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SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Entry of *Porphyromonas gingivalis* Membrane Vesicles Impairs Cellular Function

Gram-negative bacteria secrete outer membrane vesicles (MVs), which are proposed to be bacterial "bombs" containing various virulence factors. Here, Furuta et al. (p. 4761–4770) analyzed cellular functional impairment following entry of periodontal pathogen *Porphyromonas gingivalis* MVs into host epithelial cells. *P. gingivalis* MVs swiftly entered host epithelial cells via an endocytosis pathway, and then MV-associated proteases degraded the cellular transferrin receptor and integrin-related signaling molecules, which resulted in depletion of intracellular transferrin and inhibition of cellular migration. *P. gingivalis* MVs may be potent vehicles for transmission of virulence factors into host cells.

Ehrlichia chaffeensis Infections in *Drosophila melanogaster*

Ehrlichia chaffeensis is an obligate, intracellular bacterium, vectored by *Amblyomma americanum* ticks, and is the causative agent of human monocytic ehrlichiosis (HME). Luce-Fedrow et al. (p. 4815–4826) demonstrate that *E. chaffeensis* replicates in adult *D. melanogaster,* activates the cellular and humoral defenses, and involves the Toll and Imd pathways. The ability to utilize *D. melanogaster* to study *E. chaffeensis* infections will enable the use of an array of molecular and genetic tools to better understand these arthropod-borne bacteria and the genetic requirements for the replication of *E. chaffeensis* in both vertebrate and invertebrate hosts.

Interleukin-17 Receptor, a Genetic Determinant in Host Susceptibility to *Chlamydia* Infection

Chlamydia species cause many chronic inflammatory diseases. In both humans and mice, genetic susceptibility to *Chlamydia* infection determines the outcome of infection. Zhou et al. (p. 5059–5070) examined the immune responses in the susceptible C3H/HeN mice and the resistant C57BL/6 mice, with particular attention to interleukin-17 (IL-17) and Th17 responses. They demonstrate heightened activity of IL-17 and IL-17 receptors (RA and RC) being closely associated with enhanced susceptibility in C3H mice. Of interest, they identify IL-17RC as an inherent genetic factor responsible for the profound inflammatory responses observed in C3H mice.

Cross-Reactive Antibodies to Anthrax Edema and Lethal Factors Are Surprisingly Ineffective at Toxin Neutralization

Protective Antigen (PA) is the primary component of the current U.S. anthrax vaccine. The need for five initial injections and yearly boosters for protection has prompted searches for additional targets. Edema factor (EF) and lethal factor (LF) bind to the receptor-binding moiety PA to access host cells. Nguyen et al. (p. 4714–4723) hypothesized that cross-reactive B-cell epitopes of LF and EF would occur in their PA-binding domains and afford cross-protection against intoxication. Surprisingly, cross-reactive antibodies that were identified failed to neutralize either toxin. Nevertheless, whole recombinant EF immunization led to EF neutralizing antibodies and protection of mice from EF-plus-PA lethal challenge. This response mapped to both PA- and calmodulin-binding domains of EF.