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ORIGINAL PAPER

S. Costanzo · I. Simko · B. J. Christ · K. G. Haynes

QTL analysis of late blight resistance in a diploid potato family of *Solanum phureja* × *S. stenotomum*

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Abstract Field resistance to *Phytophthora infestans* (Mont.) de Bary, the causal agent of late blight in potatoes, has been characterized in a potato segregating family of 230 full-sib progenies derived from a cross between two hybrid *Solanum phureja* × *S. stenotomum* clones. The distribution of area under the disease progress curve values, measured in different years and locations, was consistent with the inheritance of multigenic resistance. Relatively high levels of resistance and transgressive segregations were also observed within this family. A genetic linkage map of this population was constructed with the intent of mapping quantitative trait loci (QTLs) associated with this late blight field resistance. A total of 132 clones from this family were genotyped based on 162 restriction fragment length polymorphism (RFLP) markers. The genome coverage by the map (855.2 cM) is estimated to be at least 70% and includes 112 segregating RFLP markers and two phenotypic markers, with an average distance of 7.7 cM between two markers. Two methods were employed to determine trait–marker association, the non-parametric Kruskal–Wallis test and interval mapping analysis. Three major QTLs were detected on linkage group III, V, and XI, explaining 23, 17, and 10%, respectively, of the total phenotypic variation. The present study revealed the presence of potentially new genetic loci in this diploid potato family contributing to general resistance against late blight. The identification of these QTLs represents the first step toward their introgression into cultivated tetraploid potato cultivars through marker-assisted selection.

Keywords Linkage map · Quantitative trait loci · Late blight · *Phytophthora infestans* · *Solanaceae* · Resistance genes

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S. Costanzo · B. J. Christ (✉)
Department of Plant Pathology,
The Pennsylvania State University,
University Park, PA 16802, USA
E-mail: ebf@psu.edu
Tel.: +1-814-8632068
Fax: +1-814-8637217

I. Simko
Department of Natural Resource Sciences
and Landscape Architecture,
University of Maryland, College Park, MD, 20742

Present address: S. Costanzo · I. Simko · K. G. Haynes
Vegetable Laboratory, USDA–ARS–PSI,
10300 Baltimore Avenue, Beltsville, MD 20705, USA

Introduction

Since its devastating appearance in Ireland and other parts of Europe in 1845, late blight, caused by the oomycete *Phytophthora infestans* (Mont.) de Bary, has remained the most important disease in potato cultivation. Without preventive control measures, this disease can cause severe damage to both foliage and tubers, which can lead to complete crop loss. The occurrence of new, aggressive, metalaxyl-resistant isolates of *P. infestans* (Goodwin et al. 1995; Fry and Goodwin 1997) has intensified breeding efforts worldwide to incorporate durable genetic resistance into new potato varieties. Moreover, because no modern commercial cultivars of potato (*Solanum tuberosum* L.) have adequate resistance to late blight, the development of resistant potato varieties is currently a high priority in every potato breeding program.

Two types of genetic resistance to late blight have been identified in potato germplasm: (1) race-specific, controlled by individual resistance (R) genes (Black et al. 1953; Malcolmson and Black 1966) initially introgressed into potato from the Mexican hexaploid species *S. demissum*, and (2) general, field resistance, or race non-specific resistance that is partial and polygenic in nature (Umaerus et al. 1983), and regarded as effective against all strains of the pathogen. Despite initial success, the introduction of race-specific resistance has proven to confer only a transient and limited protection against late blight due to the ability of *P. infestans* to overcome rapidly such

resistance. Therefore, breeders have now directed their attention to enhancing general resistance against this pathogen since this type of resistance is thought to be more durable.

Despite the improved understanding, over the past several decades of quantitative trait genetics, only limited progress has been made in practical plant breeding due to difficulties in manipulating multiple loci simultaneously. More recently, the advent of molecular marker technology, particularly DNA-based markers, has provided new tools that are helping to determine the number, positions, and individual effects of resistance loci with quantitative inheritance. The genetic dissection of polygenic resistance against *P. infestans* in potato, *Solanum* spp., using various types of DNA-based markers, has been conducted primarily on diploid mapping populations (Leonards-Schippers et al. 1994; Collins et al. 1999; Oberhagemann et al. 1999; Ewing et al. 2000; Sandbrink et al. 2000; Ghislain et al. 2001), and only in two cases was a tetraploid population employed (Meyer et al. 1998; Naess et al. 2000). Results from these various studies have provided us with an improved understanding of the genetic control of resistance. Genetic components controlling race non-specific resistance against *P. infestans* appear to be located on almost all potato chromosomes, thus confirming the multigenic nature of general resistance. The most frequently reported quantitative trait loci (QTLs) for late blight are those detected on linkage groups V, III, and IV (Collins et al. 1999; Oberhagemann et al. 1999; Gebhardt and Valkonen 2001; Simko 2002). No individual QTL, however, was detected consistently in all genetic backgrounds. This can be explained by the presence of different alleles at any given QTL in the genetic materials analyzed, which may have different effects on resistance and which may interact differently with each other and in response to the environment.

