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UNDERSTANDING BACTERIAL WILT RESISTANCE IN TOMATO THROUGH THE USE OF DNA GENETIC MARKERS

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INTRODUCTION

One of the most devastating plant diseases in the tropics and throughout the world is bacterial wilt, caused by the bacterium, *Pseudomonas solanacearum*. This pathogen disrupts water transport, alters physiology and induces a severe, usually fatal, wilting in susceptible host plants (Hayward, 1991). One plant species that is seriously affected by bacterial wilt is tomato, *Lycopersicon escuientum*, and efforts to grow tomatoes widely in the tropics have generally been hampered by the disease. Often, farmers grow tomatoes in a limited area until bacterial wilt becomes too severe, then move onto a new field until the disease reappears, with the cycle repeating itself again and again (L. Sequeira, Madison, Wisconsin, personal communication). The critical importance of bacterial wilt in international agriculture is reflected by this volume and in the recent international conference on bacterial wilt at the Asian Vegetable Research and Development Center (AVRDC) in Taiwan.

Even though bacterial wilt is a disease of major international importance, very little is known about the cellular mechanisms or genetics underlying host plant resistance. This contrasts sharply with current knowledge about pathogenecity in *P. solanacearum*, where numerous genes, with specific biochemical and physiological roles in disease development and host range, have been characterized in detail (Denny, et al., 1990; Boucher, et al., 1992). Yet this unfortunate situation need not continue, because the tools for understanding host plant resistance are now available. In the case of tomato, genetic resistance has previously been described and some forms have been shown to be effective under various environmental conditions (Opeña, et al., 1987). However, the fact that bacterial wilt resistance is genetically complex

has generally meant that efforts to breed resistant tomato cultivars or analyze the underlying genetics have usually been unsuccessful (R. Opeña and G. Hartman, AVRDC, Taiwan, personal communication).

DNA genetic markers, such as *restriction fragment length polymorphisms* (RFLPs) (Botstein, et al., 1980) and *random amplified polymorphic DNAs* (RAPDs) (Williams, et al., 1990), promise to increase our understanding of bacterial wilt resistance dramatically. DNA markers make it possible to monitor inheritance throughout an entire plant genome at high resolution and dissect the contributions of specific genetic loci to complex characters. The concept of DNA genetic markers and their application to plant improvement and genetics has been reviewed previously (Soller and Beckmann, 1983; Tanksley, et al., 1989). This chapter focuses on specific strategies for using DNA markers to characterize the underlying genetics of bacterial wilt resistance in tomato.

To devise effective strategies for studying bacterial wilt resistance with DNA genetic markers, critical questions need to be addressed. Can DNA markers be used successfully to map the chromosomal locations of partial resistance genes for bacterial wilt? If so, can the knowledge gained from mapping be used to develop resistant cultivars quickly and efficiently? Is it possible to characterize relationships between bacterial wilt resistance and host plant physiology and developmental stage using DNA markers? Do specific genes provide resistance to different races of *P. solanacearum*, and if so, can DNA markers help to elucidate these relationships? Finally, can DNA marker technology provide a basis for molecular cloning of bacterial wilt resistance genes? In this chapter, the answers to many of these questions will be addressed based on research done in our laboratory. We have been

analyzing bacterial wilt resistance in tomato using RFLP and RAPD genetic markers and our results appear very promising. For many of the questions raised above, the results indicate that DNA markers will, indeed, be useful in dissecting the genetics of bacterial wilt resistance. Moreover, results to date suggest that improved resistant cultivars can be developed through the use of this technology.

MAPPING GENES FOR PARTIAL RESISTANCE TO BACTERIAL WILT

The first step in using DNA marker technology to characterize bacterial wilt resistance is to map the chromosomal locations of the underlying resistance genes. Mapping plant resistance genes with DNA markers has been an area of active research. Recently, resistance genes for the tomato pathogens, tomato mosaic virus (Young, et al., 1988), *Pseudomonas syringae* (Martin, et al., 1991), *Fusarium oxysporum* (Sarfatti, et al., 1991), *Cladosporium clavum* (Jones, et al., 1992) and *Meloidogyne incognita* (Messeguer, et al., 1991) have all been mapped with RFLP or RAPD markers. In each of these examples, however, resistance was conditioned by a single gene only. By contrast, bacterial wilt resistance in tomato appears to be complex and polygenic (Opeña et al., 1987). Fortunately, a strategy for using DNA markers to map complex characters, including polygenic resistance, has been demonstrated (Paterson, et al., 1988). Previously, this strategy has been used to map genes underlying agronomic traits (Paterson et al., 1988; Keim, et al., 1990 Doebley, et al., 1991), but has been used to study of complex resistance characters infrequently (Nienhuis, et al., 1987; Romero-Severson, et al., 1989).

