Resistance and Susceptibility of *Arabidopsis thaliana* to Bacterial Wilt Caused by *Ralstonia solanacearum*

Chang-Hsien Yang and Gan-Der Ho

Graduate Institute of Agricultural Biotechnology, National Chung Hsing University, Taichung, Taiwan 40227 Republic of China.
Accepted for publication 8 January 1998.

ABSTRACT

Yang, C.-H., and Ho, G.-D. 1998. Resistance and susceptibility of *Arabidopsis thaliana* to bacterial wilt caused by *Ralstonia solanacearum*. Phytopathology 88:330-334.

Tomato bacterial wilt caused by *Ralstonia solanacearum* is a model system for studying plant-bacterial interactions, because it is genetically one of the best characterized plant diseases. We demonstrate here that four different strains of *R. solanacearum*, two from radishes (Rd4 and Rd15) and two from tomato (Ps21 and Ps95), can infect 27 different ecotypes of *Arabidopsis thaliana*, causing different responses. All ecotypes tested were highly susceptible to strain Rd15, which caused symptoms similar to those observed in tomato plants. For example, leaf drooping and discoloration developed just 3 days after inoculation, and plants completely wilted within 1 week. Strains Rd4 and Ps95 were less infectious than Rd15. With these two strains, a variety of disease responses were observed among different ecotypes at 2 weeks after inoculation; both susceptible and resistant ecotypes of *A. thaliana* were identified. Ps21 was the least infectious of the four strains and caused almost no symptoms in any of the ecotypes of *Arabidopsis* tested. Direct bacterial isolation and plant skeleton hybridization analysis from infected plants indicated that bacterial colonization was correlated with the severity of symptoms. Growth of bacteria was limited to the infection site in resistant plants, whereas the bacteria spread throughout susceptible plants by 1 week after inoculation.

Arabidopsis thaliana, a small flowering plant, has been successfully used as a model system to study host-pathogen interactions for several bacterial, viral, and fungal pathogens during the past few years (2,8,12,14,20). Bacterial pathogens such as *Xanthomonas campestris* and *Pseudomonas syringae* have been clearly demonstrated to cause disease on *Arabidopsis* (12,20). Symptom variation in different *Arabidopsis* ecotypes caused by *X. campestris* has been observed (6). The susceptible ecotype develops systemic necrosis, whereas the resistant ecotype remains asymptomatic after infection with *X. campestris*. This has lead to the identification of several independent nuclear genes, such as *RXC1* and *RXC2*, that determine resistance to *X. campestris* (6). Research on the interactions between *A. thaliana* and *P. syringae* has provided more information about resistance. Genes such as *RPS2* and *RPM1*, which confer resistance to *P. syringae*, have been identified and cloned in *Arabidopsis* (5,9,17). Nucleotide sequence comparisons reveal a high degree of similarity in functional motifs between these genes and other genes for resistance to viral and fungal pathogens, suggesting a common function (4,26). In addition to the identification of resistance genes, other studies using *A. thaliana* and bacterial pathogens have indicated that resistance responses may be due to the direct expression of defense genes such as pathogenesis-related proteins (PRPs) or pathogen-inducible genes (3,22,23). Since the mechanisms for these disease resistances are still unclear, further investigation of the interactions between *A. thaliana* and bacterial pathogens is needed to determine their genetic and biochemical bases, which should lead to a deeper understanding of the mechanisms involved.

Bacterial wilt caused by *Ralstonia solanacearum* is a serious plant disease in the tropics and in warm climates throughout the world and causes severe losses to many different agricultural crops (10). Tomato bacterial wilt is the most destructive disease of tomato; however, it is also a model system for studying plant-bacterial interactions, as it is genetically one of the best characterized plant diseases. Several bacterial wilt–resistant tomato varieties have been identified, and genetic studies have revealed that the resistance is controlled by quantitative trait loci (1,7,19,27,28). In this work, we used *A. thaliana* as a host plant to study the interactions between *Arabidopsis* and *R. solanacearum*. We used four strains of *R. solanacearum* to infect different ecotypes of *Arabidopsis* and to determine which ones are resistant or susceptible to *R. solanacearum*. Our results demonstrate that a race-ecotype interaction may occur between *Arabidopsis* and *R. solanacearum*. Future genetic and biochemical analyses may reveal the mechanism of this resistance.

