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SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Genome Sequence Analysis and Transposon Mutagenesis of *Mycoplasma arthritidis* Reveal Pathogenic Features

With their limited coding capacity, mycoplasmas are invaluable model organisms for studying host-pathogen interactions. Dybvig et al. (p. 4000–4008) determined the sequence of the 820-kb genome of the murine pathogen *Mycoplasma arthritidis* and through transposon mutagenesis knocked out 218 of the 635 predicted protein-coding regions. Two large families of genes were identified as coding for phase-variable proteins that might contribute to disease chronicity through the avoidance of host defenses. Mutants that have disruptions in the gene coding for the secreted superantigen MAM were isolated and are being evaluated to assess virulence. Also dispensable were genes coding for ribosomal proteins S15, S18, and L15, suggesting that the mycoplasma ribosome may differ slightly from that of most organisms.

Suppression of Innate Immunity by a Commensal/Probiotic Bacterium

One of the most profound issues with innate immunity is how animals can tolerate their own normal flora when these have the same signature molecules, recognized as threatening by the immune system, as pathogens. The study by Cosseau et al. (p. 4163–4175) demonstrates that *Streptococcus salivarius* can suppress innate immune responses to the pathogen *Pseudomonas aeruginosa* and the Toll-like receptor agonist flagellin. Using a system biology approach to compare host epithelial cell responses to this commensal/probiotic *Streptococcus* sp. with those for three other pathogens, it was shown that this organism down-regulates the innate immune responses of human epithelial cells, suppressing NF-κB, while promoting host-microbe homeostasis.

"Arresting" *Salmonella enterica* Serovar Typhimurium on the Intestinal Epithelial Border

Secretory immunoglobulin A (SIgA) antibodies against the O antigen of lipopolysaccharide are the primary determinants of mucosal immunity to enteric pathogens, including *Salmonella enterica* serovar Typhimurium. However, the underlying mechanism by which SIgA interferes with bacterial colonization and invasion of intestinal epithelial cells is poorly understood. In this issue, Forbes et al. (p. 4137–4144) demonstrate that Sal4, an anti-O5-specific monoclonal IgA previously shown to protect mice against oral challenge with *Salmonella* serovar Typhimurium, is a potent inhibitor of flagellum-based motility and *Salmonella* pathogenicity island 1-mediated invasion of epithelial cells. These data reveal a novel mechanism by which SIgA may interfere with microbial pathogenesis at mucosal surfaces.

The IbeA Protein of Extraintestinal Pathogenic *Escherichia coli* Participates in Adhesion and Invasion by Modulating Type 1 Fimbria Expression

Extraintestinal pathogenic *Escherichia coli* (ExPEC) strains are responsible for a wide range of infections. The *ibeA* gene was previously characterized as necessary for entry of ExPEC into eukaryotic cells. Cortes et al. (p. 4129–4136) investigate in further detail the role of IbeA in the adhesion of ExPEC. They suggest that, rather than directly mediating adhesion, IbeA instead regulates the expression of recombinases involved in the phase variation mechanisms controlling type 1 fimbria expression and thus type 1 fimbria-mediated adhesion.

Proteome of *Vibrio cholerae* in Human Stool

Vibrio cholerae remains an important cause of diarrheal disease globally. Since animal models of cholera are suboptimal, studies of *V. cholerae* directly in the human host are useful for understanding pathogenesis and for identifying targets of protective immunity. LaRocque et al. (p. 4145–4151) used mass spectrometry to characterize the *V. cholerae* proteins present in the stool samples of 32 cholera patients. More than 900 *V. cholerae* proteins were identified in the patients' samples, including many of the known *V. cholerae* virulence factors. This work represents the first proteome of a pathogenic bacterium recovered from a natural host.

Urinary Tract Infections Are More Severe in Diabetic Mice than in Nondiabetic Mice

Women with type 1 diabetes are at high risk for urinary tract infection (UTI) and often face serious sequelae. Combining two well-described mouse models, Rosen et al. (p. 4290–4298) established a diabetic mouse model of UTI that closely mimics human infection. Diabetic mice were more susceptible than nondiabetic mice to infection by various uropathogens, with a 50% infective dose of less than 100 CFU for *Escherichia coli*, a 290-fold increase in susceptibility. Diabetic mice also developed severe pyelonephritis, with biofilm-like collections of bacteria in kidney tubules. This model will be useful in elucidating the mechanisms of diabetes-related susceptibility to UTI.