The genetics of host–pathogen interaction

At a recent meeting of the Genetical Society*, the intimate relationships between pathogens and their hosts were explored in detail, both in plant and in animal models. It was apparent that, in terms of the sophistication and depth of our knowledge, and the power of the tools available to the investigator, plants can have great advantages as model systems. The following brief summary highlights a few examples of the talks presented to illustrate just how far these studies have progressed.

Avirulence and resistance

In many plant–pathogen interactions, the rapid onset of a plant defence response relies on a recognition process controlled by avirulence (Avr) genes in the pathogen and the corresponding resistance (R) genes in the plant. This kind of gene-for-gene interaction is something that animal geneticists can only dream of! Most of the R genes, which are often extremely polymorphic, encode putative receptor proteins that contain nucleotide-binding/leucine-rich repeats, which share homology with gene products involved in apoptotic cell death in animals (including Apaf-1 and Ced4). Jonathan Jones (John Innes Centre, Norwich, UK) is working with RPP genes in Arabidopsis [conferring resistance to downy mildew (Peronospora parasitica)] and Cf genes in tomato (conferring resistance to Cladosporium fulvum). He described the wealth of knowledge that is being accumulated about these loci and how this polymorphism is sustained by frequency-dependent selection. This mechanism involves the slow shuffle of a patchwork of sequence fingerprints between the resistance gene family members – ‘trench warfare’ rather than an ‘arms race’, which involves gene elimination.

As part of the plant’s response to infection, the interaction between the Avr and R gene products triggers a metabolic switch that leads to salicylic acid accumulation, the biosynthesis of pathogenesis-related (PR) proteins and the initiation of systemic acquired resistance (SAR). Jeff Dangl (The University of North Carolina, NC, USA) is looking at the interaction between Pseudomonas syringae and Arabidopsis, described a ‘forward genetics’ expression-profiling method that accelerates pathway-specific gene discovery. Global gene expression changes were monitored in Arabidopsis under 14 different SAR-inducing or SAR-repressing conditions using a DNA microarray of 10 000 ESTs. The results (a group of genes with a common regulation pattern) containing PR1, a reliable marker for SAR induction in Arabidopsis, also contained other genes that might be involved in SAR and infection. These genes contain a common promoter element that is recognized by members of a plant-specific family of transcription factors, the WRKY proteins1.

Gene silencing and pathogen defence

The introduction of a DNA sequence that is homologous to a plant gene causes post-transcriptional gene silencing (PTGS; Fig. 1), which is manifested as a reduction in the steady-state level of the specific mRNA involved, probably by some kind of RNA-interference, although the exact mechanism is unknown. David Baulcombe (John Innes Centre, Norwich, UK) illustrated his group’s current working model, showing that this RNA-mediated defence is initiated by the presence of ~25-nucleotide double-stranded RNA in the infected cell, and given specificity by an antisense RNA molecule that is targeted against the viral genome. This defence involves the systemic spread of a silencing signal that directs sequence-specific RNA degradation2.

Many RNA and DNA viruses encode pathogenicity components that suppress this defence mechanism, and it appears that there has been dynamic co-evolution of the genes encoding the host and viral components involved. PTGS can be monitored easily by infecting plants that have already been transformed with green fluorescent protein (GFP) with Agrobacterium tumefaciens carrying Ti plasmids containing a GFP-promoter insert. The characterization of mutants that are impaired in PTGS will further enable the identification of host components that interact with viral suppressors.

Virus-induced gene silencing (VIGS)3 is being used as a functional genomics tool in forward genetics screens to look for the genes required for resistance responses, revealing components that act downstream of the R genes, for instance, in N-mediated resistance to tobacco mosaic virus (TMV). VIGS exploits the fact that the expression of most plant genes can be suppressed by infecting the plant with virus vectors carrying the corresponding mRNAs. By cloning a normalized Nicotiana benthamiana cDNA library into a potato virus vector and then inoculating individual clones into TMV-resistant plants, loss of resistance can be screened for by subsequent inoculation with GFP-tagged TMV (which will accumulate in non-resistant plants). This powerful, high-throughput technique has already revealed three novel defence-related genes.

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Meeting report

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References


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Fig. 1. Nicotiana benthamiana infected with potato virus X, carrying an insert based on an unknown endogenous gene. The infection initiates virus-induced gene silencing that is targeted against the host gene corresponding to the insert. The symptoms on the plant are equivalent to a loss of function phenotype of the corresponding endogenous gene – akin to ‘forward epigenetics’.

Photograph courtesy of David Baulcombe.