Mapping Late Blight Resistance in *Solanum microdontum* Bitter

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ABSTRACT

A diploid *Solanum* population was developed with the objective of mapping quantitative trait loci (QTL) associated with resistance to *Phytophthora infestans* (Mont.) de Bary, the causal organism of late blight. The mapping population was a cross between a late blight resistant selection of *Solanum microdontum* Bitter and a susceptible diploid breeding clone. The progeny of 109 clones and the parents were field tested for foliar late blight reaction in 1999 and 2000 and for vine maturity in 2000 and 2001. Parents and progeny were genotyped with isozymes and simple sequence repeats (SSR) markers. A total of 161 pairs of SSR primers were screened, from which 74 amplified polymorphic bands in Metaphor agarose or polyacrylamide gels yielding a total of 109 SSR loci. The isozyme and SSR combined analyses resulted in a total of 118 marker loci in the population, of which 54 were heterozygous (band present) in *S. microdontum* and 64 in MSA133-57. High phenotypic correlation ($r = 0.89$, $P < 0.0001$) was found for late blight reaction between years and no correlation was found between late blight and vine maturity. *Solanum microdontum* transmitted high levels of resistance to more than 50% of the offspring. There was a major QTL linked with the SSR marker STM0020b associated with foliar late blight resistance located at the same position in both years of field testing explaining over 60% of the phenotypic variance. This major QTL is suitable for marker-assisted selection to introgress a new source of resistance to *P. infestans* to the cultivated tetraploid germplasm of potato.

In the mid-1990s, the USA and Canada experienced a late blight epidemic caused by new, more aggressive, and metalaxyl-resistant races (Goodwin et al., 1995; Peters et al., 2001). The development of genetic resistance has become a major strategy for late blight control in cultivated ($2n = 4x = 48$) potatoes (*Solanum tuberosum* L.) and a priority in many breeding programs (Colon et al., 1995a), with horizontal (field, partial, or general) resistance being the only durable type of resistance to late blight (Colon et al., 1995b; Umaerus et al., 1983; Kamoun et al., 1999).

Diploid ($2n = 2x = 24$) and tetraploid populations have been used for QTL associated with late blight resistance introgressed from a variety of wild species of potato. Genetic mapping at the diploid level avoids interpretation problems associated with tetrasomic inheritance (Meyer et al., 1998). The results of mapping QTL associated with late blight resistance showed that those QTL could also be associated with other traits. This is the case of chromosome V on which QTL were mapped for foliar and tuber late blight resistance, vine maturity and vigor (Oberhagemann et al., 1999), and foliar late blight resistance in other populations (Collins et al., 1999; Sandbrink et al., 2000). Association between QTL conferring foliar late blight resistance, tuberization, and vine maturity was found in four out of five chromosomes (Ewing et al., 2000). The knowledge about late blight resistance associations with undesirable traits is also of value before introgressing a new source of resistance.

The South American diploid species *S. microdontum* has shown high levels of resistance to late blight (Colon and Budding, 1988; Colon et al., 1995a, 1995c; Douches et al., 2001). Among 618 clones representing 24 accessions, 56 clones were selected as highly resistant to the US8 genotype of *P. infestans*, from which 27 clones represented three accessions of *S. microdontum* (Douches et al., 2001). Dominant gene action was identified in some crosses between *S. microdontum* and susceptible clones (Colon et al., 1995c). Strong hypersensitive reaction or infection efficiency, lesion growth rate, and sporulation time were associated with high levels of resistance in *S. microdontum* (Colon et al., 1995a).

The purposes of this research were to map QTL associated with late blight resistance from a *S. microdontum*-derived population and to examine the association between late blight resistance and late maturity.

