Articles of Significant Interest in This Issue

The Manganese-Responsive Transcriptional Regulator MumR Protects *Acinetobacter baumannii* from Oxidative Stress

*Acinetobacter baumannii* is an emerging pathogen and a leading cause of hospital-acquired infections. However, many of the mechanisms utilized by this pathogen to cause disease remain poorly understood. In this issue, Green et al. (e00762-19) characterize a mechanism by which *A. baumannii* coordinates a response to manganese (Mn) starvation and reactive oxygen species (ROS), two stressors encountered by pathogens during infection. They find that *A. baumannii* Mn utilization facilitates resistance to ROS and uncover a relationship between Mn homeostasis, metabolism, and oxidative stress in this organism, highlighting the interconnectedness of these processes and exposing a promising target for antimicrobial development.

Major Histocompatibility Complex Class II-Restricted, CD4+ T Cell-Dependent and -Independent Mechanisms Are Required For Vaccine-Induced Protective Immunity against *Coxiella burnetii*

*Coxiella burnetii* is an obligate intracellular Gram-negative bacterium that causes Q fever in humans. The mechanism of vaccine-induced protective immunity against *C. burnetii* infection remains unclear. This study (e00824-19) provides novel evidence to support that major histocompatibility complex class II (MHC-II)-restricted, CD4+ T cell-dependent and -independent mechanisms are required for vaccine-induced protective immunity against *C. burnetii* infection and that the MHC-II-dependent mechanism of protection partially depends on Tbet, CD4+ T cells, and interferon gamma. Additionally, this study highlights differences in the primary and secondary immune response, which should be considered when designing future vaccines against Q fever.

Poor B Cell Memory May Contribute to Recurrent *Clostridioides difficile* Infection

In this study, Amadou Amani and colleagues (e00829-19) use a murine model of recurrent *Clostridioides difficile* disease to demonstrate that initial and repeat infections are equally severe and associated with poor establishment of toxin- or bacteria-specific antibody and B cell memory. In contrast, subcutaneous immunization established protective antibody and B cell memory. However, infection was unable to restimulate the immunization-induced B cell memory compartment. These results may have implications for understanding *C. difficile* disease recurrence and potential limitations of vaccination.

The Twin-Arginine Translocation System of *Citrobacter rodentium* Plays an Important Role in Gut Infection

Enteric bacterial pathogens elicit gut inflammatory responses and colonize the gut lumen. Otake et al. (e00892-19) show that the twin-arginine translocation (Tat) system is involved in the gut infection with *Citrobacter rodentium*, a murine enteric pathogen. Mouse infection experiments demonstrate that the Tat mutant of *C. rodentium* displays reduced inflammatory responses, resulting in prolonged gut colonization. This mutant also becomes hypersensitive to bile acids. Furthermore, increased fecal levels of bile acids foster *C. rodentium* clearance from the gut lumen. These data indicate that the Tat system and luminal bile acids might be promising therapeutic targets for infections with enteric pathogens.