A diploid hybrid population of *S. phureja* × *S. stenotomum* has shown relatively high levels of resistance to late blight under field conditions (Haynes and Christ 1999). From this original population, two diploid hybrid clones, BD142-1 (highly susceptible to late blight) and BD172-1 (partially resistant), were selected and crossed using BD142-1 as the seed parent. A full-sib family (BD410) of 230 segregating progeny was evaluated for late blight resistance in different locations in Pennsylvania between 1999 and 2002 (Costanzo et al. 2004). The objectives of the present study were to identify QTLs for late blight resistance in this population and compare them with those previously reported in other QTL mapping projects.

Material and methods

Plant material

Parental clones for the mapping population (BD410) were identified and selected in 1997 (Liu et al. 1998a) among 281 clones derived from 72 families of a diploid random-mated hybrid population of *S. phureja* × *S. stenotomum* (Haynes and Christ 1999). In both field and lab trials, clone BD142-1 was consistently highly susceptible to late blight, whereas clone BD172-1 had moderate but significant levels of resistance to it. Moreover, preliminary analysis indicated that DNA polymorphisms between these two clones were adequate for mapping purposes (Liu et al. 1998b). BD142-1, used as the female parent, was crossed with the male parent BD172-1, and a progeny of 230 individuals was generated. A subset of 132 randomly selected clones from the BD410 population was grown under greenhouse conditions and used for DNA isolation, restriction fragment length polymorphism (RFLP) marker analysis, and linkage map construction.

RFLP analysis and map construction

Total genomic DNA was extracted from approximately 5–10 g of leaf tissue from each of the parental clones and a total of 132 full-sib individuals, using a modified protocol derived from two procedures described by Porebski et al. (1997) and Doyle and Doyle (1987). Genomic DNA was digested with five restriction enzymes including *Hin*dIII, *Eco*RI, *Dra*I, *Xba*I, and *Taq*I and subjected to gel electrophoresis. Genomic blots were prepared and hybridized with 120 genomic or cDNA clones and ten resistance gene analogue (RGA) markers detecting polymorphism between the two parents. Probes were labeled with [α^{-32}P]-dCTP using the random hexamer procedure (Oligolabelling Kit, Amersham Pharmacia Biotech). Agarose gel electrophoresis, Southern blotting, hybridization, and autoradiography were as described by Sambrook et al. (1989). A genetic linkage map was constructed using a total of 132 markers including two morphological characters (flower and tuber skin color). Multipoint linkage analysis of the entire set of RFLP and phenotypic markers was performed using the software package JoinMap (version 3.0), and a genetic linkage map was constructed. The critical $\log_{10}$ of odds ratio (LOD) value of 2.0 or more was used to assign marker loci to linkage groups. The Kosambi mapping function (Kosambi 1944) was used to convert recombination frequencies to map distances in centiMorgans. Linkage groups were identified to represent specific chromosomes on the basis of the position of known RFLP "anchor" markers used in previously published potato and tomato maps (Gebhardt et al. 1991; Leister et al. 1996; Tanksley et al. 1992). The charts of genetic linkage and the overall chromosomal maps were produced using the computer package MapChart (Voorrips 2002).

