Qian Yang et al., Virol Mycol 2018, Volume 7
DOI: 10.4172/2161-0517-C2-026

Persistent TGEV infection enhances ETEC K88 adhesion by promoting epithelial-mesenchymal transition in intestinal epithelial cells

Qian Yang and **Weigang**
Nanjing Agricultural University, China

Transmissible gastroenteritis virus (TGEV) is a coronavirus, characterized by diarrhea, high morbidity, and the mortality is 100% in piglets less than 2 weeks old. Pigs infected with TGEV often suffer from secondary infection with other pathogens, which aggravates the severity of diarrhea, but the mechanisms remain unknown. Here, we hypothesized that persistent TGEV infection stimulates the epithelial–mesenchymal transition (EMT), thereby generating cells that more easily adhere to enterotoxigenic Escherichia coli (ETEC). Intestinal epithelial cells are the primary targets of TGEV and ETEC infection. We found that TGEV can persistently infect porcine intestinal columnar epithelial cells (IPEC-J2), and cause EMT, consistent with multiple changes in key cell characteristics. Infected cells display fibroblast-like shapes, exhibit increases in mesenchymal markers with a corresponding loss of epithelial markers, have enhanced expression of IL-1β, IL-6, IL-8, TGF-β, and TNF-α mRNAs, and demonstrate increases in migratory and invasive behaviors. Additional experiments showed that activation of the PI3K/Akt and ERK signaling pathways via TGF-β are critical for the TGEV-mediated EMT process. Cellular uptake is also modified in cells that have undergone EMT. TGEV-infected cells have higher levels of integrin α5 and fibronectin and exhibit enhanced adhesion of ETEC K88. Reversal of EMT reduces ETEC K88 adhesion and inhibits the expression of integrin α5 and fibronectin. Overall, these results suggest that TGEV infection induces EMT in IPEC-J2 cells, increasing the adhesion of ETEC K88 in the intestine and facilitating dual infection.

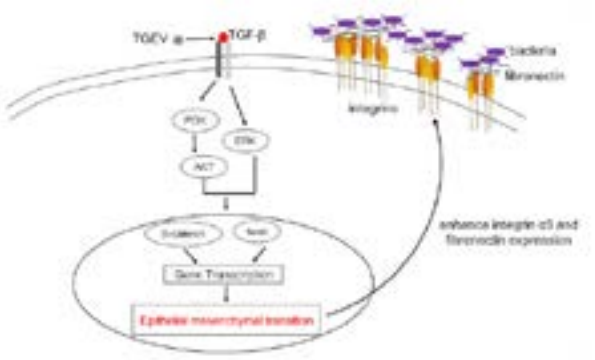

Figure 1: Schematic diagram showing a persistent TGEV infection induces EMT and promotes bacterial adhesion. TGEV infection induces the production of TGF-β, activates the P13K/Akt and ERK signaling pathways and induces the EMT markers. During TGEV- induced EMT, the expression of integrin α5 and fibronectin is enhanced, leading to increased bacterial adherence.

Recent Publications

1. Yin Yinyan, Qin Tao, Wang Xiaoqing, Lin Jian, Yu Qinghua and Yang Qian (2015) CpG DNA assists the whole inactivated H9N2 influenza virus in crossing the intestinal epithelial barriers via transepithelial uptake of dendritic cell. Mucosal Immunology 8(4):799-814.

2. Liu H, Xu W, Yu Q, Yang Qian et al. (2017) 4,4′ -Diaponeurosporene-producing Bacillus subtilis increased mouse resistance against Salmonella typhimurium infection in a CD36-dependent manner. Frontiers in Immunology 8:483.

3. Xia L, Dai L, Yu Q, et al. (2017) Persistent TGEV infection enhances ETEC K88 adhesion by promoting epithelial-mesenchymal transition in intestinal epithelial cells. Journal of Virology DOI:10.1128/JVI.01256.

4. Qin Tao, Yin Yinyan, Yu Qinghua, Huang Lulu, Wang Xiaoqing, Lin Jian and Yang Qian (2015) CpG oligodeoxynucleotides facilitate delivery of whole inactivated H9N2 influenza virus via transepithelial dendrites of dendritic cells in nasal mucosa. Journal of Virology 89(11):5904-5918.

Biography

Qian Yang worked as a Professor in College of Veterinary Medicine. Her research work focuses on the mucosal immunity in domestic animals. At first, several mucosal adjuvants were studied to increase the efficiency of inactivated viruses (influenza). Secondly, some delivery vehicles (*Lactobacillus and Bacillus subtilis*) for antigens for oral immunization have been studied because of degradation of the antigen by gastric acid and proteases present in the gastrointestinal tract. The other fields includes: the interaction between epithelium and pathogens and the relevant pathogenic mechanism. She has published more than 80 original and review articles in scientific journals.

zxbyq@njau.edu.cn

Notes: