

Sainsbury Laboratory Research Update

An evolutionary arms race between bacterial virulence proteins and plant defence mechanisms.

Two early career researchers from the Sainsbury laboratory have recently published work which increases our understanding of the battle fought between plants and their potential pathogens at the molecular level.

Vardis Ntoukakis and Selena Gimenez-Ibanez, working in John Rathjen's group, studied the bacterial pathogen *Pseudomonas syringae* pv. *tomato* which causes bacterial speck disease on susceptible tomato cultivars.

Bacteria produce a range of effectors; protein molecules which contribute to virulence in the plant. In susceptible cultivars, these effectors can block the plant defence response by attacking the host signalling machinery. For example, AvrPtoB, an effector from *Pseudomonas syringae*, targets the plant receptor FLS2, which recognises bacterial flagellin.

Plants can also recognise the presence of potential fungal pathogens. The Arabidopsis receptor CERK1, for example, recognises chitin from fungal cell walls. Work by Gimenez-Ibanez *et al*, recently published in Current Biology, showed that, in addition to its role in fungal perception, CERK1 contributes to restricting growth of bacteria in *Arabidopsis*. This is a newly-discovered pathway for plant immunity against bacteria. Bacteria expressing AvrPtoB, however, are able to suppress this CERK1-mediated immunity. The effector targets CERK1 and adds ubiquitin residues, thus tagging the protein for degradation. This discovery reveals a role for AvrPtoB in suppressing plant basal defenses.

These molecular interactions can be understood as an evolutionary "arms race", in which the plant evolves receptors to recognise potential pathogens and the pathogens evolve ways to evade recognition or suppress defence. The case of CERK1 is interesting in that a single receptor in the plant is being used to recognise both bacteria and fungi. In the interaction between CERK1 and fungi, the plant recognises fungal pathogens and mounts a defence response, thus the plant wins and the fungi lose. However, bacteria have evolved the effector AvrPtoB, to overcome recognition by both CERK1 and FLS2, so in these interactions the bacteria win the race.

Resistant plant cultivars have evolved to recognise effectors responsible for suppression of basal defenses, leading to a second layer of defense known as effector-triggered immunity. This type of immunity is often cultivar specific. One example of this is the recognition of AvrPtoB in tomatoes containing the protein kinase Pto.

AvrPtoB has been shown to interact *in vitro* with tomato protein kinases such as Fen and Pto. Whilst Fen becomes tagged for degradation via ubiquitination, Pto resists degradation and in this case, the interaction of AvrPtoB with Pto (together with an associated resistance protein, Prf) leads to the activation of immunity. Work published in Science by Ntoukakis *et al*

showed that Pto resists degradation by recognising and phosphorylating AvrPtoB. This inactivation of the virulence protein allows tomato cultivars containing Pto to resist disease. Thus, tomatoes have evolved a mechanism to counteract the action of AvrPtoB and this second layer of defence enables them to win the arms race – for the time being.

These papers illustrate the complexity of the molecular battles going on all the time between plants and pathogens in the field. Much remains to be discovered in this exciting field of research.

AvrPtoB targets the LysM receptor kinase CERK1 to promote bacterial virulence on plants.

Gimenez-Ibanez S, Hann DR, Ntoukakis V, Petutschnig E, Lipka V, Rathjen JP.
Curr Biol. 2009 Mar 10;19(5):423-9.

Host Inhibition of a Bacterial Virulence Effector Triggers Immunity to Infection

Ntoukakis V, Mucyn TS, Gimenez-Ibanez S, Chapman HC, Gutierrez JR, Balmuth AL, Jones AME, Rathjen JP
Science 2009 May 8; 324 (5928): 784-7