Trait evaluation

The entire family, two parental clones, and three standard commercial cultivars ('Atlantic', 'Kennebec', and 'Katahdin') were included in the experimental design

and evaluated for reaction to late blight under field conditions. Three field experiments were conducted between 1999 and 2002 in Pennsylvania, using a randomized complete block design with two replications. Plots consisted of five plants of a clone planted 22 cm apart within rows, and the space between rows was 91 cm with a 1.2 m break between plots within rows. Each treatment plot had an adjacent row of the highly susceptible cultivar 'Russet Burbank' that was inoculated and acted as a spreader row. Approximately 5 ml of a sporangial suspension of *P. infestans* was inoculated onto plants adjacent to each plot and allowed to infect naturally the treatment plots. Three isolates of *P. infestans* US-8 (A2 mating type) were used to inoculate the spreader rows. These isolates had been previously tested on a set of potato late blight differentials possessing a single or a combination of the following R genes: *R1*, *R2*, *R3*, *R4*, *R5*, *R6*, *R8*, *R9*, *R10*, and *R11*. All of the tested R genes were clearly overcome by the *P. infestans* isolates used in the present study (B.J. Christ and R. Valluru, unpublished). Equal amounts of the three isolates were pooled together to constitute the inoculum, and subsequently, the sporangial concentration was adjusted to 1×10^4 sporangia per milliliter. Disease was assessed by estimating the percentage of necrotic tissue observed within each plot. Assessments were made at least once a week for a total of three or four times. Area under the disease progress curve [(AUDPC) Shaner and Finney 1977] was calculated for accurate assessments of quantitative disease resistance or susceptibility.

QTL analysis

Two analytical approaches were used to identify and validate putative QTLs against late blight segregating in the full-sib family using the established linkage map and the observed phenotypic traits. First, the non-parametric Kruskal–Wallis *K*-test: this method can be regarded as the non-parametric equivalent of the one-way analysis of variance (Hollander and Wolfe 1973; Lehmann and D'Abrera 1975), which measures the association between marker genotype and disease index. For the *K*-test, an association was indicated when the mean values of the marker classes were significantly different at $P<0.01$. The second approach employed for QTL mapping was the maximum likelihood interval mapping method (Lander and Botstein 1989). This method is an extension of one marker analysis, extending the analysis from an individual marker to any genomic region flanked by two markers. The LOD scores, i.e., the likelihood for the presence of a segregating QTL for each position on the genome, were obtained from MapQTL (version 4.0) software (van Ooijen et al. 2002). These LOD scores were used to construct QTL-likelihood plots of detected QTLs using the computer package MapChart (Voorrips 2002). In order to determine the significance threshold of the LOD score, the 1,000-times permutation test was calculated. This is a resampling method to obtain empirical significance threshold values (Churchill and Doerge 1994). A 5% genome-wide error rate was chosen to identify the LOD interval threshold. Based on the result of the permutation test, a LOD score of 2.4 or above was used to declare the presence of a segregating QTL. LOD peaks were used to determine the estimated position of QTLs on the map. The QTL analysis, with both methods, was implemented using the software package MapQTL, which also provided estimates of the percentage of the total phenotypic variation explained (PVE) by the most significant marker for each QTL. The Multiple QTL model (Jansen 1993) feature of MapQTL was used for inclusion of background markers into the QTL analysis as cofactors. Because addition of the background markers did not significantly affect the map location and the LOD values of detected QTLs, only results from the simple interval mapping are presented.

Results

Phenotypic data

Trait data for late blight were assessed and collected from three replicated field experiments conducted in central and southeast Pennsylvania between 1999 and 2002 (Costanzo et al. 2004). For each location, the average AUDPC value for each clone was calculated. The data were square-root transformed to improve the normality of their distributions, and analyses of variance were performed on individual and combined datasets. Among all genotypes, mean AUDPC values ranged between 140 and 820, with an average of 423, and exhibited a continuous and normal distribution consistent with polygenic inheritance (characteristic of a quantitative trait) (Fig. 1). The standard commercial varieties 'Atlantic', 'Katahdin', and 'Kennebec' had average AUDPC values of 800, 679, and 596, respectively. The mean AUDPC value across two years for the female parent (BD142-1) was 823 and 259 for the male parent (BD172-1). Transgressive segregation, particularly towards resistance to late blight, indicated the presence of positive and negative genetic factors in the parental lines for this character. Association between markers and resistance scores were tested on 132 progenies, separately for each individual location and again on a new dataset derived from the three combined experiments.

