IN BRIEF

STRUCTURAL BIOLOGY

The 3-dimensional structure of a hepatitis C virus p7 ion channel by electron microscopy

Luik, P. *et al. Proc. Natl Acad. Sci. USA* **106**, 12712–12716 (2009)

Viroporins are small, hydrophobic proteins that are encoded by many enveloped RNA viruses. The 42 kDa, hexameric p7 viroporin is essential for the release of infectious hepatitis C virus particles *in vivo* and, as such, has been identified as a potentially important new drug target. Luik *et al.* determined the three-dimensional structure of the complete p7 ion channel at 16 Å resolution by single-particle electron microscopy using random conical tilt reconstruction. The channel complex adopts a conical, flower-shaped conformation with six distinct 'petals' branching off from a compact base, which creates a wide pore at the top of the channel that the authors calculated should face the lumen of the endoplasmic reticulum.

BACTERIAL PATHOGENICITY

Antibiotic treatment of *Clostridium difficile* carrier mice triggers a supershedder state, spore-mediated transmission, and severe disease in immunocompromised hosts

Lawley, T. D. *et al. Infect. Immun.* 29 Jun 2009 (doi:10.1128/IAI.00558-09)

In recent years, *Clostridium difficile* has become one of the most important causes of health care-associated infection. The infection is spread through the excretion of highly resistant spores by infected individuals. Trevor Lawley and colleagues now report a new mouse model of the *C. difficile* transmission and infection cycle. The *C. difficile* strain M68, which is virulent in humans, establishes a persistent, asymptomatic infection in mice. Although carrier mice constantly shed low levels of *C. difficile* spores, they are not contagious. However, the authors found that the treatment of carrier mice with the antibiotic clindamycin induced a supershedder state, in which there was a 10^6-fold increase in *C. difficile* shedding levels. Currently, infection control efforts are mainly focused on symptomatic patients; these results suggest that "…it may be prudent to monitor patients undergoing treatment with antibiotics for evidence of *C. difficile* spore shedding."

QUORUM SENSING

A quorum-sensing antagonist targets both membrane-bound and cytoplasmic receptors and controls bacterial pathogenicity

Swem, L. R. *et al. Mol. Cell* **35**, 143–153 (2009)

One potential new antimicrobial therapy that has generated much interest is the disruption of quorum sensing. In Gram-negative bacteria, which use acyl homoserine lactones (AHLs) for quorum sensing, AHL receptors can be cytoplasmic (belonging to the LuxR family) or membrane bound (belonging to the LuxN family). Previously, 15 small-molecule antagonists of the *Vibrio harveyi* membrane-bound LuxN receptor had been identified. In a recent issue of *Molecular Cell*, Bassler and colleagues now report that one of these molecules can also antagonize CviR, a LuxR-type cytoplasmic receptor from *Chromobacterium violaceum*. This antagonist was able to protect against *C. violaceum* quorum sensing-dependent killing in a *Caenorhabditis elegans* infection model. The authors conclude that their results "…make a strong case … that an anti-quorum-sensing strategy is a valid alternative to traditional antibiotics for Gram-negative bacteria …".