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Use of *Arabidopsis thaliana* and *Pseudomonas syringae* in the Study of Plant Disease Resistance and Tolerance[1]

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Abstract: The interaction between *Arabidopsis thaliana* and the bacterium *Pseudomonas syringae* is being developed as a model experimental system for plant pathology research. Race-specific ("gene-for-gene") resistance has been demonstrated for this interaction, and pathogen genes that determine avirulence have been isolated and characterized. Because certain lines of both *Arabidopsis* and soybean are resistant to bacteria carrying the avirulence genes *avrRpt2* and *avrB*, extremely similar pathogen recognition mechanisms are apparently present in these two plant species. Isogenic bacterial strains that differ by the presence of single avirulence genes are being used to analyze plant resistance. Plant resistance genes have been identified in crosses between resistant and susceptible lines. The extensive map-based cloning tools available in *Arabidopsis* are being used to isolate these resistance genes. In a related project, ethylene-insensitive *Arabidopsis* mutants are being used to examine the role of ethylene in disease development. Ethylene apparently mediates symptom formation in susceptible plants and is not required for resistance, suggesting possible strategies for enhancement of disease tolerance in crops.

Key words: Arabidopsis thaliana, avirulence, bacterium, ethylene, *Glycine max, Pseudomonas syringae,* resistance, tolerance.

Research concerning plant–pathogen interactions has frequently focused on economically important crop plants and their pathogens. This bias has many positive attributes. For example, a synergistic effect can occur when many researchers investigate a given organism. The clustering of human and financial resources around these economically relevant interactions leads to a broad knowledge base. In addition, the knowledge gained, while not always of immediate practical value, applies directly to a significant agricultural problem. The primary shortcoming in study of these organisms arises if they prove to be cumbersome for experimentation.

As an alternative, researchers in diverse areas of biology often study "model organisms" to expedite experimental progress. *Escherichia coli, Saccharomyces cerevisiae, Drosophila melanogaster,* and *Mus muscula* are the most prominent examples. The synergism of widespread attention occurs for these organisms as well, and because of their experimental suitability gains tend to come rapidly. Moreover, practical advances are spurred through adoption of basic research findings that, for instance in medicine, would not be available if researchers restricted their attention to humans and human pathogens. As long as the scientific and technical community as a whole continues to study a wide array of organisms, a focus on model organisms should continue to be a highly productive research strategy.

Organisms that have more recently achieved prominence as research models include the nematode *Caenorhabditis elegans* and the small cruciferous plant *Arabidopsis thaliana.* Several workers, including ourselves, are using *Arabidopsis* to study interactions with various viral, bacterial, fungal, and nematode plant pathogens (9,16,18, 25,26,28,30). Our primary interest is the molecular basis of specificity during recognition of pathogens by resistant plants. To maximize our ability to manipulate both the host and the pathogen genetically, Gram-negative bacterial pathogens are be-

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ing utilized. Using this approach, we have isolated and characterized single pathogen genes (avirulence genes) that affect virulence in a host-genotype-dependent manner. We have also identified corresponding plant loci that control avirulence-gene-dependent resistance and are attempting to isolate these genes. Furthermore, we have utilized this experimental system to examine the role of the plant hormone ethylene in plant disease development.

Gene-for-Gene Disease Resistance

One of the most common forms of plant disease resistance is gene-for-gene resistance, also known as major gene resistance, qualitative resistance, or race-specific resistance (4,11,15). Hallmarks of this form of resistance are i) the central role played by single dominant resistance genes in the host and single dominant avirulence genes in the pathogen, ii) the functional specificity of these gene pairs (i.e., a given resistance gene is generally effective against a single pathogen avirulence gene only), and iii) the induction of active resistance responses, in particular the hypersensitive response. Gene-for-gene interactions have been identified in resistance to viruses, bacteria, fungi, and nematodes (4,15,29). Resistance genes have been deployed in crop lines, not only because of their convenience for breeding (because they are single Mendelian loci), but also because of the strong nature of the resistance response they control.

Evidence is accumulating to support an elicitor–receptor mechanism of gene-for-gene resistance. In this model (4,15,29), the action of an avirulence gene causes the pathogen to be recognizable due to its production of extracellular "elicitor" molecules. Plants are resistant if they contain a resistance gene with functional specificity for the given pathogen avirulence gene; resistance genes are frequently postulated to control production of a receptor with specificity for the elicitor produced by avirulence gene action. Pathogen avirulence genes from viruses, bacteria, and fungi have been cloned and characterized, and in some cases the molecular species responsible for host-genotype-specific elicitation have been identified (5,6, N. Keen, pers. comm.). No gene-for-gene plant resistance genes have been isolated, however, and a significant effort is now underway in many labs to accomplish this task. In addition, the signal transduction mechanisms that activate defense responses following pathogen recognition remain essentially unknown and are the focus of current attention.

Choice of Plant and Pathogen

We selected *Arabidopsis* for the reasons alluded to in the introductory section. The positive attributes of *Arabidopsis* have been extensively reviewed (19,20) and include rapid generation time, small physical size, self-fertility with outcrossing by hand pollination, plentiful and easily harvested seed set, small genome size, and low occurrence of repetitive DNA. *Arabidopsis* tissues are well suited for nondisruptive microscopy because of their relative transparency (25). In addition, numerous mutant lines have been isolated and characterized (21). Extensive genetic maps (based on morphological markers and on restriction fragment length polymorphism [RFLP], random amplified polymorphic DNA [RAPD], and functionally characterized cDNA markers) have been developed (3, 17,22,23). Yeast artificial chromosome (YAC) and cosmid genomic DNA libraries are available and are being ordered with respect to the genetic map to expedite map-based gene cloning (13). Most important, the large and interactive community of *Arabidopsis* researchers is constantly generating new resources and expertise.

In order to maximize our ability to manipulate both plant and pathogen, our search for a suitable *Arabidopsis* pathogen focused on Gram-negative bacterial species. Many of the molecular genetic methods developed for *E. coli* can be utilized with these bacteria. *Pseudomonas syringae* bacteria are the causal agents of a variety

of leaf-spotting diseases, and individual pathovars typically exhibit a high degree of host specificity (24). Strains from *P. syringae* pvs. *tomato* and *maculicola* were identified that exhibited strong virulence on *Arabidopsis* (31).

Survey of Naturally Occurring Isolates for Disease Outcome

In order to study disease resistance, identification of virulent and avirulent bacterial strains and resistant and susceptible host lines was necessary. Of course a single plant line can be resistant to some strains and susceptible to others, and bacterial strains can be virulent on some plant lines and avirulent on others. We were also interested in learning about the extent of natural variation of resistance and avirulence specificity. More than 30 *P. syringae* pv. *tomato* strains were surveyed, and over 30 *Arabidopsis* ecotypes (isolates from natural populations) were evaluated. At least three different race-level resistance specificities were identified in a survey that did not exhaust even half of all possible strain–ecotype combinations (31).

Pathogen Avirulence Genes

Initial isolation and characterization: Having identified virulent and avirulent pathogen strains, we next attempted to isolate a single gene that could convert a virulent strain to an avirulent one on specific host genotypes. This problem was approached by the standard method of functional complementation (27). A genomic library was made using DNA from the *P. syringae* pv. *tomato* strain 1065 (avirulent on *Arabidopsis* ecotype Col-0), and individual cosmid clones from the library were moved into the *P. syringae* pv. *tomato* strain DC3000 (virulent on Col-0). These exconjugant DC3000 strains were then inoculated into *Arabidopsis* ecotype Col-0 leaves; one of the few hundred cosmids tested conferred a clearly avirulent phenotype. The locus carried on this cosmid was given the name *avrRpt2* (31). Interestingly, the *avrRpt2* locus was independently isolated two other times in a survey of a genomic library made from the *P. syringae* pv. *tomato* strain T1. Table 1 lists important aspects of the avirulent phenotype conferred by *avrRpt2* (note in particular the host-genotype-specificity of the avirulence phenotypes).

