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SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Francisella tularensis Mutants with Cell-Type-Specific Virulence Defects

Previous studies have shown that *Francisella tularensis* evades early killing inside human neutrophils by preventing NADPH oxidase assembly at the phagosome. Schulert et al. (pp 1324–1336) screened a random transposon library to identify bacterial genes that affect neutrophil activation. The first candidates identified were *carA*, *carB*, and *pyrB*, and these uracil auxotrophic mutants were rapidly killed by neutrophil oxidants. More importantly, further characterization of these organisms revealed that they exhibited pleiotropic and cell-type-specific virulence defects that differentially affected cytokine secretion, phagosome escape, and bacterial growth and survival in primary human macrophages as well as macrophage- and epithelial cell-like cell lines.

Lack of Toll-Like Receptor 2 Is Associated with Impaired Host Resistance and Increased Inflammatory Response in Group B Streptococcal Infection

Group B streptococci (GBS) are an ever-growing cause of serious invasive infections in nonpregnant adults, with septic arthritis being one of the clinical manifestations of disease. Little is known regarding the role played by Toll-like receptor 2 (TLR2) during GBS infection in adults. Puliti et al. (pp 1524–1531) used TLR2 knockout mice to demonstrate that TLR2 contributes to regulating bacterial clearance and subsequent inflammatory responses in disease caused by GBS. This work suggests that TLR2 is an essential signaling molecule both for sepsis and for induction of arthritis.