MATERIALS AND METHODS

Selection of Resistant Parent and Mapping Population

A total of 175 clones representing six accessions of *S. microdontum* were tested in the greenhouse with US8 genotype/A2 mating type of *P. infestans* in 1997. The most resistant clones were retested in 1998 and 27 highly resistant clones were selected (Douches et al., 2001), from which one clone (PI595511-5) was selected as the late blight resistant parent to develop the mapping population. The selected clone was very distinct from cultivated potato, from *S. berthaultii* (Hawkes), and from other clones of *S. microdontum* with reported late blight resistance (Bisognin and Douches 2002). The diploid mapping population chosen was a cross between the Michigan State University (MSU) late blight susceptible breeding clone MSA133-57 [(*S. tuberosum* × *S. chacoense*) × *S. phureja*] with *S. microdontum* PI595511-5. A progeny of 109 clones that produced tubers at greenhouse conditions (16-h day-length with supplemental lighting from high pressure sodium lamps) was used for field phenotypic evaluations and molecular analysis. Greenhouse tubers were used to plant the late blight trial of 1999 at the MSU Muck Soils Research Farm, Bath, MI, and the seed increase field at the MSU Montcalm Research Farm, Entrican, MI. Late blight trial of 2000 and maturity trials were planted with field-increased seed tubers.

Late Blight Reaction in Field Tests

The *P. infestans* isolates collected in Michigan (MS94-1, MS94-4, MS95-7, and MS97-2) were characterized as US8 genotype/A2 mating type as described in Bisognin et al. (2002). Those isolates were infectious on all R-gene differentials ex-

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cept R9 in detached-leaf assays. In the field, the isolates were weakly pathogenic only on the R8 and R9 of Black's differential clones and strongly pathogenic on all others.

The field tests were performed at the MSU Muck Soils Research Farm in a randomized complete block design. No fungicides were applied to the plants. Parents and progeny were planted in three replications of three-hill plots on 27 May and 9 June and inoculated on 22 and 26 July in 1999 and 2000, respectively. Inoculation was done through a permanent sprinkle irrigation system in the early evening and high humidity was maintained in the canopy through periodic irrigations throughout the season. A visual estimation of the percentage of stem and leaf infected area was scored at 3- to 5-d intervals from inoculation until the most susceptible clones reached 100% infection. The area under the disease progress curve (AUDPC) was calculated as described in Kirk et al. (2001) and divided by the maximum AUDPC (e.g., 3300 for 33 d after inoculation) converting the value to relative AUDPC (RAUDPC), with 1.0 being the maximum RAUDPC value.

Vine Maturity Evaluations

Parents, progeny, and check varieties were planted in nonreplicated three-hill plots and two-replicated five-hill plots at the MSU Montcalm Research Farm on 22 and 14 May 2000 and 2001, respectively. Vine maturity was evaluated using a 1-to-5 scale of increasing lateness in comparison with the cultivar Atlantic (Bisognin et al., 2002). Vine maturity was evaluated on 24 Sep. 2000 when the cultivar Atlantic had vine maturity rating of 1 and on 4 Sept. 2001 when the cultivar Atlantic had vine maturity rating of 2.

Marker Analysis

Parents and progeny were genotyped with isozymes and SSR markers. The isozyme analysis was performed with crude protein extraction from a newly expanded leaflet (approximately 120 mg), resolved in a horizontal 10% (w/v) starch gel by electrophoresis with two buffer systems. Tissue processing, electrophoresis, staining, and nomenclature were performed as described in Douches and Quiros (1988). Nine isozyme loci from four enzyme systems were scored according to Douches and Quiros (1988) and Douches and Ludlam (1991). Malate dehydrogenase (*MDH*) and phosphoglucose isomerase (*PGI*) were resolved with a histidine-citrate pH 5.7, and glutamate oxaloacetate transaminase (*GOT*) and phosphoglucomutase (*PGM*) were resolved with a lithium-borate pH 8.3 buffer systems (Stuber et al., 1988).

Total genomic DNA used as a template for SSR analysis was isolated from young leaves of greenhouse-grown plants. Tissue was harvested, freeze-dried, and then ground with glass beads. DNA was isolated from 20 mg of tissue with the DNeasy Plant Mini Kit (Qiagen Inc., Germany) following the manufacturer's protocol.

A total of 161 pairs of SSR primers were used. All primer pair sequences have been previously published in Provan et al. (1996), Milbourne et al. (1998), Sandbrink et al. (2000), and Ashkenazi et al. (2001). These primer pairs were synthesized at MSU and screened for polymorphism between the parents. DNA amplification for SSR primers was performed as described in Bisognin and Douches (2002).

