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Review

Old and Novel Enteric Parvoviruses of Dogs

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Abstract: Parvovirus infections have been well known for around 100 years in domestic carnivores. However, the use of molecular assays and metagenomic approaches for virus discovery and characterization has led to the detection of novel parvovirus species and/or variants in dog. Although some evidence suggests that these emerging canine parvoviruses may act as primary causative agents or as synergistic pathogens in the diseases of domestic carnivores, several aspects regarding epidemiology and virus-host interaction remain to be elucidated.

Keywords: dog; enteric viruses; emerging parvoviruses; protoparvoviruses; bocaparvoviruses; chaphamaparvoviruses

1. Introduction

Gastroenteritis is considered a common clinical problem worldwide in domestic carnivores, chiefly in animals younger than one year of age and living in high-density animal environments, such as catteries and canine shelters. Viruses may be detected in 40-60% of diarrheal fecal samples from domestic dogs and cats, and they represent the main cause of gastroenteritis in these animal species [1–5]. Various viral agents, including astroviruses, adenoviruses, noroviruses, sapoviruses, rotaviruses, vesiviruses, kobuviruses, circoviruses, have been detected in the stools of dogs and/or cats, associated with diarrhea either alone or in mixed infections, occasionally resulting in severe clinical signs. However, the most widely recognized primary causes of viral gastroenteritis in domestic carnivores are parvoviruses and enteric coronaviruses [3–7].

In recent years, using molecular assays and metagenomic approaches for virus discovery and characterization, different research groups have detected novel variants, lineages and species of parvoviruses in domestic carnivores, leading to a change in the classification of the family *Parvoviridae* [7–18]. Parvoviruses (family *Parvoviridae*) are small (23 to 28 nm in diameter), nonenveloped viruses surrounded by an icosahedral capsid. They have a linear, single- stranded DNA genome, ranging from 4.5 to 5.5 kb (kilobases) in length, with complex hairpin structures at the 5′ and 3′ ends that are essential for viability [9]. The genome contains two open reading frames (ORFs). The first ORF encodes two non-structural (NS) proteins, NS1 and NS2, whilst the second ORF encodes two structural proteins, viral protein (VP) 1 and VP2 [8]. NS1 is a pleiotropic nuclear phosphoprotein [19], essential for viral replication and responsible for inducing cell apoptosis [9]. VP2 is the major capsid protein and determines the viral tissue tropism and the host range [20–23]. Parvoviruses are considered members of the same species if their NS1 proteins contain more than 85% amino acid (aa) sequence identity, in accordance with the the International Committee on Taxonomy of Viruses (ICTV) Classification standard. They can be classed as the same taxon if their protein sequences cluster as a strong monophyletic lineage based on their full NS1 protein sequence at the subfamily level and their SF3 helicase domains at the family level.Moreover, NS1 proteins of members of the same genus should share at least 35–40% aa sequence identity, with a coverage of >80% between any two members. Along with the sequence identity-based criteria, common genus affiliation can also be justified based on a similar genome organization, i.e., presence or absence of certain auxiliary-protein-encoding genes, genome length, and/or transcription strategy [10,11,24–26].

The *Parvoviridae* family is classified into three subfamilies, according to current ICTV guidelines: *Parvovirinae* and *Densovirinae*, which infect vertebrates and arthropods, respectively, and the new subfamily *Hamaparvovirinae*, which infects both [10–12,25]. ICTV categorizes the subfamily Parvovirinae into eleven genera: Amdoparvovirus, Artiparvovirus, Aveparvovirus, Bocaparvovirus, Copiparvovirus, Dependoparvovirus, Erythroparvovirus, Loriparvovirus, Protoparvovirus, Sandeparvovirus and Tetraparvovirus (Table 1) [10–12,25,27]. Unknown parvoviruses have been found in a variety of domestic and wild species including dogs, sea otters, seals, bats, camels, horses, and pigs [28–34].

Table 1. Emerging parvoviruses detected in dogs and their current classification.

Subfamily	**Genus**	**Species**	**Common names used in literature**	**Country of first identification**	**Year**	**Detection source**	**Reference**
Parvovirinae	*Protoparvovirus*	*Protoparvovirus Carnivoron 1*	Canine Parvovirus type 2 (CPV-2 and variant a, b, c)	Texas	1977	Stools	[35]
			Feline Panleukopenia Virus (FPV-like)	Germany	1992	Thymus, bone marrow	[36]
		Protoparvovirus Carnivoron 3	Canine Bufavirus (CBuV)	Italy	2018	Stools, respiratory samples	[32]
	Bocaparvirus	*Bocaparvovirus Carnivoron 1*	Canine Parvovirus type 1 (CPV-1), Canine Minute Virus (CnMV/MCV), Canine Bocavirus 1 (CBoV-1)	USA	1967	Stools	[37]
		Bocaparvovirus Carnivoron 2	Canine Bocavirus 2 (CBoV-2)	USA	2012	Stools, respiratory samples	[38,39]
		Bocaparvovirus Carnivoron 7	Canine Bocavirus 3 (CBoV-3)	USA	2013	Liver	[40]
Hamaparvovirinae	*Chaphamaparvovirus*	*Carnivore chaphamaparvovirus 1*	Canine chaphamaparvovirus-1 (CChPV-1)	USA	2017	Stools	[41]

There is little information on the epidemiology and genetic variability of these novel parvoviruses, and it is uncertain if these viruses may operate as enteric infections in dogs or what effect they have on canine health.

The purpose of this study is to offer an update on canine paroviruses that have recently been found in relation with gastrointestinal signs.

2. Protoparvoviruses

The species *Protoparvovirus Carnivoron 1* (Table 1), within the genus *Protoparvovirus*, includes genetically and antigenically related viruses such as canine parvovirus type 2 (CPV-2), feline panleukopenia virus (FPV), and parvoviruses of wild animal, all which cause severe diseases, especially in young animals [7,10–12,27,42,43]. The phylogenetic relationships between CPV-2 isolates from dogs and the viruses isolated from cats (FPV), mink (mink enteritis virus, MEV), raccoon (raccoon parvovirus, RPV), raccoon dogs (raccoon dog parvovirus, RDPV) and blue foxes (blue fox parvovirus, BFPV) showed that all these viruses belong to the species *Protoparvovirus Carnivoron 1* and derive from a single common ancestor [44,45]. Due to a >99% nucleotide (nt) genomic identity with FPV, it was suggested that CPV-2 might be originated from a close relative of FPV through accumulation of punctate mutations [44]. Key amino acid (aa) residues (80R, 93N, 103A, 323N, 564S, and 568G) in VP2 protein distinguish CPV-2 from FPV [42,46–50]. CPV-2 likely arose by acquiring mutations that allowed binding to the canine transferrin receptor (TfR) type-1 [45,51,52]. Several studies have demonstrated that TfR plays a key role in the susceptibility of cells to infection by these viruses [20,53,54]. The evolutionary pathways and inter-species jump of protoparvoviruses in carnivores have stimulated a high scientific interest worldwide.

2.1. CPV-2 variants

CPV emerged as a pathogen of dogs in the late 1970s when it was responsible for a global panzootic in dogs of all ages, which at that time were naïve to the infection [42]. CPV is considered extremely contagious and cause high morbidity with increased incidence in shelters, pet stores and breeding kennels. A rapid clinical course characterizes the disease with death often occurring 2–3 days after the onset of signs in nonprotected animals [55,56]. The infection is generally acquired by the fecal-oral route through contact with feces from infected dogs or contaminated surfaces [5]. The virus mainly infects mitotically active tissues, such as the lymphoid tissues, intestinal epithelium and bone marrow, and the heart in neonatal pups. Following an incubation period of 3–7 days, an enteric form is observed that is characterized by vomiting, hemorrhagic diarrhea, depression, loss of appetite, fever, and dehydration in younger dogs [23,55–57]. The disease can affect dogs at any age, but severe infection is most common in puppies between 6 weeks and 6 months of age [58], with all breeds being susceptible to the disease [58,59].

The original strain was named CPV-2 to distinguish it from the genetically and antigenically unrelated canine parvovirus type 1 (CPV-1, also known as canine minute virus, CnMV or MCV), which has been reclassified as *Bocaparvovirus Carnivoron 1* (genus *Bocaparvovirus*) (Table 1) and is associated with neonatal mortality [42,60]. A few years after its emergence, CPV-2 gave origin to a first antigenic variant, named CPV-2a, which differs from the original type-2 in 5–6 aa positions of the major capsid (VP2) protein. A second antigenic variant, CPV-2b, displayed a further mutation in the VP2 protein (from asparagine to aspartic acid at aa residue 426) [61–63]. In 2000, a third antigenic variant, CPV-2c, was detected, which displayed the aa change asparagine/aspartic acid (N/D) to glutamic acid (E) at residue 426 of the VP2 protein [64]. A single aa change among CPV-2a, -2b and -2c confers different antigenic properties, as evidenced by the different reactivity to specific monoclonal antibodies [65]. However, the variants lack a clear monophyletic segregation due to accumulating other point mutations in different parts of their genome and encoded proteins [66].