Corresponding author: C.-H. Yang; E-mail address: chyang@dragon.nchu.edu.tw

Publication no. P-1998-0209-01R

MATERIALS AND METHODS

Bacteria and plants. Two *R. solanacearum* strains from radishes (Rd4 and Rd15) and two strains from tomatoes (Ps21 and Ps95) from Taiwan were provided by S.-T. Hsu and K.-C. Tzeng (Department of Plant Pathology, National Chung Hsing University, Taichung, Taiwan). Rd4 and Rd15 are both fully virulent in radishes, whereas Ps21 and Ps95 are both fully virulent in tomatoes. All four strains are virulent in plants such as sweet pepper, eggplant, and tobacco. They are all race 1. Seeds of *A. thaliana* ecotypes were obtained from the *Arabidopsis* Biological Resource Center, The Ohio State University, Columbus.

Plant growth conditions. *Arabidopsis* seeds planted on $^1/_2$ Murashige-Skoog medium (18) were grown in growth chambers under long-day conditions (16-h light/8-h dark) for 10 days before being transferred to the greenhouse. The light intensity of the growth chambers was 150 μE m^{-2} s^{-1}. Seedlings were transplanted to soil and grown in greenhouses 17 days before bacterial inoculation. The greenhouses were maintained at 22°C with 16 h of supplemental lighting.

Bacterial inoculation. Bacteria were grown on a tetrazolium chloride (TZC) selective medium (11) at 28°C for 4 days before inoculation. The bacteria were inoculated by stabbing a colony with

a needle and then puncturing the base of the inflorescence stem of 17-day-old plants. The number of bacteria introduced into the wound was determined by dilution-plating and was approximately 10^8 cells per plant. The infected plants were grown under long-day conditions (16-h light/8-h dark) at 25°C in a growth chamber until symptoms appeared. The symptoms were rated on a scale from 0 to 4, based on their severity at 15 days after inoculation, in which 0 = no symptoms, 1 = only cauline leaves near the inoculation site drooped slightly, 2 = symptoms appeared on other cauline and rosette leaves, 3 = the whole plant was wilted and stunted; and 4 = death. The experiment was done three times, and at least five plants from each ecotype were inoculated with each strain in each experiment.

Bacterial culture assay. At various times after inoculation, the inflorescence stems were surface-sterilized and cut into eight pieces of equal size (1.5 to 2 cm). All the pieces were placed on TZC medium at 28°C for 1 day. The appearance of bacteria in the medium was recorded and served as an indication of the movement of bacteria in the infected plants.

Dot blot analysis. The presence of bacteria in infected plants was confirmed by dot blot analysis by using a DNA probe specific to *R. solanacearum.* The DNA probe was a 1.1-kb genomic DNA fragment isolated from a polymerase chain reaction (PCR) amplified from *R. solanacearum* strain Ps21 genomic DNA (30). The sequences for the primers used in the PCR were 5′-GACGACATCATTTCCACCGGGCG-3′ for the sense primer and 5′-GGGTGAGATCGATTGTCTCCTTG-3′ for the antisense primer. These were provided by Y.-A. Lee (Department of Biology, Fu Jen University, Taiwan).

Plant skeleton hybridization. Whole *Arabidopsis* plants were subjected to skeleton hybridization by a method modified from that of previous reports (15,16,25). Shoots were immersed in 95% ethanol and gently shaken overnight at 25°C. The ethanol was then replaced by solution A (0.1 mM NaN_3, 0.1% sodium dodecyl sulfate [SDS], and 0.05 mg of proteinase K per ml) and gently shaken overnight at 37°C. The following day, the plants were treated with 0.15 N HCl for 20 min, followed by 0.5 M NaOH and 1.0 M NaCl for 35 min, and finally with a neutralization solution (0.5 M Tris-Cl and 1.5 M NaCl, pH 7.6) for 40 min. After being rinsed twice in 2× SSC (1× SSC is 0.15 M NaCl plus 0.015 M sodium citrate), the plants were stored or subjected to hybridization immediately after being air-dried at room temperature. The plants were prehybridized for 30 min and hybridized with a ^{32}P-labeled 1.1-kb DNA probe specific to *R. solanacearum* overnight in the same solution (0.25 M Na_2HPO_4, pH 7.2, and 7% SDS), and then washed twice each in solution 1 (20 mM Na_2HPO_4, pH 7.2, and 5% SDS) and solution 2 (20 mM Na_2HPO_4, pH 7.2, and 1% SDS) for 30 min per wash. The plants were then air-dried, covered with plastic wrap, and autoradiographed.

RESULTS

Relative virulence of different strains of *R. solanacearum* on *Arabidopsis*. The symptoms of bacterial wilt on mature solanaceous crops (e.g., tomatoes) are normally leaf wilting and discoloration, leaf drop, stunting, and finally permanent wilting and death. Four strains of *R. solanacearum* caused different symptom development on *Arabidopsis* plants. Strain Rd15, originally isolated from radishes, caused the most severe symptoms in *Arabidopsis*. Just 3 days after infection by strain Rd15, all *Arabidopsis* ecotypes began to show symptoms similar to those observed in tomato plants. The cauline leaves near the inoculation site drooped suddenly, and the color of the leaves changed from green to yellow-brown (Fig. 1A). This symptom quickly appeared on other cauline and rosette leaves and finally on the flower stalks and siliques. All ecotypes tested were susceptible to Rd15 and completely wilted within 1 week of inoculation (Fig. 1A and B; Table 1). Strain Rd4, also isolated from radishes, was much less infectious than Rd15. At 7 days after inoculation, no symptoms were observed in any of the eco-

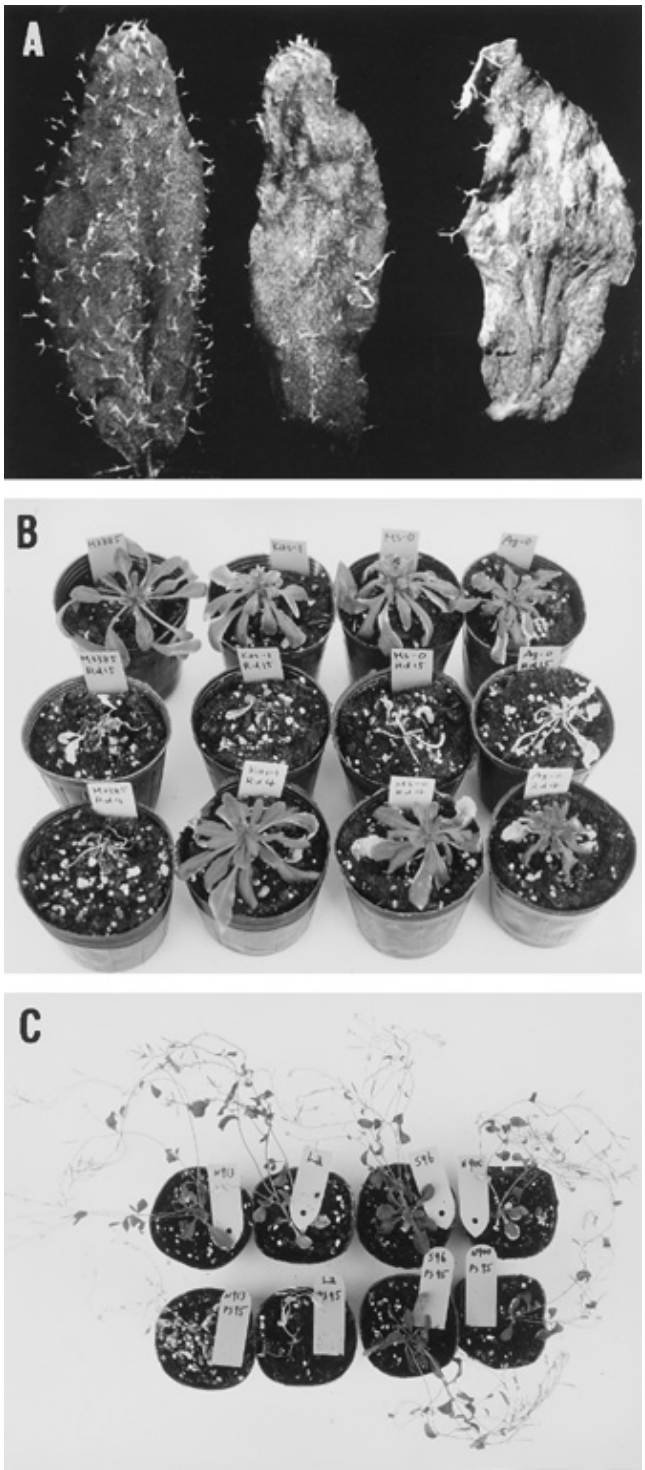

Fig. 1. Variation in symptom development produced in resistant and susceptible *Arabidopsis thaliana* ecotypes. **A,** Left: a cauline leaf from an uninoculated plant; middle: a cauline leaf from a plant 3 days after inoculation with Rd15; and right: a cauline leaf from a plant 7 days after inoculation with Rd15. **B,** Four ecotypes of *Arabidopsis* (from left to right: M3385, Kas-1, Ms-0, and Ag-0) 2 weeks after inoculation with Rd15 and Rd4. Top row: uninoculated plants; middle row: Rd15-inoculated plants; and bottom row: Rd4-inoculated plants. Results show that all four ecotypes are susceptible to Rd15 inoculation, and only M3385 is susceptible to Rd4 inoculation. **C,** Four ecotypes of *Arabidopsis* (from left to right: N913, Landsberg, S96, and N900) 2 weeks after inoculation with Ps95. Top row: uninoculated plants; and bottom row: Ps95-inoculated plants. Results show that N913 and Landsberg are susceptible, whereas S96 and N900 are resistant to Ps95 inoculation.

types. However, after 7 days, a variety of disease responses were observed. Some ecotypes started to show typical symptoms in the cauline leaves, but they developed much more slowly than in Rd15-inoculated plants. Although severe wilting was observed in some ecotypes (e.g., N913, N902, and M3385), most ecotypes either exhibited relatively mild or no symptoms 2 weeks after inoculation (Fig. 1B; Table 1).

Strain Ps95, originally isolated from tomatoes, also caused mild symptoms in *Arabidopsis*. In general, symptom development in Ps95-inoculated plants was similar to that observed in Rd4-inoculated plants. A variety of disease responses was observed among different ecotypes at 7 days after inoculation. Although the typical symptoms such as the drooping of the cauline and rosette leaves were observed in some ecotypes (e.g., N913, Landsberg, and M3385) and the plants eventually completely wilted 2 weeks after inoculation (Fig. 1C; Table 1), symptom development was relatively slower than in Rd15-inoculated plants. Most ecotypes, however, either developed mild symptoms or no symptoms at all by 15 days after inoculation (Fig. 1C; Table 1). Ps21, another strain isolated from tomatoes, was the least virulent of the four strains and caused mild symptoms in only 11 ecotypes (e.g., N913). This was seen as drooping of the cauline leaves near the inoculation site at 2 weeks after infection. No further symptoms developed during the life span of the plants.

Differential responses of *A. thaliana* ecotypes to strains of *R. solanacearum*. As shown in Table 1, some ecotypes such as N913 were highly susceptible to all four strains tested, although their response time differed. Other ecotypes exhibited differential responses when inoculated with different strains of *R. solanacearum*. For example, ecotype No-0 was highly susceptible to the two strains from radishes (Rd15 and Rd4), but exhibited relatively mild symptoms when inoculated with the strains from tomato (Ps21 and Ps95). Ecotypes such as Shahdara, En-t, and Sn(5)-1 were highly susceptible to Rd15, but developed only very mild symptoms when inoculated with the other three strains. Some ecotypes (e.g., S96, Ber, and C24) were only susceptible to Rd15, but were highly resistant to the other three strains. Despite the fact that all ecotypes tested are susceptible to Rd15, some ecotypes were more resistant than others to the other three strains. As shown in Table 1, the ecotypes listed toward the top tended to be susceptible, whereas the ecotypes listed toward the bottom tended to be resistant to different *R. solanacearum* strains.

Detection of bacterial movement in plants. To determine whether bacterial colonization was correlated with symptom development, two ecotypes, N913 and S96, were inoculated with the four strains of *R. solanacearum*, and the distribution of bacteria in the plants was analyzed using direct bacterial culturing and plant skeleton hybridization. Isolation of bacteria at different distances from the site of inoculation indicated that the most severe strain, Rd15, moved throughout both N913 and S96 plants very fast. As shown in Figure 2, the presence of strain Rd15 was detected in the second piece above the inoculation site only 1 day after inoculation and had colonized the entire inflorescence (all eight pieces) by 5 to 7 days after inoculation. Strains Rd4, Ps21, and Ps95, however, were limited to the second sampled piece above the infection site in S96-inoculated plants, even after 2 weeks following inoculation (Fig. 2). In ecotype N913, the growth of strains Ps95 and Rd4 was similar and was limited to the infection site at 1 day after inoculation. The bacteria gradually spread to the third and fourth pieces above the infection site within 1 week, and eventually reached the

TABLE 1. Disease severity ratings of *Arabidopsis thaliana* ecotypes at 15 days after inoculation with four strains of *Ralstonia solanacearum*[a]

	Strain			
Ecotypes	Rd15	Rd4	Ps21	Ps95
N902	4[b]	4	0	4
N913	4	4	2	4
M3385	4	4	0	4
Landsberg	4	2	0	4
Columbia	4	3	2	3
N907	4	2	0	3
NW20	4	3	1	3
No-0	4	4	2	2
Cs928	4	1	1	2
H55	4	3	0	2
Litva	4	1	0	1
Aa-0	4	0	0	1
N1601	4	1	1	1
Ws	4	0	1	1
Shahdara	4	1	1	1
N906	4	1	0	1
En-t	4	1	1	1
Sn(5)-1	4	1	1	1
Wei-0	4	1	1	1
N900	4	1	0	0
S96	4	0	0	0
Ber	4	0	0	0
C24	4	1	0	0
Est	4	1	0	0
Ms-0	4	0	0	0
Kas-1	4	0	0	0
Ag-0	4	0	0	0

[a] Results were obtained from three independent experiments. At least five plants from each ecotype were inoculated with a strain of bacteria in each experiment.

[b] Indicates the degree of symptoms in which 0 = no symptoms; 1 = only cauline leaves near the inoculation site drooped slightly; 2 = symptoms appeared on other cauline and rosette leaves; 3 = the whole plant was wilted and stunted; and 4 = death.

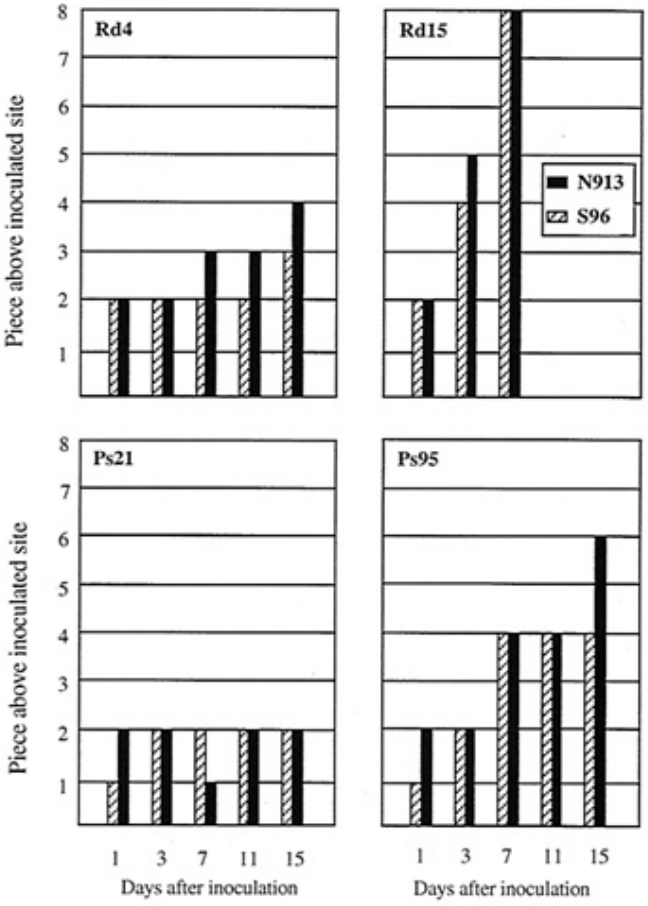

Fig. 2. Distribution of *Ralstonia solanacearum* in inoculated plants after different days of inoculation. Bars indicate the presence of bacteria in 1.5- to 2.0-cm sections (average number of five plants for each treatment) of the inflorescence stem taken at various distances above the inoculation site.

sixth to eighth pieces at 14 days after inoculation (Fig. 2). Compared with Rd4 and Ps95, the growth of Ps21 in ecotype N913 was relatively slow. It took more than 10 days to reach the second piece above the infection site in low density, and bacteria never spread beyond the third or fourth piece above the infection site after 2 weeks following inoculation (Fig. 2).

Plant skeleton hybridization analysis confirms the above finding that symptom development was correlated with bacterial colonization. That result indicated that bacteria were distributed throughout susceptible ecotype N913 at 11 days after inoculation with Ps95 (Fig. 3A). A relatively weaker signal, similar to that in uninoculated plants (Fig. 3B), was detected in the resistant ecotype S96 inoculated with strain Ps95 (Fig. 3C). Strain Ps21 caused mild symptoms on ecotype N913, and a relatively weaker signal was detected in N913 plants inoculated with strain Ps21 (Fig. 3D) than in N913 plants inoculated with strain Ps95 (Fig. 3A).

DISCUSSION

We have demonstrated in this study that *A. thaliana* is a host plant for *R. solanacearum*, where this pathogen produces symptoms similar to those described in other solanaceous species. Differences in symptom development in *Arabidopsis* plants produced by different *R. solanacearum* strains suggest variation in their virulence. That Rd15 was the most virulent strain in *Arabidopsis* is not surprising, since Rd15 was isolated from radishes, a cruciferous species related to *A. thaliana*. However, this was not true for Rd4, the other strain isolated from radishes. Since both strains caused severe wilt in radishes, these different responses in *Arabidopsis* might further differentiate them into different races. It is interesting that two strains, Ps21 and Ps95, that caused severe wilt in tomatoes also caused symptoms in *Arabidopsis*, although in a relatively mild manner compared with Rd15. The ability of *R. solanacearum* to cause disease in *Arabidopsis* suggests that *Arabidopsis* can supply all host factors needed for *R. solanacearum* to cause disease.

The appearance of strain Rd15 throughout the shoot of the susceptible plants in a short period of time was correlated with the high pathogenicity of this strain. This indicates that the ability of Rd15 to multiply and move through the plant is a major factor in disease development. The longer time needed for strains Rd4 and Ps95 to spread throughout susceptible plants and the limitation of these strains to the inoculation site in resistant plants indicates that the ability of the plants to retard either growth or movement of bacteria delays symptom development.

There are several possibilities to explain this observation. The mechanism of this resistance may be due to the expression of the existing resistance genes directly. The bacterial growth is either reduced in the plant or limited to the site of inoculation by a resistance response. A similar result has been observed in the interactions between *A. thaliana* and *X. campestris* (6), in which bacterial growth is 5- to 28-fold more in the susceptible ecotype Ler than in the resistant ecotype Col leaf tissue. The resistance of Col is probably due to suppression of bacterial growth and is correlated with the presence of *RXC* genes (6). The resistance mechanism may also function similarly to that of the *Arabidopsis RPS2* and *RPM1* genes, which enable a hypersensitive response to *P. syringae* that limits bacterial multiplication at the site of inoculation (5,9,17). The fast growth and spread of *R. solanacearum* in susceptible ecotypes could, therefore, be due to the lack of the resistance genes.

Some negative-acting host factors that have also been identified in *Arabidopsis*, such as lipid transfer proteins (LTPs), sulfotransferase (ST), PRPs, and *avr*-induced genes (AIGs) (13,21,24,29), may also be involved in the inhibition of bacterial growth in our system. LTPs have been reported to inhibit growth of several bacterial pathogens, including *R. solanacearum* and *Clavibacter michiganesis* (24). The expression of ST increases in response to an avirulent bacterial pathogen, causing a hypersensitive reaction (13). PRPs and AIGs, which have been characterized as defense gene products, have also been shown to be involved in the resistance of *Arabidopsis* to bacterial pathogens (21,29). The expression of pathogenesis-related PR-1 gene is altered in susceptible *Arabidopsis* after infection with several bacterial pathogens (22). PR-1 mRNA accumulation was reduced to an approximately 10% wild-type level in *Arabidopsis* mutant enhanced disease susceptibility, which was susceptible to *P. syringae* (22). The *Arabidopsis* phytoalexin camalexin (3-thiazol-2′-yl-indole), another PRP, inhibits the growth of *P. syringae* by disrupting bacterial membranes directly (23). Two *Arabidopsis* AIGs, *AIG1* and *AIG2*, have been shown to exhibit *RPS2*- and *avrRpt2*-dependent induction after inoculation with a *P.*

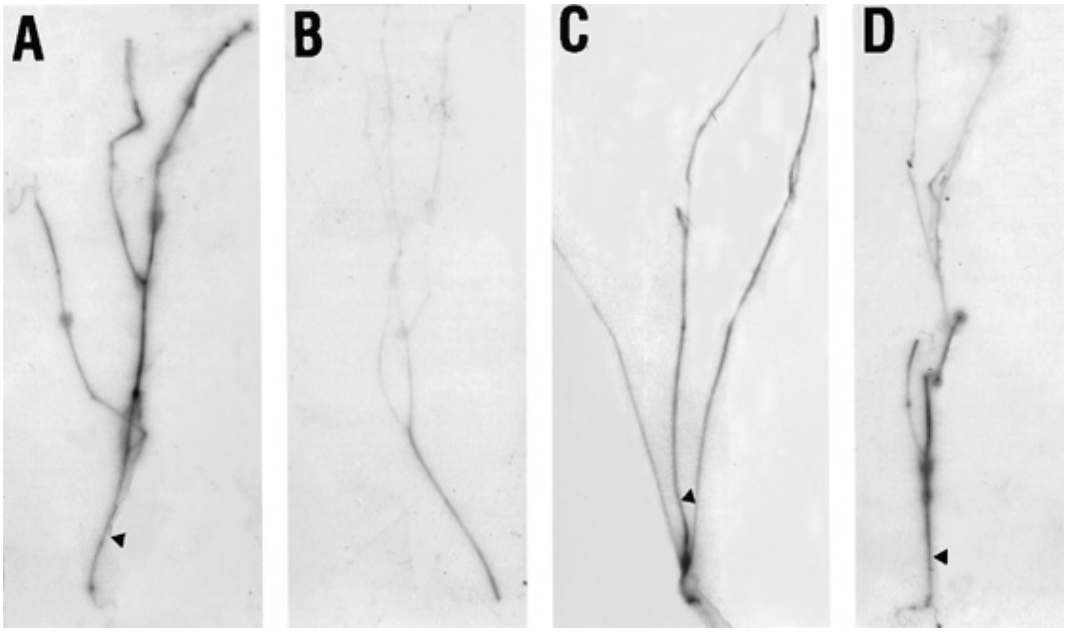

Fig. 3. The distribution of *Ralstonia solanacearum* in inoculated plants was demonstrated by plant skeleton hybridization of inflorescence shoots 11 days after inoculation. Arrowheads indicate the inoculation sites. **A,** An N913 plant inoculated with Ps95. **B,** An uninoculated N913 control plant. **C,** An S96 plant inoculated with Ps95. **D,** An N913 plant inoculated with Ps21.