To map partial resistance genes for bacterial wilt in tomato, we examined

the progeny of a cross between a highly susceptible tomato cultivar (CLN286) and a moderately resistant wild tomato relative (L285). In previous work, L285 had been found to be one of the most resistant lines to bacterial wilt in tests at AVRDC (G. Hartman and R. Opeña, AVRDC, personal communication). Associations between genomic regions and resistance loci were uncovered by scoring F2 plants for bacterial wilt response and comparing those results with the DNA genotype at 58 loci distributed throughout the tomato genome. Genomic regions containing DNA markers that showed a statistically significant association with bacterial wilt resistance were considered candidates for resistance loci.

More specifically, 71 F2 progeny were tested for their bacterial wilt response by inoculating two or more cuttings from each F2 individual with a highly virulent *P. solanacearum* isolate (UW364, race 1, biovar 4, a generous gift of L. Sequeira, Madison, Wisconsin). Disease scoring of the F2 cuttings was repeated on four separate occasions, twice through a leaf axil and twice through wounds in the roots. Fourteen days after inoculation, plants were scored on a scale from zero (no symptoms) to five (plant death). In parallel with the disease scoring, DNA was extracted from leaves for each F2 plant and used to prepare Southern blots for analysis with 47 RFLP and a total of 11 RAPD reactions. Results were then converted into a numeric format, entered into a computer and compared with the bacterial wilt score for each F2 individual. Because of the large number of associations examined, a relatively stringent level of significance ($p < 0.005$) was used to guard against false positives due to multiple comparisons.

Our results indicated that three genomic regions were associated with bacterial wilt resistance in L285. One region, located near marker TG180b on chromosome six, showed the strongest association, accounting for nearly 40%

of the total variation in resistance ($p = 0.0001$). This putative resistance locus appeared to act in a dominant manner because heterozygotes were nearly as resistant as homozygotes for the L285 allele (Figure 1). A second genomic region associated strongly with bacterial wilt resistance was located on chromosome 10 near TG230. This putative resistance locus accounted for approximately 23% of the total variation ($p = 0.0001$) and seemed to act in a partially dominant manner, with heterozygotes at TG230 being slightly less resistant than homozygotes for the L285 allele.

Finally, a third putative resistance locus, located near RFLP marker TG51b did not reach the cutoff for significance (0.0121), but is included for reasons described in detail below. The chromosomal location of TG51b, and its corresponding resistance locus, are still uncertain. This putative resistance locus differed from the other two mentioned above in an important way — it was the allele from the susceptible parent (CLN286) that was associated with greater levels of resistance in the F2 population.

Together, the two major partial resistance loci (TG180b and TG230) explained more than 54% of the total variation in bacterial wilt resistance. This is actually a very large fraction of the resistance when one considers that total variation includes genetic, environmental, experimental, and interactive effects. Thus, 54% of the total variation is actually a low estimate for the genetic influence of these two loci on bacterial wilt resistance.

ORGAN SPECIFIC RESISTANCE REVEALED WITH DNA MARKERS

Previous research indicated that tomatoes respond differently to bacterial wilt invasion depending upon the site of infection (Winstead and Kelman,

1952). With this in mind, we analyzed the results from the L285 x CLN286 F2 population to see if loci associated with bacterial wilt resistance differed according to infection site. As noted above, DNA marker genotypes for 58 loci throughout the tomato genome were determined. Statistical associations between these 58 loci and the bacterial wilt reactions were then compared between stem- and root-inoculated plants. The results indicate that at least one genetic locus was associated with bacterial wilt resistance at markedly different levels of significance depending upon the site of *P. solanacearum* infection.

For both TG180b and TG230, the association between DNA marker genotype and bacterial wilt resistance was statistically significant for both root and stem inoculations. In the case of TG180b, the association was stronger in root inoculations, where more than 45% of the total variation was explained by TG180b ($p = 0.0001$), compared to the stem, where only 14% of the variation was explained ($p = 0.0004$). For TG230, opposite results were observed. In the root, TG230 could explain only 15% of the total variation in bacterial wilt response ($p = 0.0051$), while in the stem, it accounted for more than 23% ($p = 0.0001$).