Genetic linkage mapping

Among the 162 markers originally selected and used for a survey of RFLPs between BD142-1 and BD172-1, 151 (93%) detected polymorphism between the two parental clones. There were no relevant differences for percentage of polymorphism among the different types of markers surveyed (cDNA vs genomic vs RGA). Of these last 151 probes, 23 (14%) did not produce a segregation banding

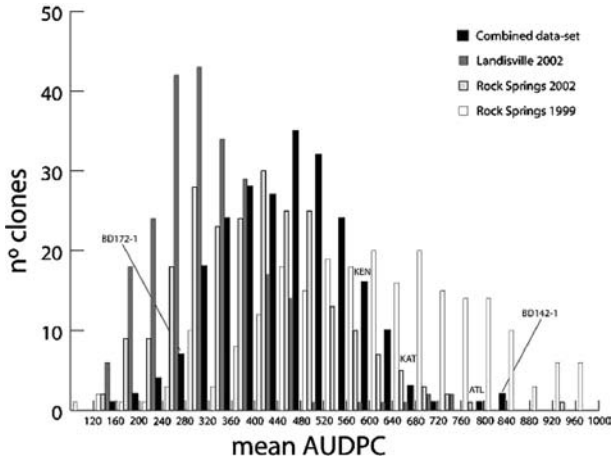

Fig. 1 Distribution by area under the disease progress curve (*AUDPC*) in three individual locations and averaged over the three locations of the 235 clones, including parental and standard cultivars, evaluated for percentage foliar late blight in Pennsylvania in 1999 and 2002

pattern in the progeny blots that could be clearly scored and were subsequently eliminated. Of the 31 probes derived from tomato, 29 of them (94%) detected polymorphism in the parental survey blots. Among them, seven produced unresolvable bands in the progeny blots and were therefore eliminated. Of the total 128 polymorphic markers identified, 114 (89%) met the minimum requirement for linkage at LOD≥2.0 and were used for map construction. Twenty-seven RFLP probes (21%) detected two or more marker loci. As a result of using non-inbred parents, five types of single-locus segregation were found: 1:1 ♂, 1:1 ♀, 3:1, 1:2:1, and 1:1:1:1. Distorted segregation of markers was determined using the χ^2 goodness-of-fit analysis. A genetic linkage map was constructed based on 112 segregating markers on a subset of 132 individuals using the computer program JoinMap (Stam 1993; van Ooijen and Voorrips 2001). A total of 126 loci were identified and placed into 14 linkage groups that could be matched to the 12 potato chromosomes (chromosomes X and XII were each represented by two separate linkage groups). This map covers, in total, 855.2 cM, in map units defined by Kosambi (1944), with an average interval length of 7.7 ± 0.63 cM between markers and with approximately ten loci per chromosome. Chromosome VIII had the largest linkage group (85.5 cM), followed by chromosomes IV and I (77.9 cM and 77.5 cM, respectively), whereas chromosome XII had the smallest one (33.1 cM), followed by chromosomes X and VII (44 cM and 58.1 cM, respectively). Genome coverage by the map is estimated to be at least 70%, based on mapping of subtelomeric repeats (Gebhardt et al. 1995). The overall length of the map and the linear order of the markers were in good agreement with previously published RFLP maps of potato (Bonierbale et al. 1988; Gebhardt et al. 2001; Tanksley et al. 1992). Considerable differences were found among relative genetic distances within certain marker intervals. Furthermore, some differences were also observed in the individual chromosome lengths between the compared maps (Table 1). Skewed segregation ratios (χ^2 test, $P < 0.005$) were observed; the strongest distortion was found on linkage group I and is assumed to be caused by the activity of the self-incompatibility locus (Gebhardt et al. 1991).

QTL analysis

Analysis on the trait dataset from the estimated multiple locations average

Results from the non-parametric Kruskal–Wallis test suggested the existence of QTLs for late blight resistance on linkage groups III and V (Table 2). A *P*-value of less than 0.01 was used as a threshold criterion for QTL detection. The QTL on linkage group III has its highest

Table 1 Comparison of map length (in centiMorgans) with previously published potato restriction fragment length polymorphism maps

Mapping population[a]				
Chromosome	*phu* × (2 × *tbr* × *chc*)[b]	(2 × *tbr* × 2 × *tbr*) × 2 × *tbr*[c]	(2 × *tbr* × *ber*) × *ber*[d]	*stn-phu* × *stn-phu*[e]
I	63	127	76	78
II	46	76	50	61
III	47	62	64	69
IV	55	102	65	78
V	47	78	56	72
VI	59	83	54	72
VII	55	92	59	58
VIII	64	79	49	86
IX	50	97	50	69
X	53	49	51	44
XI	21	96	48	74
XII	46	93	63	33
Total span	606	1,034	684	855
Total markers	135	492	261	132
Total loci	101	304	150	122

[a] *phu* = *Solanum phureja*, 2 × *tbr* = haploid *S. tuberosum*, *chc* = *S. chaocoense*, *ber* = *S. berthaultii*, *stn-phu* = selected clone from *S. stenotomum*–*S. phureja* bulk population
[b] Bonierbale et al. (1998)
[c] Gebhardt et al. (1991)
[d] Tanksley et al. (1992)
[e] This study