Electrophoretic separation for SSRs was done in a 3% (w/v) Metaphor Agarose (FMC Bioproducts, Rockland, ME) or a 5% (w/v) polyacrylamide gels (Sigma-Aldrich Co., St. Louis, MO) depending on the size of amplified fragment. Metaphor agarose gels were run at 100 V from 3.5 to 4.5 h at 10°C, stained with ethidium bromide (1 μg mL^{-1}) for 45 min, visualized under UV light, and photographed for permanent records. Polyacrylamide gels (34.5 $\times$ 50 cm) were run at 90 W for 2 h and 30 min in a Sequi-Gen GT Sequencing Cell (Bio-Rad, Richmond, VA) and stained with Silver Sequence DNA (Promega, Madison, WI) following the respective manufacturer's protocol. Fragment sizes were estimated using a 10- or 25-bp DNA ladder (Gibco BRL, Grand Island, NY) in each gel. Multiple loci of SSR markers were labeled with a letter after the marker designation.

Statistical Analysis

The phenotypic data of foliar late blight reaction and vine maturity was submitted to analysis of variance for both years of evaluations. Pearson correlation analysis was performed to compare late blight and maturity data and different years. Descriptive statistics were used to characterize population distribution for both evaluated traits. All analyses were done by the statistical package GENES (Cruz 2001).

Since the parents of the mapping population were noninbred diploid potato clones, data collection, linkage analysis, and QTL mapping were based on the presence or absence of SSR or isozyme bands in the segregating population. The χ^2 test for goodness-of-fit was used to test for deviations of the expected Mendelian segregation ratio of 1:1 (presence vs. absence). Linkage analysis was done with JoinMap V2.0 (Stam 1993) setting minimal LOD score as 3.0 and maximum recombination fraction as 0.49. Map distances were calculated in centimorgans (cM) using the Kosambi mapping function (Kosambi 1944). The locations of QTL associated with the traits were determined by the program package QTL-CARTOGRAPHER (Basten et al., 1999). The model 6 of the Zmapqtl program, which employs a composite interval mapping method (Jansen and Stam, 1994; Zeng, 1994), was used for all analyses. The number of markers for the background control was set to 5, meaning that the five most significant markers outside the interval under analysis were fitted to the model. The markers used for the background control were detected through forward and backward stepwise regression using the program SRmapqtl. The likelihood value for the presence of a QTL was expressed as LOD score $log_{10}(L_1/L_0)$, where L_1 was the likelihood of the model with the putative QTL and L_0 was the likelihood of the model without the QTL. The threshold of the LOD score for declaring a putative QTL significant was determined by 1000 permutations of each trait data variable (Churchill and Doerge 1994). The position of the QTL was estimated as the point of maximum LOD score in the region under consideration. Year was treated as a separate trait for the analysis of year $\times$ QTL interaction using JZmapqtl program. The likelihood value for the presence of year $\times$ QTL interaction was expressed as a LOD score.

QTL Introgression to Cultivated Potato

Solanum microdontum is a South American diploid species that can be directly crossed with cultivated potato via unilateral sexual polyploidization (4*x*–2*x* crosses) using 2*n* gametes (Hermsen 1994, p. 515–538; Hutten et al., 1994). One late blight resistant clone of the progeny (DLB1-150) was identified also as producing 2*n* pollen on the basis of pollen grain size. DLB1-150 was crossed with the late blight susceptible tetraploid clones MSF313-3 and NorValley. Greenhouse grown seedlings of each progeny were used for DNA isolation. The SSR marker Stm0020 was used to identify clones of the unilateral sexual polyploidization having the late blight resistant allele from *S. microdontum*.

