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Isolation and molecular characterization of canine and feline parvovirus strains - an updated review

Abstract

Canine parvovirus is the most important and significant contagious viral cause of enteritis in pups, adult dogs and wild carnivores characterized by acute haemorrhagic enteritis, myocarditis and leucopenia. These viruses target the rapidly dividing cells. Phylogenetic analysis showed that all CPV isolates descended from a single ancestor which emerged during mid-1970s and were which was closely related to the long-known feline panleukopenia virus (FPV) that infected cats, mink, and raccoons but not dogs or cultured dog cells. FPV and CPV differ only by 0.5% in their DNA sequences. Inspite of their similarity, CPV-2 was not able to replicate in cat cells. FPV is not the only parvovirus species which infects cats, in addition to MEV, the new variants of canine parvovirus, CPV-2a, 2b and 2c have also penetrated the feline host-range, and they are able to infect and replicate in cats, causing diseases indistinguishable from feline panleukopenia. There are five to six amino acid (87, 101, 300, 305, 426 & 555) differences between CPV-2, CPV-2a and its variants. Amplification of full length VP2 gene followed by sequencing of the PCR products can give ample information about the antigenic and genetic differences between the original prototype CPV-2 and its variants.

Keywords: canine parvovirus, canine parvovirus variants, vp2 gene, sequencing

Volume 3 Issue 5 - 2016

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Received: March 25, 2016 | **Published:** July 22, 2016

Introduction

Canine parvovirus is the most important and significant contagious viral cause of enteritis in pups, adult dogs and wild carnivores characterized by acute haemorrhagic enteritis, myocarditis and leucopenia.[1,2] Now CPV infection is considered to be most threatening especially to the puppies between the time of weaning and six months of age. These viruses target the rapidly dividing cells.

Molecular characteristic of canine parvovirus type 2

Canine parvovirus belongs to the genus *Protoparvovirus*, family *Parvoviridae*. The virion is non-enveloped having icosahedral symmetry of 26nm diameter. It has a single stranded negative sense genome of 5.2kb in length. This single-stranded virus has a DNA genome and use cellular replication machinery, their rate of nucleotide substitution is closer to that of RNA viruses than to that of double-stranded DNA viruses**.** Phylogenetic analysis showed that all CPV isolates descended from a single ancestor which emerged during mid-1970s and were which was closely related to the long-known feline panleukopenia virus (FPV) that infected cats, mink, and raccoons but not dogs or cultured dog cells.[3] FPV and CPV differ only by 0.5% in their DNA sequences. Inspite of their similarity, CPV-2 was not able to replicate in cat cells. FPV is not the only parvovirus species which infects cats, in addition to MEV, the new variants of canine parvovirus, CPV-2a, 2b and 2c have also penetrated the feline host-range, and they are able to infect and replicate in cats, causing diseases indistinguishable from feline panleukopenia.[4] CPV comprises of two open reading frames, one codes for structural proteins VP1 and VP2 while the other codes for non-structural proteins NS_1 and NS_2. VP_3 is a capsid protein which is formed by proteolytic cleavage of VP2 capsid protein. The VP2 is the major capsid protein and plays an important role in determining the antigenicity and the host range of CPV. In 1978, CPV-2 emerged as a cause of a new disease in dogs throughout the world. The first documentation of the virus as CPV-2 was done in Canada.[1] Canine parvovirus (CPV) and feline panleukopenia virus (FPV) capsids bind to the transferrin receptors (TfRs) of their hosts and use these receptors to infect cells. The binding is partially host specific, as FPV binds only to the feline TfR, while CPV binds to both the canine and feline TfRs.[5]

Variants of canine parvovirus

After the emergence of canine parvovirus in late 1970s[1,6] due to rapid evolution, new antigenic types were evolved as CPV-2a, CPV-2b and CPV-2c (CPV2a with Asn426Glu) which have completely replaced the original CPV-2.[7,8] Currently the original CPV-2 was not found in dog population but present only in vaccine formulations[8,9] (Figure 1).

