Review

Canine Parvovirus: Epidemiology, Pathogenesis, Diagnosis and Disease Control. PVC: Epidemiology, Pathogenesis, Diagnosis and Control

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Article

Canine Parvovirus: Epidemiology, Pathogenesis, Diagnosis and Disease Control. PVC: Epidemiology, Pathogenesis, Diagnosis and Control

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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. This study provides an updated and integrative overview of epidemiology, pathogenesis, diagnosis, and control of CPV through a systematic qualitative review conducted following PRISMA guidelines. A comprehensive literature search was carried out using major academic databases, including PubMed, SciELO, MDPI, and Google Scholar, 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 detailed analysis. The results confirm the global circulation of multiple variants (CPV-2a, CPV-2b, and CPV-2c), as well as the persistence of the virus in diverse environments and its impact on susceptible populations. Molecular diagnostic techniques, particularly PCR and qPCR, remain the most reliable tools for early detection, although their availability is still uneven across regions. 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; CPV-2a; CPV-2b; CPV-2c; viral evolution; qPCR diagnosis; enteric infection; immunization

1. Introduction

Canine parvovirus (CPV) remains one of the most significant viral pathogens affecting domestic dogs worldwide, particularly in young and unvaccinated populations, due to its high environmental resistance and rapid transmission [1,2]. Since its emergence in the late 1970s, CPV has undergone rapid genetic evolution, leading to the emergence of multiple antigenic variants that continue to circulate globally and contribute to the persistence of the disease [3–5]. Its efficient fecal–oral transmission and prolonged environmental stability allow the virus to spread rapidly in both urban and rural environments, representing an ongoing challenge for veterinary practice and disease control [6,7].

From a biological perspective, CPV is a small, non-enveloped, single-stranded DNA virus belonging to the genus *Protoparvovirus* within the family *Parvoviridae* [8,9]. Despite its relatively simple genome, the virus exhibits a remarkable capacity for adaptation, with mutation rates comparable to those of RNA viruses, facilitating the emergence of variants such as CPV-2a, CPV-2b,

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and CPV-2c [24,38,70]. These variants differ in their epidemiological distribution, host interaction, and potential implications for vaccine effectiveness [10,11].

The pathogenesis of CPV is closely associated with its tropism for rapidly dividing cells, particularly those of the intestinal epithelium and bone marrow, leading to severe clinical manifestations such as hemorrhagic enteritis, leukopenia, dehydration, and, in severe cases, death [2,12]. In addition, the destruction of immune cells predisposes infected animals to secondary infections, further complicating clinical outcomes [13,14].

Advances in molecular diagnostics have significantly improved the detection of CPV, with techniques such as polymerase chain reaction (PCR) and quantitative PCR (qPCR) offering high sensitivity and specificity [12,15]. However, their implementation in routine clinical practice remains uneven, particularly in resource-limited settings [16]. At the same time, despite the availability of effective vaccines, outbreaks continue to be reported, often associated with incomplete vaccination schedules, viral evolution, or failures in biosecurity measures [13,17].

In Latin America, and particularly in Ecuador, recent studies have reported the circulation of multiple CPV variants, including CPV-2a, CPV-2b, and CPV-2c, highlighting the importance of continuous molecular surveillance and updated control strategies [18,19]. This regional evidence underscores the need to better understand the dynamics of CPV infection in different epidemiological contexts.

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 but also to identify gaps and emerging challenges. Therefore, the aim of this study was to provide a comprehensive and updated overview of the epidemiology, pathogenesis, diagnosis, and control strategies of CPV through a systematic qualitative review of recent literature.

2. Materials and Methods

This study was designed as a qualitative systematic review aimed at integrating current scientific evidence on canine parvovirus (CPV), with particular emphasis on its epidemiology, pathogenesis, diagnostic approaches, and control strategies. The methodological approach followed established principles for systematic reviews in biomedical research, allowing a structured and transparent selection of the available literature.

A comprehensive search was conducted in recognized academic databases, including PubMed, SciELO, Scopus-indexed sources, and Google Scholar, to identify relevant studies published between 2020 and 2026. These databases were selected due to their broad coverage of veterinary and biomedical research. 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 [20–22]. The distribution of retrieved records across databases is summarized in Table 1.

Table 1. Databases used for the literature search.

Database	Results
PubMed	66
Google Scholar	135
MDPI	96
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 only open-access articles and to restrict the publication period to the selected timeframe.

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The initial search yielded a total of 312 records. Following the application of inclusion and exclusion criteria, as well as the removal of duplicates, 70 studies were selected for detailed analysis. The selection process was conducted according to the PRISMA framework, ensuring transparency in the identification, screening, eligibility, and inclusion stages, as shown in Figure 1.

