

## Genome Sequence of Canine Parvovirus Strain SC02/2011, Isolated from a Puppy with Severe Diarrhea in South China

## Chunmei Ju, Yi Cheng, Yikuan Ji, Yu Wang, Leilei Sun, and Jiaxin Huang

College of Veterinary Medicine, South China Agricultural University, Wushan, Guangzhou, Guangdong, China

A widespread hemorrhagic gastroenteritis in young dogs occurred in South China. A virulent field canine parvovirus (CPV) strain, SC02/2011, was isolated from a puppy showing enteric signs in Guangdong, China. The genome of CPV strain SC02/2011 was sequenced and analyzed, which will promote a better understanding of the molecular epidemiology and genetic diversity of CPV field isolates in South China.

Canine parvovirus type 2 (CPV-2), a nonenveloped singlestranded linear DNA virus in the *Parvoviridae* family, causes hemorrhagic gastroenteritis and myocarditis in young dogs (1). Currently, the antigenic variants, termed CPV-2a, CPV-2b, New CPV-2a, New CPV-2b, and CPV-2c, have completely replaced the original CPV-2 (2, 4, 6, 7). In recent years, the symptoms have been characterized by lethargy, vomiting, fever, and diarrhea (usually bloody), and a high mortality rate in puppies has emerged frequently in South China. A virulent field CPV strain, SC02/2011, was isolated from a puppy with severe diarrhea in Guangdong Province in October 2011. In order to identify the genotype of field strain SC02/2011, its genome sequence was determined.

The genome, excluding a partial inverted terminal repeat (ITR) sequence, was amplified by PCR with two primer pairs. The PCR products were gel purified using the QIAquick gel extraction kit (Qiagen) and sequenced on a 3730 DNA analyzer (Applied Biosystems). The genomic sequence of SC02/2011 comprises 4,699 nucleotides (nt) containing two open reading frames (ORFs). ORF1 (nt 151 to 2157) encoded two nonstructural proteins (NS1 and NS2), and ORF2 (nt 2164 to 4419) encoded two structural proteins (VP1 and VP2) through alternative splicing of the same mRNAs.

The genome sequence of SC02/2011 shares 96.13%, 98.51%, and 99.4% nucleotide sequence identity with those of CPV-d (NC\_001539), CPV-Y1 (D26079), and CPV-b (M38245), respectively (3, 5, 8). One deletion region (nt 4659 to 4781) was found in strain SC02/2011 compared to CPV-d isolated from the United States. There is an insertion region (nt 4433 to 4473) in strain SC02/2011 compared to CPV-Y1 isolated from Japan. These insertion and deletion sites are all in the 3' untranscribed region (UTR). Compared with CPV-b, SC02/2011 has 28 single nucleotide sequence variations, of which 16 variant loci were identified in the VP2 gene and resulted in 10 amino acid substitutions in the VP2 protein.

CPV-2 evolves rapidly, showing genomic substitution rates similar to those of RNA viruses, with values of about  $10^{-4}$  substitutions per site per year. CPV-2 variants differ from the original strain CPV-2 in 5 to 7 amino acid (aa) residues of the VP2 capsid protein interacting with the host-cell transferring receptor (TfR). According to the variation of these amino acid residues, strain SC02/2011 was classified as New CPV-2a. Phylogenetic analysis of the SC02/2011 VP2 sequence indicated that it was different from foreign and other domestic strains. The full-length VP2 coding sequence (1,755 bp) of strain SC02/2011 had maximum identity to Thailand strain GQ379048. Two mutations were first observed in the VP2 gene of SC02/2011. One was at nucleotide position 575, where variation C $\rightarrow$ T was observed, which resulted in amino acid substitution Ser $\rightarrow$ Thr. Another was at nucleotide position 1752, which is a synonymous mutation (T $\rightarrow$ C). The present study will promote a better understanding of the molecular epidemiology and genetic diversity of CPV field isolates in South China and help with the prevention and control of CPV infection in the future.

**Nucleotide sequence accession number.** The genome sequence of CPV strain SC02/2011 has been deposited in GenBank under accession no. JX660690.

## ACKNOWLEDGMENT

This work was supported by a grant from the National Natural Science Foundation of China (no. 31001074).

## REFERENCES

- 1. Appel MJ, Scott FW, Carmichael LE. 1979. Isolation and immunisation studies of a canine parvo-like virus from dogs with haemorrhagic enteritis. Vet. Rec. 105:156–159.
- Buonavoglia C, et al. 2001. Evidence for evolution of canine parvovirus type-2 in Italy. J. Gen. Virol. 82:1555–1560.
- 3. Horiuchi M, Goto H, Ishiguro N, Shinagawa M. 1994. 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. 75:1319–1328.
- 4. Ohshima T, et al. 2008. Chronological analysis of canine parvovirus type 2 isolates in Japan. J. Vet. Med. Sci. 70:769–775.
- Parrish CR. 1991. Mapping specific functions in the capsid structure of canine parvovirus and feline panleukopenia virus using infectious plasmid clones. Virology 183:195–205.
- 6. Parrish CR, et al. 1991. Rapid antigenic-type replacement and DNA sequence evolution of canine parvovirus. J. Virol. 65:6544–6552.
- 7. Parrish CR, O'Connell PH, Evermann JF, Carmichael LE. 1985. Natural variation of canine parvovirus. Science 230:1046–1048.
- 8. Reed AP, Jones EV, Miller TJ. 1988. Nucleotide sequence and genome organization of canine parvovirus. J. Virol. 62:266–276.

Received 17 September 2012 Accepted 18 September 2012 Address correspondence to Chunmei Ju, juchunmei@scau.edu.cn. Copyright © 2012, American Society for Microbiology. All Rights Reserved. doi:10.1128/JVI.02532-12