The virus has undergone mutations from the original CPV-2 to CPV-2a and CPV-2b. A third mutant, CPV-2c has been discovered in Italy.[10] The variants CPV-2a and CPV-2b differ from the original type CPV-2 by a few amino acid changes in the VP2 protein. The latter accounts for an extended host range *in-vivo* the reported rate of substitution refers only to the VP2 gene and for an increased affinity to canine transferrin receptors.[3] The difference between CPV types 2a and 2b is the presence of two single nucleotide polymorphisms (SNPs) in the capsid protein gene sequence, which determines amino acid changes in the major antigenic sites of the viral capsid.[11]

Currently circulating type 2a strains possess Val instead of Ile at 555 positions exactly as CPV-2b; thus, the difference is now restricted to a single residue at position 426. SNP alanine/cysteine (A/C) encountered at position 4449 determines the presence of the amino acid isoleucine (Ile) for CPV type 2a or valine (Val) for CPV type 2b at residue 555.[8] Recently, an antigenic variant of CPV-2 has been reported in Italy, with the amino acid substitution aspartic acid to glutamic acid (Asp→Glu) at residue 426.[10] This is due to the

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nucleotide change threonine (T) 4064, and alanine with respect to type 2b. This mutation affects the major antigenic region located over the 3-fold spike of the CPV-2 capsid. Monoclonal antibodies (Mabs) capable of distinguishing between CPV type 2b and CPV-2c has been developed (Table 1).[12]

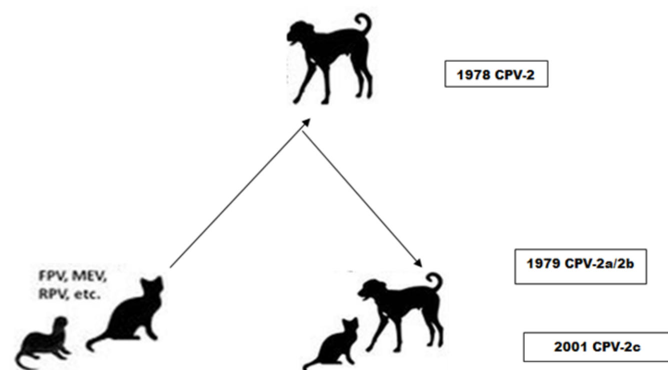

Figure 1 Evolution of canine parvovirus.

Table 1 Amino acid variations in the VP2 protein differentiating between FPV and CPV-2

Amino acid	80	93	103	323	375	564	568
FPV	Lys	Lys	Val	Asp	Asp	Asn	Ala
CPV-2	Arg	Asn	Ala	Asn	Asn	Ser	Gly

Amino acid variations in the vp2 protein differentiating between FPV and CPV-2

Residue 426 had undergone two mutations since the emergence of CPV, first from asparagine to aspartate and more recently to glutamate, amino acid changes which constituted an important genetic marker for the new variants of CPV and the only coding change capable of distinguishing the CPV-2a strains from CPV-2b/2c strains.[13] A novel CPV mutant, CPV-2c is widely distributed and co-existing with other CPV types in Europe, North and South America countries. There was a single report of occurrence of CPV-2c in North India (Table 2).[14]

Table 2 Amino acid variations in the VP2 protein differentiating the strains of CPV-2

Amino acid	87	297	300	305	375	426	555
CPV-2	Met	Ser	Ala	Asp	Asn	Asn	Val
CPV-2a	Leu	Ser	Gly	Tyr	Asp	Asn	Ile
CPV-2b	Leu	Ser	Gly	Tyr	Asp	Asp	Val
New CPV-2a	Leu	Ala	Gly	Tyr	Asp	Asn	Val
New CPV-2b	Leu	Ala	Gly	Tyr	Asp	Asp	Val
CPV-2c	Leu	Ala	Gly	Tyr	Asp	Glu	Val

Epidemiology

Parvoviruses have been characterised by a rapidly changing epidemiology in dogs and cats. Feline panleukopaenia has been recognised as a disease of cats for over 100years, and the virus (FPLV) was first isolated and associated with the disease in 1965.[15] Twenty-three years later, in 1978, a variant of an FPLV-like virus called canine parvovirus CPV-2 emerged in the canine population, and appeared to spread rapidly worldwide with profound clinical consequences.[16] The precise origin of this virus remains the source of some speculation but possibilities include the evolution of the virus in a wildlife intermediate such as a fox.[17]

