88.2% specificity compared with qPCR (Abousenna et al., 2024).

Table 3. Comparison of immunological and molecular tests used in the diagnosis of canine parvovirus

Diagnostic method	Target detected	Reported diagnostic performance	Main limitations	Clinical use context	Reference
Rapid antigen test (lateral flow)	CPV viral antigen in feces	Sensitivity 96.4% and specificity 88.2% compared with qPCR and viral shedding stage.	May not accurately reflect sensitivity in clinical fecal samples; experimental kit	Rapid screening; point-of-care; useful as an initial test	Abousenna et al. (2024)
ELISA (antigen detection)	CPV viral antigen	Sensitivity ranges from 16% to 80% depending on the commercial assay and reference standard.	Lower sensitivity than molecular methods	Complementary diagnostic support; epidemiological studies	Li et al. (2025)
Conventional PCR	CPV viral DNA	High sensitivity and specificity reported across different laboratory protocols.	Does not quantify viral load; requires laboratory infrastructure	Confirmatory diagnosis when qPCR is not available	Li et al. (2025); Segev et al. (2022)
qPCR	CPV viral DNA (quantifiable)	Very high sensitivity and specificity; reference molecular method	Requires specialized equipment and trained personnel	Confirmatory diagnosis and viral load quantification	Loor-Giler et al. (2024); Li et al. (2025)
ddPCR	Viral DNA (absolute quantification)	Higher analytical sensitivity and greater precision (shown in related viruses such as CDV)	No specific clinical validation metrics for CPV; mainly used in the literature.	Advanced research; potential for detection of low viral loads	Iribarnegar y et al. (2024)

ELISA: Enzyme-linked immunosorbent assay; qPCR: Real-time qPCR; ddPCR: Droplet digital PCR. CDV: Canine distemper virus; Diagnostic performance values shown in this table correspond to representative studies or commercial assays and should not be interpreted as universal performance estimates for each diagnostic method. Test performance varies according to the assay, sample type, stage of infection, and reference standard used.

Interpretation of diagnostic results should take vaccination history into account. Modified-live virus vaccines may result in transient positive antigen or molecular test results for up to approximately 28 days following vaccination, potentially complicating diagnostic interpretation (Decaro et al., 2020). Conversely, false-negative results may occur when viral concentrations in feces are below the detection threshold of the assay or when virus particles are neutralized by intestinal antibodies, reducing antigen availability for detection (Li et al., 2025). Consequently, when animals with clinical signs suggestive of CPV infection yield negative results on rapid antigen tests, complementary molecular testing is strongly recommended to confirm or exclude infection and to minimize the risk of viral spread (Decaro et al., 2020; Nandi et al., 2022).

Among molecular techniques, qPCR is currently regarded as the diagnostic reference standard because of its high sensitivity, specificity, and ability to detect low viral loads while simultaneously providing quantitative information on viral burden (Segey G et al., 2022; Li et al., 2025). In addition, qPCR is particularly valuable for confirming cases with negative antigen test results and for epidemiological surveillance of circulating strains (Table 3). Droplet digital PCR (ddPCR) has recently emerged as a promising molecular platform capable of absolute nucleic acid quantification and enhanced analytical sensitivity compared with conventional qPCR (Iribarnegaray et al., 2024). Nevertheless, despite its potential advantages, the clinical application of ddPCR for routine CPV diagnosis remains limited and has not yet been fully validated for widespread veterinary use, only is used for canine distemper virus (Iribarnegaray et al., 2024). Overall, molecular diagnostic techniques enable the detection of CPV during the early stages of infection and play a critical role in disease surveillance, outbreak control, and the implementation of effective preventive measures (Table 3).

In addition to laboratory diagnosis, the identification and molecular characterization of circulating CPV-2 genotypes constitute essential epidemiological and immunological tools (Li et al., 2025). Genotyping, particularly through the analysis of the VP2 gene, provides valuable information regarding viral evolution, transmission dynamics, geographic distribution, and the coexistence of distinct antigenic variants (Li et al., 2025). Furthermore, molecular surveillance enables the detection of emerging mutations that may influence host range, viral fitness, antigenicity, and potentially vaccine performance (Muñoz et al., 2021). Recent studies conducted in Ecuador have demonstrated the concurrent circulation of multiple CPV-2 variants, as well as the occurrence of mixed-genotype infections, highlighting the importance of continuous molecular monitoring to improve disease surveillance and support evidence-based prevention and control strategies (De la Torre et al., 2018; Loo-Giler et al., 2024).

TREATMENT

The CPV infection is a globally distributed disease for which treatment remains primarily supportive and aimed at correcting the physiological disturbances associated with infection (Horecka et al., 2020). Persistent vomiting and acute hemorrhagic gastroenteritis are among the most characteristic clinical manifestations of the disease (Muñoz et al., 2021). Viral destruction of intestinal crypt epithelial cells compromises the integrity of the gastrointestinal barrier, facilitating bacterial translocation and predisposing affected animals to sepsis, endotoxemia, and systemic inflammatory responses (Gerlach et al., 2020; Dos Santos et al., 2023).