In sharp contrast to these two RFLP loci, the association between TG51b and bacterial wilt resistance was completely sensitive to site of *P. solanacearum* inoculation. When TG51b was analyzed for statistical association with bacterial wilt resistance in the stem, nearly 17% of the variation was associated TG51b genotype at $p = 0.0001$. However, the association between TG51b and bacterial wilt resistance in the root was not statistically significant. Instead, less than 3% of the variation was explained by TG51b, with a statistically insignificant probability of $p = 0.245$. Based on these

results, it appears that there may be a gene for bacterial wilt resistance located near TG51b that is important only if plants are inoculated through the stem — the same locus seems to play no role in resistance if plants are inoculated through the root.

MARKER-ASSISTED SELECTION FOR BACTERIAL WILT RESISTANCE

A powerful application of DNA marker technology in agriculture is the selection of optimal individuals for crossing as part of breeding programs (Tanksley et al., 1989). In disease resistance breeding, DNA markers can provide a method for selecting resistant individuals without the need for pathogen inoculation. In many situations, this should save time and make it possible to select for resistance genes against several plant pathogens at the same time, even if one or more of the resistance traits are recessive. DNA markers should also make it possible to breed for resistance to pathogens that are quarantined in certain locations.

Another important application of DNA makers is in breeding for complex, polygenic characters. Often, it is difficult to retain all the genes involved in a complex resistance character throughout the course of a breeding program. Retaining resistance loci can be especially challenging if creating an artificial epidemic is difficult or if the disease is associated with a specific environmental condition or plant developmental stage. However, DNA markers tightly linked to the underlying resistance loci can be used as a basis for selection, thereby ensuring that the genes are retained during the course of the breeding program. Knowing the locations of resistance loci in terms of DNA markers also provides another benefit — the ability to select against

other parts of the genome that may carry undesirable genes. This is often the case when resistance comes from a wild or unadapted relative, as in L285, which is actually a *cerasiforme* type of tomato with very tiny fruit and other undesirable agronomic characters.

To identify the best individual for breeding in the F2 population of the L285 x CLN286 cross, we adopted the following strategy. The most desirable individual would be one that retained the resistant (L285) alleles in the regions around TG180b and TG230, plus a minimum of L285 germplasm everywhere else in the genome (remember that the resistance gene near TG51b comes from the recurrent parent, CLN286). To make this selection, we utilized a Macintosh software program known as "HyperGene" (Young, et al., 1989a), which can prepare drawings of an F2 plant's genome based on its DNA marker data and also makes it possible to select among the F2 individuals for those with specific combinations of genomic segments.

Among the 71 F2 individuals examined, two plants, numbers 49 and 57, were found to be best suited for subsequent breeding. Each plant retained the desirable (L285) alleles near TG180b and TG230 and carried a relatively low proportion of L285 DNA elsewhere in the genome (Figure 2). In the case of plant #57, TG180b was heterozygous and TG230 was homozygous for L285, while the remainder of the genome was less than 35% composed of L285 germplasm (compared to 52% for the F2 population as a whole). In the case of plant #49, both TG180b and TG230 were both homozygous for the L285 allele, while the remainder of the genome was 45% derived from L285. These plants have since been backcrossed to CLN286 and the progeny will be analyzed with DNA markers to identify the best progeny individuals for the next round of crossing.

DISSECTING EFFECTS OF INDIVIDUAL BACTERIAL WILT LOCI

Just as DNA markers can be used to select the best individuals for breeding, they can also be used to select the best plants for examining the roles of individual resistance loci. By selecting a plant that is fixed (homozygous) for all putative resistance loci but one, the role of that one remaining locus can be examined individually. As an example, consider plant #49 described above (Figure 2). Note that this individual is homozygous for the L285 allele at both TG180b and TG230, and would therefore be expected to be homozygous for resistance at these loci after selfing. However, the region around TG51b is heterozygous, and this is the locus associated with organ-specific resistance to bacterial wilt. By selfing plant #49 and then examining the resistance properties of the progeny in relation to their TG51b genotype, the biology of the putative resistance locus in this genomic region can be studied further. For example, the relationship between TG51b and organ-specificity, as well as different strains of *P. solanacearum* and various environmental conditions, can now be examined in detail.

HIGH RESOLUTION MAPPING AROUND PARTIAL RESISTANCE GENES

While genetic mapping of complex polygenic characters with DNA markers determines the number and approximate genomic location of the underlying genes, the technique does not precisely locate them. There is an inherent "fuzziness" in using DNA markers to map quantitative trait loci (such as partial resistance genes). Nevertheless, selecting individuals with a minimum amount of linkage drag around target genes (Young, et al., 1989b),

as well as map-based gene cloning (Young, 1990), in which genes of interest are cloned based solely on their chromosomal location, both require high resolution genetic mapping. Fortunately, there is a strategy based on marker-assisted selection that enables high resolution mapping, even for quantitative trait loci such as the partial resistance genes.