Table 2 Kruskal–Wallis test on three locations averaged late blight severity dataset and summary of quantitative trait loci (QTLs) detected for late blight resistance

[a]Linkage group
[b]Kruskal–Wallis test statistic

Locus	LG[a]	K^b	*P*-value	Locus	LG[a]	K^b	*P*-value
CP15	III	13.829	0.0005	TG69	V	21.379	0.00001
CP198-1	III	15.528	0.00001	GP22	V	11.504	0.001
CP167-1	III	14.269	0.0005	Sol179A1	VII	7.211	0.01
CP14-1	V	7.248	0.01	GP193	VII	6.692	0.01
GP284	V	7.782	0.01	CP117	XI	6.836	0.01
CP49	V	10.338	0.005				

value in correspondence with marker GP198-1 (*P*-value of 0.00001). The QTL identified on linkage group V appears to be spread across a large number of markers, extending approximately 12 cM along the proximal end of this linkage group. The highest *P*-value (0.00001) was found associated with marker TG69. In the case of linkage groups VII and XI, *P*-values of 0.01 indicated the potential presence of two additional, but less statistically supported, QTLs, one near the GP179A1-GP193 marker interval on linkage group VII and one located in correspondence of the CP117 marker on the central section of linkage group XI. All of the detected QTLs, with the sole exception of the one present on chromosome XI, contributed toward increased resistance by alleles present in both parents. The QTL on chromosome XI was instead contributed by an allele from the susceptible parent.

The interval-mapping analysis identified three QTLs with LOD scores above the threshold value of 2.4 located on linkage groups III, V, and XI (Table 3). The genetic location of QTLs and their correspondent LOD values are shown in Fig. 2. The QTL on linkage group III had its highest LOD score (4.12) associated with marker GP198-1 and explained 23.4% of the total phenotypic variation. On linkage group V, the QTL had a likelihood peak in the position of marker GP22 with an LOD value of 5.17 and explained 17.3% of the total phenotypic variation. The QTL with the smallest effect was found in correspondence with marker GP232 on linkage group XI (LOD = 2.63, PVE = 9.4%). Furthermore, the presence of a QTL in this chromosomal region was found to be not statistically significant in the Kruskal–Wallis test. The LOD values on this linkage group present two significant peaks within a 23-cM interval, one in correspondence of marker GP232 and one centered near GP255-3. In addition to these QTLs, interval-mapping analysis determined relatively high LOD scores associated with marker GP121 (2.21) and GP179A1 (2.28) on linkage group VII. These values fell just below the threshold value of 2.4 for the detection of a QTL, but were considerably high with respect to the overall QTL analysis.

Analysis on individual trait datasets from the three locations

For the QTL analysis conducted on the three separate datasets from the individual locations both types of analysis, Kruskal–Wallis test and interval mapping, revealed similar results (data not shown). The analysis on the Rock Springs 1999 dataset identified two QTLs on linkage groups III and V, accounting for 19.8% and 13.9% of the total phenotypic variation, respectively. These QTLs were mapped in comparable positions along the two chromosomes, as found in the combined analysis. Specifically, on linkage group V, the highest LOD value shifted from marker TG69 to GP22, which were separated on the map by only 3.7 cM. From the Rock Springs 2002 dataset analysis, four QTLs were detected on linkage groups III, V, XI, and XII-A. The QTL with the greater effect (30.6% PVE) was located on the distal section of linkage group III. The LOD values on this linkage group present a significant peak in correspondence of marker GP198-1. QTLs on linkage groups V and XI have similar effects and positions as in the 1999 Rock Springs dataset. The QTL with the smallest effect (10.8% PVE), on linkage group XII-A, had the highest LOD score associated with marker GP99. This QTL is also a contribution of alleles present in both parents. Results from the Landisville 2002 experiment showed a single LOD peak (2.49) positioned

Table 3 Interval mapping test on three locations averaged late blight severity dataset and summary of QTLs detected for late blight resistance