RESULTS AND DISCUSSION

Phenotypic Evaluations

The analysis of variance showed significant differences ($P \leq 0.0001$) among clones for foliar late blight reaction in the field tests in 1999 and in 2000, between years and also clones × years interaction. The 1999 test had a higher mean and median RAUDPC, for parents and progeny, and progeny range RAUDPC than the 2000 test (Table 1). The RAUDPC of *S. microdontum* PI595511-5 was 0.021 and 0.019 compared with 0.529 and 0.175 of MSA133-57, respectively for 1999 and 2000. Some clones in the progeny showed a RAUDPC value 1.5-fold higher than MAS133-57 in both years of testing and the standard deviation in 1999 was three times higher than in 2000. Even with higher artificial epidemic of *P. infestans* in the field in 1999 than in 2000, *S. microdontum* showed almost the same RAUDPC values in both years, confirming its high resistance found in greenhouse tests (Douches et al., 2001). This *S. microdontum* clone also had only 10% infection in a late blight nursery in Toluca Valley, Mexico, during the 2000 season compared to 100% for the cultivar Alpha, used as susceptible check (Lozoya-Saldaña, personal communication).

The analysis of variance showed significant differences among clones for vine maturity in 2001. There was transgressive segregation for early vine maturity (rating 1) in the progeny and MSA133-57 had a similar rating as the progeny mean in both years of evaluations (Table 1). Standard deviation for 2000 was almost 2-fold higher than in 2001. Foliar late blight reaction and vine maturity showed skewed distributions in both years of evaluation. A set of data with this level of skewness would usually be transformed to normality, but as highly contrasting parents were used to develop the population, a mixture of distributions, as expected, was observed in the progeny (Doerge et al., 1997).

There was a high correlation ($r = 0.82$, $P \leq 0.0001$) for late blight reaction between the two years of testing and there was no correlation between late blight reaction and vine maturity in either year. An intermediate correlation ($r = 0.42$, $P \leq 0.0001$) was found between years for maturity evaluation. The nonreplicated trial in 2000 associated with later maturity assessment probably contributed to a smaller correlation coefficient. More importantly, no correlation was found between late blight reaction and vine maturity. A significant correlation between these two traits is probably the most undesirable association from a late blight resistant source as found by Ross (1986) and Umaerus et al. (1983). Since the high level of resistance to late blight in this clone of *S. microdontum* was not correlated with late maturity, this clone should be an excellent source of germplasm to consider for breeding late blight resistant cultivars.

Marker and Linkage Analyses

From four isozyme systems, nine isozyme loci were polymorphic in the population. A total of 161 pairs of SSR primers were screened with DNA template from the parents and 125 were successfully amplified and produced readable bands. A total of 46 pairs of primers amplified polymorphic bands in Metaphor agarose gels and resulted in 61 SSR loci, with fragment sizes varying from 80 to 500 bp. Twenty-eight additional pair of primers produced polymorphic bands on polyacrylamide gels and resulted in 48 SSR loci, with fragment sizes varying from 115 to 350 bp. The SSR analyses resulted in 109 loci. The isozyme and SSR combined analyses resulted in a total of 118 marker loci in the population, of which 54 were heterozygous (band present) in *S. microdontum* and 64 in MSA133-57. Eight pairs of SSR primers produced unique homozygous bands in both parents and were used to confirm the pedigree and F_1 status of the mapping population (data not shown).

A total of 95 markers (eight isozymes and 87 SSRs) were grouped into 28 linkage groups (LGs), leaving 23 markers unlinked. Nineteen LGs were assigned to known chromosomes. Assuming that the assigned linkages are correct, 10 of the 12 chromosomes have associations with 19 of the 28 LGs. Only chromosomes X and XI have no assigned LGs. We need to further saturate the map to completely assign the LGs and unlinked markers. The map covered 876 cM of the potato genome, with an average distance of 9.2 cM between markers and an average of 3.4 markers per linkage group. The length of the potato linkage maps varies from 690 cM (Gebhardt et al., 1989) to 1120 cM (Jacobs et al., 1995). Previously published *S. microdontum* maps were constructed from six subpopulations with total genome coverage length varying from 341 cM to 683 cM (Sandbrink et al., 2000).