The three variants are distributed worldwide, and they are undergoing dynamic changes. The original CPV-2 or CPV-2-like virus was completely replaced in the field by CPV-2 variants after 1980, while it is still present in some vaccine formulations [49,65,67–81]. A recent study revealed that the CPV-2c proportion has been increasing gradually, replacing CPV-2a as the new dominant variant since 2020 [82]. In addition, CPV-2b maintained a low epidemic relevance with a peak of circulation in 2003. However, the dynamic changes in CPV-2 variants may differ geographically. In Asia, CPV-2a has long been the dominant strain but it was replaced by CPV-2c in 2020. In Europe, CPV-2a/2b/2c are co-epidemic, accounting for a similar proportion, and the circulation rate of CPV-2/2-like viruses is relatively low. In 2004, CPV-2a replaced CPV-2c as the dominant variant in South America. In Oceania, there was a gradual change from CPV-2a to a co-endemic status for CPV-2a and CPV-2b. Interestingly, before 2014, CPV-2 variants in North America co-circulated without an apparent trend, but CPV-2c steadily became predominant from 2014 onwards. Likewise, based on the temporal dynamic patterns, CPV-2c after 2017 appeared to replace CPV-2a also in Asia, South America and Africa, but not in Europe and Oceania [82–84].

CPV-2 VP2 is the main component of the viral envelope. Several VP2 amino acids are related to antigenicity and host range [85], and antigenic drift may account for vaccine failure [86,87]. Previously, it was reported that three aa changes in VP2 (F267Y, Y324I and T440A) are of particular concern for vaccine failures [88]. Monitoring of the patterns of variation of these three aa residues has shown that strains with the mutations 267Y and 324I have become predominant. In contrast, the frequency of 440A did not exceeded that of the original residue 440T, which peaked in 2014 and then declined gradually thereafter [82].

Recent analyses of aa mutations in the VP2 gene of CPV-2c Asian strains revealed two frequent mutations, A5G and Q370R [82,89]. A5G was first reported in China in 2015, although its potential functional consequences remain to be determined [90]. Q370R first appeared in CPV-2a strains isolated from giant pandas in Sichuan, China, and subsequently became the dominant mutation of CPV-2c [91]. It is unclear, to date, if the vaccines used for CPV prophylaxis provide complete protection against the 5G and 370R mutant strains [82].

In summary, it is important to monitor the evolution of VP2 to identify readily the emergence of new variants of CPV-2. Due to the rapid evolution of CPV-2 and the continuous emergence of new variants, some immunized dogs still develop the disease [51,66,92,93]. Overall, understanding the molecular and biological characteristics and epidemiological trends of CPV-2 can help to prevent and control parvovirus disease.

Compared with the original CPV-2 strain, the variants exhibit better adaptation to the canine hosts, and have re-gained the feline host range, causing subclinical infection or diseases indistinguishable from feline panleukopenia induced by FPV in cats [24,43].

2.2. *FPV and FPV-like*

Transmission of CPV-2 to cats and FPV to dogs has been an interesting topic for researchers, and many *in-vivo* and *in-vitro* experiments have been conducted to provide more details regarding the host ranges of these viruses [94]. Based on in vitro results, FPV efficiently replicates only in feline cells, whereas CPV-2 can replicate in both canine and feline cells [22,36,57,94–96].

The host ranges of FPV and CPV-2 are quite sophisticated in *vivo*. Specifically, FPV can replicate in feline tissues, including the thymus, spleen, lymph nodes, and intestinal epithelial cells, and high viral loads are shed in feces. In dogs, the virus is found only in the thymus and bone marrow, but it is not detected in the mesenteric lymph nodes or in the enteric tract [36].