Consequently, intensive supportive care remains the cornerstone of CPV treatment (Muñoz et al., 2021). Fluid therapy is considered the most critical component of patient management, with intravenous crystalloid solutions, such as Lactated Ringer's solution, commonly administered to correct dehydration, electrolyte imbalances, and hypovolemia (Horecka et al., 2020). Potassium supplementation should be adjusted according to individual patient requirements and laboratory findings (Schmitz, 2021). In cases of severe hypoproteinemia, colloid support, plasma transfusions, or other blood products may be considered to restore oncotic pressure and improve circulatory stability (Gerlach et al., 2020).

Additional therapeutic measures include the administration of antiemetics such as maropitant-cerenia (1 mg/kg subcutaneously every 24 hours), metoclopramide-primperan (0.2-0.5 mg/kg intravenous route every 6-8 hours) to control vomiting and facilitate nutritional support, as well as broad-spectrum antimicrobial therapy to reduce the risk of secondary bacterial infections resulting from intestinal barrier disruption and neutropenia (Mazzaferro, 2020). Once emesis is adequately controlled, early enteral nutrition is generally recommended by the American Animal Hospital Association (AAHA), as it promotes intestinal mucosal recovery, maintains gastrointestinal integrity, and may improve clinical outcomes (Mazzaferro, 2020; Ozaeta et al., 2025).

Different adjunctive therapies have also been investigated, including immunomodulators, probiotics, antioxidants, hyperimmune plasma, and other supportive interventions (Chethan et al., 2023; Ozaeta et al., 2025). However, the clinical evidence supporting many therapies, such as probiotics (Schmitz, 2021) and antioxidants, is limited, and their use should be considered complementary to standard supportive treatment rather than a replacement for established therapeutic protocols (Muñoz et al., 2021; Chethan et al., 2023). Despite significant advances in supportive care, no universally accepted antiviral treatment is currently available for routine clinical management of CPV infection (Decaro

et al., 2020). Therefore, early diagnosis, aggressive supportive therapy, and appropriate monitoring remain the most effective strategies for reducing morbidity and mortality associated with this disease (Decaro et al., 2020).

Although no breed has been consistently identified as having the highest treatment failure rate in canine parvoviral enteritis, Rottweilers, Doberman Pinschers, German Shepherd Dogs, and American Pit Bull Terriers are recognized as breeds with increased susceptibility to severe disease (Decaro et al., 2020). Previous studies indicated that factors such as low body weight, disease severity at admission, and inadequate immune status are stronger predictors of mortality than breed alone (Decaro et al., 2020).

At present, the management of CPV infection relies primarily on supportive care aimed at correcting dehydration, electrolyte imbalances, and secondary bacterial complications (Mazzaferro, 2020). However, in animals presenting with high viral loads or severe systemic disease, supportive therapy alone may be insufficient to effectively control viral replication (Decaro et al., 2020). Consequently, recent studies have focused on the development of targeted antiviral strategies designed to improve clinical outcomes and reduce mortality associated with CPV infection (Decaro et al., 2020).

Current antiviral studies can be broadly categorized into three major areas. The first involves interferon-based therapies, particularly interferons (IFN I), which have demonstrated immunomodulatory and antiviral effects, including reductions in viral shedding and improvements in survival rates in some experimental and clinical studies (Reyes-Cruz et al., 2021; Kim et al., 2022). The second area focuses on antibody-based therapies, including hyperimmune plasma and monoclonal antibodies, which aim to neutralize circulating virus particles and limit viral replication during the acute phase of infection (Lopes et al., 2024). Recent investigations have reported that the administration of a human immunomodulator (0.5 mg/day $\times$ 5 days) to puppies under six months of age promises to reduce disease severity and enhance recovery in affected animals (Muñoz et al., 2021).

The third area encompasses small-molecule antiviral compounds targeting different stages of the viral replication cycle (Decaro et al., 2020). Different compounds, such as mRNA alternatives, have demonstrated antiviral activity against CPV *in vitro* by interfering with viral replication, nucleic acid synthesis, or virus-host interactions (Uhl and Fasina, 2025). Nevertheless, most small-molecule mRNA remains at the experimental stage, and additional studies are required to establish their safety, efficacy, pharmacokinetics, and clinical applicability in naturally infected dogs (Ulas et al., 2024).

Collectively, the antiviral strategies highlight the progress achieved in the search for targeted therapies against CPV. However, the magnitude of their reported benefits varies considerably among studies. While recombinant feline IFN I has demonstrated improvements in survival and clinical recovery in experimentally infected dogs (Reyes-Cruz et al., 2021), other antiviral candidates, including metoclopramide-primperan and maropitant-cerenia (Mazzaferro, 2020), and RNA interference-based approaches (Uhl and Fascina, 2025), have mainly shown reductions in viral replication under laboratory conditions, with limited evidence from clinical patients. This discrepancy suggests that promising *in vitro* antiviral activity does not necessarily translate into equivalent therapeutic efficacy *in vivo*. Consequently, despite the growing body of evidence supporting these novel interventions, robust clinical trials involving larger populations are still required to determine their therapeutic value, safety profile, and feasibility in routine veterinary medicine (Lopes et al., 2024).