[a]Likelihood ratio statistic
[b]Percentage of the variance explained by the QTL

Locus	LG	LOD[a]	Variance	Percentage explained[b]	Locus	LG	LOD[a]	Variance	Percentage explained[b]
CP15	III	2.85	5.84385		GP121	VII	2.21	5.95499	
GP198-1	III	4.12	4.94920	23.4	Sol179A1	VII	2.28	5.90539	8.6
GP167-1	III	3.92	4.97982		CP117-1	XI	2.56	5.84249	
CP49	V	4.14	5.56139		GP232	XI	2.63	5.84904	9.4
TG69	V	4.54	5.50574		GP255-3	XI	2.54	5.76887	
GP22	V	5.17	5.30091	17.9					

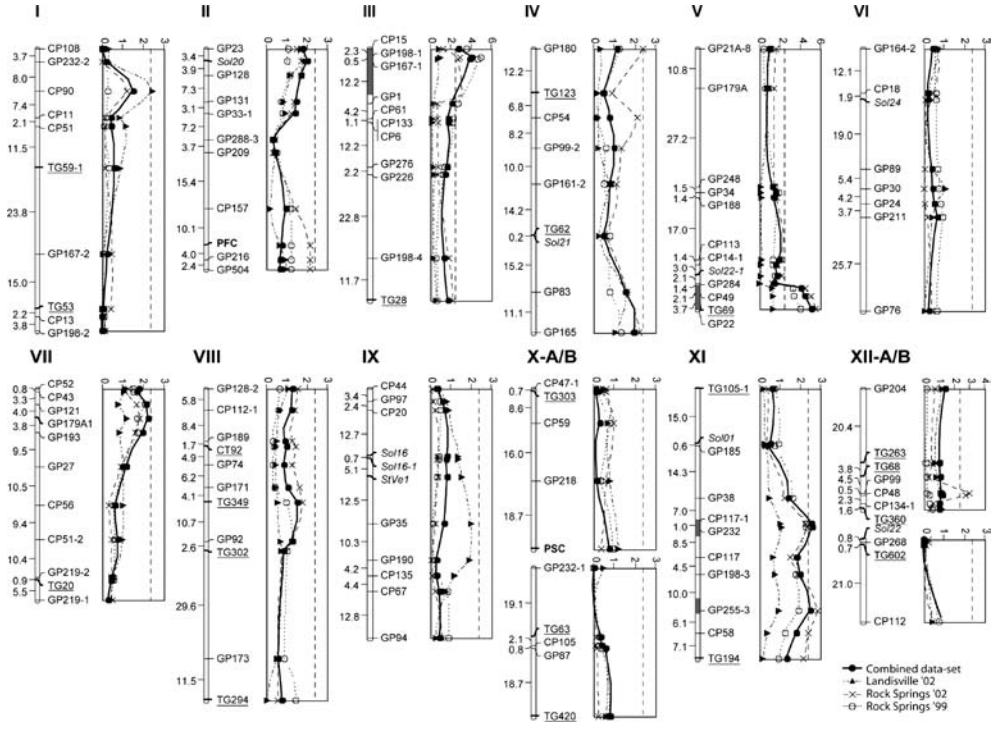

Fig. 2 Quantitative trait loci (QTL) and linkage map of a full-sib family of a cross between clones BD142-1 and BD172-1 from a diploid *Solanum phureja* × *S. stenotomum* population. QTL analysis based on interval mapping method of trait dataset from individual location and averaged over three locations. The names of the markers and map distances in centiMorgans are shown on the *right side* and *left side* of the chromosomes, respectively. Multiple loci homologous to a single marker probe are suffixed with designations *−1*, *−2*, etc., beside the specific marker name. Tomato-derived markers (*TG*, *CT*) are *underlined*, resistance gene analogue (*Sol*) markers are in *italics* and morphological markers are in *boldface*. QTL likelihood plots are shown on the *right side* of the chromosome, indicating $\log_{10}$ of odds ratio (LOD) scores for potato late blight resistance. The *height* of the curve indicates the strength of the evidence for the presence of a QTL at each position. The *horizontal dotted line* at a height of 2.4 indicates the threshold that the LOD score must cross to allow the presence of a QTL to be inferred. Areas along the linkage groups marked in *gray* had a corresponding LOD value above the 2.4 threshold value in the averaged dataset

on linkage group I, centered on marker CP90, explaining 8.4% of the total phenotypic variation and contributed from alleles present in both parents. A potential QTL could be located on linkage group IX, where the LOD plot reached its highest peak in proximity of marker GP35 with a LOD value of 2.00 and 7.9% of phenotypic variation explained. This QTL was contributed by an allele only present in the heterozygous state in the male resistant parent.