In 1979 and 1980, an antigenic variant of CPV-2 was identified in several different countries using monoclonal antibodies and the variant was termed canine parvovirus type 2a.[18] By employing monoclonal antibodies (mAbs) and restriction enzymes (RE) for antigenic and genomic analysis of this virus, ample evidences were found that original CPV-2 was replaced in the field by viruses designated as CPV-2a[7] and CPV-2b[8] between 1979 and 1981. In the middle of 1980s, the virus underwent a further antigenic change, and the new variant was referred to as canine parvovirus type 2b, CPV-2b.[19]

The isolation rates proved that CPV-2a and CPV-2b types increased yearly from 1979 to 1982 and in Japan all 50 isolates and in the United States 48 of 51 isolates collected after 1983 were CPV-2a and CPV-2b types.[20] Another new antigenic variant was detected in CPV-2 strains isolated from domestic dogs in Italy during 2001[10] and was designated as CPV-2c. CPV-2c was broadly distributed in Italy, where it was co circulating with types 2a and 2b.[21]

Presently the variants of CPV(CPV-2a and CPV-2b) were the predominant strains distributed worldwide and the original prototype CPV-2, no longer circulated in dog population.[22–26]

CPV-2c had also been reported in Vietnam,[12] Spain,[23] Germany,[27] United Kingdom,[28] South America,[24] United States of America,[29] Central Portugal,[30] Argentina,[31] Greece,[32] North India,[14] Brazil[33] and Uruguay.[34,35] Recent reports revealed the predominant CPV strain in India as new CPV-2a (Ser 297Ala) with one new CPV-2b (Ser 297Ala) and another new CPV-2a (Ser 297Gly) variant strain.[36]

usceptible hosts/ reservoirs of CPV-2

The main source of infection is faeces from the infected dogs. More than 10^9 virus particles per gram of faeces are shed during the acute phase in the enteric form. The faeces are the suitable clinical material for the detection of virus in the enteric form of disease.[37] The CPV spreads by faeco-oral route[11,38,39] and is shed for 8-12days post infection via faeces.[9,38]

CPV-2 can affect dogs of any age, however, severe clinical manifestation is seen in puppies between 6weeks and 4months old. All breeds of dogs are susceptible.[3] The crossbreds are less susceptible in comparison to pure breeds like Rottweiler, Doberman Pincher, English Springer, Spaniels and German Shepherd, the exception to this being Toy Poodles and Cocker Spaniels.[40] Several species of wild carnivores, red foxes, wolves, raccoons and coyotes are susceptible to canine parvovirus infection.[17] Canine parvovirus infection has also been reported in the bat eared fox, honey badger, cheetah, African wild cat and Siberian tiger.[41]

CPV infection in cats

In the late 1970s, CPVs evolved from FPV after crossing species barriers by acquiring five or six amino acid changes in the capsid protein gene.[42] Within 1 year, the first CPV (CPV-2) changed to the current subtypes, CPV-2a and CPV-2a derived strains. Whereas CPV-2 was not able to infect cats, the subtypes can cause clinical signs that cannot be distinguished from those caused by FPV.[43–45]

CPV had been isolated from a variety of domestic cats, as well

Citation: Brindhalakshmi B, Mukhopadhyay HK, Antony PX, et al. Isolation and molecular characterization of canine and feline parvovirus strains - an updated review. *J Dairy Vet Anim Res.* 2016;3(5):164–169. DOI: 10.15406/jdvar.2016.03.00093

as other more-or-less related carnivores, including raccoons, Asiatic raccoon dogs, cheetahs, mountain lions, leopard cats and a red panda.[41,46–49] CPV-2a and CPV-2b were likely to act as newly emerged viruses in cats, some cat-specific positive selection(s), such as relative *in vivo* fitness and immune surveillance, could have operated as a driving force of CPV evolution.[50] CPV had presumably started a new process of readapting in feline hosts and confirmed the importance of viral host switching as a mechanism for the emergence of new viruses.[4] They also reported two CPV infections in 24 cat samples in Italy, one with CPV-2c and another with mixed infection of both CPV-2a and FPV.

This mixed infection pointed out the predominant epidemiological role of cats as

a) Carriers of carnivore parvovirus since it was susceptible to both the viruses and as

b) Sources of new variants of parvoviruses.[50]

Isolation of CPV/FPV in cell cultures: The host ranges of FPV and CPV variants have been extensively studied *in vitro* by several authors. In general, CPV-type viruses replicate efficiently in feline and canine cell lines, while most FPLV and FPLV-like viruses can replicate efficiently only in feline cells.[51,52] The isolation of CPV from clinical samples by using various cell cultures has been used for the diagnosis of canine parvovirus.

