

真菌医学研究センター Monthly セミナー

平成27年7月30日(木) 16:00~17:00

東京大学医科学研究所 2号館2階 小会議室
(東京メトロ南北線・都営地下鉄三田線 白金台駅下車)

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Title: Oxygen modulation of *Shigella flexneri* virulence and neutrophils physiology

Abstract:

Enteric pathogens must survive in the anaerobic environment of the large intestine, and many of them use shared mechanisms to sense lack of oxygen and to enhance their virulence (e.g. Type Three Secretion Systems, T3SSs). *Shigella* spp. are the leading cause of bacillary dysentery, and are able to invade non-epithelial cells in the gastrointestinal (GI) track. During the infectious process, *Shigella* has to resist to neutrophils bactericidal activity, as they represent the most abundant immune cell population recruited during *Shigella* invasion.

Exploiting *in vivo* models (rabbit, guinea pig) and *in vitro* approaches (epithelial cells culture), we have shown that *Shigella* infection is associated with hypoxia induction within the colonic mucosa. We observed that neutrophils recruitment and activation is the leading cause of hypoxia. Additionally, we have shown that neutrophils viability is maintained under anoxic conditions, which promote their bactericidal functions upon infiltration in infected tissues.

We characterized the adaptation of *Shigella* to low oxygen conditions in order to successfully colonize the colonic mucosa. We demonstrated that *Shigella* virulence is modulated by oxygen abundance, through the regulation of the T3SS function (Marteyn et al. 2010, Nature). We also identified a novel cell-division protein, named ZapE, which is essential for *Shigella* growth under low oxygen conditions (Marteyn et al. 2014, mBio).

Altogether, we demonstrate that *Shigella* and the innate immune system adapt to low oxygen environments. We aim at deciphering respective benefits of such physiological conditions on bacteria virulence and host response efficiency.

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