Articles of Significant Interest Selected from This Issue by the Editors

Salmonella enterica Serovar Dublin Isolates That Repress Flagella Expression: Mechanism To Promote Invasiveness?

Salmonella enterica serovar Dublin is adapted to cattle but also infrequently infects humans, causing an invasive disease with high levels of morbidity and mortality. The bacterial factors responsible for this invasive phenotype are unknown. *Salmonella*-induced intestinal inflammatory responses may prevent bacterial dissemination to systemic sites. Yim et al. (p. 1465–1476) report the isolation of several *S.* Dublin strains completely lacking flagella, obtained from human bloodstream infections. These aflagellate isolates were impaired for triggering inflammation in a human intestinal epithelial cell line and in the ceca of mice. These findings suggest that this mechanism for reduced intestinal inflammation may contribute to enhanced systemic dissemination of *S.* Dublin.

CARD9 Is a Critical Signaling Molecule for Innate Host Defense to *Cryptococcus neoformans*

CARD9 is a critical adaptor molecule signaling through C-type lectin receptors. Yamamoto et al. (p. 1606–1615) show that CARD9 knockout mice are highly susceptible to pulmonary infection with *Cryptococcus neoformans*. Gamma interferon (IFN-γ) production was strongly reduced on day 3 postinfection in these mice, due to impaired accumulation of IFN-γ-producing NK and memory phenotype T cells. The reduced accumulation of these cells was correlated with lowered CCL4, CCL5, CXCL9, and CXCL10 synthesis. These results suggest that the susceptibility of mice lacking CARD9 to *C. neoformans* infection is likely due to the reduced accumulation of IFN-γ-expressing NK and memory phenotype T cells early in infection.

Type I Interferons Promote Lethal Disease in a Mouse Model of Shock-Like Illness Caused by the Rickettsial Pathogen *Ixodes ovatus Ehrlichia*

Rickettsiae are arthropod-born intracellular bacteria that cause mild to severe shock-like illnesses. Zhang et al. (p. 1698–1709) demonstrate that *Ixodes ovatus Ehrlichia* infection elicits type I interferons (alpha interferon [IFN-α] and IFN-β), whereas infection with a less virulent strain, *Ehrlichia muris*, does not. Type I interferons promote severe disease and death and suppress IFN-γ production, thought to be essential to control *Ehrlichia* growth. Surprisingly, IFN-γ is dispensable for protection in the absence of type I interferons. These data demonstrate that type I interferon signaling in nonhematopoietic tissues may promote tissue damage and death independent of bacterial growth, revealing a novel mechanism by which type I interferons drive bacterial pathogenesis.

Human Lyme Disease Patients Develop Lasting Strain-Specific Immunity

Many people suffer from Lyme disease on multiple occasions, suggesting that previous exposure to the causative agent *Borrelia burgdorferi* does not elicit a protective immune response. However, a given strain is almost never detected multiple times from the same patient, suggesting that *B. burgdorferi* exposure may elicit strain-specific immunity. Khatchikian et al. (p. 1408–1413) perform an analysis using mathematical and statistical models parameterized with data on the occurrence and frequency of the occurrence of different *B. burgdorferi* strains in human patients. This analysis demonstrates that humans treated for early Lyme disease develop protective immunity that is strain specific and lasts for at least 6 years.

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