PREVENTION AND CONTROL

The prevention and control of CPV rely primarily on systematic and timely vaccination, which remains the most effective strategy for reducing infection rates, disease severity, and outbreak occurrence (Ford et al., 2022). Modified-live virus (MLV) vaccines are generally considered the vaccines of choice because they induce a rapid, robust, and long-lasting immune response (Ford et al., 2022). In contrast, inactivated vaccines, although safe, typically exhibit lower immunogenicity and require multiple doses to achieve comparable protection; therefore, their use is generally restricted to specific situations or the evaluation of novel vaccine formulations (Squires et al., 2024).

Puppies should receive their first vaccination between 6 and 8 weeks of age, followed by booster doses administered every 3-4 weeks until at least 16-20 weeks of age to ensure adequate protection during the period of declining MDA (Table 4). The vaccination schedule is designed to minimize the impact of the so-called window of susceptibility, a critical period during which maternal antibodies may be insufficient to prevent infection while still interfering with active immunization (AAHA, 2022). Following completion of the primary vaccination series, a booster is generally recommended at one year of age and subsequently at intervals of up to three years, in accordance with current international vaccination guidelines (Squires et al., 2024).

The use of vaccines capable of providing broad protection against circulating CPV variants is essential for maintaining population immunity (Pearce et al., 2023). Although many commercial vaccines were originally developed using CPV-2 or CPV-2b strains, substantial evidence indicates that vaccines confer effective cross-protection against the

currently circulating CPV-2a, CPV-2b, and CPV-2c variants (Al-Saadi et al., 2025). Furthermore, novel vaccine approaches are being investigated to improve immunogenicity in the presence of high levels of maternal antibodies, potentially enabling effective immunization at earlier ages (Zhou et al., 2024). Nevertheless, sporadic cases of CPV continue to occur despite vaccine availability, often as a consequence of incomplete vaccination schedules, persistent maternal antibody interference, improper vaccine handling or storage, and exposure to high environmental viral loads in endemic settings (Ford et al., 2022).

Table 4. Canine parvovirus vaccines currently available in Ecuador, including vaccine type, vaccination schedule according to age, and recommended immunization protocols

Animal age	Commercial vaccine	Vaccine type	Protocol	Maternal antibody interference	Biosafety and contraindications
Puppies (4-6 weeks)	Nobivac Canine-1Pv	Modified live virus	Start > 6 weeks; 1 mL SC or IM; boosters every 2-4 weeks	Interference may occur due to maternal antibodies	Vaccinate only healthy animals; do not vaccinate pregnant females; store at 2-8 °C; do not freeze
	Nobivac Puppy DP	Modified live virus	4-6 weeks; 1 mL SC	No significant interference reported	Store at 2-8 °C; protect from light; avoid heat exposure
	Vibix C Puppy Advanced	Inactivated virus	≥ 6 weeks; 1 mL SC	No maternal antibody interference	Deworm at least 10 days before vaccination; store at 2-8 °C; protect from light
	Nobivac Parvo-C	Live attenuated virus	2-4 weeks; 1 mL SC; revaccinate every 3-4 weeks until 3 months	Interference may occur; recommended vaccination at 6-9 weeks	Store at 2-8 °C; protect from light
Juvenile and adult dogs (> 6 weeks)	Vanguard Plus	Modified live virus	Start at 6 weeks; boosters at 9 and 12 weeks; ≥ 12 weeks: repeat dose after 3 weeks	No significant interference reported	Avoid vaccination in parasitized, weakened, or pregnant animals; store at 2-7 °C; do not freeze
	Nobivac DHPPi	Modified live virus	6-9 weeks; 1 mL SC	Possible interference; final dose recommended at 12 weeks	Store at 2-8 °C; protect from light
	Parvigen	Live attenuated virus	Two doses: 8-12 weeks and 3-4 weeks later (not before 12 weeks); annual revaccination	Maternal antibodies may reduce the response	Store at 2-8 °C; do not freeze; protect from light
	Nobivac Parvo-C	Live attenuated virus	6-9 weeks; 1 mL SC	No significant interference reported	Store at 2-8 °C; protect from light
	Vanguard Plus (Zoetis)	Modified live virus	Start at 6 weeks; boosters at 9 and 12 weeks; ≥ 12 weeks: repeat dose after 3 weeks; 1 mL SC	No significant interference reported	Avoid vaccination in parasitized, weakened, or pregnant animals; store at 2-7 °C; do not freeze

SC: Subcutaneous; IM: Intramuscular; DP: Distemper-parvovirus

In Ecuador, where multiple CPV variants circulate and CPV-2b has been identified as the predominant genotype in recent studies, continuous monitoring of circulating strains is important to support vaccination programs and epidemiological surveillance (Lloor-Giler et al., 2025).

