INFECTION AND IMMUNITY, Sept. 2007, p. 4189 Vol. 75, No. 9
0019-9567/07/$08.00+0 doi:10.1128/IAI.00967-07

SPOTLIGHT

Articles of Significant Interest Selected from This Issue by the Editors

Modulation of Host Cell Death by Obligate Intracellular *Coxiella burnetii*

Apoptosis is a highly regulated mode of cell death that can be triggered or inhibited during infection by intracellular parasites. Obligate intracellular *Coxiella burnetii*, which causes human Q fever, persistently infects mammalian cells with minimal cytopathic effect. Voth et al. (p. 4263–4271) demonstrate that *C. burnetii* actively inhibits apoptosis of macrophages, its in vivo target cell, by a mechanism involving decreased caspase activation and modulation of apoptosis-related gene expression. *C. burnetii* likely employs its potent antiapoptotic activity as a pathogenic strategy to maintain the host cell for the duration of its lengthy infectious cycle.

Inhibition of Host Cell Apoptosis without Activation of NF-κB

The apicomplexan parasites *Toxoplasma gondii* and *Theileria* spp. prevent host cell apoptosis by mechanisms that include activation of the host transcription factor NF-κB to upregulate prosurvival genes. Herman et al. (p. 4255–4262) show that the related parasite *Neospora caninum* is similarly capable of inhibiting apoptosis, but it does so without apparent translocation of NF-κB into the nuclei of infected cells. Therefore, the establishment of an antiapoptotic state by *N. caninum* does not appear to require activation of the NF-κB pathway. These findings highlight the diversity of mechanisms employed by parasitic protozoa to prevent host cell death.

Old Dog, New Trick: Novel Role for an ABC Transporter in *Streptococcus pyogenes* Intracellular Survival

Many gram-positive bacteria produce lantibiotic antimicrobial compounds, providing a competitive advantage in polymicrobial environments. Self-immunity to the lantibiotic is most often provided through an ABC transporter. Phelps and Neely (p. 4541–4551) report that a *Streptococcus pyogenes* transporter protein with high homology to lantibiotic immunity proteins and encoded within a lantibiotic operon was found to be required for intracellular survival in macrophages. Survival in intracellular environments has only recently been demonstrated for *S. pyogenes*. The SalY transporter may provide an example of the ability of *S. pyogenes* to adapt and evolve new survival strategies that allow dissemination and growth in previously uninhabitable sites.

Cholecystokinin and Mast Cells Connect Immunity and Pathogenesis during *Giardia intestinalis* Infection

Intestinal infections with *Giardia* spp. lead to cramps, nausea, and malabsorptive diarrhea, although no clear mechanisms have been identified. Mast cell responses are known to be required for elimination of this infection. Li et al. (p. 4514–4518) show that in infected mice, mast cells also control changes in gut motility. Moreover, they show that the peptide hormone cholecystokinin is responsible for triggering this mast cell-mediated response. Given that cholecystokinin is important in regulating levels of bile in the intestinal lumen and that bile plays key roles in parasite growth and differentiation, these findings suggest an elegant example of host-parasite coevolution.

Host Contact-Dependent Expression of Bacterial Virulence Genes

Intimate contact of *Pseudomonas aeruginosa* with host cells is an inducing signal for expression of a type III secretion system (T3SS). Most details of T3SS gene regulation have been deduced in vitro by using calcium-depletion as the inducing signal. The response to calcium depletion involves a regulatory cascade that is activated by secretion of a negative regulator of T3SS gene transcription. Urbanowski et al. (p. 4432–4439) demonstrate that translocation of this same regulator is also required to trigger this cascade in response to the more physiologically relevant host cell-contact signal.