Quantitative Trait Locus Analysis

Simple linear regression showed that the SSR loci ST56 ($P < 0.01$) on LG 3, Stm1056 ($P < 0.01$) on LG 8, and Stm2013 ($P < 0.05$) on LG 25 were linked with vine maturity in 2000. In 2001, the SSR loci STIIKA ($P < 0.05$) and Stm1025 ($P < 0.05$) on LG 12, ST6162 ($P < 0.05$) on LG 9, Stm1064 ($P < 0.05$) on LG 11, LECAB9 ($P < 0.05$) on LG 19, and Stm1024 ($P < 0.05$) on LG 27 were linked with vine maturity. In the joint

Table 1. Phenotypic values of the parents, progeny size and descriptive statistics for foliar late blight reaction and vine maturity of a *S. microdontum* (resistant parent) and MSA133-57 (susceptible parent) derived population.

	Phenotypic values		Progeny	Descriptive statistics				
Evaluated traits	*S. microdontum*	MSA133-57	Size	Mean	Median	Skewness	Range†	SD
RAUDPC 1999‡	0.021	0.529	109	0.259	0.176	0.80	0.007–0.883	0.24
RAUDPC 2000‡	0.019	0.175	109	0.107	0.066	0.56	0.010–0.273	0.08
Maturity 2000§	5.0	2.0	109	2.0	1.0	1.23	1.0–5.0	1.32
Maturity 2001§	4.0	2.0	102	2.1	2.0	0.67	1.0–4.5	0.78

† Progeny range: lowest and highest value of the trait
‡ Relative area under the disease progress curve as a measurement of foliar late blight reaction (maximum RAUDPC = 1)
§ Scale 1 to 5 of increasing lateness.

analysis, five SSR loci were linked to vine maturity, of which the two loci that mapped on LG 3 and LG 25 also linked with vine maturity in 2000. No QTL was identified in this population associated with vine maturity.

For foliar resistance to late blight, the SSR loci ST13STb ($P < 0.01$), ST3334b ($P < 0.001$), and Stm0020b and Stm0020a ($P < 0.0001$) on LG 21 were linked with the late blight reaction in both years of field testing and joint analysis. There were two other SSR loci linked with the late blight reaction in 1999, ST6162 ($P < 0.05$) on LG 6 and ST1920 ($P < 0.05$) on LG 16. The allozyme *Got-2*[5] ($P < 0.05$) on LG 4 was linked with the late blight reaction in 2000. None of these loci were linked with vine maturity indicating that these two traits were not associated in the *S. microdontum* clone.

There was a QTL in *S. microdontum* associated with foliar late blight resistance on LG 21 in both years of field testing and joint analysis located almost at the same position. On the basis of composite interval mapping analyses, this QTL explained over 40% of the phenotypic variance in 1999 within the interval between 38 and 48 cM, over 60% of the phenotypic variance in 2000 within the interval between 40 and 46 cM, and over 50% of the phenotypic variance in the joint analysis within the interval between 42 and 44 cM (Fig. 1). There was a significant interaction (LOD 9.8) between this QTL and years of field testing. The SSR marker Stm0020b was linked in coupling to late blight resistance in both years and joint analysis being located within the region of high LOD scores of the QTL. A total of 31 and 32% of the phenotypic variance was explained by the QTL at the position of the marker Stm0020b in 2000 and 2001, respectively. The average RAUDPC of resistant clones having the band linked with resistance was 0.156 and 0.070 and with susceptibility was 0.407 and 0.160, respectively in 1999 and 2000 field tests.

Quantitative trait loci associated with late blight resistance have been mapped to different positions of the genome in wild *Solanum* species and in hybrids among wild species. In the cultivar Stirling, for which *S. demissum* is the source of late blight resistance, a QTL was mapped to chromosome IV (Meyer et al., 1998; Pande et al., 2001). In *S. bulbocastanum* Dunal a major QTL was localized to chromosome VIII (Naess et al., 2000). In *S. berthaultii* late blight resistance QTL were mapped on chromosomes I, III, VII, VIII, and XI (Ewing et al., 2000), and in *S. microdontum* late blight resistance QTL were identified on chromosomes IV, V, and X (Sandbrink et al., 2000). In other wild species hybrids, QTLs were mapped to chromosomes III, IV, V, VI, IX, and XI. A major region conferring late blight resistance and late maturity was located on chromosome V (Collins et al., 1999; Oberhagemann et al., 1999). There were also QTL associated with late blight susceptibility and early maturity that map to other regions of the genome (Ewing et al., 2000).