Environmental biosecurity represents another essential component of disease control. Canine parvovirus is exceptionally resistant to environmental degradation and may remain infectious in contaminated environments for extended periods, often exceeding one year under favorable conditions (Mia and Hasan, 2021). Consequently, contaminated areas should undergo thorough cleaning followed by disinfection using sodium hypochlorite solutions in a concentration of 0.37% for 15 minutes or other disinfectants such as iodine, glutaraldehyde with proven efficacy against non-enveloped viruses (Zhang, 2026). Immediate removal of fecal material, isolation of infected animals, and strict control of personnel, equipment, and clothing movement between contaminated and clean areas are critical measures for reducing indirect viral transmission (Decaro et al., 2020; Rivera and Ayora, 2024). Infected dogs should be isolated to minimize viral transmission. Although fecal viral shedding decreases substantially after clinical recovery, dogs may continue shedding CPV for up to 2-4 weeks following infection (Ozaeta et al., 2025). Consequently, isolation for at least

14 days after the resolution of clinical signs is recommended, particularly in multi-dog environments where the risk of transmission is high (Decaro et al., 2020).

Finally, owner and veterinary staff education plays a fundamental role in the prevention of canine parvoviral disease (Horecka et al., 2020). Avoiding the exposure of unvaccinated puppies to public environments, strengthening vaccination programs in shelters and breeding facilities, and maintaining accurate health records contribute substantially to reducing disease incidence (WOAH, 2025). In addition, molecular surveillance of circulating strains, combined with timely reporting of cases, facilitates the early detection of epidemiologically relevant viral changes and supports evidence-based adjustments to local prevention and immunization strategies (Loor-Giler et al., 2025).

Collectively, vaccination, environmental disinfection, isolation of infected animals, epidemiological surveillance, and educational initiatives constitute the most effective integrated approach for the prevention and control of canine parvoviral disease in both urban and rural populations (Ozaeta et al., 2025). More recently, subunit vaccines based on the VP2 protein have shown protective efficacy against heterologous CPV2 strains, suggesting that updated vaccine formulations may enhance protection against emerging viral variants (Jantafong et al., 2022). A summary of the vaccines currently available in Ecuador is presented in Table 4.

CONCLUSION

Canine parvovirus remains a major infectious disease of dogs worldwide due to its high transmissibility, environmental persistence, and ongoing genetic evolution. The emergence and global distribution of CPV variants have important implications for epidemiological surveillance and disease control. In Ecuador, there are three variants (CPV-2a, CPV-2b, and CPV-2c), and CPV-2b was most common in 2022–2023 in different provinces. Furthermore, vaccination remains the most effective preventive strategy, with MLV vaccines providing cross-protection against circulating variants; supportive care (fluid therapy, antiemetics, antimicrobials) is the mainstay of treatment; no antiviral agent is yet approved for routine use.

The marked tropism of CPV for rapidly dividing cells explains the severity of gastrointestinal and hematological manifestations, which can rapidly progress to fatal outcomes if not diagnosed and managed in a timely manner. In this context, advances in diagnostic technologies, particularly molecular techniques such as PCR and qPCR, have significantly improved the accuracy and speed of detection, enabled earlier intervention and improved clinical outcomes.

From a preventive perspective, vaccination remains the most effective strategy for controlling CPV infection. Nevertheless, factors such as maternal antibody interference, incomplete vaccination schedules, and the circulation of multiple viral variants continue to challenge vaccine effectiveness in certain contexts. The emergence and co-circulation of CPV-2 variants highlight the need for continuous molecular surveillance and the periodic evaluation of vaccine formulations to ensure sustained protection.

Future studies should focus on characterizing CPV, the evolution of circulating variants, and identifying mutation hotspots, particularly in the VP2 gene, which are critical for antigenic variation. Such studies are essential to evaluate the potential impact of emerging strains on vaccine efficacy and to guide the development of updated, strain-matched vaccines. Additionally, expanded epidemiological studies in regions such as Ecuador are needed to improve understanding of local viral dynamics and to support the implementation of more effective national prevention and control strategies.

DECLARATIONS

Acknowledgments

The authors would like to thank the support of UPEC–Carrera de Veterinaria, Carchi, Ecuador during the preparation of the present study.

Authors' contributions

Pamela Rodríguez Huera was responsible for conceptualization, investigation, writing of the original draft, visualization, and project administration. David Martínez Martínez contributed to data curation, validation, supervision, and writing, review, and editing. All authors have read and approved the final edition of the manuscript before publication in the present journal.

Availability of data and materials

All relevant data generated during the study are included in the article and are available upon reasonable request from the corresponding author.

Competing interests

The authors have declared no conflict of interest.

Ethical considerations

Ethical issues, including plagiarism, duplicate publication, redundant publication, and authorship misconduct, have been carefully considered and checked by all authors. This manuscript is based exclusively on published scientific literature and did not involve the use of animals, human participants, clinical samples, confidential data, or AI tools in writing and preparation. The authors accept full responsibility about the content of this review.

Funding

The present study was financially supported by the State Polytechnic University of Carchi, Carchi, Ecuador.

REFERENCES

- Abousenna M, Sayed R, Shasha, El Sawy FS, and Darwish D (2024). Sensitivity of lateral flow technique for diagnosis of canine parvovirus. *Scientific Report*, 14(1): 5060. DOI: <https://www.doi.org/10.1038/s41598-024-55548-x>
- Afumba R, Liu J, and Dong H (2022). Apoptosis mechanisms induced by parvovirus infections. *Acta Virologica*, 66(2): 101-109. DOI: https://www.doi.org/10.4149/av_2022_210
- Ain-Fatin R, Nur-Fazila SH, Yasmin AR, Sifa-Shaida AH, Ayuni WN, and Muhamad-Alif Z (2020). Canine parvovirus (CPV) infection in a Great Dane puppy: A case report. *Advanced Animal Veterinary Science*, 8(10): 1075-1078. DOI: <http://www.doi.org/10.17582/journal.aavs/2020/8>
- Al-Saadi M, Nubgan A, and Abbas AH (2025). Comparison of the sequences of the viral capsid protein 1 and viral capsid protein 2 encoded genes in symptomatic and asymptomatic cases of canine parvovirus 2 in dogs. *Veterinary World*, 18(1): 8-14. DOI: <https://www.doi.org/10.14202/vetworld.2025.8-14>
- American animal hospital association (AAHA) (2022). AAHA canine vaccination guidelines: Canine parvovirus (CPV-2), pp. 3-4. Available at: <https://www.aaha.org/wp-content/uploads/globalassets/02-guidelines/2022-aaha-canine-vaccination-guidelines/resources/2022-aaha-canine-vaccinations-guidelines.pdf>
- Basurto MG, Zambrano JD, and Vallejo S (2024). Risk factors associated with canine parvovirus in different climatic regions of Ecuador. *Peer-Reviewed Scientific Journal in Health Research*, 7(14): 240-253. Available at: <https://journalgestar.org/index.php/gestar/article/view/123/224>
- Chen S, Liu F, Yang A, and Shang K (2024). For better or worse: Crosstalk of parvovirus and host DNA damage response. *Frontiers in Immunology*, 15: 1324531. DOI: <https://www.doi.org/doi.org/10.3389/fimmu.2024.1324531>
- Chethan GE, De UK, Singh MK, Chander V, Raja R, Paul BR, Choudhary OP, Thakur N, Sarma K, and Prasad H (2023). Antioxidant supplementation during treatment of outpatient dogs with parvovirus enteritis ameliorates oxidative stress and attenuates intestinal injury: A randomized controlled trial. *Veterinary and Animal Science*, 21: 100300. DOI: <https://www.doi.org/10.1016/j.vas.2023.100300>
- Cho KN, Ouh IO, Park YM, Park MH, Min KM, Kang HJ, Yun SY, Song JY, Hyun BH, Park CK et al. (2022). A plant-produced porcine parvovirus 1-82 VP2 subunit vaccine protects pregnant sows against challenge with a genetically heterologous PPV1 strain. *Vaccines*, 11(1): 54. DOI: <https://www.doi.org/10.3390/vaccines11010054>
- De la Torre D, Mafla E, Puga B, Erazo L, Astolfi-Ferreira C, and Ferreira A (2018). Molecular characterization of canine parvovirus variants (CPV-2a, CPV-2b, and CPV-2c) based on the VP2 gene in affected domestic dogs in Ecuador. *Veterinary world*, 11(4): 480. DOI: <https://www.doi.org/10.14202/vetworld.2018.480-487>
- Decaro N, Buonavoglia CB, and Barrs VR (2020). Canine parvovirus vaccination and immunisation failures: Are we far from disease eradication?. *Veterinary Microbiology*, 247: 108760. DOI: <https://www.doi.org/10.1016/j.vetmic.2020.108760>
- Dines B, Fossum TW, Skidgel E, Singh A, Brown DC, and Smarick SD (2023). Case report: Long-term survival in puppies assessed with echocardiography, electrocardiography and cardiac troponin I after acute death in littermates due to parvoviral myocarditis. *Frontiers Veterinary Science*, 10: 1229756. DOI: <https://www.doi.org/10.3389/fvets.2023.1229756>
- Dos Santos TG, Orlandín JR, de Almeida MF, Scassiotti RF, Oliveira VC, Santos SIP, Pereira A, Avelino P, Pinto F, Clésio M et al. (2023). Ozone therapy: Protocol for treating canine parvovirus infection. *Brazil Journal of Veterinary Medicine*, 45: e004622. DOI: <https://www.doi.org/10.29374/2527-2179.bjvm004622>
- Ford RB, Larson LJ, Schultz RD, Welborn LV, and the AAHA Canine Vaccination Guidelines Task Force (2022). 2022 AAHA canine vaccination guidelines. *Journal of the American Animal Hospital Association*, 58(5): 213-230. <https://www.doi.org/10.5326/JAAHA-MS-7292>
- Flores B, Mairena J, Gutiérrez J, Sheleby-Elías J, Fuertes H, and Halaihel N (2020). Identification of canine parvovirus type 2C in puppies from Nicaragua. *Journal MVZ Córdoba*, 25(2): 11-16. DOI: <https://www.doi.org/10.21897/rmvz.1788>
- Franzo G, Mira F, Schirò G, and Canuti M (2023). Not Asian anymore: Reconstruction of the history, evolution, and dispersal of the Asian lineage of CPV-2c. *Viruses*, 15(9): 1962. DOI: <https://www.doi.org/10.3390/v15091962>
- Gerlach M, Proksch AL, Dorfelt R, Unterer S, and Hartmann K (2020). Therapy of canine parvovirus infection – review and current insights. *Tierarztl Prax Ausg K Kleintiere Heimtiere*, 48(1): 26-37. DOI: <https://www.doi.org/10.1055/a-1020-3341>
- Gjurovski I, Bozinovski S, Murdzevska E, Novakov T, Bozkurt MF, and Ristoski T (2023). Immunohistochemical diagnostic characteristics of parvovirus infection in dogs. *Macedonian Veterinary Review*, 46(2): 193-198. DOI: <https://www.doi.org/10.2478/macvetrev-2023-0024>
- Grecco S, Condon E, Bucafusco D, Bratanich AC, Panzera Y, and Pérez R (2024). Comparative genomics of canine parvovirus in South America: Diversification patterns in local populations. *Infection, Genetics and Evolution*, 123: 105633. DOI: <https://www.doi.org/10.1016/j.meegid.2024.105633>
- Horecka K, Porter S, Amirian ES, and Jefferson E (2020). A decade of treatment of canine parvovirus in an animal shelter: A retrospective study. *Animals*, 10(6): 939. DOI: <https://www.doi.org/10.3390/ani10060939>
- Hyeon LS, Yang DK, Kim EJ, Park YR, Lee HJ, and Hyun BH (2023). Evaluation of commercial immunochromatography test kits for diagnosing canine parvovirus. *Korean Journal of Veterinary Research*, 63(2): 19-21. DOI: <https://www.doi.org/10.14405/kjvr.20230016>