Discussion

The hybrid population derived from two diploid potato clones, BD142-1 and BD172-1, was evaluated in the field for foliar resistance in three different environments between 1999 and 2002. QTL analyses for this trait were performed on four datasets—separately for each of the three locations and on the estimated multiple location average.

Dataset from the estimated multiple locations average

On the combined dataset, the analysis identified three QTLs on chromosomes III, V, and XI, which together accounted for a significant portion of the total phenotypic variation. Furthermore, the smaller effect (8.6% PVE) of a fourth QTL on chromosome VII could also be detected. The most important QTL effect was clearly observed on chromosome III (Fig. 2). On this chromosome, a QTL was detected, centered on marker GP198-1, with an individual effect on the phenotypic variation of 23.4%. The location and effect of this QTL is in accordance with previous research conducted on different diploid potato populations (Leonards-Schippers et al. 1994; Ewing et al. 2000; Visker et al. 2003). In particular, in a population derived from *S. tuberosum* and *S. berthaultii*, Ewing et al. (2000) reported the presence of an epistatic interaction between the QTL on chromosome III and those located on chromosomes VII and XI, which were mapped in positions close but not coincident to the one detected in this present QTL analysis. In the same study, the potential presence at the same locus of two closely linked QTLs with differential effect on late blight resistance was also suggested.

A second significant QTL located on the proximal section of chromosome V accounted for 17.9% of the total phenotypic variation. Its position, the interval between marker TG69 and GP22, does not strictly coincide

with any previously reported late blight resistance locus, although QTLs for late blight resistance were identified in the proximal region of chromosome V in two other studies that were also based on diploid potato populations (Oberhagemann et al. 1999; Sandbrink et al. 2000). Furthermore, the root cyst nematode (*Globodera rostochiensis*) resistance loci *H1* (Pineda et al. 1992; Gebhardt et al. 1993) and *GroV1* (Jacobs et al. 1996) have been mapped in proximity of those same QTLs. This could be indicative of the presence in this region of a cluster of genes involved in plant defense response mechanisms. It should be noted that this QTL is definitively distinct from the most prominent and consistent QTL identified in the majority of the studies located on chromosome V and tightly linked to the anchor marker GP179 (Leonards-Schippers et al. 1994; Collins et al. 1999; Oberhagemann et al. 1999; Ewing et al. 2000; Ghislain et al. 2001). This major QTL is known to affect leaf as well as tuber resistance (Collins et al. 1999; Oberhagemann et al. 1999; Gebhardt and Valkonen 2001). Tightly linked to the same marker GP179, on the distal section of chromosome V, are the *R1* locus conferring race specific resistance to late blight and a major QTL for plant maturity (Collins et al. 1999; Oberhagemann et al. 1999). Neither QTL mapping method, in the present analysis, found any significant association between marker GP179 and race non-specific resistance against late blight present in this population.

The third most significant QTL was identified on chromosome XI (PVE = 9.4%). The likelihood for the presence of a QTL on this linkage group was below the significant threshold criterion in the correspondent Kruskal–Wallis test. On this chromosome, the QTL appeared to extend across a large section in the middle portion of this linkage group (23 cM), and two LOD peaks were identified with values of 2.63 and 2.54 and individual PVE values of 9.4% and 10.7%, respectively. The presence of multiple nearby significant likelihood peaks on this chromosome, however, does not necessarily imply the presence of multiple QTLs in this region, but this possibility cannot be ruled out from the present study. The location of this complex locus on chromosome XI is comparable with those described by Leonards-Schippers et al. (1994) and Ewing et al. (2000). A previous study also determined that the proximal end of chromosome XI harbors a cluster of three R genes (*R3*, *R6*, and *R7*) with different pathogen race specificity to *P. infestans* (El-Kharbotly et al. 1994, 1996). Moreover, on the same chromosome, the presence of a large number of specific resistance genes for virus, bacteria, nematodes, and fungi have been reported (Gebhardt and Valkonen 2001). From the results of the Kruskal–Wallis analysis, it appears that this QTL was contributed by the susceptible parent. Such an observation has been previously reported in potato and other species (DeVicente and Tanksley 1993; Leonards-Schippers et al. 1994). It is not uncommon, in the case of late blight, to identify QTLs for resistance present in the heterozygous state in the susceptible parent. It has been repeatedly observed that QTL alleles, which increase susceptibility to late blight, are often dominant over alleles that increase resistance, thus explaining why during backcross breeding to susceptible cultivars, field resistance to late blight is frequently lost (Leonards-Schippers et al. 1994; Gebhardt and Valkonen 2001; Ghislain et al. 2001; Visker et al. 2003). This observation illustrates also the specific ability of QTL marker analysis in identifying loci that make a relevant contribution toward resistance that otherwise would have been masked by the overall negative effect of other genetic traits of the susceptible parent.

