Resistance to *Phytophthora infestans* in *Lycopersicon pennellii*

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ABSTRACT

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To determine if the desert tomato, *Lycopersicon pennellii*, possesses resistance to late blight, caused by *Phytophthora infestans*, two plant populations were analyzed. Resistance was identified through assessments of disease progress in an F₂ mapping population (*L. esculentum* × *L. pennellii*) and in a series of introgression lines (*L. pennellii* into *L. esculentum*). Levels of resistance varied widely among individuals within each population. However, the response of individuals to different strains of *P. infestans* was consistent. In the mapping population, a quantitative trait locus (QTL) was detected near marker T1556 on chromosome 6. This QTL accounted for 25% of the phenotypic variance in the population. The occurrence of this QTL was confirmed from analysis of the introgression lines (ILs), where IL 6-2 (containing marker T1556) was the most resistant IL in 2002 and the second most resistant IL in 2001. The identification of an additional QTL for resistance to late blight in tomato will aid in the development of durable resistance to this devastating disease.

Additional keyword: potato

The oomycete pathogen *Phytophthora infestans* causes the late blight disease of potato, tomato (7), and other Solanaceous plants (2). Host resistance should be an important component of a sustainable disease management system for both potato and tomato, but is not yet widely used. Fortunately, some resistant or tolerant cultivars with commercially acceptable horticultural traits are becoming available. The potato cv. Defender was released in 2004, and possesses both foliar and tuber resistance to late blight infection (28). In Europe, a study of potato cultivars grown in an organic agriculture setting in four countries identified many cultivars that were far less susceptible than the more popular cultivars currently grown (34). Tomato breeding lines with resistance against multiple clonal lineages of *P. infestans* also have been released (20). Even with these exciting successes, the number of resistant cultivars available is still quite limited and there is a need to continue to identify new sources of resistance.

Initial efforts to identify resistance focused on single genes with large effects (*R* genes). Such resistances first were described in potato (38), but also have been reported (as *Ph* genes) in tomato (15).

These include *Ph-1* on chromosome 7 (6), *Ph-2* on chromosome 10 (26), and *Ph-3* on chromosome 9 (5). However, except possibly for a gene recently discovered from *Solanum bulbocastanum* (33), this type of resistance is not effective against all *P. infestans* genotypes. Pathogen types unaffected by an *R* gene are described as “compatible.”

Selection for compatible genotypes in the pathogen population leads to the “breakdown” or “instability” of resistance (35). Because of selection imposed by the most popular cultivars of potato and tomato, such *R*-gene-containing cultivars quickly “become” susceptible—a classic example is described by Malcolmson (24) and the more general situation is described by Wastie (38). Thus, *R*-gene-mediated resistance to late blight has had a much smaller role than is desired. Researchers have identified several broad-spectrum resistance genes from the wild *Solanum* spp. *S. bulbocastanum* and *S. phureja*. The *S. bulbocastanum* genes *RB* (27) and *Rpi-blb1* (37) have been cloned, and are known to be identical and located on chromosome 8. The second *S. bulbocastanum* gene, *Rpi-blb2*, also has been cloned and is located on chromosome 9 (36). A major locus, *Rpi-phu1*, has been identified in *S. phureja* on chromosome 9 and also confers broad-spectrum resistance to late blight (31).

Another method for the development of durable resistance is to utilize many genes, each of which confers some resistance to the pathogen. The expectation is that polygenic resistance will be stable. Several examples of stable resistance to late blight

have been published (17,18). One way to dissect chromosomal regions responsible for a stable reduction of disease is to analyze segregating populations and identify quantitative trait loci (QTLs) for resistance, which then would be used in breeding programs. It may be that several QTLs will need to be combined to obtain a sufficient level of resistance. This tactic is feasible in diploid plants such as tomato, where QTLs for resistance to late blight from *Lycopersicon pimpinellifolium* (11) and from *L. hirsutum* (3,4) have been reported.

Although there are many uses of the term resistance, in this study the type of resistance we identify is not the complete absence of disease. Rather, when studying populations, we consider the entire spectrum from susceptibility to resistance; therefore, resistance is identified by comparison with a plant that is more susceptible (that has more disease). The goal of this study was to determine whether *L. pennellii* possessed useable resistance to *P. infestans*. In an initial greenhouse screening, *L. pennellii* was found to be more resistant than the *L. esculentum* genotypes tested (*unpublished data*). This wild tomato species, also known as the desert tomato, was optimal for our study for two important reasons. First, a mapping population of a *L. esculentum* × *L. pennellii* cross with a high-density molecular marker map was available (14); and, second, introgression lines (ILs) containing large chromosomal segments of *L. pennellii* introgressed into *L. esculentum* also were available (8). Our approach was to conduct controlled field experiments with tomato-specialized strains (22,32) of *P. infestans* and identify QTLs that could be utilized in breeding programs.