Under natural conditions, all CPV-2 variants have been identified, although sporadically, from cats with feline panleukopenia with several independent reports from different countries [43,51,97–101]. On the opposite, reports on FPV in dogs are less frequent. In 1993, an isolate was made from a typical clinical parvoviral infection in a dog, but the virus possessed properties more similar to FPV [100]. More recently, transmission of FPV to dogs with enteric clinical signs has been reported in Pakistan, Thailand, Vietnam, China and in Italy [79,94,95,102–104]. These viruses have been characterized as FPV after either partial or complete sequence analysis of the gene encoding for the VP2. A unique K93N aa mutation involved in host range control was observed in an FPV-like strain in Thailand [103] and the I101T mutation was found in dog-associated FPV strains from Vietnam [94], China [95] and Italy [104]. The pathogenetic role, if any, of FPV in dogs remains unclear. The adoption of cost-effective sequencing strategies in recent years has demonstrated that residual circulation of FPV or FPV-like viruses occurs in dogs in some settings but, overall, FPV seems to infect dogs only occasionally. Apical domain residues in host TfR seem critical for controlling parvovirus binding [20,53,105,106]. Sequencing of the canine TfR from FPV-infected dogs could help to understand the ability of FPV to infect some dogs [50]. Likewise, genome sequencing could be useful to monitor the spread, evolution, and potential host jumping of *Protoparvovirus Carnivoron 1* variants in domestic and wild carnivores [104].

2.3. *Canine bufavirus*

In 2018, a new protoparvovirus strain was detected in a litter of five-month-old puppies involved in an Italian outbreak of canine infectious respiratory disease (CIRD) [32]. This virus was provisionally named canine bufavirus (CBuV) and displayed low aa 19.3%–51.4% identity in the NS1 to members of the species *Protoparvovirus Carnivoron 1*, while the closest relatives to CBuV (47.2%–51.4% aa identity in NS1) were protoparvoviruses identified in human and non-human primates, commonly termed as bufaviruses (BuVs) [107–109]. In agreement with ICTV classification criteria, the canine protoparvovirus could be considered members of a new species, *Protoparvovirus Carnivoron 3* (Table 1), within the genus *Protoparvovirus* [32,110].

In humans and more recently in wild animals (wolves and foxes) BuVs have been identified almost exclusively in the enteric tract [111,112]. However, investigations in dogs [32,113], monkeys [109], shrews [114], and sea otters [33] suggest possible extraintestinal and/or systemic infections of BuVs. In 2019, in China, canine BuVs were detected in sera from dogs with signs of CIRD [113].

Presently, the circulation of CBuV has been reported in Italy [32,115], China [113,116], and India [117], but its genetic and pathobiological features are still unclear [118].

In Italy, the detection rate of this virus was 7.7% (16/207), with a higher frequency (8.8%) in diarrheic dogs, but CBuV infection was non statistically correlated with gastrointestinal disease [115]. In China, CBuV was found in Shanghai, Guangxi province and Henan province, and the positive rates were 42.15% (51/121), 2.5% (5/200), and 1.74% (2/115), respectively [113,116,119]. Another study in the Chinese province of Anhui revealed a CBuV prevalence of 2.5% (3/120) [118]. In a more recent investigation, CBuV was detected with a proportion of 4.3% (8/186) from both diarrheic puppies (< 1 year old) and adult dogs (> 1 year old) [117]. The nearly complete genome sequence (strain 407/PVNRTVU/2020) obtained in that study shared a 93.4-98.8% nucleotide (nt) identity to other available CBuV sequences. Furthermore, the strain 407/PVNRTVU/2020 was most closely related to other Chinese CBuV strains, forming a distinct lineage [117].

The primary clinical sign caused by a member of *Protoparvovirus* in carnivores is diarrhea [5]. Some studies have shown a positive correlation between CBuV infection and diarrhea, and CBuV DNA was also detected in the serum samples of dogs with gastroenteritis [116]. Genome sequencing of CBuV strains has demonstrated genetic heterogeneity and suggested that recombination may be important factors in the virus evolution [115].

In most cases, CBuV genome has been co-detected in dogs with other viral pathogens including CPV-2 [115,118], canine coronavirus (CCoV), canine kobuvirus (CKoV) [115], canine adenovirus type 1 (CAdV-1) and type 2 (CAdV-2) [117], suggesting that CBuV could be considered as a common component of the canine fecal virome. Although the patho-biological role of CBuV in dogs remains still unclear, a possible role of this virus in the etiology of canine enteritis can be hypothesized. Synergistic effects of co-infection with other enteric viruses could lead to more serious clinical signs.