Isolation of FPV in CRFK cell line: MDCK and CRFK was used for the isolation and cultivation of canine parvovirus.[45] Antigenic and genetic properties of isolates from pups were elucidated. Three parvoviruses that were recently isolated from a domestic cat and 2 cheetahs in cell culture, detected by means of the polymerase chain reaction were shown to be typical feline parvoviruses. Crandell feline cell cultures inoculated with material derived from the cat faeces showed mild cellular degeneration in comparison with the cell controls after 5days.

Isolation of CPV in CRFK cell line: CRFK was used for the isolation and cultivation of canine parvovirus from the faeces of infected dogs and adapted the isolate to grow in CRFK cell lines. Rounding and degenerative changes were the cytopathic changes observed at third passage level 72hours post infection. The harvesting was done by three alternate cycles of freezing and thawing. Presence of virus in the harvested cell culture supernatant was detected by PCR.[53–56] These studies shows that Canine parvovirus variants obtained from dogs can be isolated from CRFK cell line which is of feline origin.

Isolation of CPV in A-72 cell line: The A-72 cell line was widely used for cultivation of CPV in cell culture and the first CPV-2c was isolated in A-72 cell line in Uruguay.[10,34,57–59] A-72 cell lines was used for isolation of CPV from samples found positive by PCR, CPE was observed in A-72 cell line from second passage onwards showing greater sensitivity of A-72 cell line over CRFK cell line.[60] Rounding, increased granularity and detached cells were the cytopathic changes observed from the second passage onwards.[61]The studies show that CPV could be efficiently isolated from A-72 cell line which of canine origin when compared to CRFK cell line (feline origin). The isolation of CPV in cell culture cannot be applied to a spoiled sample in which viruses have been inactivated. The sensitivity of different cell lines may also be different.

Molecular characterisation of CPV/FPV by vp2 gene sequencing

Amplification and subsequent sequencing of VP2 gene was useful in characterization of CPV by identifying the types of CPV currently present in the field.[24–26,56]

22 CPVs isolates from 27 faecal samples collected from various states of India.[62] The isolates were characterized by nucleotide sequencing of the VP2 gene and found that 18 were CPV-2a and 4 were CPV-2b. The genetic analysis of canine parvovirus was done in a total of 13 CPV vaccines, which had been licensed in Korea since late 1980s, and a field isolate of CPV from a dog with CPV infection. The partial VP2 gene of CPV was amplified and sequenced from 13 vaccine strains and one field isolate. The results showed that of the 13 vaccine strains, 10 strains belong to the CPV-2, 2 strains to CPV-2b, the remaining one isolate to CPV-2a type, respectively. Several mutations of amino acids were detected at residues of the critical region of the commercial vaccine strains.[63] This suggested that CPV-2 contained in most licensed vaccines had been replaced by antigenic variants designated CPV-2a or CPV-2b/c in the worldwide dog population.

PCR amplification with sequence-specific primers was used to detect canine parvovirus DNA in 38 rectal swabs from Argentine dogs with canine parvoviral disease symptoms. Twenty-seven out of 38 samples analyzed were CPV positive. The classical CPV-2 was not detected in any of the samples, but nine samples were identified as CPV-2a variant and 18 samples as CPV-2b variant. Further sequence analysis revealed a mutation at amino acid 426 of the VP2 gene (Asp426Glu), characteristic of the CPV-2c variant, in 14 out of 18 of the samples identified initially by PCR as CPV-2b.

