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Benfang Lei

Montana State University, USA

Molecular mechanisms of hypervirulent Group A *Streptococcus* to evade innate immune responses and to invade the vascular system in mouse model of pulmonary infection

Group A *Streptococcus* (GAS) causes common pharyngitis and occasional severe invasive infections. There is a significant knowledge gap on why noninvasive upper respiratory GAS infections usually do not result in lower respiratory infections while certain GAS strains can cause pneumonia and how invasive GAS disseminates systemically. A pulmonary murine infection model is used to address these questions. Paryngeal GAS isolates induced robust neutrophil recruitment and was effectively cleared in a NADPH Oxidase-dependent mechansim by neutrophils. In contrast, invasive isolates with mutations in virulence regulators CovRS and/or RopB inhibited neutrophil recruitment and caused pulmonary infections. Natural GAS RopB mutants caused infection only in the alveolar region whereas natural CovS and RopB double GAS mutants invade the perivascular interstitium, disrupts smooth muscle and endothelial layers of the blood vessels, and penetrates into the lumen of endothelial layer and the systemic circulation. Correction of the CovS mutation abolished the capacity of GAS to invade the vascular system. To identify virulecence factors that are critical for GAS innate immune evasion and vascular invasion, we tested single and double deletion mutants of CovRS-controlled virulence genes of hypervirulent GAS. Only a surface protein was found to be critical for the vascular invasion, and the inhibition of neutrophil recruitment requires both streptolysin S and the platelet-activating factor acetyl hydroslase Sse. Thus, Streptolysin S- and Sse-dependent evasion of neutrophil response is critical for the capacity of GAS to cause pulmonary infection, and GAS invasion of the vascular system requires the surface protein

Biography

Benfang Lei has completed his PhD from University of Houston, Texas and postdoctoral study at the Rocky Mountain Laboratories, NIAID, NIH at Hamilton, Montana. He is an Associate Professor at Department of Microbiology and Immunology, Montana State University. He has published 70 primary research papers and has been serving as an academic editor of PloS One and an editorial board member of Infection and Immunity.

blei@montana.edu

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