IN BRIEF

VIROLOGY

Expression of the 1918 influenza A virus PB1-F2 enhances the pathogenesis of viral and secondary bacterial pneumonia

McAuley, J. L. *et al. Cell Host Microbe* **2**, 240–249 (2007)

Why was the 1918 influenza A virus so remarkably virulent and strongly associated with secondary bacterial disease? McAuley and colleagues examined the contribution of the viral accessory protein PB1-F2. They showed that PB1-F2 enhances inflammation during primary viral infection and facilitates secondary bacterial infections in mice. An isogenic strain of the virus, which was engineered to express the PB1-F2 protein from the 1918 pandemic strain, was compared with a wild-type strain in mice, and was found to be more virulent, induce more pulmonary immunopathology and lead to more severe secondary bacterial pneumonia. These results help to explain the virulence of the 1918 strain and the high incidence of fatal pneumonia during the pandemic.

BACTERIAL PATHOGENESIS

Intestinal adherence associated with type IV pili of enterohemorrhagic *Escherichia coli* O157:H7

Xicohtencatl-Cortes, J. *et al. J. Clin. Invest.* 18 Oct 2007 (doi:10.1172/JCI30727)

The only factor that has been shown to have a role in the adherence of enterohaemorrhagic *Escherichia coli* (EHEC) to intestinal epithelial cells is intimin, an outer-membrane protein adhesin. Here, the authors examined the role of pili in EHEC O157:H7 colonization of the intestine. They found that EHEC O157:H7 produces and assembles the type IV pilin subunit HcpA into long bundles of pili, which the authors call haemorrhagic coli pilus (HCP). HCP were found to form physical bridges between bacteria that adhered to human and bovine host cells, and were involved in the adherence of EHEC O157:H7 to various intestinal and nonintestinal cell lines, epithelial cells and gut explants. In addition, HCP-mediated adherence and cytotoxicity were independent events. These results show that EHEC O157:H7 HCP are intestinal colonization factors.

VACCINES

Adjuvanting a DNA vaccine with a TLR9 ligand plus Flt3 ligand results in enhanced cellular immunity against the simian immunodeficiency virus

Kwissa, M. *et al. J. Exp. Med.* **204**, 2733–2746 (2007)

In this report, Kwissa and colleagues examined the efficacy of Toll-like receptor (TLR) ligands on augmenting the immunogenicity of a DNA prime–boost vaccine against simian immunodeficiency virus (SIV). They injected rhesus macaques with FMS-like tyrosine kinase 3 ligand (FLT3L) to expand dendritic cells (DCs), and primed them with a DNA vaccine encoding immunodeficiency virus antigens mixed with ligands for TLR9 or TLR7 and TLR8. The animals were then boosted with DNA and twice with recombinant modified vaccinia virus Ankara that expressed the same antigens. Activating DCs with TLR9 ligand during the initial immunization with a DNA vaccine resulted in an enhanced antigen-specific $CD8^+$ T-cell response and improved control of viral loads after challenge with SIV.