The *avrRpt2* locus was further localized to a 1.4-kb stretch of DNA, and we determined that an insertional disruption in the middle of this region eliminated phenotypic expression of *avrRpt2* (31). More recently, the *avrRpt2* region was sequenced and found to contain a single large open reading frame (Innes, Bent, Kunkel, Bisgrove, and Staskawicz, unpublished). Expression of mRNA from this open reading frame was also demonstrated. The DNA sequence of *avrRpt2* did not contain significant similarity to sequences present in the Genbank or EMBL sequence databases, but sequences immediately 5′ to the putative start of *avrRpt2* transcription shared extensive homology with the so called "*hrp box*" regulatory element found upstream of other *P. syringae* avirulence genes and pathogenicity genes (10,14). Transcription of *avrRpt2* was found to be dependent on the same *hrp* loci that control expression of other genes carrying the 5′ *hrp* box sequence (10, Innes, Bent, Kunkel, Bisgrove, and Staskawicz, unpubl.).

Similarities to resistance in soybean: Although avirulence genes are typically thought of as responsible for controlling host range within species at the cultivar level, evidence is accumulating that these genes can also limit host range at the spe-

Table 1. Phenotypes conferred by *avrRpt2* in originally virulent *Pseudomonas syringae* strains.

On resistant *Arabidopsis thaliana* genotypes:
Absence of visible disease lesions on leaves.
Induction of the plant hypersensitive response.
Fifty-fold or greater reduction of pathogen population size in leaves
On susceptible host genotypes (*A. thaliana*, tomato soybean):
Disease lesions produced.
No hypersensitive response.
No reduction of pathogen growth.

cies level (15). To explore this possibility, the cloned *avrRpt2* locus was moved into the soybean pathogen *P. syringae* pv. *glycinea*. In one set of tests, a race 4 strain of *P. syringae* pv. *glycinea* was virulent on four soybean cultivars, while race 4 carrying *avrRpt2* was virulent only on two of the four cultivars. This and other experiments revealed that *avrRpt2* functioned as an avirulence gene with respect to soybean (31), providing another demonstration that taxonomically diverse plant species can share resistance based on identical avirulence genes (15). This similarity between resistance in *Arabidopsis* and soybean is especially stimulating, as it suggests the possibility of using resistance genes identified in *Arabidopsis* to enhance the resistance of crop plants, either by direct transformation with the *Arabidopsis* gene or by using the *Arabidopsis* gene to identify similar resistance loci with different avirulence gene specificities.

Additional avirulence genes: One of the most important uses of cloned avirulence genes is in construction of genuinely isogenic pathogen lines, so that virulence or avirulence is governed by the presence or absence of a single gene. This attribute allows researchers to isolate variables (in our case, the causal factor that triggers plant responses) while still working with a true-to-life system involving live pathogens and intact plants.

We have learned that pathogens carrying the well-characterized avirulence gene *avrB* are also recognized by *Arabidopsis* (Innes, Bent, Bisgrove, and Staskawicz, unpubl.). Similarly, Dr. Jeff Dangl and his co-workers have identified a third avirulence gene, *avrRpm1*, that is recognized by *Arabidopsis* (7). The availability of these genes and *avrRpt2* places us in position to compare the transduction mechanisms triggered by three distinct recognitional determinants, and to study the respective plant genes required for resistance in each case.