syringae strain carrying *avrRpt2* (21). All the host factors described above in *Arabidopsis* have the potential to cause a resistance response to *R. solanacearum.*

Alternatively, the inhibition of the bacterial growth may be simply due to the failure of the resistant plants to provide the host factors (i.e., nutrients) necessary for the multiplication of the pathogen. Susceptible plants that provide these host factors may allow bacteria to grow rapidly and cause disease.

Whether the resistance mechanisms discussed above influence only bacterial growth, movement, or both remains uncertain and needs further study. Since no host movement factors have been reported in any bacterial disease, we hypothesize that limitation of bacterial multiplication should be the major factor in resistance. The mechanisms of this resistance may involve products from only one single gene or from many genes. Further genetic research into the basis of resistance and defense gene expression in *Arabidopsis* is in progress.

ACKNOWLEDGMENTS

This work was supported by a grant to C.-H. Yang from the National Science Council, Taiwan, Republic of China, grant number NSC86-2321-B-005-021-B13. We thank S.-T. Hsu and K.-C. Tzeng for providing strains of *Ralstonia solanacearum* used in this work and for their helpful discussion. We also thank Y.-A. Lee for providing the polymerase chain reaction primers specific to *R. solanacearum* and the *Arabidopsis* Biological Resource Center for providing seed stocks used in this research.