The primers CPV2655-F and CPV3511-R; CPV3381-F and CPV4116-R[64] and 555(F)/555(R)[10] were used for both amplification and sequencing of full length VP2 gene in 27 samples. The findings revealed that 100 CPVs belong to type 2b strains and 6 as type 2a strain which shows CPV-2b predominates in South Africa whereas all the 30 Nigerian samples were CPV-2a strains.[65]

Molecular characterisation of CPV/FPV by partial VP2 gene sequencing

Canine parvovirus strains were detected and characterized in Brazil by screening 144 dog faecal samples by PCR for CPV-2, and 29.2%(42/144) of them were positive. Sequencing of the 583bp fragment of the VP2 gene from the 42 positive strains revealed 78.6%(33/42) as type 2c, 19%(8/42) as type 2b and 2.4% (1/42) as type 2a.[66]

The predominant CPV strain in India as new CPV- 2a (Ser 297Ala) with one new CPV-2b (Ser 297Ala) and another new CPV -2a (Ser 297Gly) variant strain by partially sequencing the VP2 gene of CPV which was reported by Mukhopadhayay et al.[36]

The full-length VP2 coding sequences from 11 CPV-2c strains, 11 CPV-2a strains and 2 CPV-2b strains revealed remarkable genetic diversity that included the circulation of all three variants (2a, 2b and 2c) and the existence of divergent evolutionary groups. CPV-2c was the most prevalent variant (54.47%), confirming the spread of this variant in America.[67]

By sequence and phylogenetic analysis of VP2 gene reveals that out of 28 Taiwanese CPV 2 isolates, 15 were identified as new CPV 2a, and 13 were identified as new CPV 2b.[68] Many researchers carried out molecular characterization by sequencing the full length VP2 gene of CPV/FPV which is more reliable. The molecular characterization also reveals that the predominantly circulating strain which is causing infection in dog is CPV-2a then followed by CPV-2b. Recently CPV-

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2c strain has also been isolated from India. The full length sequencing of VP2 gene gives ample information about the biological properties of the viruses which helps in characterization.

Conclusion

Canine parvovirus (CPV) is one of the most important infectious agents of domestic dogs and wild carnivores worldwide which causes severe gastroenteritis with a high case fatality rate, particularly in puppies less than 3 months of age. Early and differential diagnosis of CPV is very essential for the effective clinical management of the affected dogs. Surveillance and genotyping of CPV from various geographical locations help in detecting the prevalent CPV types/strains. The CPV is now known to infect both wild and domesticated feline species. Since the cats are susceptible to both CPV and FPV viruses, superinfection and co-infection with multiple parvovirus strains occur facilitating recombination and high genetic heterogenicity leading to evolution of new virulent strains/virus. The cats also act as carrier and a source of infection to dogs. Considering the contradictory reports about vaccine efficacy, there is also an urgent need to consider developing vaccines incorporating the strains/types currently circulating in a particular geographical region. Moreover isolation of CPV in the susceptible cell line is very much essential for antigenic, molecular and phylogenetic analysis.

The main disadvantage of viral isolation in cell line is low sensitivity. It has been demonstrated in natural and experimental infections that CPV-2 is detectable by viral isolation only for a few days post infection. The strain currently circulating in the dog population causing disease is New CPV-2a then followed by New CPV-2b. Recently CPV-2c strain was isolated from dog in India. Amplification of full length VP2 gene followed by sequencing of the PCR products gives ample information about the antigenic and genetic differences between the original prototype CPV-2 and its variants which helps in molecular characterization.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

