中文題目:腸道移生大腸桿菌引發之移生阻力抵抗黏膜沙門氏菌感染

英文題目: Commensal *E. coli* induced colonization resistance against intestinal *Salmonella* infection

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Background: *Salmonella* is a leading cause of gastrointestinal bacterial infections worldwide. During mucosal infection, *Salmonella* must first establish a niche in the gastrointestinal tract in order to initiate infection. Colonization resistance (CR), defined as the ability of intestinal microbiota to protect host against pathogens, is the first line defense against intestinal infection. The complex intestinal microbiota is capable of constraining the extracellular growth of *Salmonella* in the gut lumen and mucus layers to prevent the SPI-1/2 type three secretion systems (TTSS) dependent intracellular growth phase of the infection. The mechanism for commensal bacteria mediated CR has been studied for a long time; however, only a few studies have explored how microbiota can induce CR against mucosal *Salmonella* infection.

Methods: We have identified a murine commensal *E. coli XZ* and oral gavaged C57BL6 mice with wide type, SPI-1 or/and 2 mutant *Salmonella* in the presence or absence of *E. coli XZ* precolonization. In this study we dissect how commensal *E. coli XZ* contributes host innate immunity against intestinal *Salmonella* infection in the presence or absence of complex mucosal microbiota.

Result: We have identified a murine commensal *E. coli* strain (*E. coli XZ*) that mediates colonization resistance against mucosal *Salmonella Typhimurium* infection, which is independent of the inflammasome, TLR5, MyD88, and adaptive immunity. We demonstrate that *E. coli* XZ does not directly compete with *Salmonella* growth *in vitro*, but is fully capable of inducing colonization resistance *in vivo*. *E. coli* XZ limits the early phase of *Salmonella*'s invasion into the murine gut ecosystem. *Salmonella Typhimurium* requires the SPI-2 TTSS to overcome *E. coli XZ* mediated colonization resistance in the presence of complex intestinal microbiota.

Conclusion: Commensal *E. Coli XZ* interacts with the host through a novel pathway to induce colonization resistance against mucosal *Salmonella* infection, *which* is not TLR5, inflammasome, MyD88/Trif, and adaptive immunity dependent.