- Iribarnegaray V, Godiño G, Larrañaga C, Yamasaki K, Verdes JM, and Puentes R (2024). Droplet digital PCR enhances sensitivity of canine distemper virus detection. *Viruses*, 16(11): 1720. DOI: <https://www.doi.org/10.3390/v16111720>
- Jantafong T, Ruenphet S, Garner HR, and Ritthipichai K (2022). Tracing the genetic evolution of canine parvovirus type 2 (CPV-2) in Thailand. *Pathogens*, 11(12): 1460. DOI: <https://www.doi.org/10.3390/pathogens11121460>
- Jyothi VP, Bhaskaran MS, and Gundi VAKB (2024). Epidemiology, molecular prevalence and prevention on canine parvovirus in India: A review. *Bioinformation*, 20(5): 536-546. DOI: <https://www.doi.org/10.6026/973206300200536>
- Kim D, Han S, Go H, Kim D, Kim J, Lee J, Seung Y, Chang S, Sang W, and In Soo C (2022). Antiviral activity of canine interferon lambda 3 expressed by a recombinant adenovirus against canine coronavirus, canine parvovirus, and canine distemper virus. *Veterinary Research Communications*, 46: 1363-8. DOI: <https://www.doi.org/10.1007/s11259-022-10000-1>
- Kurucay H, Tamer C, Muftuoglu B, Elhag A, Gozel S, Cicek-Yildiz Y, Demirtas S, Ozan E, Harun A, Gumusova S et al. (2023). First isolation and molecular characterization of canine parvovirus-type 2b (CPV-2b) from red foxes (*Vulpes vulpes*) living in the wild habitat of Turkey. *Virology Journal*, 20(1): 27. DOI: <https://www.doi.org/10.1186/s12985-023-01988-2>
- Li H, Li S, Pan Y, and Shi Q (2025). Decoding canine parvovirus: Biomarkers for diagnosis and advances in vaccine development to address emerging challenges. *Frontiers in Veterinary Science*, 12: 1624275. DOI: <https://www.doi.org/10.3389/fvets.2025.1624275>
- Liu C, Chen Y, Cui N, Yang Y, Ding H, Wu H, and Xu X (2025). Molecular detection of canine viral infectious diseases in China: An investigation from 2018 to 2024. *Frontiers in Veterinary Science*, 12: 1709294. DOI: <https://www.doi.org/10.3389/fvets.2025.1709294>
- Loor-Giler A, Castillo-Reyes S, Santander-Parra S, Campos M, Mena-Pérez R, and Prado-Chiriboga S (2024). Molecular detection and quantification of canine parvovirus 2 using a fast and sensitive SYBR® green-based quantitative polymerase chain reaction assay in dogs affected with gastroenteritis. *Veterinary World*, 17(10): 2286-2294. DOI: <https://www.doi.org/10.14202/vetworld.2024.2286-2294>
- Loor-Giler A, Santander-Parra S, Castillo-Reyes S, Campos M, Mena-Pérez R, Prado-Chiriboga S, and Nuñez L (2025). Characterization, quantification, and molecular identification of co-infection of canine parvovirus (CPV-2) variants in dogs affected by gastroenteritis in Ecuador during 2022-2023. *Veterinary Science*, 12(1): 46. DOI: <https://www.doi.org/10.3390/vetsci12010046>
- Lopes TS, Lunge VR, and Streck AF (2024). Antiviral alternatives against important members of the Parvovirinae subfamily: A review. *Archives of Virology*, 169: 52. DOI: <https://www.doi.org/10.1007/s00705-024-05995-8>
- López-Astacio RA, Adu OF, Lee H, Hafenstein SL, and Parrish CR (2023). The structures and functions of parvovirus capsids and missing pieces: The viral DNA and its packaging, asymmetrical features, nonprotein components, and receptor or antibody binding and interactions. *Journal of Virology*, 97(7): e0016123. DOI: <https://www.doi.org/10.1128/jvi.00161-23>
- Loza-Vega AJ (2025). Evolution of canine parvovirus and the emergence of variant 2c in South America: Virological and epidemiological perspectives. *Journal of the Selva Andina Animal Science*, 12(1): 1-5. DOI: <https://www.doi.org/10.36610/j.jsaas.2025.120100001>
- Maganga GD, Labouba I, Milendz Ikapi SZ, Nkili-Meyong AA, Ngonga Dikongo AM, Boundenga L, and Kumulungui BS (2023). Molecular characterization of canine parvovirus variants CPV-2a and CPV-2c, associated with vaccinated dogs at Libreville, Gabon. *Viruses*, 15(5): 1169. DOI: <https://www.doi.org/10.3390/v15051169>
