Articles of Significant Interest Selected from This Issue by the Editors

Bile Salt Taurocholate Stimulates *Vibrio cholerae* Biofilm Dispersal and Virulence Induction

Ingestion of biofilm-like particles may be a clinically important route of *Vibrio cholerae* transmission. Little is known about the interaction of these bacterial aggregates and host signals as they transition into the host intestine. Hay and Zhu (p. 317–323) find that certain host-derived bile salts promote biofilm dispersal through abiotic degradation of the matrix and release of free-living cells. The bile salt taurocholate, which is known to activate virulence, is only able to do so after promoting cell detachment from the biofilm. This study suggests that *V. cholerae* has coopted host signals to coordinate biofilm dispersal and colonization early in infection.

Multiscale Computational Modeling of Macrophage Polarization in Granulomas Suggests Potential Pharmacological Target for Tuberculosis Infection Control

Macrophages are the most abundant cells in tuberculosis granulomas and have been shown to perform many key immunological functions throughout the course of infection. Very little is known about how these processes are regulated, what controls macrophage microenvironment-specific polarization and plasticity, or why some granulomas control bacterial replication while others permit bacterial dissemination. Marino et al. (p. 324–338) predict that temporal dynamics of the ratio of pro- to anti-inflammatory macrophages can determine granuloma outcome and suggest manipulation of the dynamics of NF-κB signaling as a viable therapeutic target for promoting bacterium-controlling proinflammatory polarization early during infection.

Intestinal Alkaline Phosphatase Maintains Immune Sensitivity to Bacterial Lipopolysaccharide and Prevents Lipopolysaccharide-Mediated Weight Gain

Previous work has revealed that the zebrafish intestinal alkaline phosphatase (ALPI) is induced by bacterial lipopolysaccharide (LPS) and functions to detoxify LPS and prevent excessive inflammatory responses to the gut microbiota. Yang et al. (p. 247–258) report that the mouse ALPI gene *Akp3* is also upregulated by microbiota, although not through the same LPS-dependent mechanism. *Akp3* appears to be dispensable for gut homeostasis or during *Yersinia pseudotuberculosis* infection but is required for maintaining immune sensitivity to LPS. Importantly, long-term ALPI deficiency in $Akp^{-/-}$ mice that is associated with LPS desensitization results in increased diet-induced weight gain through an LPS-dependent mechanism.

Accumulation-Associated Protein Is Essential for *Staphylococcus epidermidis* Central Venous Catheter Infection

Staphylococcus epidermidis is one of the most common causes of hospital-acquired infections, due largely to its ability to form robust biofilms. Accumulation-associated protein (Aap) is involved in bacterial accumulation and also binds epithelial cells. Schaeffer et al. (p. 214–226) demonstrate that Aap is essential for infection in a central venous catheter model following systemic inoculation of *S. epidermidis*. Binding assays with recombinant Aap domains confirm that the N-terminally located A domain facilitates initial attachment to abiotic surfaces, likely explaining the requirement for Aap *in vivo*. These data demonstrate that Aap contributes to virulence and suggest functions for the protein during both commensal growth and infection.

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