References

1. Appel MJ, Scott FW, Carmichael LE. Isolation and immunization studies of a canine parvo-like virus from dogs with haemorrhagic enteritis. *Vet Rec.* 1979;105(8):156–159.
2. Decaro N, Elia G, Campolo M, et al. New approaches for the molecular characterization of canine parvovirus type 2 strains. *J Vet Med B Infect Dis Vet Public Health.* 2005;52(7–8):316–319.
3. Hueffer K, Parker JS, Weichert WS, et al. The natural host range shift and subsequent evolution of canine parvovirus resulted from virus-specific binding to the canine transferrin receptor. *J Virol.* 2003;77(3):1718–1726.
4. Battilani M, Balboni A, Ustulin M, et al. Genetic complexity and multiple infections with more Parvovirus species in naturally infected cats. *Vet Res.* 2011;42:43.
5. Palermo LM, Hueffer K, Parrish CR. Residues in the apical domain of the feline and canine transferrin receptors control host-specific binding and cell infection of canine and feline parvoviruses. *J Virol.* 2003;77(16):8915–8923.
6. Kelly WR. An enteric disease of dogs resembling feline panleukopenia. *Aust Vet J.* 1978;54(12):593.
7. Parrish CR, O'Connell PH, Evermann JF, et al. Natural variation of canine parvovirus. *Science.* 1985;230(4729):1046–1048.
8. Parrish CR, Aquadro CF, Strassheim ML, et al. Rapid antigenic-type replacement and DNA sequence evolution of canine parvovirus. *J Virol.* 1991;65(12):6544–6552.
9. Decaro N, Elia G, Desario C, et al. A minor groove binder probe real-time PCR assay for discrimination between type 2-based vaccines and field strains of canine parvovirus. *J Virol Methods*. 2006;136(1–2):65–70.
10. Buonavoglia C, Martella V, Pratelli A, et al. Evidence for evolution of canine parvovirus type 2 in Italy. *J Gen Virol.* 2001;82(Pt 12):3021–3025.
11. Decaro N, Desario C, Campolo M, et al. Clinical and virological findings in pups naturally infected by canine parvovirus type 2 Glu-426 mutants. *J Vet Diagn Invest*. 2005;17(2):133–138.
12. Nakamura M, Tohya Y, Miyazawa T, et al. A novel antigenic variant of canine parvovirus from a Vietnamese dog. *Arch Virol.* 2004;149(11):2261–2269.
13. Martella V, Decaro N, Buonavoglia C. Evolution of CPV-2 and implication for antigenic/genetic characterization. *Virus Genes*. 2006;33(1):11–13.
14. Nandi S, Chidri S, Kumar M, et al. Occurrence of canine parvovirus type 2c in the dogs with haemorrhagic enteritis in India. *Res Vet Sci.* 2010;88(1):169–171.
15. Johnson RH, Margolis G, Kilham L. Identity of feline ataxia virus with feline panleukopenia virus. *Nature.* 1967;214(5084):175–177.
16. Appel MJG, Cooper BJ, Greisen H, et al. Status report: canine viral enteritis. *J Am Vet Med Assoc*. 1978;173:1516–1518
17. Truyen U, Müller T, Heidrich R, et al. Survey on viral pathogens in wild red foxes (*Vulpes Vulpes*) in Germany with emphasis on parvoviruses and analysis of a DNA sequence from a red fox parvovirus. *Epidemiol Infect.* 1998;121(2):433–440.
18. Carman S, Povey C. Successful experimental challenge of dogs with canine parvovirus-2. *Can J Comp Med.* 1984;46(1):33–38.
19. de Ybañez RR, Vela C, Cortés E, et al. Identification of types of canine parvovirus circulating in Spain. *Vet Rec*. 1995;136(7):174–175.
20. Senda, M, Parrish CR, Harasawa R, et al. Detection by PCR of wild type canine parvovirus which contaminates dog vaccines. *J Clin Microbiol.* 1995;18:110–113.
21. Martella V, Cavalli A, Patella A, et al. A canine parvovirus mutant is spreading in Italy. *J Clin Microbiol.* 2004;42(3):1333–1336.
22. Decaro N, Elia G, Campolo M, et al. New approaches for the molecular characterization of canine parvovirus type 2 strains. *J Vet Med B Infec Dis Vet Public Health.* 2005;52(7–8):316–319.
23. Decaro N, Elia G, Martella V, et al. Characterization of the canine parvovirus type 2 variants using minor groove binder probe technology. *J Virol Methods.* 2006;133(1):92–99.
24. Perez R, Francia L, Romero V, et al. First detection of canine parvovirus type 2c in South America. *Vet Microbiol*. 2007;124(1–2):147–152.
25. Kang BK, Song DS, Lee CS, et al. Prevalence and genetic characterization of canine parvoviruses in Korea. *Virus Genes.* 2008;36(1):127–133.
26. Ohshima T, Hisaka M, Kawakami K, et al. Chronological analysis of canine parvovirus type 2 isolates in Japan. *J Vet Med Sci.* 2008;70(8):769–775.

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27. Shackelton LA, Parrish CR, Truyen U, et al. High rate of viral evolution associated with the emergence of carnivore parvovirus. *Proc Natl Acad Sci USA*. 2005;102(2):379–384.

28. Decaro N, Desario C, Addie DD, et al. Molecular Epidemiology of Canine Parvovirus, Europe. *Emerg Infect Dis*. 2007;13(8):1222–1224.