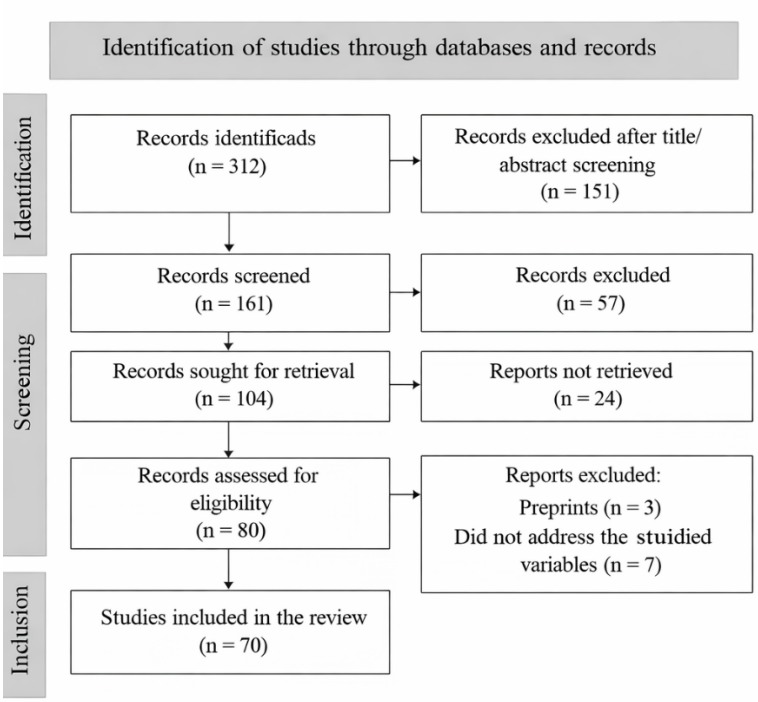

Figure 1. Identification of studies through databases and records.

3. Molecular Classification

The family *Parvoviridae* comprises a diverse group of viruses characterized by a small, non-enveloped icosahedral capsid enclosing a single-stranded DNA genome of approximately 4 to 6 kilobases. The viral particle typically measures between 18 and 26 nm in diameter [23]. Due to their DNA nature, parvoviruses replicate within the nucleus of actively dividing host cells, relying on the host cellular machinery for genome replication.

Members of the *Parvoviridae* family infect a wide range of hosts and are currently classified into three main subfamilies. The subfamily *Densovirinae* includes viruses that infect invertebrates, whereas *Parvovirinae* comprises viruses infecting vertebrate hosts, such as mammals, birds, and reptiles. More recently, a third subfamily, *Hamaparvovirinae*, has been described, including viruses associated with both crustaceans and insects. This classification is largely based on the sequence homology of conserved genomic regions, particularly within the helicase domain [24].

Within the subfamily *Parvovirinae*, viruses are further divided into several genera according to host specificity and genetic characteristics. This classification reflects the evolutionary diversity and adaptability of parvoviruses across different species. A summary of the main genera, their associated hosts, and susceptible species is presented in Table 2.

Table 2. Parvoviridae: genera, hosts, and susceptible species.

Genus	Hosts	Susceptible Species
Amdoparvovirus	Carnivores	Foxes (Aleutian mink disease virus), Minks (Aleutian disease virus)
Aveparvovirus	Galliform birds	Chickens (Chicken parvovirus), Turkeys (Turkey parvovirus)

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Bocaparvovirus	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)
Copiparvovirus	Ungulates	Bovines (Bovine adeno-associated virus), Caprines (Caprine adeno-associated virus)
Dependoparvovirus	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)
Erythroparvovirus	Ungulates	Bovines (Bovine parvovirus 3)
	Carnivores	Canines (Canine parvovirus), Felines (Feline parvovirus), Minks (Mink enteritis virus), Raccoons, Arctic wolves
Protoparvovirus	Primates	Humans (Bufavirus 1a, 1b, and 2)
	Rodents	Rats (Kilham rat virus, Rat parvovirus 1), Mice (Minute virus of mice Mp, Mm, Mi, Mc; Mouse parvovirus 1–5), Hamsters
	Ungulates	Swine (Porcine parvovirus type 1, Kresse strain, NADL-2)
	Chiroptera	Fruit bats (*Eidolon helvum* parvovirus)
Tetraparvovirus	Primates	Humans (Human parvovirus 4 genotypes G1–G3), Chimpanzees
	Ungulates	Bovines (Bovine hokovirus 1 and 2), Swine (Porcine parvovirus type 2 and 3), Sheep (Ovine hokovirus)

Structure and Genome

Parvoviruses are small, non-enveloped viruses, a characteristic that contributes to their remarkable stability under adverse environmental conditions. The viral particle consists of an icosahedral capsid with an approximate diameter of 18–26 nm, composed of 60 copies of structural viral proteins (VP), among which VP2 is the predominant component [25]. This protein plays a central role in capsid assembly, host receptor interaction, and antigenic variability. In canine parvovirus, a third capsid protein, VP3, has also been described, likely originating from post-translational modification of VP2 during infection [26].