Precise linkage mapping requires crossovers near a gene to define its exact location. In an F2 population, where multiple partial resistance genes are all segregating simultaneously, individuals with recombinations occurring near one gene will be expected to show variation in their resistance phenotype due to segregation at the remaining, unlinked resistance loci. Thus, it is more difficult to map genes underlying a polygenic character than major, single locus genes due to genetic variation from the other loci affecting the trait (plus any environmental variation). Nevertheless, DNA markers can be used to select individuals that are especially informative for high resolution mapping in a manner similar to selecting individuals for dissecting the roles of individual resistance loci.

Specifically, individuals that are fixed at all resistance loci but one and which also contain crossovers at different points in the region of a segregating resistance locus can be selected with DNA markers. These informative individuals can then be selfed or backcrossed, and progeny examined for disease response. By comparing their disease scores with the results of several tightly linked DNA markers in the genomic region of interest, the location of the target resistance gene can be determined with a precision approaching that of major, single locus genes. An approach similar to this has previously been used successfully for high resolution mapping of tomato fruit loci (Paterson, et al., 1990).

In the case of the partial resistance loci for bacterial wilt in L285, several potentially informative individuals have been uncovered through DNA marker analysis. Because of the very large proportion of the total variation attributed to TG180b after root inoculation, individuals with recombinations in the region of this locus, but fixed for the susceptible allele at TG230, have been selfed and backcrossed for further analysis. At the same time, additional DNA marker loci that are known to map to the area of interest on chromosome six, near TG180b, are being examined. Eventually, this line of research may lead to the precise localization of the partial resistance locus in this region of the genome, perhaps to a degree sufficient to initiate physical mapping and map-based cloning.

DNA MARKER MAPPING AND RESISTANCE IN THE FIELD

The results described in this chapter demonstrate that partial resistance genes for bacterial wilt can be mapped using DNA genetic markers. The results also show that this kind of mapping information can be used for breeding and genetic dissection of bacterial wilt resistance in tomato. For example, two major partial resistance loci have been uncovered and another resistance locus that appears to be organ-specific in its action has been characterized. The best F2 lines for breeding, as well as for distinguishing the individual roles and precise map locations of partial resistance genes, have also been identified with DNA markers. However, a serious limitation to these studies, thus far, has been that they are based on disease assays in highly controlled, growth chamber experiments with a single strain of *P. solanacearum*.

In the future, experiments to examine the genetic basis of L285 resistance in the field with different strains of *P. solanacearum* will be essential. Mapping bacterial wilt resistance as a quantitative character with DNA markers, as described in this chapter, should be entirely suitable for experiments involving field scoring. Nevertheless, we will still need a new type of mapping population to examine the genetics of resistance with several different *P. solanacearum* strains in several different locations. The best type of population for this purpose is known as a *recombinant inbred* population (Burr, et al., 1991). Recombinant inbreds are sibling inbred lines, derived by single seed descent from individual F2 plants and advanced to the F7 or F8 generation. At this point, each recombinant inbred line is homozygous for most of the genome and is also composed of a unique mosaic of linkage blocks from the original parents. As such, a population of recombinant inbred lines provides a permanent mapping resource that need only be analyzed in terms of DNA genetic markers once, and can then be tested in an unlimited number locations or with many different pathogen strains. A set of recombinant lines, derived from the cross between L285 and CLN286, is already being developed by collaborators at AVRDC and will soon be ready for testing.

CONCLUSIONS

DNA genetic markers offer many opportunities for the study of complex disease resistance in crop plants. In the past, it was difficult to breed for polygenic disease resistance, and analyzing the role of individual loci underlying a polygenic resistance character was nearly impossible. Recently, DNA marker technology has begun to make major strides in the genetic

analysis of complex agronomic characters (Stuber, et al., 1987; Paterson, et al., 1991). Now, the power of DNA marker technology needs to be applied to the many agriculturally important situations involving polygenic disease resistance. The results in this chapter indicate that bacterial wilt resistance in tomato is an excellent place to start.

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FIGURE LEGENDS

Figure 1. Bar chart illustrating relationship between the RFLP marker, TG180b, and bacterial wilt resistance. Seventy-one F2 progeny from a cross between a bacterial wilt resistant tomato line (L285) and a susceptible cultivar (CLN286) were assayed for bacterial wilt response and DNA genotype at the RFLP locus, TG180b. The F2 plants were split into three classes according to their TG180b genotype and the mean bacterial wilt response (± standard error) for each genotypic class was calculated. The differences were statistically significant at $p = 0.0001$.