MATERIALS AND METHODS

Plant populations and pathogen isolates. Two different plant populations were used. The first was an F₂ mapping population (83 individuals) of the cross *L. esculentum* (LA925) × *L. pennellii* (LA716) (14). The second population was a set of 50 ILs derived from a separate cross between *L. esculentum* (M82) and *L. pennellii* (LA716) (8). Each IL has a single segment of *L. pennellii* chromosome introgressed into the *L. esculentum* background. The 50 lines together provided complete coverage of the tomato genome (8). The F₂ mapping population had been maintained as plantlets in tissue culture at

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Cornell University. Seed of each of the 50 ILs were provided by Greg Martin (Plant Pathology Department, Cornell University and Boyce Thompson Institute, Ithaca NY).

The field experiments occurred at the Homer C. Thompson Research Farm of Cornell University, near Freeville, NY. All plants were grown in Cornell soilless mix containing equal parts peat and vermiculite with nitrogen, phosphorus, and potassium, each added at the rate of 0.4 kg/m³ of mix. Plants were grown in a greenhouse with temperatures ranging from 24 to 29°C under natural sunlight which, in Ithaca, NY, was approximately 15 h per day from mid-May to mid-June. Plants were fertilized once per week with liquid fertilizer (nitrogen at 300 ppm). Prior to planting in the field, tissue culture plantlets of the F2 mapping population were transplanted and grown in the greenhouse for 2 weeks. Seed of the *L. pennellii* ILs were sown and grown in the greenhouse for 4 to 5 weeks. Plants were moved from the greenhouse to a cold frame 5 days prior to planting on 27

June 2001. The mapping population was planted in one location in the field and the ILs planted at another location in the same field. In the mapping population, each component of the genome was replicated many times; thus, we analyzed one replication of the mapping population in single-plant plots. The spacing among plants was 1 m in all directions. One replication of the ILs was planted in two-plant plots. Prior to transplanting, the soil had received N at 100 kg/ha, P at 100 kg/ha, and K at 100 kg/ha. Herbicides and hand weeding were used to suppress weeds. On 26 May, Treflan HFP (trifluralin) at 1.5 pt/A (Dow Agrosiences LLC, Indianapolis, IN) was applied and, on 26 June, Trilin (tifluralin) at 1.5 pt/A (Griffin/Dupont, Wilmington, DE) was applied. Insects were controlled with applications of Provado 1.6 F (imidacloprid) nitrogen 3.75 fl oz/A (Bayer Crop Science, Research Triangle Park, NC) on 6 July and 10 August.

In 2002, the IL population was analyzed again. Seedlings were transplanted on 20

June using two-plant plots in three randomized complete blocks. Fertilization was applied at planting at a rate of N, P, and K at 100 kg/ha each. Herbicides and hand weeding were used to suppress weeds. Treflan HFP (trifluralin) at 1.5 pt/A (Dow Agrosiences LLC) and Sencor DF (metribuzin) at 0.5 lb/A (Bayer Crop Science) were applied on 19 June.

Two different isolates of *P. infestans* were used. The first (used in 2001) was US980025 from the Cornell University *Phytophthora* culture collection. It was a member of the US-11 clonal lineage, which had been obtained originally from infected tomato in California in 1998 (16). The other (used in 2002) was US970001 (American Type Culture Collection no. MYA-2350), a member of the US17 clonal lineage (see Fry and Goodwin for a description of these clonal lineages; 13). Both isolates were specialized to tomato (21,22,32).

Field evaluations of disease development. Plots were inoculated on the evenings of 17 August 2001 and 24 August 2002. In each case, a sporangial suspension was sprayed onto plants that had been wetted via sprinkler irrigation (2 h at the rate of 0.2 cm/h). In each year, 450 sporangia (3 ml at 150 sporangia/ml) were applied per plot. Sporangia for inoculation were washed with water from sporulating lesions on tomato leaves about 4 h prior to inoculation. Sporangia from lesions are more robust for field studies than are sporangia from culture plates (25). The concentration of sporangia was measured using a hemacytometer for subsequent adjustment of the inoculum (to 150 sporangia/ml). Prior to dilution, the suspension was incubated at 4°C for 2 h to facilitate zoospore release. After inoculation, the plots again were irrigated (30 min at the rate of 0.2 cm/h). Subsequently, the plots were sprinkler irrigated three to four times each week for 1 to 2 h at 0.2 cm/h in the evening.