The genome of parvoviruses is composed of a single-stranded linear DNA molecule, typically ranging from 4.5 to 5.5 kilobases in length, as illustrated in Figure 2. A distinctive feature of this genome is the presence of terminal palindromic sequences that fold into hairpin structures [27]. These secondary structures are essential for viral replication, as they serve as primers for DNA synthesis and enable the formation of replication intermediates.

Replication follows a mechanism known as rolling-hairpin replication, in which the genome undergoes continuous strand displacement and folding processes that allow efficient DNA synthesis. The palindromic terminal regions, together with adjacent nucleotide sequences, contain the necessary signals for genome replication and packaging [25,28]. In addition, these hairpin structures participate in the resolution of genome ends through the activity of viral proteins such as NS1, which introduce site-specific nicks and generate new 3′-OH termini that are subsequently extended by the host cell replication machinery [29].

Importantly, viral replication is tightly linked to the host cell cycle and occurs predominantly in cells that are actively dividing. This dependency is associated with the requirement for cellular factors expressed during the S phase, which are essential for DNA synthesis and viral genome amplification.

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This feature explains the strong tropism of parvoviruses for rapidly proliferating tissues, such as intestinal epithelium and bone marrow.

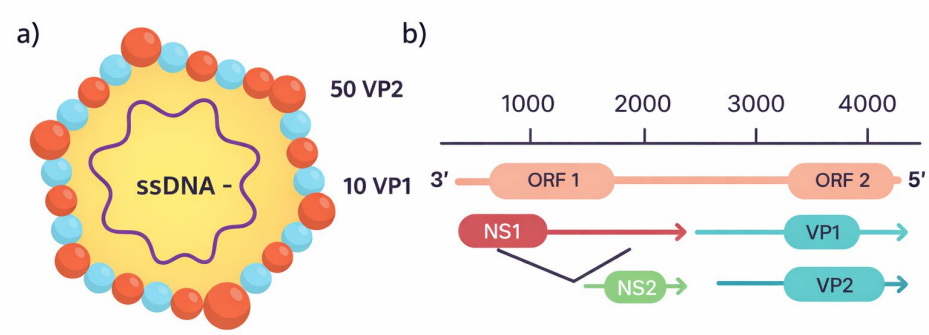

Figure 2. Representation of the structure of parvoviruses. (a) Viral capsid. (b) ORF1 and ORF2 regions showing structural and non-structural proteins of the parvovirus. Adapted from: https://www.youtube.com/watch?v=Fhi8vQOBxis.

The parvoviral genome contains two major open reading frames (ORFs), which are responsible for encoding both non-structural and structural proteins essential for viral replication and assembly. The left ORF encodes the *rep* gene, which directs the expression of non-structural proteins, including NS1. This protein plays a central role in viral replication and is also associated with cytopathic effects, such as necrosis induced by the accumulation of reactive oxygen species (ROS), facilitating viral release. In addition, NS1 exhibits multiple enzymatic activities, including endonuclease (nickase) and helicase/ATPase functions, and is involved in the initiation of viral DNA replication, interaction with host replication machinery, and regulation of transcription [30].

Furthermore, NS2 has been implicated in processes related to apoptosis and may modulate NS1-associated cytotoxic effects, although its precise role remains less clearly defined [31].

The second open reading frame (ORF2) encodes the *cap* gene, which contains the genetic information required for the synthesis of the structural capsid proteins VP1 and VP2. VP1 is characterized by the presence of arginine- and lysine-rich regions associated with nuclear localization signals and includes a unique N-terminal region (VP1u), which is essential during viral entry. In contrast, VP2 constitutes the major structural component of the capsid and plays a role in host interaction and antigenic variation. Notably, VP1 contains a phospholipase A2 (PLA2) domain that enables the virus to escape from the endosomal/lysosomal pathway following cell entry [32]. These structural features contribute to viral adaptability, including the emergence of new variants and changes in host range [5].

Additionally, ORF2 expression is regulated through specific mRNA processing mechanisms, including alternative splicing and the use of alternative start codons, which control the relative proportions of VP1 and VP2 within the viral particle. Consequently, the organization and sequence variability of ORF2 are critical not only for viral biology but also for molecular surveillance and vaccine development strategies [33].

