Porcine Deltacoronavirus Outbreaks in the World

Hee-Chun Chung and Bong-Kyun Park*

Department of Veterinary Medicine Virology Lab, College of Veterinary Medicine and Research Institute for Veterinary Science, Seoul National University DaeHakRo 1, GwanAk-Gu, Seoul 151-742, Korea

*Corresponding author: Bong Kyun Park, Department of Veterinary Medicine Virology Lab, College of Veterinary Medicine and Research Institute for Veterinary Science, Seoul National University DaeHakRo 1, GwanAk-Gu, Seoul 151-742, Korea, Tel: +82-2-880-1255; Fax: +82-2-885-0263; E-mail: parkx026@snu.ac.kr

Received Date: September 14, 2017; Accepted Date: September 20, 2017; Published Date: September 25, 2017

Copyright: © 2017 Chung HC, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.


Opinion

Porcine deltacoronaviruses (PDCoV) belong to Deltacoronavirus genus of the Coronaviridae family [1]. In February 2014, a new PDCoV, similar to a coronavirus detected in Hong Kong in 2012, has been recognized first in the United States, where the virus has been identified in pig farms in Iowa with a history of clinical acute severe diarrhea [1]. Since then, PDCoVs have been detected in more than 20 states in the USA and Canada, South Korea, Thailand, and China [2,3]. The recent study conducted in the USA with newly emerged PDCoV revealed the U.S. PDCoV possesses unique genetic characteristics and share a close relationship with PDCoVs of Hong Kong and Korea. In Korea, the PDCoV strains (KOR/KNU14-04/2014, SL, DH) were also reported in feces from diarrheic piglets in Korea in April 2014-2017. The Korean strains showed 99-99.7% homology with U.S. strains [3].

Recently, PDCoVs have also been detected in 20 out of 143 samples collected in five Chinese provinces: Heilongjiang, Liaoning, Tianjin, Handong, and Jiangsu [4]. Sequence analysis suggests that U.S. PDCoV originated from Hong Kong strains, and further studies required demonstrating any possibility of virulence in U.S. PDCoV isolates due to an existence of unique amino acid substitutions.

Overall clinical diseases associated with PDCoV infection are highly similar with those caused by porcine epidemic diarrhea virus (PEDV) and Transmissible gastroenteritis coronavirus (TGEV) infection. But the mortality rates induced by PDCoV infection are lower (40-50%) than those for PEDV and TGEV (90-100%) [4]. Similar to TGEV and PEDV, PDCoV heavily replicates in the small intestine and high levels of genomic RNA were detected in feces, intestine, and tissues, and moderate levels of PDCoV RNA were detected in blood and extra intestinal tissues. Antigen-positive cells were detected in large number in all sections of the small intestine [4,5]. It is expected that further analysis of very recently performed experiments will provide a better understanding of the pathogenesis and clinical symptoms associated with PDCoV infection. Pathogenesis and virulence were seen and severe, watery diarrhea and vomiting appeared in 48-72 hour post infection in gnotobiotic and conventionally raised pigs [5].

In many farms, PEDV and PDCoV were simultaneously detected and further research is needed to establish its role in swine disease. The understanding of data from the field has limitations since co-infections with PEDV or other intestinal pathogens are common. PDCoV has been reported in a few countries and limited studies have been done so far. Based on the currently available information, it seems that PDCoV would have a lower impact than PEDV, however, further studies should require to the emergence of novel PDCoV in other countries and should be considered the possibility of further disease outbreak and future impact on swine industry worldwide.

Acknowledgments

This study was supported by the BioGreen 21 Program, Rural Development Administration (grant no. PJ011184).

References