- Magouz A, Elhaig MM, Algebaly HA, and El Zowalaty ME (2023). Molecular typing of canine parvovirus type 2 by VP2 gene: Prevalence and mutations. *Frontiers in Microbiology*, 14: 1123456. DOI: <https://www.doi.org/10.3389/fmicb.2023.1254060>
- Mazzaferro EM (2020). Update on canine parvoviral enteritis. *Small Animal Practice*, 50(6): 1307. DOI: <http://www.doi.org/10.1016/j.cvsm.2020.07.008>
- Mia MM and Hasan M (2021). Update on canine parvovirus infection: A review from the literature. *Veterinary Sciences: Research and Reviews*, 7(2): 92-100. DOI: <https://www.doi.org/10.17582/journal.vsr/2021.7.2.92.100>
- Miranda C and Thompson G (2016). Canine parvovirus: The worldwide occurrence of antigenic variants. *Viruses*, 14(8): 1759. DOI: <https://www.doi.org/10.1099/jgv.0.000540>
- Muñoz AI, Vallejo-Castillo L, Fragozo A, Vázquez-Leyva S, Pavón L, and Pérez (2021). Increased survival in puppies affected by Canine Parvovirus type II using an immunomodulator as a therapeutic aid. *Science Reports*, 11: 19864. <https://www.doi.org/10.1038/s41598-021-99357-y>
- Nandi S, De D, and Panda SK (2022). Diagnostic evaluation of canine parvovirus infection using conventional and molecular methods. *Journal of Veterinary Science and Technology*, 13(2): 1-8. DOI: <https://www.doi.org/10.35248/2157-7579.22.13.002>
- Ozaeta DS, Williman MM, Negrelli P, Echeverría MG, Metz GE, Serena MS, and Williams S (2025). Parvoviruses: A review of the epidemiology, pathogenesis, diagnosis, and control of parvovirus diseases in domestic animals. *Revista Veterinaria*, 36(1): 1-16. DOI: <https://www.doi.org/10.30972/vet.3618084>
- Page MJ, Moher D, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, Shamseer L, Tetzlaff JM, Akl EA, Brennan SE et al. (2021). PRISMA 2020 explanation and elaboration: Updated guidance and exemplars for reporting systematic reviews. *The BMJ*, 372: n160. DOI: <https://www.doi.org/10.1136/BMJ.N160>
- Parrish, CR and Sykes JE (2021). Canine parvovirus infections and other viral enteritides. *Greene's infectious diseases of the dog and cat*, pp. 341-351. DOI: <https://www.doi.org/10.1016/B978-0-323-50934-3.00029-X>
- Pearce J, Spibey N, Sutton D, and Tarpey I (2023). Development of a novel canine parvovirus vaccine capable of stimulating protective immunity in four-week-old puppies in the face of high levels of maternal antibodies. *Vaccines*, 11(9): 1499. DOI: <https://www.doi.org/10.3390/vaccines11091499>
- Pénzes JJ, Pham HT, Chipman P, Smith EW, McKenna R, and Tijssen P (2023). Bipartite genome and structural organization of the parvovirus *Acheta domesticus* segmented densovirus. *Natural Community*, 14(1): 3515. DOI: <https://www.doi.org/10.1038/s41467-023-38875-x>
- Purwitasari MS, Soma IG, and Batan IW (2022). Healing of enteric hemorrhage in domestic puppy positively infected with canine parvovirus: A case report. *Journal Kajian Veterinary*, 10(1): 67-81. DOI: <https://www.doi.org/10.35508/jkv.v10i1.6290>
- Qi S, Zhao J, Guo D, and Sun D (2020). A mini-review on the epidemiology of canine parvovirus in China. *Frontiers in Veterinary Science*, 7: 5. DOI: <https://www.doi.org/10.3389/fvets.2020.00005>
- Reyes-Cruz T, Martínez-Gómez D, Verdugo-Rodríguez A, Bustos-Martínez J, López-Islas J, and Méndez-Olvera ET (2021). Participation of interferon type I during canine parvovirus infection. *Polish Journal of Veterinary Sciences*, 24(3): 375-384. DOI: <https://www.doi.org/10.24425/pjvs.2021.138728>
- Rivera G and Ayora J (2024). Exploring new trends in the treatment of canine parvovirosis: A literature review. *MQRInvestigar*, 8(4): 2706-2738. DOI: <https://www.doi.org/10.56048/MQR20225.8.4.2024.2706-2738>