Due to their small genome size and limited coding capacity, parvoviruses rely on multifunctional structural and non-structural proteins to complete their replication cycle. These proteins participate in a wide range of interactions with host cellular components, allowing the virus to efficiently regulate infection, dissemination, and persistence within the host environment.

Cellular Tropism

Parvoviruses exhibit a highly restricted cellular tropism that depends on both the presence of specific surface receptors and the differentiation state and cell cycle phase of the host cell. In the case of protoparvoviruses, such as canine parvovirus (CPV), viral entry is primarily mediated through the transferrin receptor type 1 (TfR1), which is abundantly expressed in rapidly dividing epithelial cells, particularly in the intestinal tract [34].

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Viral entry is initiated by receptor binding followed by clathrin-mediated endocytosis. Upon endosomal acidification, conformational changes in the capsid expose the N-terminal region of VP1 (VP1u), which contains a phospholipase A2 (PLA2) activity. This enzymatic function is essential for endosomal membrane disruption, allowing the release of the viral particle into the cytosol [35].

Following endosomal escape, the viral particle is transported toward the nucleus, where the single-stranded DNA genome is released. Replication then occurs using the host cell DNA polymerase through a rolling-hairpin mechanism that depends on inverted terminal repeats (ITRs) located at the 5′ and 3′ ends of the viral genome. The non-structural protein NS1 plays a critical role in this process by recognizing these sequences and functioning as a nickase and helicase, as well as recruiting host replication and repair factors [36].

In addition, alternative transcription and mRNA splicing generate the transcripts required for the synthesis of both non-structural (NS) and structural (VP) proteins. These proteins coordinate viral replication, particle assembly, and modulation of host antiviral responses. At later stages of infection, parvoviruses induce apoptosis through multiple pathways, facilitating the release and dissemination of newly formed viral particles [37].

Parvovirus in Wildlife

Canine parvovirus (CPV) emerged as a canine pathogen in the late 1970s, likely originating from a cross-species transmission event involving parvoviruses circulating in wild carnivores. It has been proposed that CPV evolved from a virus closely related to feline panleukopenia virus (FPV), rather than directly from porcine parvovirus (PPV), through a process of host adaptation. Although definitive evidence remains limited, this hypothesis is supported by the identification of intermediate viral strains between FPV and CPV in wildlife, as well as the inability of FPV to efficiently infect canine hosts [38].

Wild carnivores play an important role in the ecology and maintenance of CPV. In regions such as Turkey and Italy, wild canids—including red foxes (*Vulpes vulpes*), Eurasian badgers, and wolves—have been identified as potential reservoirs, contributing to the persistence and transmission of the virus to domestic dog populations [39,40]. This interface between wildlife and domestic animals represents a critical point for viral circulation and epidemiological dynamics.

CPV-2 was first identified in 1978 as a highly virulent pathogen in dogs and rapidly spread worldwide. Its emergence is associated with a host-switching event from FPV-like viruses infecting wild felids, followed by rapid global dissemination. The genomic evolution of CPV has been notably fast, with mutation rates estimated to be comparable to those of certain RNA viruses, which may explain the continuous emergence of antigenic variants with distinct biological and epidemiological characteristics [28,41].

Serological studies have demonstrated a high prevalence of antibodies against FPV in wild felid populations, suggesting exposure through contact with domestic animals or shared environments [42]. These findings highlight the complexity of viral transmission cycles involving both wild and domestic hosts.

In addition to carnivores, wild boars (*Sus scrofa*) have been recognized as important reservoirs of various viral pathogens, including porcine parvovirus. Several studies conducted in Europe and the Americas have documented the presence and prevalence of PPV in wild populations, emphasizing their potential role in the transmission of infections to domestic livestock [43–46]. These observations reinforce the importance of considering wildlife in the epidemiology and control of parvoviral infections.

Parvovirus in Ecuador

In Ecuador, recent molecular and epidemiological studies on canine parvovirus type 2 (CPV-2) have confirmed the circulation of the three classical antigenic variants, namely CPV-2a, CPV-2b, and CPV-2c. Evidence from a multicenter study conducted between 2022 and 2023, which included clinical samples from six provinces (Carchi, Chimborazo, Guayas, Imbabura, Pichincha, and Santo Domingo de los Tsáchilas), identified CPV-2b as the predominant variant in the analyzed cases [47]. This study employed quantitative PCR (qPCR) and molecular characterization of the VP2 region,

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allowing not only the detection of viral presence but also the estimation of viral load and the identification of co-infections among different variants.

