FMT reduces the mortality of BALB/c mice caused by *Listeria monocytogenes* (EGD-e) infection

Qing Liu and Liang Guo
University of Shanghai for Science and Technology, China

*Listeria monocytogenes* (Lm) is a kind of food pathogenic bacteria with strong pathogenicity that has been shown previously to cause infection via the gastrointestinal (GI) tract. External pathogens can cause changes in gut microbiota, and such a change can promote or confer resistance to the infection of pathogenic bacteria. However, the changes in the microbiota during Lm through the GI tract and infect the body is unknown. Eight-week-old mice's were inoculated orally with *L. monocytogenes* EGD-e, and portions of the liver, spleen and cecal contents were removed, homogenized and plated, and feces were collected on 0 day, first day and third day. After that, different concentrations of FMT were used to treat Lm infected mice. *L. monocytogenes* culture confirmed that the content of Lm in cecum after intragastric inoculation reached the highest level on first day, and then remained at a low level. The content of Lm in spleen and liver reached the highest level on third day. The percentage of the *Proteobacteria* spp, *Bacteroidetes* and *Cyanobacteria* on first day remained significantly higher than that of the 0 day (P<0.01), while the proportion of *Lactobacillus* and *Staphylococcus* on first day was significantly lower than that of the 0 day (P<0.01). Compared to first day, the *Coprococcus*, *Blautia* and *Eubacterium* increased significantly on third day. In addition, the mortality of infected mice was reduced by 28% after FMT treatment compared with PBS treatment. Finally, we showed that inoculated with EGD-e significantly altered the gut microbiome in mice in different times and the potential probiotics increased in infected mice like the *Blautia* may be developed as new probiotics to enhance resistance against *L. monocytogenes* infections. The gut microbiome of healthy mice can significantly reduce the mortality of infected mice by reducing the inflammatory response and rebuilding the dysbacteriosis.

Biography
Qing Liu is a Director of institute for foodbone pathogen harmless and management school of medical instrument and food engineering and Professor at University of Shanghai for Science and Technology, China

liuq@usst.edu.cn

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