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Polygenic Resistance of Tomato Plants to Bacterial Wilt in the French West Indies

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Analysis of wilting symptoms using F_2 clones and F_3 families of plants derived from a cross between the resistant tomato cultivar Hawaii7996 and the susceptible *Lycopersicon pimpinellifolium* line WVa7OO inoculated with a strain of bacteria (GMI8217, derived from an endemic race 1 pathogen strain) permitted the detection of six quantitative trait loci (QTL) that may be important for field resistance to *Ralstonia solanacearum*. Three QTL, one on chromosome 6 and two QTL on chromosome 4, confirmed results previously obtained in tests done in a culture chamber (P. Thoquet, J. Olivier, C. Sperisen, P. Rogowsky, H. Laterrot, and N. Grimsley, MPMI 9:826-836, 1996). Two putative new QTL were found on chromosomes 3 and 8. In addition, a weak putative QTL previously detected on chromosome 10 was again observed in the field. One QTL on chromosome 11 was found to be specific to F_2 clones.

Additional keywords: Pseudomonas solanacearum, Burkholderia solanacearum.

While bacterial wilt caused by *Ralstonia solanacearum* is a disease that affects many species worldwide in warm climates, the severity of attacks caused by this soil-borne vascular pathogen is known to vary considerably according to climate, cropping practices, soil type, and geographic location (Hayward 1991). Five races of the pathogen, comprising hundreds of different strains, have been classified according to host range (Buddenhagen 1962). In Guadeloupe, where race 1 strains of the pathogen prevail (Prior and Steva 1990), the most severe damage occurs in vegetable plants, particularly in tomato, potato, and eggplant.

As the bacterium resides in the soil, chemical methods for control are largely ineffective and environmentally unacceptable. Breeding for resistance, more appropriately described as tolerance (Grimault and Prior 1993), to *R. solanacearum* remains the principal means of controlling the disease, but is painstakingly slow and has met with limited success due to the nature of the disease. In tomato, it has been bred into some cultivars from *L. pimpinellifolium* or from *L. esculentum* var. *cerasiforme* (reviewed by Laterrot 1989). *Lycopersicon* spp., with a densely marked RFLP map (Tanksley et al. 1992) and on which diverse studies on plant pathology have been conducted (Agrios 1988), is thus particularly suitable as a model for investigation of loci involved in resistance to bacterial wilt. Resistance depends on the environment, varying according to geographic location (Prior et al. 1994), probably as a result of different endemic bacterial strains, the physiology of the plant, the type of soil, and the climate.

In the preceding paper (Thoquet et al. 1996) the molecular and clonal analysis of an F_2 population was described, which permitted localization of QTL important for resistance to *R. solanacearum* on chromosomes 3, 4 and 6, using molecular markers. Our main objectives in this study were to (i) genetically map loci important for resistance to bacterial wilt in Guadeloupe and (ii) to compare these loci with those found using the same strain as an inoculum in a controlled environment growth chamber.

RESULTS

Analysis of clonally propagated F_2 plants in the field.

One series of cuttings from the F_2 population in Toulouse (Thoquet et al. 1996) was sent to Guadeloupe by air courier for a field test. Once the plants had acclimatized, they were inoculated and transferred to the field. Wilting symptoms appeared slowly, but at the end of the test levels of symptoms were comparable to those seen in the F_3 population (see Fig. 1 and below). Several putative QTLs were identified at a low

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level of confidence (Table 1) using the Kruskal-Wallis test option of MapQTL (van Ooijen and Maliepaard 1996). Most markers on chromosome 6 were associated the phenotype (P = 0.05) and the association was more significant around K4c (P = 0.005). A very significant effect was observed on chromosome 11 at marker O10 ($P < 0.001$). These results confirm the presence of a QTL on chromosome 6, as found in Toulouse (Thoquet et al. 1996), and give additional support to the presence of a locus on chromosome 11.

Analysis of segregation in an F_3 population Hawaii7996 × WVa700.