Earlier studies conducted in Ecuador, based on smaller sample sizes and more limited geographic coverage, reported different patterns of variant distribution, with CPV-2a being the most prevalent, followed by CPV-2b [48]. These discrepancies may be explained by methodological differences, including reduced sample numbers, localized sampling strategies, and the use of conventional sequencing techniques without large-scale quantitative analysis.

Age-related prevalence patterns indicate that dogs between 3 and 12 months represent the most affected group, with a progressive decrease in infection probability as age increases [49]. This trend is consistent with the relative immaturity of the immune system in young animals, which increases susceptibility to infection. In addition, most reported cases in Ecuador correspond to domestic dogs presenting with hemorrhagic gastroenteritis, with puppies under six months of age being particularly vulnerable to severe disease due to declining maternal antibody levels [50].

The detection of co-circulating variants and co-infections highlights the need for continuous molecular surveillance, as well as the periodic evaluation and updating of vaccination strategies. The high prevalence, elevated viral loads, and genetic variability observed in Ecuadorian populations may have important implications for vaccine effectiveness and disease control efforts [51].

Epidemiology: Susceptible Hosts

Parvoviruses are highly contagious pathogens that primarily affect both domestic and wild canids, with a worldwide distribution and a high morbidity rate. Susceptibility is particularly associated with animals that are unvaccinated or have incomplete vaccination schedules. In addition to immunization status, host-related factors such as breed, age, and health condition play a significant role in disease susceptibility.

Epidemiological studies have identified certain dog breeds as being more prone to severe infection, including Doberman Pinschers, Rottweilers, American Pit Bull Terriers, Labrador Retrievers, and German Shepherds, as well as their crossbreeds. Although the underlying reasons for this increased susceptibility are not fully understood, genetic and immunological factors are likely involved [52].

The risk of morbidity and mortality is further increased in animals with predisposing clinical conditions, such as enteritis or myocarditis, and in those affected by concurrent infections. Co-infections with other viral pathogens—including canine distemper virus, canine enteric coronavirus, rotavirus, astrovirus, adenovirus, calicivirus, and emerging viruses such as norovirus, kobuvirus, sapovirus, and circovirus—can compromise the immune response and facilitate CPV infection [52]. This highlights the importance of considering parvovirus infection within a broader context of multifactorial disease processes.

Although adult dogs can become infected, they often remain asymptomatic carriers, shedding the virus through feces and contributing to environmental contamination and transmission [53]. The primary route of transmission is fecal–oral, either through direct contact with infected animals or indirectly via contaminated fomites. Notably, the environmental persistence of CPV is a critical factor in its epidemiology, as the virus can remain viable for extended periods due to its resistance to common disinfectants and adverse environmental conditions [54].

Pathogenesis

The primary route of transmission of canine parvovirus type 2 (CPV-2) is fecal–oral, occurring through the ingestion of contaminated feces or contact with infected surfaces. In high-density environments such as kennels, body secretions from infected animals may also contribute to viral spread among susceptible hosts.

CPV-2 exhibits a marked tropism for rapidly dividing cells. Following initial infection, the virus replicates in lymphoid tissues of the oropharyngeal region, particularly within lymphocytes. It then disseminates via the bloodstream, reaching the bone marrow, where it infects and destroys hematopoietic cells, leading to severe leukopenia. This early replication in lymphoid tissues results in viremia, which typically develops within the first five days post-infection [55].

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In puppies born to non-immunized dams, infection during the first weeks of life may lead to viral myocarditis due to replication of the virus in cardiac muscle cells. However, this clinical presentation has become relatively uncommon, largely due to the presence of maternally derived antibodies (MDA), which provide early protection in most neonates [56].

As the infection progresses, the virus spreads from infected leukocytes to the epithelial cells of the small intestine, particularly targeting the crypts of Lieberkühn. These cells are responsible for continuous epithelial renewal; therefore, their destruction disrupts the regeneration of intestinal villi. The resulting structural damage leads to villous atrophy and collapse, impairing nutrient absorption and causing acute diarrhea [57].

In addition, the destruction of circulating and tissue lymphocytes results in lymphopenia and neutropenia, predisposing affected animals to secondary bacterial infections. The loss of intestinal epithelial integrity, combined with inflammation and crypt necrosis, facilitates bacterial translocation, which may ultimately lead to septicemia [58].

Infected animals begin to shed detectable viral particles in feces approximately 4 to 7 days after exposure, contributing to the rapid dissemination of the virus, particularly in environments where animals are housed in proximity [41].

Clinical Signs

The incubation period of canine parvovirus (CPV) infection typically ranges from 4 to 7 days [51]. Early clinical signs in companion animals commonly include anorexia, fever, depression, and lethargy, which rapidly progress to vomiting, hemorrhagic diarrhea, loss of appetite, and severe dehydration [27,52].

CPV infection causes significant fluid and protein loss through the gastrointestinal tract, leading to marked dehydration and, in severe cases, hypovolemic shock [51]. The small intestinal epithelium represents the primary site of viral replication, resulting in extensive cellular destruction, hemorrhage, and villous atrophy in the duodenum, jejunum, and ileum [38,59].

Two main clinical forms of CPV-2 infection have been described: myocarditis and enteritis [60]. The myocardial form primarily affects puppies younger than two months of age and is often associated with sudden death due to inflammation and necrosis of cardiac muscle, typically occurring between the third and fourth week after infection [12]. Histopathologically, CPV-induced myocarditis is characterized by multifocal inflammation, myocardial fiber degeneration, and necrosis, and in some cases, lesions may extend to other organs such as the lungs, liver, and kidneys [12,55].

In contrast, dogs older than two months are more frequently affected by the enteric form of the disease [61]. This form is mainly observed in animals older than six weeks, when the mitotic activity of intestinal crypt cells is increased, making them more susceptible to viral replication and damage [51]. Clinically, enteritis is characterized by hemorrhagic diarrhea, vomiting, fever, apathy, generalized weakness, and anorexia [62].

From a pathological perspective, lesions include gastric erosion, duodenal hemorrhage, intestinal petechiae, and the presence of serous to granular exudates ranging in color from yellowish to dark brown on the mucosal surface. In advanced cases, extensive ulceration, fusion of intestinal villi, and gallbladder distension may be observed [8].

Recent studies investigating acute diarrhea in dogs have reported a significant association between CPV and canine coronavirus (CCoV), although prevalence varies depending on the age of the animals [29,32]. In addition, CPV clinical signs may be misinterpreted as those of other diseases, including canine distemper, viral enteritis of different etiology, hookworm infections, and coccidiosis [8,20]. According to Lubis et al. [63], parasitic infections such as *Toxocara canis* and *Ancylostoma caninum* should also be considered in the differential diagnosis, as they can present with similar clinical features, including hemorrhagic diarrhea, pale mucous membranes, and vomiting.

Diagnosis

Due to the similarity of clinical signs with other causes of hemorrhagic gastroenteritis, early and accurate diagnosis of canine parvovirus (CPV) infection is essential to prevent disease spread,

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particularly in veterinary clinics and environments with high animal density [24]. Notably, infected animals may begin shedding the virus in feces prior to the onset of clinical signs, increasing the risk of environmental contamination and transmission [11].

Samples commonly used for diagnosis include feces, oropharyngeal swabs, and whole blood. Confirmatory diagnosis can be achieved through several laboratory techniques, including antigen detection by enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR), electron microscopy, hemagglutination assays, and viral isolation in cell culture [11,46]. In clinical practice, rapid immunochromatographic tests and commercial ELISA kits are widely used due to their ease of application and relatively high specificity (≥90%). However, their sensitivity may vary considerably, ranging from 16% to 80% when compared to PCR, which is considered the reference method for CPV detection [64].

Vaccination status should also be considered when interpreting diagnostic results. Modified live virus vaccines may lead to false-positive results for up to approximately 28 days after immunization [11]. Conversely, false-negative results may occur when viral load in fecal samples is low or when intestinal antibodies neutralize viral particles, limiting their detection [6]. For this reason, in animals with compatible clinical signs and negative rapid test results, confirmatory molecular testing—particularly PCR—is strongly recommended to avoid misdiagnosis and reduce the risk of viral dissemination [11,46].

Molecular techniques, especially real-time PCR (qPCR), are currently considered the gold standard for CPV diagnosis due to their high sensitivity and specificity, allowing detection of low viral loads and confirmation of cases with negative antigen-based results (Table 2) [30]. In addition, droplet digital PCR (ddPCR) has emerged as a promising technique, offering higher analytical sensitivity and absolute quantification of viral DNA. However, its application in routine clinical diagnosis of CPV remains limited and is primarily restricted to research settings [23].

Overall, the implementation of sensitive and reliable diagnostic methods is essential for early detection, effective disease control, and prevention of CPV transmission in both clinical and field conditions.

