Molecular Microbiology (2012) 85(2), 195–200 ■
doi:10.1111/j.1365-2958.2012.08119.x
First published online 14 June 2012

MicroCommentary

Multiple lessons from the multiple functions of a regulator of type III secretion system assembly in the plant pathogen *Pseudomonas syringae*

Hai-Lei Wei and Alan Collmer*
Department of Plant Pathology and Plant-Microbe Biology, Cornell University, Ithaca, NY 14853, USA.

Summary

The assembly of type III secretion systems (T3SSs), which inject bacterial effector proteins into the cytosol of animal and plant hosts, is a highly regulated process. Animal pathogens use a length-control protein to produce T3SS needles of fixed length and then a second regulator, such as YopN in *Yersinia* spp, to mediate host contact-dependent effector delivery. For *Pseudomonas syringae* and other plant pathogens, regulation of the assembly process differs because the T3SS pilus must grow through variably thick plant cell walls before contacting the host plasma membrane. In this issue of *Molecular Microbiology*, Crabill *et al*. (2012) report evidence that the YopN homologue HrpJ is a multifunctional regulator of T3SS assembly in DC3000. A *hrpJ* mutant hyper-secretes pilus protein and no longer secretes four translocator proteins in culture, and it fails to inject effectors *in planta*. As with other proteins in this class, HrpJ is itself a T3SS substrate, but secretion-incompetent forms retain regulatory function. However, HrpJ is unusual in suppressing innate immune responses within host cells, as demonstrated with transgenic plants. The multiple capabilities of HrpJ appear to couple host contact sensing with pilus length control and translocator secretion while also contributing to immunity suppression early in the interaction.

Type III secretion systems (T3SSs) are essential to the virulence of many Gram-negative bacterial pathogens of plants and animals (Cornelis, 2006). T3SSs inject large repertoires of effector proteins that suppress innate immunity and otherwise remodel host cells for pathogen benefit. T3SS injectisomes can be divided into three functional modules (Enninga and Rosenshine, 2009; Deane *et al*., 2010). The first is a basal body that spans the inner and outer membranes and shares conserved components in plant and animal pathogen T3SSs and flagellar biogenesis systems; the second is a needle/pilus that serves as a conduit for effector transfer; and the third is a translocon complex that forms a pore in the host plasma membrane. Needle/pilus and translocon proteins travel through the basal body in all T3SSs, but their identity, assembly and associated regulators show fundamental differences between animal and plant pathogens (Buttner and He, 2009; Deane *et al*., 2010). The necessity for the plant pathogen pilus to deliver effectors through the plant cell wall likely underlies these differences and also complicates study of the T3SS assembly process in these pathogens.

Extensive work with animal pathogens in the genera *Yersinia*, *Salmonella*, *Shigella* and *Escherichia* has revealed two major steps and associated regulators governing assembly of the extracellular T3SS modules (Deane *et al*., 2010). The first step is assembly of the needle, whose respective lengths in these bacteria are determined by the length-control proteins YscP, InvJ (SPI-1 T3SS), Spa32 and Orf16 respectively (Deane *et al*., 2010). This step is followed by the assembly of the translocon complex, which is comprised of a tip protein and two pore proteins (LcrV and YopB/D, respectively, in *Yersina*) and entry into a poised/standby mode until contact with host cells relieves a block on effector delivery (Enninga and Rosenshine, 2009; Deane *et al*., 2010). The standby mode is controlled by the gatekeeper proteins YopN/TyeA, InvE, MxiC and SepL, respectively, in these pathogens. Representatives of the proteins regulating both of these steps in T3SS assembly have been shown to travel the T3SS themselves, although export may not be universal or required for function (Deane *et al*., 2010).

The mechanism by which needle length is controlled is an active area of investigation. YscP has been proposed to act as a molecular ruler whose function depends on

Accepted 25 May, 2012. *For correspondence. E-mail arc2@cornell.edu; Tel. (+1) 607 255 7843; Fax (+1) 607 255 4471.