As observed for CPV-2 and FPV, there is evidence that CBuV can also infect the feline host [110,120].

The ability of viruses in the genus *Protoparvirus* to determine severe clinical signs in dogs, as well as their multi-host nature, must be considered in the implementation of individual and collective prophylaxis plans, in order to limit the spread of these viruses not only between individuals of the same species, but also between individuals of different species sharing the same environment.

3. Bocaparvoviruses

The genus *Bocaparvovirus* (BoVS), subfamily *Parvovirinae*, includes virus that cause diseases in humans and in various animals, including porcine BoVs [121,122], bovine parvovirus [123], California sea lion BoV [124], bat BoV [125], rabbit BoV [126], rodent BoV [127], pine marten BoV [128], mink BoV [129], canine BoVs [37-39], feline BoVs [13,14,38,130], gorilla BoV [131,132] and human BoVs (HBoVs) [133,134], suggesting a potentially wide host range of these viruses.

Viruses in this genus are monophyletic and share >30% NS1 aa identity. However, <30% identity values are allowed between certain viruses to accommodate disparities between current and previous analytical methods. BoVs are unique among parvoviruses since they possess an additional ORF (ORF3), located between the non-structural (ORF1) and structural (ORF2) coding regions of their genome (5.5 kb ssDNA). ORF3 encodes the NP1, a highly phosphorylated protein that differs from proteins seen in other parvoviruses and is involved in in RNA processing. NP1 regulates VP-encoding RNAs splicing and read-through of the proximal polyadenylation [27,123,135-139]. The great majority of these viruses have been identified using metaviromic strategies and they have not been adapted to culture systems [10,11,27].

The genus was originally named according to its initial two members, bovine parvovirus (BPV) and CnMV (formerly known as CPV-1) [140]. Based on ICTV classification criteria, BoVs are classified into thirty-two species, of which at least six species have been detected in domestic carnivores and classified as *Bocaparvovirus Carnivoron* (CBoV) 1 to 5, while a sixth species (CBoV-6) has been found in minks (Table 1) [11,12,27,129]. Currently, BoVs identified in domestic dogs have been classified within the species CBoVs-1, 2 and 3 [13,14,38] and they have been associated with different clinical manifestations.

As previously mentioned, CnMV, is an autonomous parvovirus of dogs that is genetically and antigenically unrelated to CPV-2 [42]. CnMV is currently classified as species *Bocaparvovirus Carnivoron-1* [141]. CnMV was first isolated in 1967 from the feces of a clinically healthy military dog [35,142] and it seems common worldwide in domestic dogs of different ages. Its clinical significance and virulence are uncertain. It determines mild to inapparent infections in puppies, and it is weakly pathogenic in adults [140]. Pneumonitis, myocarditis, lymphadenitis, and hepatitis have been reported in dogs with CnMV infection [60,143,144]. CnMV may cross the placenta, causing early fetal death, birth defects and neonatal mortality [60,143,145].

Bocaparvovirus Carnivoron -2 (CBoV-2) was identified in 2012 in association with canine respiratory disease in a metagenomic study [39]. The NS, NP, and VP genes of CBoV-2 share less than 63%, 62%, and 64% aa identity with CnMV (CBoV-1), respectively [39] CBoV-2 infection also has been associated with massive enteritis in a litter of dogs with atrophied and fused villi, severe crypt regeneration, and severe bone marrow and lymphoid atrophy [28]. In addition, interstitial pneumonia has been reported to be a feature of CBoV-2 infection [146].

Variants of CBoV-2 have been also detected in fecal, nasal, urine and blood samples collected from dogs in Hong Kong [38], thus suggesting possible extraintestinal and/or systemic infections. A more recent investigation has identified a novel strain of CBoV-2 in a litter of puppies that died in Thailand from acute dyspnea and hemoptysis, using a metagenomic approach [147]. This strain was most closely related to previously identified CBoV-2 strains from South Korea [146] and Hong Kong [28].