Table 2. 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	Ref
Rapid antigen test (lateral flow)	CPV viral antigen in feces	Sensitivity 96.4% and specificity 88.2% compared with qPCR	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% compared with PCR	Lower sensitivity than molecular methods	Complementary diagnostic support; epidemiological studies	(Li et al., 2025)
Conventional PCR	CPV viral DNA	High sensitivity and specificity; detects virus in clinical infections	Does not quantify viral load; requires laboratory infrastructure	Confirmatory diagnosis when qPCR is not available	(Li et al., 2025)
qPCR (real-time PCR)	CPV viral DNA (quantifiable)	Very high sensitivity and specificity; reference molecular method	Requires specialized equipment and trained personnel	Confirmatory diagnosis and viral load quantification	(Segev et al., 2022; Li et al., 2025)
ddPCR (droplet digital PCR)	Viral DNA (absolute quantification)	Higher analytical sensitivity and greater precision	No specific clinical validation metrics for CPV; mainly used in research	Advanced research; potential for detection of low viral loads	(Iribarnegaray et al., 2024)

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	(shown in related viruses such as CDV)

In addition to diagnostic confirmation, the identification of circulating CPV-2 genotypes represents a critical epidemiological and immunological component. This approach provides valuable insights into viral transmission dynamics, the co-circulation of variants, and their potential impact on vaccine effectiveness. In Ecuador, recent studies have reported the simultaneous circulation of multiple variants, as well as the presence of genotypic co-infections, highlighting the importance of continuous molecular surveillance for effective disease control [65].

Treatment

Canine parvovirus (CPV) infection has a worldwide distribution, and its current management is primarily focused on controlling clinical signs rather than targeting the virus directly. Persistent vomiting and acute hemorrhagic gastroenteritis are among the most characteristic manifestations of the disease [31]. CPV causes severe damage to the epithelial cells of the gastrointestinal tract, promoting bacterial translocation and increasing the risk of sepsis and endotoxemia [27,33].

For this reason, therapeutic management is based on intensive supportive care, in which fluid therapy, antiemetic drugs, and broad-spectrum antibiotics represent the main pillars of treatment. Standard clinical protocols include the intravenous administration of crystalloid solutions, such as Ringer's lactate, often supplemented with potassium chloride according to the individual needs of the patient. In cases of marked hypovolemia or hypoproteinemia, the use of synthetic colloids may be indicated to restore circulatory volume and oncotic pressure [18,27,66].

During the initial 24–48 hours of treatment, enteral feeding is generally withheld, while antiemetics are administered every 6–8 hours and antibiotics are given at regular intervals to prevent or control secondary bacterial infections. In addition, complementary therapies have been explored to enhance clinical recovery, including ozone therapy, immunomodulators, probiotics, and antioxidant supplementation, although their efficacy may vary depending on the clinical context [20,45].

It is important to note that, although these supportive measures significantly improve clinical outcomes and survival rates, they do not constitute a specific antiviral treatment against CPV. Therefore, prevention through vaccination and early intervention remains essential for effective disease control.

Antiviral Agents

Currently, the management of canine parvovirus (CPV) infection relies primarily on supportive symptomatic therapy. However, in animals with high viral loads, conventional treatments are often insufficient to effectively control viral replication. For this reason, recent research has increasingly focused on the development of specific antiviral agents aimed at improving future therapeutic strategies against CPV [11,53].

Current antiviral research can be broadly grouped into three main approaches. First, interferon-based therapies have been investigated due to their immunomodulatory and antiviral properties, with several studies reporting improved survival rates in treated animals [26,53]. Second, antibody-based therapies have shown potential in limiting viral replication by neutralizing viral particles and reducing viral load [32]. Third, a variety of pharmacological compounds have been evaluated for antiviral activity in vitro, demonstrating inhibitory effects on viral replication, although their clinical applicability remains under investigation [67].

Taken together, these approaches represent important advances in the search for targeted antiviral therapies against CPV. Nevertheless, further studies are required to validate their efficacy, safety, and practical application under clinical conditions [47].

Prevention and Control

The control of canine parvovirus (CPV) is primarily based on timely and systematic vaccination, which remains the most effective strategy for preventing infection and limiting outbreaks. Modified live virus (MLV) vaccines are generally recommended due to their ability to induce a strong and long-lasting immune response. In contrast, inactivated vaccines, although safer in certain clinical

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conditions, tend to exhibit lower immunogenicity and require multiple doses to achieve comparable protection, and are therefore reserved for specific cases or experimental applications [63].

Vaccination protocols typically recommend that puppies receive their first dose between 6 and 8 weeks of age, followed by booster doses every 3–4 weeks until 16–20 weeks of age. This schedule is designed to overcome the interference caused by maternally derived antibodies (MDA) and to close the so-called "window of susceptibility," during which vaccine-induced immunity may be insufficient (Table 3). High levels of MDA can neutralize vaccine virus particles, particularly when vaccination is initiated too early or without adequate booster intervals. Subsequent booster vaccinations are recommended annually and then every three years, according to international canine vaccination guidelines [63].

