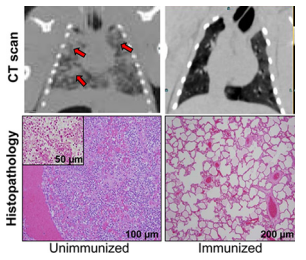
AMERICAN SOCIETY FOR MICROBIOLOGY | Clinical and Vaccine Immunology

Articles of Significant Interest Selected from This Issue by the Editors

New Vaccine against the Pneumonic Form of Plague

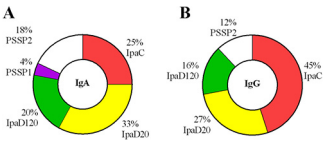
By using a replication-defective adenovirus vector system, Sha et al. (p. 586–600) introduced three protective antigen-encoding genes of *Yersinia pestis* into the viral genome. One dose of the vaccine administered by the intranasal route followed by a booster of the recombinant fusion protein consisting of these antigens provided complete protection to mice and nonhuman primates against a stringent dose of the aerosolized plague bacterium. During vaccination and after subsequent challenge with *Y. pestis*, animals did not exhibit any clinical symptoms of the disease or show histopathological lesions in various organs. The plague bacterium cleared rapidly from the blood after infection of vaccinated animals.



Adenovirus-based vaccine provides protection against pneumonic plague.

Acute Shigellosis Induces Differential Gut-Derived Antibody-Secreting Cell Responses against *Shigella* Common Protein Antigens

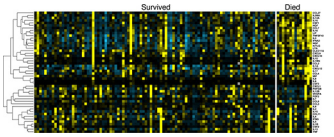
Gut-seeking ($\alpha_4\beta_7^+$) antibody-secreting cell (ASC) responses to the highly conserved invasive plasmid antigens IpaC and IpaD were detected in the majority of patients with recent-onset shigellosis. However, in the paper by Sinha et al. (p. 610–617), few if any ASC responses were observed against the recently identified pan-*Shigella* surface protein 1 (PSSP1), common to all *Shigella* species and found to be cross-protective in animal studies. These findings suggest that gut antibody responses to conserved *Shigella* antigens may wane too rapidly and/or be of insufficient magnitude to be protective against clinical disease.



Gut-derived IgA and IgG ASC responses to *Shigella* conserved protein antigens in blood from patients with recent-onset shigellosis.

Cytokine Signatures of *Salmonella* Sepsis in African Children

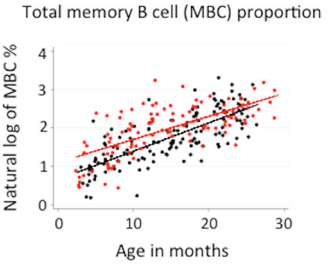
Nontyphoidal *Salmonella* (NTS) is a leading cause of sepsis and death in African children. In this study, Gilchrist et al. (p. 601–609) use multiplexed immunoassays to describe serum cytokine profiles in Malawian children with culture-proven NTS bacteremia. Cytokine signatures associated with acute NTS bacteremia were largely unaltered by common NTS-associated comorbidities (HIV infection and severe malnutrition) but were significantly perturbed in children who subsequently died during their hospital admission. The cytokine profile associated with mortality is defined by increased concentrations of molecules associated with neutrophil migration and function, highlighting the key role of these cells in NTS pathophysiology.



Heat map comparison of cytokine profiles in fatal and nonfatal NTS sepsis.

Delayed Accumulation of Memory B Cells in HIV-Exposed Uninfected Infants Does Not Alter Recall and Antibody Responses

HIV-exposed uninfected (HEU) infants born to mothers who are HIV infected often experience increased mortality and morbidity. Exposure to HIV antigens, antiretroviral drugs, or an altered placental cytokine environment may alter their developing immunity, predisposing them to postnatal infections. In this study, Nduati et al. (p. 576–585) describe B-cell phenotypes and function in HEU infants during the first 2 years of life. The results show that HIV exposure is associated with a lower proportion of memory B cells. This, however, did not affect the infants' ability to generate recall responses to previously encountered antigens or reduce their antigen-specific antibody levels at 18 months.



Delayed accumulation of memory B cells in HEU infants (red dots, HIV-unexposed uninfected infants born to HIV-uninfected mothers; black dots, HEU infants).

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