Percent disease (0 to 100%) was estimated visually every 2 to 4 days as the epidemic progressed, as previously described (9). Data were collected by the same individual throughout the experiment to remove person-to-person variation. Disease severity was estimated over the entire plot visually by determining the percent leaf tissue with late blight lesions. This was done using a modification (12) of a technique described by the British Mycological Society for the evaluation of late blight on potato (1). Previously developed standard diagrams were used with very low levels of disease (19). These data then were used to locate QTL using QGENE (27). QTL analyses were executed using both the point and interval analysis function of QGENE, using a cut-off value of $P < 0.01$ or log of the likelihood ratio (LOD) > 2.0 . These criteria for QTL detection follow the ones previously applied for QTL analysis in this same population (10).

Fig. 1. Diversity of response to *Phytophthora infestans* in the F2 mapping population in 2001. **A**, Sporulating lesions on a more resistant individual at 5 days after inoculation (dai). Note the sporulation surrounding a necrotic lesion. **B**, Sporulating lesions on a more susceptible individual at 5 dai, with sporulation across the total lesion. **C**, A more resistant plant at 14 dai. Some lesions could be found, but the plant was still alive and vigorous. **D**, A very susceptible plant at 14 dai. Most parts of the plant have been killed.

RESULTS

Disease developed rapidly on the more susceptible genotypes, with disease progression being more rapid in 2001 than in 2002. In both years, there was a wide range of resistance response to *P. infestans* in the populations. Some individuals were nearly dead within 14 days after inoculation (dai) (Fig. 1). No genotype was completely immune. The more resistant genotypes (those with the lowest percent disease) had a smaller number of lesions, and these were slower growing with sporulation limited to the periphery of lesions that had predominant necrosis (Fig. 1A). The more susceptible genotypes (those with higher percent disease) had rapidly growing lesions and, initially, necrosis was limited, with sporulation across the entire lesion (Fig. 1B). Both populations had large numbers of quite susceptible individuals, with more individuals resembling the reaction of the *L. esculentum* (M82) parent than that of the *L. pennellii* (LA716) parent (Figs. 2 and 3).

Analysis of the disease severity data for the mapping population in the 2001 experiment indicated that marker T1556 was common in the more resistant progeny. When the disease severity data were analyzed, the strongest QTL for resistance was from *L. pennellii*, chromosome 6, closest to marker T1556 (Fig. 4). This locus accounted for 25% of the phenotypic variance (based on regression analysis using Q-GENE software), with an LOD score of 3. Additionally, QTLs for susceptibility were identified in the F2 population on chromosomes 8 and 12. The QTL on chromosome 8 was linked to marker T1277 with an LOD score of 2.78, and on chromosome 12 was linked to marker CT211 (LOD = 2.19).

The response of the IL population to infection by *P. infestans* corroborated the resistance associated with marker T1556 identified in the F2 mapping population. The IL that contained the chromosomal segment from *L. pennellii* chromosome 6 with marker T1556 (IL6-2) was the second most resistant individual in 2001 (Fig. 2A) and was most resistant during the somewhat slower disease progression seen in 2002 (Fig. 2B).

One IL had resistance that was not detected in the F2 mapping population. IL1-1 was the most resistant IL in 2001 and also was highly resistant in 2002. In 2002, this IL had 48% disease at 14 dai, whereas IL6-2 had 31% disease.

DISCUSSION

We have identified a QTL for resistance to late blight from *L. pennellii*. To our knowledge, this is the first report of resistance to late blight in *L. pennellii*. This QTL is located on chromosome 6 near marker T1556. The phenotype of lesions on plants with this QTL is that of slower growth and restricted sporulation (limited

to the periphery of lesions). This phenotype is different from that of *Ph* genes, which completely prevent macroscopic lesion development of incompatible strains.

This QTL also appears to be novel based on map position. The *Ph* genes from *L. pimpinellifolium* (*Ph1*, *Ph2*, and *Ph3*) are located on chromosomes 7, 10, and 9, respectively. This QTL also is different from QTLs identified from *L. hirsutum*, which occur on chromosomes 4, 5, and 11 (4). However, there are other reports of resistance potentially on chromosome 6. Resistance to late blight was detected on chromosome 6 (precise location is not yet known) of *L. hirsutum* (3). And, in potato,

an *R* gene analogue (phenotype not yet described) has been located on chromosome 6 (23); but, again, it is not known if this locus is near marker T1556.