The use of updated vaccines containing representative strains of circulating variants, such as CPV-2a, CPV-2b, and CPV-2c, is essential to maintain protective efficacy, especially in regions where multiple genotypes co-circulate. In Ecuador, where CPV-2b has been identified as a predominant variant, this consideration becomes particularly relevant [35]. Despite the widespread availability and proven effectiveness of vaccines, CPV infections are still reported, often associated with incomplete vaccination schedules, persistent MDA interference, improper vaccine handling, or exposure to high viral loads in endemic environments [68].

Recent evidence suggests that many commercial vaccines, originally based on earlier CPV-2 or CPV-2b strains, continue to provide effective cross-protection against currently circulating variants. Additionally, new vaccine formulations are being developed to induce protective immunity even in the presence of high MDA levels, potentially allowing effective immunization at earlier ages, including as early as four weeks [68].

Environmental biosecurity represents another critical component of CPV control. Subclinically infected animals may shed the virus in feces, contributing to environmental contamination and transmission. CPV is highly resistant to adverse environmental conditions and can remain infectious for extended periods, exceeding 12 months in contaminated environments. Therefore, effective disinfection protocols are essential, including the use of sodium hypochlorite (1:30 dilution) or validated virucidal agents such as peroxides or quaternary ammonium compounds, preceded by thorough mechanical cleaning. Additional measures, such as prompt feces removal, isolation of infected animals, and control of movement of personnel, equipment, and clothing between contaminated and clean areas, are fundamental to prevent indirect transmission [11,50].

Finally, education of pet owners and veterinary personnel plays a crucial role in disease prevention. Avoiding the exposure of unvaccinated puppies to public areas, reinforcing vaccination programs in shelters and breeding facilities, and maintaining accurate health records contribute significantly to reducing disease incidence. Moreover, molecular surveillance of circulating strains, combined with timely case reporting, allows early detection of epidemiological changes and supports the adaptation of local immunization strategies [69].

Overall, the integration of vaccination, environmental disinfection, biosecurity measures, and education constitutes the most effective approach for the prevention and control of canine parvovirus in both urban and rural settings.

Classical inactivated vaccines have demonstrated effectiveness in reducing fetal death, mummification, and resorption, although they may not completely prevent viral replication or shedding following heterologous infection. More recent studies on subunit vaccines based on the VP2 protein have shown protective effects against heterologous PPV1 strains, suggesting that updated vaccine formulations may improve efficacy against emerging variants [24,25]. The vaccines currently available in Ecuador are summarized in Table 3.

Table 3. Vaccination protocols, maternal antibody interference, and biosafety considerations for canine parvovirus prevention.

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Animal Age	Commercial Vaccine	Vaccine Type	Protocol	Maternal Antibody Interference	Biosafety and Contraindications
Puppies (4–6 weeks)	Nobivac Canine-1Pv	Modified live virus (MLV)	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 (MLV)	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 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

Source: Prepared by the authors based on technical datasheets of canine parvovirus vaccines (MSD Animal Health; Intervet Central America S. de R.L., 2022; Pearce et al., 2023).

5. Conclusions

The present systematic review, conducted in accordance with PRISMA guidelines, allowed for a structured and transparent synthesis of the current scientific evidence on the epidemiology, pathogenesis, diagnosis, and treatment of canine parvovirus (CPV). The application of a systematic

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and reproducible methodology facilitated the identification of trends, recent advances, and existing limitations in the literature, highlighting the relevance of early diagnosis, preventive strategies, and appropriate clinical management.

CPV remains one of the most highly contagious and pathogenic viral agents affecting domestic dogs, particularly puppies and immunocompromised animals. Although it is not classified as a notifiable disease by the World Organisation for Animal Health (WOAH), its sanitary and economic impact is considerable due to its rapid transmission and the high mortality rates observed in the absence of adequate preventive measures. These characteristics underscore the continued importance of strengthening control strategies, especially in high-density animal populations.

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, enabling earlier intervention and better clinical outcomes.

Despite these advances, therapeutic management remains largely supportive. Intensive care protocols, including fluid therapy, antiemetics, and antimicrobial treatment, continue to represent the cornerstone of clinical management. The incorporation of complementary approaches, such as immunomodulators, probiotics, and antioxidant therapies, has shown promising results; however, their clinical efficacy requires further validation under controlled conditions.

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.

Overall, effective control of CPV requires an integrated approach that combines vaccination, early diagnosis, appropriate clinical management, and strict biosecurity measures. Future research should focus on improving antiviral therapies, optimizing vaccination strategies in the presence of maternal antibodies, and strengthening epidemiological surveillance to better understand viral evolution and transmission dynamics. These efforts are essential to reduce the impact of CPV infection and to support more effective disease control in canine populations.

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