Using deep sequencing, a third type of canine bocavirus, CBoV-3 (currently proposed as *Bocaparvovirus Carnivoron*-7) (Table 1) [27] was identified in 2013 in the liver of a dog with hemorrhagic gastroenteritis, necrotizing vasculitis, granulomatous lymphadenitis and anuric renal failure, and coinfected with a canine circovirus [40]. CBoV-3 has been classified as an additional novel species since in the NS1, NP1 and VP1 regions it shares only 49-51%, 52-57% and 56-57% aa identity, respectively, with CBoV-1 and CBoV-2 [40]. On phylogenetic analysis of the complete VP1, CBoV-3 is phylogenetically distinct from other canine bocaparvoviruses [40]. Episomal forms were detected by PCR indicating that replication may occur in hepatocytes or other liver cell types. Since circovirus infection can lead to lymphocyte depletion and immunosuppression in the host [148], the pathogenic role of CBoV-3 in the original study could not be assessed clearly [40].

Including CBoVs in the diagnostic algorithm of canine enteritis using specific molecular tools could help to understand better the enteropathogenic role of these viruses and to assess whether some CBoV species/strains possess peculiar phenotype changes.

Finally, it is noteworthy to mention that several bocaparvoviruses have been detected in cats and other wildlife carnivores [13,14,24,38,129,130,149].

4. Chaphamaparvoviruses

The genus *Chaphamaparvovirus* (ChPV), belonging to the subfamily *Hamaparvovirinae*, recently introduced into the family *Parvoviridae* (Table 1). This genus includes viruses genetically more related to invertebrate-infecting parvoviruses than to other members of the subfamily *Parvovirinae*. Detection and characterization of viruses related to this proposed taxon might eventually result in splitting the genus into more genera. Currently, however, their clustering as a single genus is the only common node characterized by significant topology support by both Bayesian and ML-based inference [11,12,25,27].

ChPV was first identified in an oropharyngeal swab sample collected from a fruit bat (*Eidolon helvum*) in Ghana (Africa) [150], ChPV-like viruses have been described in several additional animal species [25], including dogs and cats [15,16,18,41,151]. The first description of ChPV in domestic carnivore's dates back to 2017, USA, in a metaviromic study carried out on the feces of two dogs with hemorrhagic diarrhea of unknown etiology [41]. In subsequent studies, viruses genetically related to the American strains of canine ChPV (provisionally termed as cachavirus) have been detected in feces of dogs and cats in China and Italy [15,18,151]. Based on ICTV classification criteria, all strains of canine origin segregate into the new species *Carnivore chaphamaparvovirus 1* (CaChPV-1) [11]

A possible association of CaChPV-1 with enteric disease in dogs was first hypothesized in a 2019 study [41]. However, this possible association has not been demonstrated in other studies [18,151]. In a recent investigation in Thailand, a correlation was observed with the presence of viral DNA in samples of young dogs with mild enteritis but not in archival samples of deceased animals with diarrhea [152].

Although the presence of CaChPV-1 DNA in cases of canine enteric disease has been documented repeatedly [18,41,151,152], information about CaChPV-1 tropism and viral distribution in the intestine or in other organs is limited.

Overall, the potential clinical impact of ChPVs on canine health and its possible role as primary enteric pathogen remain to be clarified [18,41,151,152]. Indeed, ChPVs DNA was often detected in dogs in co-infection with other viral pathogens such as CBuV, CAdV [153], CPV-2, CCoV [18,151], and canine distemper virus (CDV) [18].

5. Conclusions

In the last twenty years, exploration of canine virome using sequence-independent protocols and consensus (pan-viral) PCR strategies, has identified several novel parvovirus species and variants in dogs with enteric and/or respiratory disease. Whether these novel canine parvoviruses may act as primary causative pathogens or synergistic agents remain to be elucidated.

A trend in the diagnostics of human infectious disease is the adoption of syndromic testing panels covering a wide spectrum of common and uncommon pathogens based on advanced microbiology technologies such as multiplex molecular assays (i.e., syndromic diagnostic tests). Including these novel canine parvoviruses in the diagnostic algorithms of canine diseases, combined with larger epidemiological studies with a multidisciplinary approach and/or with experimental infections, could help to clarify their epidemiology and their eventual association, if any, with canine diseases. Expanding our knowledge on the enteric virome of animals at the animal-human interface is, by the way, necessary to assess more properly eventual zoonotic risks and fulfill the recommendations of the One Health paradigm.

Interestingly, multi-species circulation of some of these novel parvoviruses could represent a challenge when devising measures of prophylaxis in animals of different species living/housed in the same household, shelters and clinics.

Use of vaccines, when available, could prevent the spread of many of these emerging parvoviruses, although this strategy should be complemented with detailed disinfection plans and physical separation of animals, chiefly in the case of suspected parvovirus circulation in multi-animal and multi-species environments.

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