In addition to QTLs for resistance to late blight, the *self-pruning* (*SP*) locus, which confers the indeterminate phenotype in tomato, also is found on chromosome 6 (29). A single mutation in the *SP* gene results in a recessive allele (*sp*) which, when homozygous in a plant, results in a determinate growth habit. The majority of cultivated tomato plants, particularly those for processing, have a determinate growth habit which facilitates mechanical harvest. Several previous studies have identified

Fig. 2. Distribution of 50 individual introgression lines (ILs) in terms of disease severity at 14 days after inoculation in **A**, 2001 and **B**, 2002. IL6-2 and the *Lycopersicon pennellii* parent (LA716) were among the most resistant individuals. The *L. esculentum* parent (M82) was among the most susceptible individuals.

Fig. 3. Distribution of 83 members of the F2 mapping population of the cross *Lycopersicon esculentum* (LA925) $\times$ *L. pennellii* (LA716) in terms of disease severity 10 days after inoculation in 2001. The *L. pennellii* and *L. esculentum* parents were among the most resistant and most susceptible individuals, respectively.

late blight resistance in tomato that is linked to the *SP* allele at marker TG279 (39). However, an indeterminate growth habit could confound late blight ratings because new plant growth would appear healthy until an infection period would spread the pathogen to this new growth. Thus, the QTL for resistance at marker TG279 may be conditioned by the indeterminate growth habit rather than by actual host resistance.

The populations used in our study enable the differentiation between indeterminate growth and host resistance and allow the identification of a new late blight resistance QTL for several reasons. First, the F2 mapping population used in this study does not segregate for *SP*; both parents are indeterminate (14). Second, the late blight resistance QTL identified maps to marker T1556, which is approximately 15 centimorgans from *SP* (29; Fig. 4). Finally, the *SP* gene is contained on two different individuals within the IL population we used (IL6-2 and IL6-3). An increase in resistance was identified only in IL6-2, not in IL6-3. Because the late blight epidemic was rapid and severe in the field study, the indeterminate growth habit did not influence disease ratings.

The rapid disease progress in our study also could explain why the QTLs for susceptibility to late blight that were identified in the mapping population were not observed in the IL population. Because the M82 background is highly susceptible to *P. infestans*, it was difficult to observe an increase in susceptibility (due to the introgressed *L. pennellii* chromosomal fragment) because the majority of the plants were dead at 14 dai.

The magnitude of the effect of this late blight resistance QTL seems large enough

to have potential for utility in a breeding program. It accounted for 25% of the phenotypic variance in the F2 mapping population. In that population, disease severity at 14 dai ranged from 15 to 100% (Fig. 3). In the IL population at 14 dai (2001 experiment), most ILs were more than 90% diseased, but the individual carrying the resistance QTL, IL6-2, and the parental *L. pennellii* were both at 75% disease (Fig. 2A). In 2002, IL6-2 was only 35% diseased at 14 dai, when disease severity for the entire IL population ranged from 35 to 100% (Fig. 2B). It is very promising that the effect of the QTL was evident in a *L. esculentum* (M82) background, because this suggests that the QTL also will have efficacy in other cultivated tomato backgrounds.

At this time, the "stability" of this QTL remains unknown. However, the effect of the QTL was large in both years of assessment using two different tomato-specialized isolates of *P. infestans* that represent two different clonal lineages. The two isolates are genetically quite distinct and have different pathotypes (16,30). Certainly, the effect of this QTL on many different pathotypes of *P. infestans* should be determined.

The availability of a horticulturally acceptable cultivar (M82) and a near-isogenic IL (IL 6-2) of tomato that are susceptible and partially resistant, respectively, to *P. infestans* enables many future studies. By comparing host responses with the pathogen it may be possible to identify mechanisms by which the QTL on chromosome 6 enhances resistance to this devastating pathogen. Additionally, this QTL could be combined with others previously identified from *L. pimpinellifolium* and *L. hirsutum* to generate a du-

Fig. 4. Map of chromosome 6 from *Lycopersicon esculentum* with the quantitative trait locus for late blight resistance (star) from *L. pennellii* located near marker T1556.

table multigene form of resistance to *P. infestans*.

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