Plant Resistance Genes

We have adopted two strategies for the identification of plant genes controlling resistance: screening of mutagenized populations derived from resistant plants to identify susceptible mutants, and analysis of naturally occurring *Arabidopsis* ecotypes to characterize diversity in resistance. For the first approach, refinement of screening methods was necessary. Inoculation by simple spraying of bacterial suspensions onto plants was found to be unreliable, because too many individuals escaped detectable infection. Manual introduction of bacteria into leaves with a plastic Pasteur pipette is extremely reliable, but prohibitively slow if thousands of plants are to be screened. The first inoculation method we developed was based on use of a wetting agent, the surfactant Silwet L-77 (Union Carbide, Danbury, CT; 31). This particular surfactant lowers surface tension sufficiently so that aqueous solutions will coat leaf surfaces instead of beading up and running off. Consistent inoculation of hundreds of plants in a single experiment was obtained by dipping leaf rosettes in a solution of bacteria and 0.01% L-77. With this approach, four independent mutants that had lost resistance to *Pseudomonas* strains carrying *avrRpt2* were identified in a screen of approximately 6,000 M2 seedlings derived from Col-0 seed mutagenized with diepoxybutane (Kunkel, Bent, Dahlbeck, Innes, and Staskawicz, unpubl.).

To facilitate continued isolation of recognition-response mutants, a novel selection assay developed by Drs. Guo-Liang Yu and Fred Ausubel was utilized. This method is based on the observation, first made by Dr. Michael Mindrinos, that certain *P. syringae* strains not pathogenic on *Arabidopsis* will still trigger a plant-hypersensitive response if these bacteria carry an avirulence gene recognized by the host genotype. In the selection assay, entire plantlets (grown in nutrient agar on petri plates) are infiltrated with a high concentration of these bacterial strains. Resistant plantlets undergo a systemic hypersensitive response and are killed, while plants that do not respond to the avirulence gene end-product survive because the bacteria used are nonpathogenic. Sev-

eral putative resistance mutants have been isolated using this method, and progeny from these plants are currently being retested. This rapid assay has also proven to be very useful for scoring segregating populations (e.g.,in F_3-progeny-testing for large F_2 populations).

One of the mutant lines identified in the L-77 based screen has been more extensively characterized. This line, derived from the mutant D203, carries an avirulence-gene-specific loss of resistance. The plants are fully susceptible to pathogen strains carrying *avrRpt2* (by the criteria listed in Table 1), but retain resistance to bacteria carrying *avrB* or *avrRpm1*. Experiments with the F_2 from crosses to wild-type parental lines revealed the presence of a single Mendelian locus controlling *avrRpt2*-specific resistance, and we named this locus *RPS2*. F_1 plants from Col-0 × D203 crosses were intermediately resistant, indicating that the resistance phenotype of *RPS2* is semi-dominant. Crosses to a separate resistant ecotype were utilized to place RPS2 relative to previously mapped RFLP markers, and we have determined a precise map position for this locus on *Arabidopsis* chromosome 4 (Kunkel, Bent, Dahlbeck, Innes, and Staskawicz, unpubl.). Efforts are underway to further exploit the extensive map-based cloning tools available for *Arabidopsis,* with our present effort focused on fine-structure mapping of recombinants and on identification and ordering of YAC clones with inserts that map to the *RPS2* region.

Our survey of *Arabidopsis* ecotypes also revealed lines deficient in the *RPS2* locus. The ecotype Wü-0 resembled mutant D203 in that plants were at least partially susceptible to *P. syringae* carrying *avrRpt2* but retained the capacity to express race-specific resistance against strains carrying *avrB* or *avrRpm1*. Complementation tests suggested that Wü-0 may also lack functional alleles at the *RPS2* locus (Kunkel, Bent, Dahlbeck, Innes, and Staskawicz, unpubl.).