F_3 families of 20 plants from each of the 200 F_2 individuals and 150 of each of the parental controls were tested in the field in Guadeloupe in summer 1993. Some plants were lost due to heavy rains and attacks by pathogens other that *R. solanacearum*, leaving an effective F_3 population size of about 3,500 F_3 individuals. Wilting progressed rapidly in the cultivar WVa700 and in the F_3 population between days 2 and 10, then evolved more slowly (Fig. lB). Although a scale was used to classify disease severity, at 28 days after inoculation 97.4% of the F_3 population was noted either as fully healthy or as fully wilted. The level of resistance of each F_3 family was thus assessed by the proportion of plants wilted within that family. Only a few Hawaii7996 plants showed symptoms, and none of them wilted completely. The distribution of the levels of resistance observed in the F_3 population resembled a normal distribution (data not shown), and suggests that the character is controlled by several genes, none of which has a sufficiently strong effect to permit discrete phenotypic classes to be recognized.

Using a previously established genetic map covering about three quarters of the genome with 74 markers (Thoquet et al. 1996) together with data collected from the field trial, at least six chromosomal regions showing putative QTLs associated with resistance to *R. solanacearum* in the F_3 were located by interval mapping using the software MapQTL (van Ooijen and Maliepaard 1996) (Fig. 2). As in the F_2 analyzed in Toulouse (Thoquet et al. 1996), chromosome 6 is very strongly associated between the markers *AscPer* and TG240 with a maximum LOD score (7.5) close to TG118. The putative QTL previously detected on chromosome 4 (Thoquet et al. 1996) were again found, with a maximum LOD score of 3 around marker K12 on the upper arm, and a possible QTL putatively placed on the lower arm (GP165, LOD = 2).

Additional weak putative QTL associated with the resistance, not detected in any of the previous F_2 analyses, were observed on the lower arm of chromosome 3 (LOD = 3.0), the upper extremity of chromosome 8 (LOD = 2.0), and on the lower extremity of chromosome 10 (LOD = 2.0) (Fig. 2).

Analysis of segregation in F_3 families in the culture chamber.

To examine the reproducibility of wilting tests and to compare the variation between certain F_3 families in the field and in Toulouse some tests were done using a greater F_3 family size under culture chamber conditions. Two F_3 families were chosen on the basis of their genotypes at representative loci, their phenotypes in the F_2 and F_3 population analyses, and using the available genetic material. These lines, showing a specific genotypic difference concerning the locus present on chromosome 3, were retested in growth chamber conditions. At the genomic region around GP226 they were homozygous either for H7996 alleles or for WVa700 alleles, whereas they were homozyous H7996 at other loci that were identified as contributing to the phenotypic variation (Table 2). The phenotypic effect around GP226, accounting for 5 to 10% of the total variation in the F_3 population, was not visible in the F_2 clonal analysis done in Toulouse (Thoquet et al. 1996). This observation could arise either because the locus represents a field-specific effect or because of technical limitations in the F_2 clonal analysis (such as sample size or between-cutting variation). If the latter case were true, we hypothesized that it might be possible to differentiate between these lines under culture chamber conditions by testing 100 progeny from each F3 family. Comparison of the data obtained from the two families did show that they were significantly different ($P < 0.001$ that the families are the same 21 days after inoculation, by comparing classes using a χ^2 test, Table 2).

DISCUSSION

In our material, resistance to bacterial wilt segregates in a complex way, the different tests revealing that seven different