Figure 2. Graphical genotypes for two plants (#49, top, #57, bottom) in the F2 population between L285 and CLN286. Graphical genotypes indicate the size and distribution of linkage blocks inherited from each parent of the population based on DNA marker data (Young and Tanksley, 1989a). In this figure, dark segments represent genomic blocks inherited from L285 and light segments represent genomic blocks inherited from CLN286. The locations of putative bacterial wilt resistance loci are highlighted by the name of the DNA marker showing the most significant association with resistance. Because the tomato linkage map used in the present study is still incomplete, the assignment of the linkage groups shown in this figure do not necessarily correspond to the 12 chromosomes in the tomato RFLP map described in Tanksley, et al. (1988).

FIGURE 1

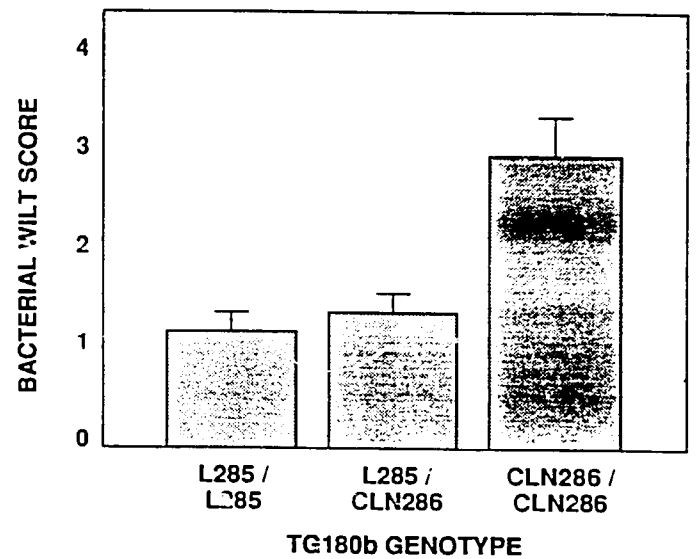
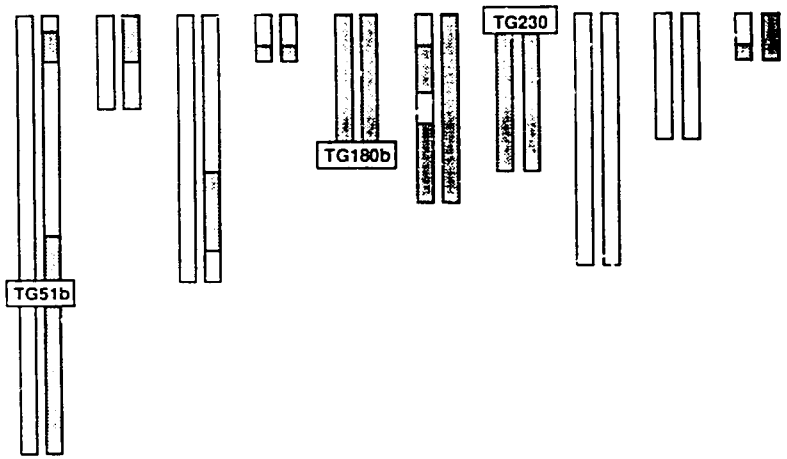
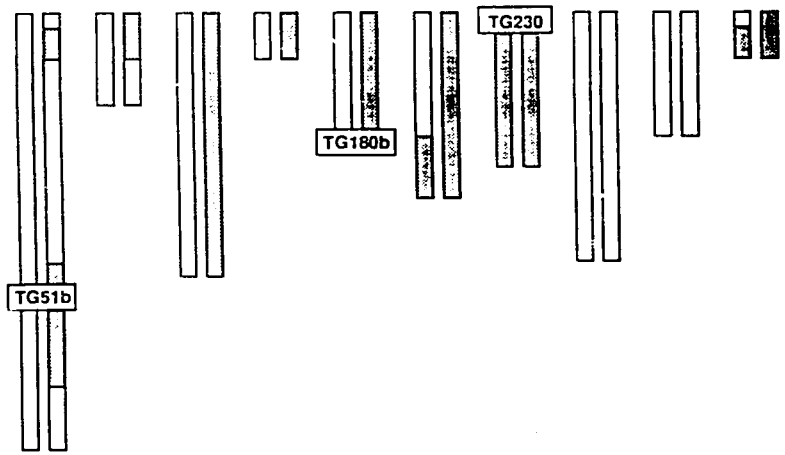


FIGURE 2

F2 PLANT #49



F2 PLANT #57