IN BRIEF

BACTERIAL PHYSIOLOGY

Hip persisters

Persisters are dormant bacterial populations that contribute to multidrug tolerance (MDT) because they are able to escape treatment with most antibiotics, which target metabolically active cells. A new study now characterizes the molecular basis for persister generation involving HipA, a kinase that inhibits protein synthesis and induces dormancy in *Escherichia coli*. HipA forms a toxin–antitoxin pair with HipB, and together they bind to multiple operator sites within the *hipBA* promoter to form a large complex. By resolving the structure of this complex, the authors found that HipA forms dimers via interactions between operator-adjacent HipA–HipB pairs and that dimerization blocks the active site of HipA. Notably, mutations in the dimerization domain were found in *E. coli* isolates causing urinary tract infections and conferred MDT; these mutations are thought to prevent dimerization, allowing HipA to remain active, which in turn induces the generation of persisters.

ORIGINAL RESEARCH PAPER Schumacher, M. A. *et al.* HipBA–promoter structures reveal the basis of heritable multidrug tolerance. *Nature* http://dx.doi.org/10.1038/nature14662 (2015)

BACTERIAL TOXINS

Toxins actin(g) up

Several bacterial pathogens, including *Vibrio cholerae*, use toxins containing actin crosslinking domains (ACDs) to interfere with the ability of the host cell to polymerize actin. The toxicity of ACD-containing toxins is thought to arise via sequestration of bulk amounts of actin as non-functional oligomers, which ultimately results in failure of the cytoskeleton. Now, a new study shows that ACD-containing toxins also work by converting actin into a toxic oligomer that interferes with formins, which are a family of proteins involved in the nucleation and elongation of actin filaments. The authors show that ACD-modified actin filaments bind with high affinity to formins and inhibit their activity. These data suggest that ACD-containing toxins act not only by sequestering actin but also by 'poisoning' proteins involved in actin polymerization.

ORIGINAL RESEARCH PAPER Heisler, D. B. *et al.* ACD toxin-produced actin oligomers poison formin-controlled actin polymerization. *Science* **349**, 535–539 (2015)

VACCINES

A protective Ebola vaccine

The Ebola epidemic in West Africa is still ongoing, but a new study reports that a ring vaccination strategy — in which contacts, and contacts of contacts, of an infected patient are vaccinated — using rVSV-ZEBOV was successful in preventing infection. The vaccine consists of a live-attenuated recombinant vesicular stomatitis virus (rVSV) expressing Ebolavirus (EBOV) glycoproteins and was administered to two groups in Guinea: the 'immediate' group (2,104 individuals) received the vaccine shortly after the original patient developed Ebola; and the 'delayed' group (2,380 individuals) was vaccinated 3 weeks after that event. After 10 days of vaccination, none of the vaccinees in the 'immediate' group developed Ebola, whereas 16 individuals from the 'delayed' group developed the disease. The 10-day cut-off was used to account for the incubation time of EBOV and to allow adaptive immune responses induced by the vaccine to develop. These data suggest that this vaccination strategy may be highly protective and could be useful in controlling Ebola outbreaks.

ORIGINAL RESEARCH PAPER Henao-Restrepo, A. M. *et al.* Efficacy and effectiveness of an rVSV-vectored vaccine expressing Ebola surface glycoprotein: interim results from the Guinea ring vaccination cluster-randomised trial. *Lancet* http://dx.doi.org/10.1016/S0140-6736(15)61117-5 (2015)