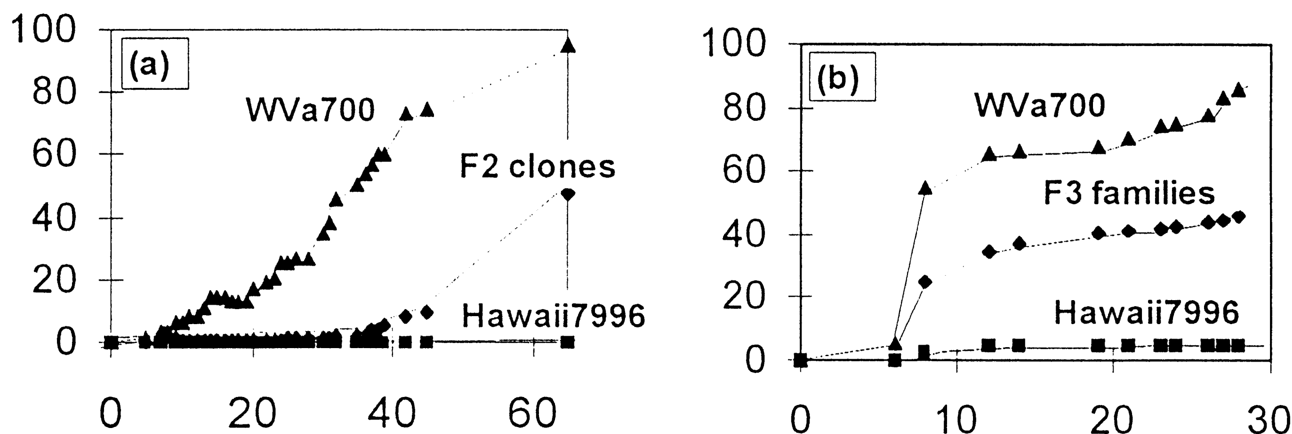

Fig. 1. Temporal progression (abscissae, days after inoculation) of percentage (ordinates) of plants wilted in the field. **A,** The F_2 population of 200 individuals is compared with 40 clones of each parental line (Hawaii7996 or WVa700). All plants were grown from cuttings. **B,** The F_3 population (3,500 plants) compared with the same parental control lines (100 of each). All plants were grown from seed.

loci may be involved. All of the loci carrying a part of the resistance come from the cultivar Hawaii7996, simplifying interpretation of the results.

Since only one field test on the clones of the F_2 population was done, these data must be considered less representative than either the nine replications previously done in Toulouse or the field trial on F_3 families. Nevertheless, the results obtained were consistent with the other tests, chromosomes 6 and 11 showing the strongest effects. Although the effects on the lower half of chromosome 4 and on chromosome 3 are too weak to be considered significant by themselves, they were found in previous tests, and thus add weight to the conclusion that QTL may be present at these locations. The remaining putative effects identified (Table 1) are too weak to be considered as QTL without any other supporting data.

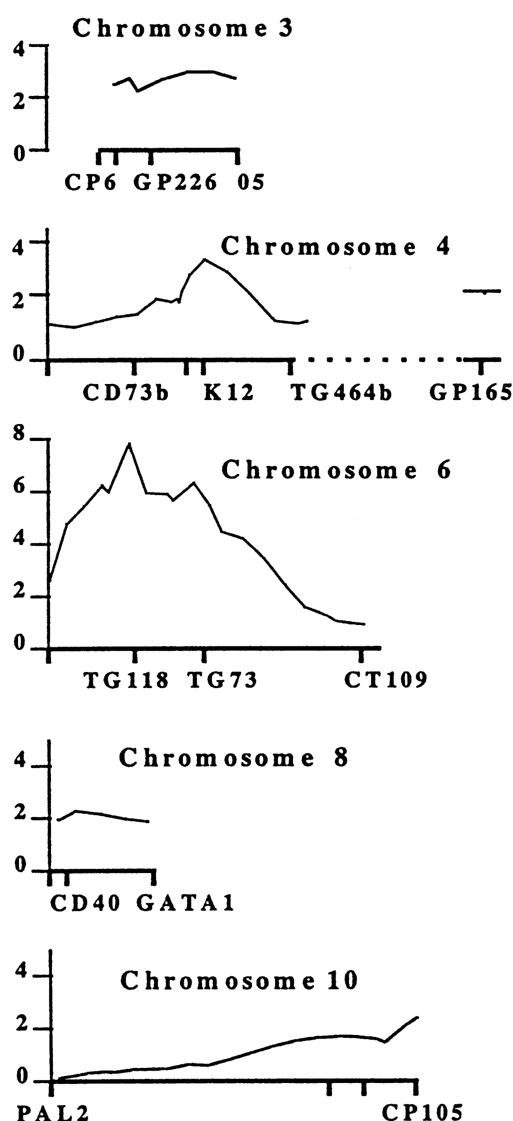

Fig 2. Interval mapping using data collected from the F_3 field trial. The LOD scores (ordinates) with which QTL were attributed to linked groups of markers (abscissae, Thoquet et al. 1996) are shown.

