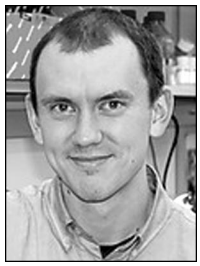
# Journal Highlights

## Just One Dose of Clindamycin Wreaks Havoc on Intestinal Flora

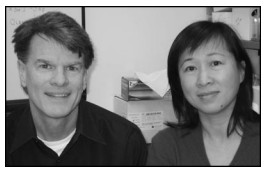


Buffie

Yet another study adds to the growing evidence that antibiotics can disrupt the balance of the intestinal flora. Eric Pamer, Charlie Buffie, and Joao Xavier of the Memorial Sloan Kettering Cancer Center, New York City, show that a single dose of the commonly used antibiotic clindamycin resulted in the loss in mouse models of roughly 90% of bacterial taxa and that these changes lasted for the experiment's duration, 28 days. "In line with this long-lasting effect of a single dose of antibiotic on the intestinal flora, mice remain susceptible to infection with *Clostrodium difficile* for at least 10 days after antibiotic treatment," says Pamer. "Our analyses also demonstrate that bacterial taxa that constitute only a small minority of the microbiota prior to antibiotic treatment become dominant members of the microbiota after clindamycin treatment, and that the composition of microbiota becomes unstable in the aftermath of antibiotic treatment. The impact of antibiotics on the normal commensal flora, which provides an important layer of protection against pathogens, is underappreciated." Pamer plans to use deep sequencing platforms to discover bacterial taxa that can enhance resistance to intestinal infection.

(C. G. Buffie, I. Jarchum, M. Equinda, L. Lipuma, A. Gobourne, A. Viale, C. Ubeda-Morant, J. Xavier, and E. G. Pamer. 2011. Profound alterations of intestinal microbiota following a single dose of clindamycin results in sustained susceptibility to *Clostridium difficile*-induced colitis. Infect. Immun. 80:62–73.)

## H5N1 Virus Targets Pulmonary Endothelial Cells



Tumpey (l) and Zeng

At roughly 60%, the case fatality rate for H5N1 virus infections in humans is orders of magnitude higher than that of seasonal influenza virus infections. Respiratory failure due to acute respiratory distress syndrome is a complication among severe H5N1 cases. Hui Zeng of the Centers for Disease Control and Prevention, Atlanta, Ga., et al. show that H5N1 virus, but not seasonal influenza viruses, can target human pulmonary endothelial cells for efficient replication and rapid induction of proinflammatory mediators. This contributes to the heavy viral load, reduced cell viability, and an overwhelming immune response, which correlates with H5N1-induced acute respiratory distress syndrome observed in humans. Zeng says that further characterization of viral factors associated with H5N1-induced pulmonary endothelial injury will lead to a better understanding of the clinical course of H5N1 virus infection in humans.

(H. Zeng, C. Pappas, J. A. Belser, K. V. Houser, W. Zhong, D. A. Wadford, T. Stevens, R. Balczon, J. M. Katz, and T. M. Tumpey. 2011. Human pulmonary microvascular endothelial cells support productive replication of highly pathogenic avian influenza viruses: possible involvement in the pathogenesis of human H5N1 virus infection. J. Virol. 86:667–678.)

ASM Articles in the Spotlight. The ASM journals *Eukaryotic Cell*, *Infection and Immunity*, the *Journal of Virology*, and *Molecular and Cellular Biology* highlight current articles of interest in their Spotlight sections:

http://ec.asm.org/current.dtl#SPOTLIGHT
http://iai.asm.org/current.dtl#SPOTLIGHT
http://jvi.asm.org/current.dtl#SPOTLIGHT
http://mcb.asm.org/current.dtl#SPOTLIGHT