29. Kapil S, Cooper E, Lamn C, et al. Canine Parvovirus Types 2c and 2b Circulating in North American dogs in 2006 and 2007. *J Clin Microbiol*. 2007;45(12):4044–4047.

30. Vieira JM, Silva E, Oliveira J, Vieira LA, Decaro N, et al. (2008) Canine parvovirus 2c infection in central Portugal. *J Vet Diagn Invest*. 2008;20(4):488–491.

31. Calderon MG, Mattion N, Bucafusco D, et al. Molecular characterization of canine parvovirus strains in Argentina. Detection of the pathogenic variant CPV-2c in vaccinated dogs. *J Virol Methods*. 2009;159(2):141–145.

32. Ntafis V, Xylouri E, Kalli I, et al. Characterization of canine parvovirus 2 variants circulating in Greece. *J Vet Diagn Invest*. 2010;22(5):737–740.

33. Castro TX, Costa E M, Leite JP, et al. Partial VP2 sequencing of canine parvovirus (CPV) strains circulating in the state of Rio de Janerio, Brazil: detection of the new variant CPV-2c. *Braz J Microbiol*. 2010;41(4):1093–1098.

34. Pintos AB, Larrama CBN, Baratta EER, et al. Isolation and characterisation of canine parvovirus type 2c circulating in Uruguay. *Ciencia Rural*. 2011:41(8).

35. Perez R, Bianchi P, Calleros L, et al. Recent spreading of a divergent canine parvovirus type 2a (CPV-2a) strain in a CPV-2c homogenous population. *Vet Microbiol*. 2012;155(2–4):214–219.

36. Mukhopadhayay HK, Matta SL, Amsaveni S, et al. Phylogenetic analysis of canine parvovirus partial VP2 gene in India. *Virus Genes*. 2013;48(1):89–95.

37. Carmichael LE, Binn LN. New enteric viruses in the dog. *Adv Vet Sci Comp Med*. 1981;25:1–37.

38. Pollock RV, Carmichael LE. Maternally derived immunity to canine parvovirus infection. transfer, decline, and interference with vaccination. *s*. 1982;180(1):37–42.

39. Carmichael LE, Schlafer DH, Hashimoto A. Minute virus of canines (MVC, canine parvovirus type-1): pathogenicity for pups and seroprevalence estimate. *J Vet Diagn Invest*. 1994;6(2):165–174.

40. Houston DM, Ribble CS, Head LL. Risk factors associated with parvovirus enteritis in dogs: 283 cases (1982-1991). *J Am Vet Med Assoc*. 1996;208(4):542–546.

41. Steinel A, Munson L, Van Vuuren M, et al. Genetic characterisation of feline parvovirus sequences from various carnivores. *J Gen Virol*. 2000;81(Pt 2):345–350.

42. Truyen U. Emergence and recent evolution of canine parvovirus. *Vet Microbiol*. 1999;69(1–2):47–50.

43. Truyen U, Gruenberg A, Chang SF, et al. Evolution of the feline-subgroup parvoviruses and the control of canine host range *in-vivo*. *J Virol*. 1995;69(8):4702–4710.

44. Truyen U. Evolution of canine parvovirus: loss and gain of the feline host. *Tierarztl Prax*. 1996;24(3):316–318.

45. Mochizuki M, Horiuchi M, Hizagi H, et al. Isolation of canine parvovirus from a cat manifesting clinical sign of feline panleukopenia. *Clin Microbiol*. 1996;34(9):2101–2105.

46. Ikeda Y, Miyazawa T, Nakamura K, et al. Serosurvey for selected virus infections of wild carnivores in Taiwan and Vietnam. *J Wildl Dis*. 1999;35(3):578–581.

47. Ikeda Y, Mochizuki M, Naito R, et al. Predominance of canine parvovirus (CPV) in unvaccinated cat populations and emergence of new antigenic types of CPVs in cats. *Virology*. 2000;278(1):13–19.

48. Kapil S, Rezabek G, Germany B, et al. Isolation of a virus related to canine parvovirus type 2 from a raccoon (*Procyon lotor*). *Vet Rec*. 2010;166(1):24–25.

49. Qin Q, Loeffler IK, Li M, et al. Sequence analysis of a canine parvovirus isolated from a red panda (*Ailurus fulgens*) in China. *Virus Genes*. 2007;34(3):299–302

50. Ikeda Y, Nakamura K, Miyazawa T, et al. Feline host range of canine parvovirus: recent emergence of new antigenic types in cats. *Emerg Infect Dis*. 2002;8(4):341–346.