The general importance of the locus on chromosome 6 for resistance to *R. solanacearum* is confirmed; it contributes the largest effect to the variation in all tests. A very broad QTL peak, with markers spanning about 56 cM of the chromosome scoring LOD > 2, is observed. In the field, nematode attacks increase the problems associated with bacterial wilt, as mechanical damage facilitates entry of the bacteria (McGuire 1960; Prior et al. 1994). It is therefore highly desirable to have these two characters in combination. Resistance to *R. solanacearum* is difficult to combine with resistance to nematodes governed by the locus *Mi* in breeding programs (Acosta et al. 1964). We therefore hypothesize that one important locus could lie close to *Mi* (Weide et al. 1993) in the region of TG178, which shows an association with wilting of LOD =

Table 1. Markers showing linkage to resistance in the F_2 clonal analysis

Marker[a]	Chromosome	K	p
GP226	3	8.6	0.05
GPI65	4	6.3	0.05
CD186	5	5.4	0.05
Ascper	6	4.2	0.05
K4c	6	8.5	0.005
TG178	6	6.4	0.05
CD67	6	8.4	0.05
TG232	6	7.9	0.05
TG325	6	8.0	0.05
TG153	6	6.5	0.05
CP18	6	6.6	0.05
TG73	6	6.7	0.05
CT225	9	7.2	0.05
GP88	9	6.0	0.05
D6b	11	4.4	0.05
O10	11	16.4	<0.001

[a] Markers with $P > 0.05$ are not shown. K: Values of the Kruskal-Wallis statistic.

Table 2. Comparison of resistance between two F_3 lines differing genetically at a putative QTL on chromosome 3

	Marker	F_3 line 41	F_3 line 114	Hawaii-7996	WVa-700
Genotype[a]					
Chromosome					
3	GP226	A	B	A	B
4	TG268	H	H	A	B
4	GP165	A	A	A	B
6	TG178	A	A	A	B
6	CP18	A	A	A	B
6	TG240	A	A	A	B
6	TG162	A	A	A	B
6	CT109	A	A	A	B
8	CP112	A	A	A	B
10	TG229	A	A	A	B
10	GP87	A	A	A	B
10	CP105	A	A	A	B
Phenotype[b]					
Observed number of plants per class					
Fully healthy		94	73	49	1
Dead or diseased		6	27	1	49
Total		100	100	50	50

[a] Markers representing genomic regions of putative QTL are shown. A: homozygous Hawaii7996; B: homozygous WVa700; H: heterozygous.
[b] Twenty nine days after inoculation with *Ralstonia solanacearum* they differed significantly (P [χ^2] = 6×10^{-5}). Whereas F_3 line 114 differed significantly to the resistant parent (P[χ^2] = 0.0008) line 41 did not (P[χ^2] =0.29)

5.3 in the F_3 population. At TG240, over 30 cM from TG178, the LOD score (4.3) remains very significant; the question of whether a second QTL lies in this region therefore remains to be resolved. The QTL on chromosome 6 observed by Danesh et al. (1994) reaches a maximum LOD around CT184, at the same map position as TG240 (Tanksley et al. 1992), also supporting the notion of another important locus here. The overriding importance of the QTL on chromosome 6 may explain to some extent why, in the F_2 population from a different cross and in certain conditions, segregations consistent with the presence of one main effect may be found (Grimault et al. 1995). However, in the absence of molecular markers, these authors could not assess the contribution of different genomic regions to the total variation observed.