LITERATURE CITED

1. Acosta, J. C., Gilbert, J. C., and Quinon, V. L. 1964. Heritability of bacterial wilt resistance in tomato. Proc. Am. Soc. Hortic. Sci. 84:455-461.
2. Adam, L., and Somerville, C. 1996. Genetic characterization of five powdery mildew disease resistance loci in *Arabidopsis thaliana*. Plant J. 9:341-356.
3. Aufsatz, W., and Grimm, C. 1994. A new, pathogen-inducible gene of *Arabidopsis* is expressed in an ecotype-specific manner. Plant Mol. Biol. 25:229-239.
4. Bent, A. 1996. Plant disease resistance genes: Function meets structure. Plant Cell 8:1757-1771.
5. Bent, A., Kunkel, B. N., Dahlbeck, D., Brown, K. L., Schmidt, R., Giraudat, J., Leung, J., and Staskawicz, B. J. 1994. *RPS2* of *Arabidopsis thaliana*: A leucine-rich repeat class of plant disease resistance genes. Science 265:1856-1860.
6. Buell, C. R., and Somerville, S. C. 1997. Use of *Arabidopsis* recombinant inbred lines reveals a monogenic and novel digenic resistance mechanism to *Xanthomonas campestris* pv. *campestris*. Plant J. 12:21-29.
7. Danesh, D., Aarons, S., McGill, G. E., and Young, N. D. 1994. Genetic dissection of oligogenic resistance to bacterial wilt in tomato. Mol. Plant-Microbe Interact. 7:464-471.
8. Dangl, J. L. 1993. Applications of *Arabidopsis thaliana* to outstanding issues in plant-pathogen interaction. Int. Rev. Cytol. 144:53-83.
9. Grant, M. R., Godiard, L., Straube, E., Ashfield, T., Lewald, J., Sattler, A., Innes, R. W., and Dangl, J. L. 1995. Structure of the *Arabidopsis RPM1* gene enabling dual specificity disease resistance. Science 269: 843-846.
10. Hayward, A. C. 1991. Biology and epidemiology of bacterial wilt caused by *Pseudomonas solanacearum*. Annu. Rev. Phytopathol. 29:65-87.
11. Kelman, A. 1954. The relationship of pathogenicity in *Pseudomonas solanacearum* to colony appearance on a tetrazolium medium. Phytopathology 44:693-695.
12. Kunkel, B. N., Bent, A. F., Dahlbeck, D., Innes, R. W., and Staskawicz, B. J. 1993. *RPS2*, an *Arabidopsis* disease resistance locus specifying recognition of *Pseudomonas syringae* strains expressing the avirulence gene *avrRpt2*. Plant Cell 5:865-875.
13. Lacomme, C., and Roby, D. 1996. Molecular cloning of a sulfotransferase in *Arabidopsis thaliana* and regulation during development and in response to infection with pathogenic bacteria. Plant Mol. Biol. 30:995-1008.
14. Lee, S., Stenger, C., Bisaro, D. M., and Davis, K. R. 1994. Identification of loci in *Arabidopsis* that confer resistance to geminivirus infection. Plant J. 6:525-535.
15. Leisner, S., Turgeon, R., and Howell, S. H. 1993. Effects of host plant development and genetic determinants on the long-distance movement of cauliflower mosaic virus in *Arabidopsis*. Plant Cell 5:191-202.
16. Melcher, U., Brannan, C. M., Gardner, C. O., Jr., and Essenberg, R. C. 1992. Diverse mechanisms of plant resistance to cauliflower mosaic virus revealed by leaf skeleton hybridization. Arch. Virol. 123:379-387.
17. Mindrinos, M., Katagiri, F., Yu, G.-L., and Ausubel, F. M. 1994. The *A. thaliana* disease resistance gene *RPS2* encodes a protein containing a nucleotide-binding site and leucine-rich repeats. Cell 78:1089-1099.
18. Murashige, T., and Skoog, F. 1962. A revised medium for rapid growth and bioassays with tobacco tissue cultures. Physiol. Plant 15:473-479.
19. Opena, R. T., Green, S. K., Talekar, N. S., and Chen, J. T. 1988. Genetic improvement of tomato adaptability to the tropics: Progress and future prospects. Pages 70-85 in: Tomato and Pepper Production in the Tropics. S. K. Green, ed. Asian Vegetable Research and Development Center Press, Shanhua, Taiwan.
20. Parker, J. E., Barber, C. E., Mi-jiao, F., and Daniels, M. J. 1993. Interaction of *Xanthomonas campestris* with *Arabidopsis thaliana*: Characterization of a gene from *X. c.* pv. *raphani* that confers avirulence to most *A. thaliana* accessions. Mol. Plant-Microbe Interact. 6:216-224.
21. Reuber, T. L., and Ausubel, F. M. 1996. Isolation of *Arabidopsis* genes that differentiate between resistance responses mediated by the *RPS2* and *RPM1* disease resistance genes. Plant Cell 8:241-249.
22. Rogers, E. E., and Ausubel, F. M. 1997. *Arabidopsis* enhanced disease susceptibility mutants exhibit enhanced susceptibility to several bacterial pathogens and alterations in *PR-1* gene expression. Plant Cell 9:305-316.
23. Rogers, E. E., Glazebrook, J., and Ausubel, F. M. 1996. Mode of action of the *Arabidopsis thaliana* phytoalexin camalexin and its role in *Arabidopsis*-pathogen interactions. Mol. Plant-Microbe Interact. 9:748-757.
24. Segura, A., Moreno, M., and Garcia-Olmedo, F. 1993. Purification and antipathogenic activity of lipid transfer proteins (LTPs) from the leaves of *Arabidopsis* and spinach. FEBS (Fed. Eur. Biochem. Soc.) Lett. 332: 243-246.
25. Simon, A. E., Li, X. H., Lew, J. E., Stange, R., Zhang, C., Polacco, M., and Carpenter, C. D. 1992. Susceptibility and resistance of *Arabidopsis thaliana* to turnip crinkle virus. Mol. Plant-Microbe Interact. 5:496-503.
26. Staskawicz, B. J., Ausubel, F. M., Baker, B. J., Ellis, J. G., and Jones, J. D. G. 1995. Molecular genetics of plant disease resistance. Science 268: 661-667.
27. Thoquet, P., Olivier, J., Sperisen, C., Rogowsky, P., Laterrot, H., and Grimsley, N. 1996. Quantitative trait loci determining resistance to bacterial wilt in tomato cultivar Hawaii7996. Mol. Plant-Microbe Interact. 9:826-836.
28. Thoquet, P., Olivier, J., Sperisen, C., Rogowsky, P., Prior, P., Anaïs, G., Mangin, B., Bazin, B., Nazer, R., and Grimsley, N. 1996. Polygenic resistance of tomato plants to bacterial wilt in the French West Indies. Mol. Plant-Microbe Interact. 9:837-842.
29. Uknes, S., Mauch-Mani, B., Moyer, M., Potter, S., Williams, S., Dincher, S., Chandler, D., Slusarenko, A., Ward, E., and Ryals, J. 1992. Acquired resistance in *Arabidopsis*. Plant Cell 4:645-656.
30. Wang, C.-C. 1996. Isolation of specific probe and detection of *Pseudomonas solanacearum* based on PCR analysis. M.S. thesis. Fu Jen University, Taiwan.