51. Horiuchi M, Goto H, Ishiguro N, et al. Mapping of determinants of the host range for canine cells in the genome of canine parvovirus using canine parvovirus/mink enteritis virus chimeric viruses. *J Gen Virol*. 1994;75 (Pt 6):1319–1328.

52. Ikeda Y, Miyazawa T, Kurosawa K, et al. New quantitative methods for detection of feline parvovirus (FPV) and virus neutralizing antibody against FPV using a feline T lymphoid cell line. *J Vet Med Sci*. 1998;60(8):973–974.

53. Kumar P, Garg SK, Gupta PK, et al. Detection of canine parvovirus DNA by polymerase chain reaction. *Indian J Anim Sci*. 2003;73:573–575.

54. Rai A, Gupta AA, Raut U, et al. Isolation of canine parvovirus in CRFK cell line. *Indian J Comp Microbiol Immunol Inf Dis*. 2004;25:51–52.

55. Panneer D, Mukhopadhyay HK, Antony PX, et al. Isolation and antigenic typing of canine parvovirus. *J Interacad*. 2009;13:178–183.

56. Mochizuki M, Ohshima T, Une Y, et al. Recombination between vaccine and field strains of canine parvovirus is revealed by isolation of virus in canine and feline cell cultures. *J Vet Med Sci*. 2008;70(12):1305–1314.

57. Decaro N, Desario C, Campolo M, et al. Clinical and virological findings in pups naturally infected by canine parvovirus type 2 Glu-426 mutants. *J Vet Diagn Invest*. 2005;17(2):133–138.

58. Cavalli A, Martella V, Desario C, , et al. Evaluation of the antigenic relationships among canine parvovirus type 2 variants. *Clin Vaccine Immunol*. 2008;15(3):534–539.

59. Decaro N, Desario C, Elia G, et al. Evidence for immunisation failure in vaccinated adult dogs infected with canine parvovirus type 2c. *New Microbiol*. 2008;31(1):125–130.

60. Samyukta ML. *Genotyping of canine parvovirus in southern India and cross antigenic evaluation of vaccine strains*. M.V.Sc. thesis submitted to Pondicherry University. 2012.

61. Nobal. *Genetic analysis and monitoring of canine parvovirus strains circulating in southern India*. M.V.Sc. thesis submitted to Pondicherry University. 2012.

62. Chinchkar SR, Mohana Subramanian B, Hanumantha Rao N, et al. Analysis of VP2 gene sequences of canine parvovirus isolates in India. *Arch Virol*. 2006;151(9):1881–1887.

63. Yang DK, Kim B, Kim Y, et al. Genetic analysis of canine parvovirus vaccine strains in Korea. *Korean J Vet Res*. 2009;49:243–248.

64. Decaro N, Desario C, Lucente MS, et al. Specific identification of feline panleukopenia virus and its rapid differentiation from canine parvoviruses using minor groove binder probes. *J Virol Methods*. 2008;147(1):67–71.

65. Bajehson DB. *Molecular characterization of canine parvovirus strains from domestic dogs in South Africa and Nigeria*. South Africa: A thesis submitted to University of Pretoria; 2010. p. 1–106.

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66. Pinto LD, Streck AF, Gonçalves KR, et al. Typing of canine parvovirus strains circulating in Brazil between 2008 and 2010. *Virus Res*. 2012;165(1):29–33.
67. Aldaz J, García-Díaz J, Calleros L, et al. High local genetic diversity of canine parvovirus from Ecuador. *Vet Microbiol*. 2013;166(1–2):214–219.
68. Lin Chao Nan, Chi Hsien Chien, Ming Tang Chiou, et al. Genetic characterization of type 2a canine parvoviruses from Taiwan reveals the emergence of an Ile324 mutation in VP2. *Virol J*. 2014:11(39).

Citation: Brindhalakshmi B, Mukhopadhyay HK, Antony PX, et al. Isolation and molecular characterization of canine and feline parvovirus strains - an updated review. *J Dairy Vet Anim Res.* 2016;3(5):164–169. DOI: 10.15406/jdvar.2016.03.00093