Two putative QTL were found on chromosome 4 in the F_3 population from Hawaii7996 × WVa700. These associations were found in several of the previous F_2 tests done in Toulouse, and a weak association was also found in the F_2 population in Guadeloupe. Several putative new QTL, which were not previously found in the tests done in Toulouse, were observed in the F_3 analysis on chromosomes 3, 8, and 10, albeit at a low threshold of detection (LOD = 2).

Although no effects were found on chromosome 7 or the upper region of chromosome 10, in contrast to previous work on another cross (Danesh et al. 1994), another putative QTL was found on the lower extremity of chromosome 10. In the F_2 in Toulouse (Thoquet et al. 1996) we did find a weak effect, but this was not considered to be a significant, because such associations could arise by chance. Since the LOD score in the F_3 analysis is low, further work is required to confirm the presence of a QTL in this region.

A putative QTL, linked to TG181, specifying resistance was previously noted in the upper region of chromosome 8 in L285 (Aarons et al. 1993), but this observation was not confirmed in a later paper (Danesh et al. 1994). Whereas Aarons et al. (1993) report that this resistance effect is derived from the sensitive CLN286 parent, in our material the resistance is derived from H7996. Thus there might be another weak QTL in this region. Interestingly, a family of genes encoding polyphenol oxidases, genes which are important in general plant defense responses (Newman et al. 1993) also maps in this region. Since these genes are characterized in detail, molecular studies can now be done to clarify their possible role in resistance to *R. solanacearum.*

Although the QTL on chromosome 3 was not found in the F_2 population in Toulouse, it was found in the F_3 population in Guadeloupe, where about 20 plants per F_3 family were tested, and again on retesting a larger number (100 individuals) of certain F_3 lines under growth chamber conditions. In the latter case, these lines were not significantly different when they were compared using the numbers of completely wilted plants, but they were clearly different when the number of plants showing low levels of disease were compared. This result is not surprising, as these lines carry a great majority of the H7996 alleles specifying resistance, and the small number of individuals found in the severely diseased classes precludes a meaningful comparison. This weak QTL was therefore not field specific, but a general effect that could not easily be found in the analysis done using F_2 cuttings. In contrast, the QTL on chromosome 11 was found in the most severe test group done on cuttings in Toulouse (Thoquet et al. 1996, represented by the probe GP162), but not in the F_3 field trial, suggesting that it may be an undefined physiological effect, since these plants were subjected to the trauma of propagation by cuttings. The xylem vessels of older plants might also have higher levels of lignification than those found in younger plants (the F_2 population was kept by clonal propagation for a period of 2 years during this study).

Overall, more of the phenotypic variation could be accounted for in the F_3 population (about 60%; chromosome 3 = 10%, chromosome 4 = 10 to 15%, chromosome 6 = 20%, chromosome 8 = 10% and chromosome 10 = 5%, neglecting between-locus interactions and ignoring overestimations that may arise by choosing those markers that show the effects) than in the different groups of the F_2 tests done in Toulouse (Thoquet et al. 1996), permitting weaker putative QTL to be detected. This increase in sensitivity probably arises because it was possible to evaluate a larger population under one set of climatic conditions in the field. The phenotypic variation remaining genetically unexplained may arise either from the presence of QTL in regions of the genome that are not covered by markers (chromosome 12, the upper part of chromosome 3, or the center part of chromosome 8) or from environmental variation.

However, several observations lead us to believe that the QTL detected account for most of the genetic variation segregating in the population. Firstly, when two F_3 families descendants from F_2 individuals having similar genotypes at all loci associated with the resistance phenotype were compared (100 individuals of each family) under culture chamber conditions in Toulouse, no significant difference was found between the lines (data not shown). Secondly, when lines that were genetically similar to the resistant parent except for the locus on chromosome 3 were compared (100 individuals of each F_3 family), their levels of resistance approached that of H7996. Thus the remaining phenotypic variation seems most likely to be environmental rather than due to the presence of other undefined QTL.