Analysis of an additional ecotype, Po-1, has proven to be more complex. Po-1 lacks resistance to strains carrying *avrRpt2, avrB,* and *avrRpm1,* and Po-1 is also susceptible to a number of wild-type isolates of *P. syringae* pv. *tomato* that elicit resistance on *Arabidopsis* ecotype Col-0. Thus, Po-1 may carry a defect in a more pleiotropic function affecting race-specific resistance. This ecotype does retain competence for disease resistance, however, since a particular strain of *P. syringae* pv. *pisi* elicits a hypersensitive response in Po-1. Testing of F_1 progeny from crosses between Po-1 and the resistant Col-0 ecotype indicated that resistance to bacteria carrying *avrRpt2* is dominant or semi-dominant. The segregation ratios observed in the F_2 and F_3 suggested that two genes are missing from Po-1, both of which are required for resistance to strains carrying *avrRpt2*. One of these loci was shown by complementation tests and genetic mapping to be allelic with the *RPS2* resistance locus already discussed. Efforts are currently underway to confirm and further characterize the second locus.

Interestingly, analysis of Po-1 × Col-0 progeny for *avrB*-specific resistance also suggested the absence of two required loci in Po-1. The identification of F_2 individuals resistant to bacteria expressing *avrB* but not resistant to bacteria expressing *avrRpt2* (and vice-versa) revealed an *avrB*-specific resistance locus in Col-0 not tightly linked to the *avrRpt2*-specific *RPS2* locus. Further evidence for an *avrB*-specific resistance locus came from the identification of F_3 families that exhibit 3:1 segregation for *avrB*-specific resistance (Bent and Staskawicz, unpubl.). Two very salient questions we are now addressing concern whether the second locus required for *avrB* resistance has a pleiotropic effect on multiple race-specific resistance pathways, and whether it is the same "second locus" required for *avrRpt2* resistance.

Ethylene and Disease Development

With the *Arabidopsis–P. syringae* model system in place, we can take advantage of plant lines isolated by other researchers. A successful first example of this has been our collaboration with Dr. Joseph Ecker,

who (with co-workers) has characterized *Arabidopsis* mutants that either overproduce or are insensitive to the plant hormone ethylene (12). The role of ethylene in plant disease remains unclear despite extensive biochemical and molecular biological research, indicating roles in both resistance and susceptibility (2,8,32). In the first set of experiments, we examined disease development in *Arabidopsis* ethylene mutants by monitoring active resistance toward *P. syringae* pv. *tomato* expressing *avrRpt2, avrB,* or *avrRpm1*. For all three gene-for-gene interactions, resistance remained functional in the ethylene-insensitive mutants (as assessed using the criteria listed in Table 1) (1). These results suggest that ethylene may have little role, if any, in resistance to infection by avirulent bacteria.

In a separate set of experiments, the ethylene-insensitive "*ein*" mutants were inoculated with virulent, disease-causing bacteria (*P. syringae* pv. *tomato, P. syringae* pv. *maculicola,* or *Xanthomonas campestris* pv. *campestris*). Although wild-type and *ein1* mutants developed disease, *ein2* mutants developed far fewer symptoms following infection. Surprisingly, bacteria grew to similar population levels in *ein2* and wild-type plants, indicating that the *ein2* defect conferred disease tolerance (reduced disease damage despite extensive pathogen growth) (1). Therefore ethylene appears to be a mediator of pathogen-induced damage. These results suggest that reduction of ethylene production or sensitivity may be an effective strategy for the development of crop plants with improved disease tolerance (1).

Conclusion

Study of a model organism that is highly amenable to experimental manipulation does not preclude pursuit of practical applications and can actually expedite such work. In the early stages, a significant amount of time can be spent "reinventing the wheel" in the new experimental system, but this investment often yields future dividends. As resistance genes are identified and work proceeds on topics such as ethylene and plant disease, we hope that study of the *Arabidopsis–Pseudomonas* interaction will not only enhance our understanding of the basic biological features of plant–pathogen interactions, but also open up new possibilities for improving resistance and tolerance in crop plants. Efforts to develop *Arabidopsis*–nematode models will undoubtedly yield similar benefits.

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