Medicine, Research Institute for Veterinary Science, and BK21 PLUS Creative Veterinary Research Center, Seoul National University, Seoul 08826, Korea; [2]National Research Laboratory of Molecular Microbiology and Toxicology, Department of Agricultural Biotechnology, and Center for Food Safety and Toxicology, Seoul National University, Seoul 08826, Korea.

Introduction: *Vibrio(V.) vulnificus*, VvpE, is an elastase responsiblefor host surface adherence and vascular permeability; however, the roles ofVvpE in the pathogenesis of *V. vulnificus*(WT) are poorly understood. In the present study, we have investigated therole of VvpE in regulation of intestinal tight junctions and the colonizationof WT.

Materials and Methods: *In vivo* roleof VvpE in tight junction disruption and intestinal colonization was assessedby the mutation and complementation of the *vvpE*gene from *V. vulnificus* in mouseinfection models. Gain- and loss-of- function approaches were used to identifyenterotoxicity and pro-inflammatory response in mice ileum tissue.

Results: We found that *vvpE* mutant prevents intestinaltight/ adherens junction dysregulation due to a WT infection and maintains thephysiological level of the epithelial paracellular permeability. Interestingly,the *vvpE* mutant exhibited defective intestinal colonization abilities, whereas WT colonization was significantly elevated in the ileum. Finally, the *vvpE*mutant negated the enterotoxicity, the breakdown of red blood cells, andpro-inflammatory responses, all of which are induced by the WT infection. In addition, we found thatVvpE contributes to WT pathogenesis in multiple ways by interacting withintestinal proteins, including β-globin, Annexin A2, Annexin A4, F-actin, andintelectin-1b.

Conclusions: These results demonstrate that VvpE plays important role inpromoting the tight junction disruption and intestinal colonization of *V. vulnificus*.

References

[1] Ashida H. et al. 2012. Bacteria and host interactions in the gut epithelial barrier. Nat Chem Biol 8, 36-45.
[2] Berkes J. et al., 2003. Intestinal epithelial responses to enteric pathogens: effects on the tight junction barrier, ion transport, and inflammation. Gut 52, 439-451.
[3] Blake P.A. et al. 1979. Disease caused by a marine Vibrio. Clinical characteristics and epidemiology. N Engl J Med 300, 1-5

P-012

Studies on pathogenesis of avian influenza viruses in mice by infection route

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Introduction: Introduction Highly pathogenic avian influenza virus (HPAI), H5N1 has been regarded as a strong pandemic candidate with high pathogenesis and mortality threatening the health of the human. Respiratory complications are general symptoms, but gastrointestinal symptoms have often been reported in human infected with HPAI H5N1.

Materials and Methods: Here we investigated whether infection route can affect the different types of pathogenesis with a mouse infection model. Mice were infected with avian influenza virus bearing three different degrees of subtypes (HPAI, low pathogenic avian influenza (LPAI), and seasonal influenza) by intranasal or oral route. The mice infected with HPAI by intranasal route showed higher mortality and viral infection in lung than those infected with seasonal influenza or LPAI.

Results: The virus was not detected in intestinal in all mice infected through intranasal route. However evident pathogenesis was observed in intestine of the mice infected with A/Vietnam/1194/2001 orally. Moreover, the virus was recovered in intestine as a 2.0 TCID50/ml viral titer.

Conclusions: This mice study supported that some orally HPAI infection might cause enteropathogenesis like A/Vietnam/1194/2001 (H5N1) strain. It means that high dose infection of HPAI through nasal route or intake HPAI contaminated poultry can cause gastrointestinal infection and spread whole body including lung and respiratory tract.

References

[1] Aleksandr S. Lipatov et al. Pathogenesis of H5N1 Influenza Virus Infections in Mice and Ferret Models Differs According to Respiratory Tract or Defective System Exposure. The Journal of Infectious Diseases. 2009. Vol. 199, No. 5, 717
[1] Kyoko Oh-Oka et al. Expressions of Tight Junction Proteins Occludin and Claudin-1 Are under the Circadian Control in the Mouse Large Intestine: Implications in Intestinal Permeability and Susceptibility to Colitis. PLoS ONE. 2014. PLoS ONE 9(5): e98016.

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Sequence Analysis of NucleocapsidGene of Porcine Epidemic Diarrhea Virus isolated in 2015

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Introduction: Porcineepidemic diarrhea virus (PEDV), a porcine enteropathogenic coronavirus, causeslethal watery diarrhea in piglets resulting in large economic losses due tohigh mortality [4]. Morbidity and mortality in infected neonatal piglets lessthan 5 days old approach 100% because