Articles of Significant Interest Selected from This Issue by the Editors

Are Cholesterol-Dependent Cytolysins Discarded or Recently Acquired by Gram-Negative Bacteria?

Cholesterol-dependent cytolysins (CDCs), also known as CDC pore-forming toxins, are encoded by many Gram-positive opportunistic pathogens. In this issue, Hotze and colleagues (p. 216–225) identified CDC pore-forming toxins in two Gram-negative species. Both species likely use the CDCs to fend off eukaryotic predators, as they exclusively inhabit anaerobic soils or sediments. Gram-positive pathogens (many are soil inhabitants) have successfully adapted CDCs to defend against immune cells, yet the CDC genes are conspicuously absent from Gram-negative pathogens. The reason for the absence is unknown but may be due to the recent acquisition of CDCs, to insufficient time to disseminate, or to the fact that they were previously tried and discarded by the pathogens due to incompatibilities with their pathogenic mechanisms.

Immunosuppression Is a Double-Edged Sword following Urinary Catheterization

The benefits of urinary catheterization are tarnished by the adverse effects of catheter-induced inflammation. Guiton et al. (p. 329–339) employed an optimized murine model of catheter-associated urinary tract infection (CAUTI) to identify key constituents of this inflammatory response in the presence or absence of *Enterococcus faecalis*, a predominant agent of CAUTI. They demonstrate that induction of cystitis in catheterized but not in noncatheterized animals promotes enterococcal infection. Yet, immunossupression following glucocorticoid treatment or neutrophil depletion in mice with catheters significantly increases bacterial burden. Thus, understanding host responses will help tailor treatments that reduce the deleterious effects of urinary catheterization without promoting infections.

Salmonella enterica Serovar Typhimurium Attenuates Growth *In Vivo* inside Nonphagocytic Host Cells

Salmonella enterica serovars are enteric pathogens causing asymptomatic and chronic infections that are still poorly understood at the cellular level. Persistent infection demands tight control of pathogen proliferation, and both the host and the pathogen can contribute to this goal. Núñez-Hernández and colleagues (p. 154–165) demonstrate that *S. enterica* serovar Typhimurium restrains growth *in vivo* within nonphagocytic cells present in the intestinal lamina propria. As previously observed in cultured fibroblasts, the bacterial PhoP-PhoQ system prevents pathogen overgrowth *in vivo*. Genome expression analyses provide the first catalogue of *Salmonella* genes upregulated in nongrowing intracellular bacteria. Phagosomal acidic pH turns out to be a signal-inducing PhoP-PhoQ system in these dormant *Salmonella* bacteria.

Transcriptome Analysis Highlights the Unheralded Complexity of Enterotoxigenic *Escherichia coli* Pathogenesis

Enterotoxigenic *Escherichia coli* (ETEC) kills hundreds of thousands of children annually in developing countries. Presently, there is no vaccine to prevent ETEC infection. Vaccine development has been narrowly focused on a few antigens, namely plasmid-encoded colonization factors and known toxins. However, studies of ETEC transcriptional modulation during pathogen-intestinal cell interactions by Kansal et al. (p. 259–270) suggest a highly orchestrated sequence of events involving a cascade of known and putative virulence molecules that might be exploited in novel approaches to the rational design of future ETEC vaccines.

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