Although the previous tests on the clonal F_2 population were done in a controlled environment culture chamber, the age and physiological state of the mother plants did vary with time, and this undoubtedly contributed to between-test variation and probably masked the genetic effects of certain QTLs, such as those on chromosomes 3, 8, and 10. However, unlike the tests done in growth chambers, in the field it was not possible to control the nature of the inoculum as precisely. Presence of the bacterial strain used for inoculations was confirmed (resistance to rifampicin and streptomycin) in all dying (stage 9) plants, and many of the resistance plants also contained this strain at the end of the test, but we cannot exclude the possibility that some plants were additionally infected by another indigenous (Prior and Steva 1990) strain. No uninoculated plants were planted in our test plant plot, but disease symptoms did develop on some uninoculated plants in another field on the same site. The F_3 field results confirm and extend observations made by analysis of the F_2 segregation data.

Since we deliberately chose a low threshold for the detection of putative QTL, it is possible that one (or more) of these weak QTL might have arisen by chance, when only the information gained in the tests done in Guadeloupe is taken into consideration. Thus, for example, by applying more stringent

threshold criteria to our data (Rebai et al. 1994), taking into account all of the linkage groups and markers studied, we find that the effects on chromosome 4 could arise by chance in about 1 in 5 cases. Technical limitations (the difficulty of disease resistance tests, an F_2 population size of only 200 individuals for the molecular analysis, and the low level of polymorphism) preclude the calculation of QTL map positions with more certainty at this stage. Presentation and discussion of such effects is, however, justifiable in the context of tests done previously (Thoquet et al. 1996) or work done in a different cross (Danesh et al. 1994), and future work which can be undertaken to refute or confirm our interpretations.

Although useful levels of resistance have been obtained in some crop species such as tobacco and groundnut, breeding of tomato and potato lines with good levels of resistance to *R. solanacearum* has so far met with only limited success. A combination of factors make breeding for resistance a long and difficult task: The great diversity of bacterial races (different places having different endemic bacterial populations), environmental effects (especially loss of resistance at high temperature), and genetic linkage of genes controlling resistance to characters of agronomic importance.

In the long term, identification of molecular markers linked to QTLs will facilitate the development of tomato genotypes adapted to different geographic locations where *R. solanacearum* is a serious barrier to crop production.

MATERIALS AND METHODS

Plant material.

Two kinds of plant material were prepared. Firstly, a set of F_2 clones from cuttings of the Toulouse F_2 population (grown in perlite to meet phytosanitary requirements) was sent by airmail courier to Guadeloupe. These plants were kept in shade for 10 days after arrival to acclimatize before transfer to the field. Secondly, 30 F_3 seed from each F_2 plant in Toulouse, treated with sodium hypochlorite solution before export to meet phytosanitary requirements, were sown in compost, and after development of the first true leaf were transferred to pots, before inoculation and transfer to the infested field.

Inoculation with *R. solanacearum*.

Bacteria (strain GMI8217, A. Trigalet, personal communication, resistant to streptomycin and rifampicin, a derivative of GT1, Prior and Steva 1990) were grown as previously described (Thoquet et al. 1996) and the plants were inoculated under shade cover, using 2 ml of bacterial suspension per plant. Plants were kept under shade for a further 2 days before transfer to an infested field.

Notation of disease symptoms.

Disease symptoms were noted in all cases on a scale of 1 (healthy plant) to 9 (completely wilted plant). F_2 plants (from cuttings) were, in general, noted daily, whereas the F_3 population was noted every 2 days. In the F_3 population, individual plants were finally classified 28 days after inoculation as wilted (classes 5 to 9) or healthy (classes 1 to 4). Each F_3 family was then scored by the proportion of wilted plants within that family. In all cases, control and test plants were assigned individual numbers and distributed randomly to minimize environmental effects.

Molecular and genetic analyses.

The map of molecular markers used was previously described (Thoquet et al. 1996). The analysis of the F_2 population propagated from cuttings was done using the Kruskal-Wallis rank-sum test (see e.g., Lehmann 1975), and interval mapping was used to locate QTL in the F_3 population. Both kinds of analyses were done using the MapQTL software (Van Ooijen and Maliepaard 1996).

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