- Sarchese V, Di Profio F, Robetto S, Orusa R, Vuillermoz B, Pellegrini F, Marsilio F, Martella V, and Di Martino B (2025). Molecular survey for major canine enteric viral pathogens in wild carnivores, northwestern Italy. *Veterinary Science*, 12(9): 814. DOI: <https://www.doi.org/10.3390/vetsci12090814>
- Schmitz SS (2021). Value of probiotics in canine and feline gastroenterology. *Small Animal Practice*, 51: 171-217. DOI: <https://www.doi.org/10.1016/j.cvs.2020.09.011>
- Segev G, Yaaran T, Maurice S, and Baneth G (2022). Effect of sampling site on the diagnosis of canine parvovirus infection in dogs using polymerase chain reaction. *Journal of Veterinary Internal Medicine*, 36(2): 591-598. DOI: <https://www.doi.org/10.1111/jvim.16373>
- Serena M, Cappuccio J, Barrales H, Metz G, Aspitia C, Lozada I, Perfumo C, Quiroga M, Piñeyro P, and Echeverría G (2021). First detection and genetic characterization of porcine circovirus type 3 (PCV3) in Argentina and its association with reproductive failure. *Transboundary Emerging Diseases*, 68(4): 1761-1766. DOI: <https://www.doi.org/10.1111/tbed.13893>
- Sherasiya R, Vagh A, Bilwal A, Damor J, Parmar V, Khorajiya J, Vinay B, and Priyanshi P (2024). Evaluation of various alterations in clinical signs and clinical parameters in canine parvoviral infection. *International Journal of Veterinary Sciences and Animal Husbandry*, 9(2): 550-3. Available at: <https://www.veterinarypaper.com/pdf/2024/vol9issue2/PartH/9-2-80-843.pdf>
- Shi J, Pei Y, Yu Q, and Dong H (2023). Progress in the study of parvovirus entry pathway. *Virology Journal*, 20(1): 61. DOI: <https://www.doi.org/10.1186/s12985-023-02016-z>
- Squires RA, Crawford C, Marcondes M, and Whitley N (2024). 2024 guidelines for the vaccination of dogs and cats—compiled by the vaccination guidelines group (VGG) of the world small animal veterinary association (WSAVA). *Journal of Small Animal Practice*, 65(5): 277-316. DOI: <https://www.doi.org/10.1111/jsap.13718>
- Suvethika P and Kumar KSV (2021). Canine parvovirus infection: A case report. *Pharma Innovation Journal*, 10(1): 141-143. Available at: <https://www.thepharmajournal.com/archives/2021/vol10issue1/PartB/9-12-62-902.pdf>
- Tuteja D, Banu K, and Mondal B (2022). Canine parvovirology: A brief updated review on structural biology, occurrence, pathogenesis, clinical diagnosis, treatment and prevention. *Comparative Immunology Microbiology and Infectious Diseases*, 82: 101765. DOI: <https://www.doi.org/10.1016/j.cimid.2022.101765>
- Uhl LK and Fasina OO (2025). Parvovirus RNA processing: Compact genomic organization and unique alternative mRNA processing mechanisms. *Viruses*, 17(7): 984. DOI: <https://www.doi.org/10.3390/v17070984>
- Ulas N, Ozkanlar Y, Ozkanlar S, Timurkan MO, and Aydin H (2024). Clinical and inflammatory response to antiviral treatments in dogs with parvoviral enteritis. *Journal Veterinary Science*, 25: e11. DOI: <https://www.doi.org/10.4142/jvs.23139>
- World organization for animal health (WOAH) (2025). The state of animal health in the world 2025. World Organization for Animal Health, Paris. Available at: <https://www.woah.org/app/uploads/2025/05/el-estado-de-la-sanidad-animal-en-el-mundo.pdf>
- Yin S, Zhang G, Lyu Y, Wang J, Liu W, and Qin T (2022). Characterization of the VP2 and NS1 genes from canine parvovirus type 2 (CPV-2) and feline panleukopenia virus (FPV) in northern China. *Frontier Veterinary Science*, 9: 934849. DOI: <https://www.doi.org/10.3389/fvets.2022.934849>
- Zhang H (2026). Prevention and control of canine parvovirus. *American Journal of Student Research*, 4(2): 522-531. DOI: <https://www.doi.org/10.70251/HYJR2348.42522532>
- Zhou H, Cui K, Su X, Zhang H, Xiao B, Li S, and Yang B (2024). Overview of recent advances in canine parvovirus research: Current status and future perspectives. *Microorganisms*, 13(1): 47. DOI: <https://www.doi.org/10.3390/microorganisms13010047>

Publisher's note: Sciencline Publication Ltd. remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Open Access: This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <https://creativecommons.org/licenses/by/4.0/>.

© The Author(s) 2026