On chromosome VII, the smaller effect (8.6% PVE) of a less-significant QTL was identified. This QTL is positioned at the distal end of the chromosome, with a LOD plot that has its highest value in correspondence of marker GP179A1 (LOD = 2.28). Despite the relatively low LOD value associated with this QTL, its position and effect is in accord with a QTL of minor effect ($0.05 < P < 0.01$) reported in two studies by Leonards-Schippers et al. (1994) and by Ewing et al. (2000). Moreover, in a more recent study (Kuhl et al. 2001), a single dominant late blight resistance locus (*Rpi1*) from *S. pinnatisectum* was mapped in the proximity of this region on chromosome VII.

Individual trait datasets from the three locations

The analysis of the QTL mapping results performed on the three locations separately reveals the presence of similar trends among the LOD plots from the three datasets, which is indicative of common genetic factors acting in concert across environments. Despite this overall agreement, a clear distinction can be made between the two experiments conducted at Rock Springs in 1999 and 2002 and the third one at Landisville in 2002. In this last case, LOD plots with consistently lower values than the other two cases can be observed in at least 6 of the 14 linkage groups analyzed. In particular, the linkage groups where significant QTLs have been identified in the two Rock Springs datasets (III, V, and XI) have LOD plots that never reach the threshold of 2.4 for the Landisville dataset. Moreover, this trend is completely reversed when we consider the LOD plots of linkage group I and IX. It can be speculated that these differences were caused by the effect of the distinct environmental conditions, in the different locations, on the expression of specific resistance components. Another important factor that most probably has contributed to these differences is the level of maturity of the plants in the Landisville experiment at the time of inoculation. Due to warmer and dryer weather conditions at this location, the plants were in the field a month longer, compared to field experiments at Rock Springs, before the inoculation of *P. infestans* spores could be applied. Clear evidence has been found that potato plants become more susceptible to infection as the foliage matures (Hodgson 1961; Collins et al. 1999; Oberhagemann et al. 1999). It is possible that this

generalized increase in plant susceptibility may have hampered the ability to clearly discriminate in the field among levels of resistance consequently effecting the QTL mapping analysis. An additional QTL was identified on linkage group XII-A in the Rock Springs 2002 dataset. There are no previous reports indicating the presence of a QTL associated with late blight resistance in the same chromosomal region. Moreover, neither of the two other datasets supported the presence of a QTL in that same position; therefore, the authenticity of this QTL remains to be confirmed.

Conclusion

The results from this study suggested the presence, in the diploid potato germplasm utilized, of three major QTLs on chromosomes III, V, and XI, which accounted for 23.4, 17.9, and 9.4% of the total phenotypic variation, respectively. Furthermore, three additional loci on chromosomes I, II, and VII were identified with a potential, but more limited, effect on the same trait. Two different approaches of QTL analysis, the non-parametric Kruskal–Wallis test and the interval mapping method, were applied. Both methods gave comparable results in identifying several QTLs with significant effect on late blight general resistance in potato. Partial discrepancy was observed between the two methods in the exact position of the QTL detected on chromosome XI, although this could be explained by the presence of potentially two small QTLs located in close proximity. Some limitations of the present QTL analysis could also be attributed to incomplete genome coverage and lack of informative molecular markers in specific linkage group sections. For this purpose, additional markers are needed to complete coverage on linkage groups X and XII. These results fully support the initial hypothesis, suggesting the presence in this population of several loci contributing enhanced general resistance against late blight. Some of these QTLs have also been identified in other studies using different intra- and inter-specific crosses. Common QTLs identified in different species, populations and environments are ideal targets for marker-assisted selection and breeding and for basic research towards cloning and characterizing genes affecting quantitative traits. This valuable information could be used in any available population to monitor the inheritance of a specific chromosomal segment or to evaluate the variation available in a specific gene pool at a particular locus. Moreover, in future studies, the resolution of QTL mapping obtained from this initial study could be improved by adding more genetic markers and by using a larger population to allow an examination of more recombinant types. These would allow the identification of markers more closely linked to the mapped QTLs. Those markers could then be transformed into PCR-based marker assay to simplify the detection of specific QTL alleles among selected genotypes.

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