

JOINT EVENT

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## A novel pathway of virus dissemination within the host: Spread of porcine epidemic diarrhea virus (PEDV) from the nasal cavity to the intestinal mucosa in swine

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Porcine epidemic diarrhea (PED) has made catastrophic impacts on the global pig industry since 2011. The causative agent, porcine epidemic diarrhea virus (PEDV), was a typical intestinal coronavirus and transmitted by the generally acknowledged fecal-oral route. However, high infectivity of airborne PEDV and quickly spread between pig farm (even over the far distance) indicated that airborne transmission may make contributions to the rapid spread of PEDV. This study demonstrated that PEDV could cause typical diarrhea in piglets through nasal spraying and the exact mechanisms involved has been well studied in vitro and in piglets. At first, PEDV was detected by immunohistochemistry test in nasal epithelium at early stages of the infection. Then, the results were further verified by establishing air liquid interface culture of pig's nasal epithelial cells (NECs) in vitro. Moreover, PEDV captured by dendritic cells (DCs) in nasal passage were observed in nasal passage and DC/NECs co-culture system, demonstrating that PEDV could recruit DCs to the nasal epithelial cells (ECs) and form transepithelial dendrites (TEDs) to capture luminal viruses. Subsequently, PEDV carried DCs could form firm adhesion with T cells and transmit the virus to CD3<sup>+</sup> T cells via virological synapse. Additionally, the virus loaded CD3<sup>+</sup> T lymphocyte could enter the blood circulation through the lymphocyte recirculation and reach the intestinal mucosa. Finally, virus caused infection in intestinal epithelium (Vero cells, susceptible cells for PEDV) by CD3<sup>+</sup> T cells mediated transfer infection. Our finding is the first to demonstrate a novel pathway of PEDV dissemination within host and illustrated the mechanism of it transport from entry site to pathogenic site, which sheds light on prevention measures and pathogenic mechanism for viruses with the same characteristics.

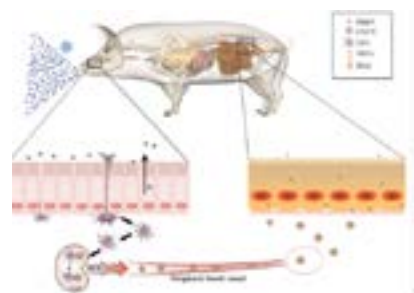


Fig. 8. Schematic of the proposed mechanism for PEDV transportation from the portal of entry (nasal mucosa) to the pathogenic site (intestinal mucosa). When airborne PEDV enters the nasal cavity, the virus accumulates and propagates in NECs and was released from the apical side of NECs. DCs may play an important role in helping the virus enter the nasal mucosa and be transferred to CD3<sup>+</sup> T cells. Furthermore, virus-carrying T cells entered the blood and reach the intestine through lymphocyte recirculation. Finally, the virus-carrying CD3<sup>+</sup> T cells can transfer the virus to intestinal epithelial cells, causing typical PED symptoms.

### Recent Publications

1. Li Y, Wang G, Wang J, Man K and Yang Q (2017) Cell attenuated porcine epidemic diarrhea virus strain Zhejiang08 provides effective immune protection attributed to dendritic cell stimulation. *Vaccine* 35(50):7033-41.
2. Li Y, Cai Y, Tao J, Kang X, Jiao Y, Guo R, Wang G, Pan Z and Jiao X (2016) Salmonella isolated from the slaughterhouses and correlation with pork contamination in free market. *Food Control* 59:591-600.
3. Li Y C, Pan Z M, Kang X L, Geng S Z, Liu Z Y, Cai Y Q and Jiao X A (2014) Prevalence, characteristics, and antimicrobial resistance patterns of Salmonella in retail pork in Jiangsu province, eastern China. *Journal of Food Protection* 77(2):236-45

### Biography

Yuchen Li has been studying in Nanjing Agricultural University as a PhD candidate since 2015. Currently, he is studying in the major of preventive veterinary medicine under supervision of Professor Qian Yang. His research focuses on the pathogenic mechanism of PEDV. He has published three research articles and interested in the research process.

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