The four SSR loci of LG 21 have not been previously mapped in potato, which does not permit us to assign linkage to any chromosome. However, this QTL should be different from the QTL mapped in the cultivar Stirling (Meyer et al., 1998) and the previous QTL mapped

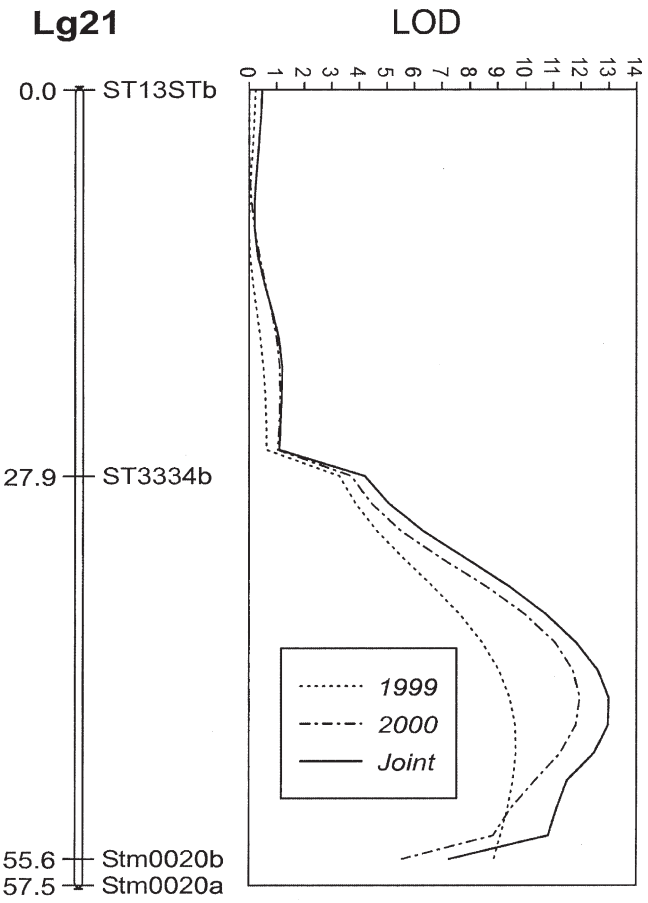

Fig. 1. Quantitative trait loci associated with foliar late blight (LB) resistance of a *S. microdontum*-derived population on linkage group 21 for 1999, 2000, and joint analysis. Primer sequences of the SSR marker ST3334 were published by Ashkenazi et al. (2001), Stm by Milbourne et al. (1998) and ST13ST by Sandbrink et al. (2000).

in *S. microdontum* (Sandbrink et al., 2000). The only SSR loci linked with late blight resistance in these previous studies (Stm3016 in the cultivar Stirling on chromosome IV and STPOAC58 in *S. microdontum* on chromosome V) were polymorphic in this population but were not linked to any of the 28 LGs. Moreover, the SSR marker ST13ST, on the top of the LG 21, was not linked with late blight resistance in Sandbrink's *S. microdontum* population. The addition of more linkage data is necessary to locate the QTL associated with foliar late blight resistance in *S. microdontum* on the potato genome to a specific chromosome and determine the relationship with late blight resistance QTL mapped in *S. bulbocastanum* (Naess et al., 2000) and *S. berthaultii* (Ewing et al., 2000).

This study indicated that high levels of late blight resistance can be transmitted to a high percentage of the offspring and can be conferred by a single QTL. This result is similar to previous mapping studies. A QTL associated with late blight resistance from *S. bulbocastanum* explaining 62% of the phenotypic variance was mapped to chromosome VIII (Naess et al., 2000). A QTL from *S. demissum* explaining about 30% of the phenotypic variance for late blight was mapped to

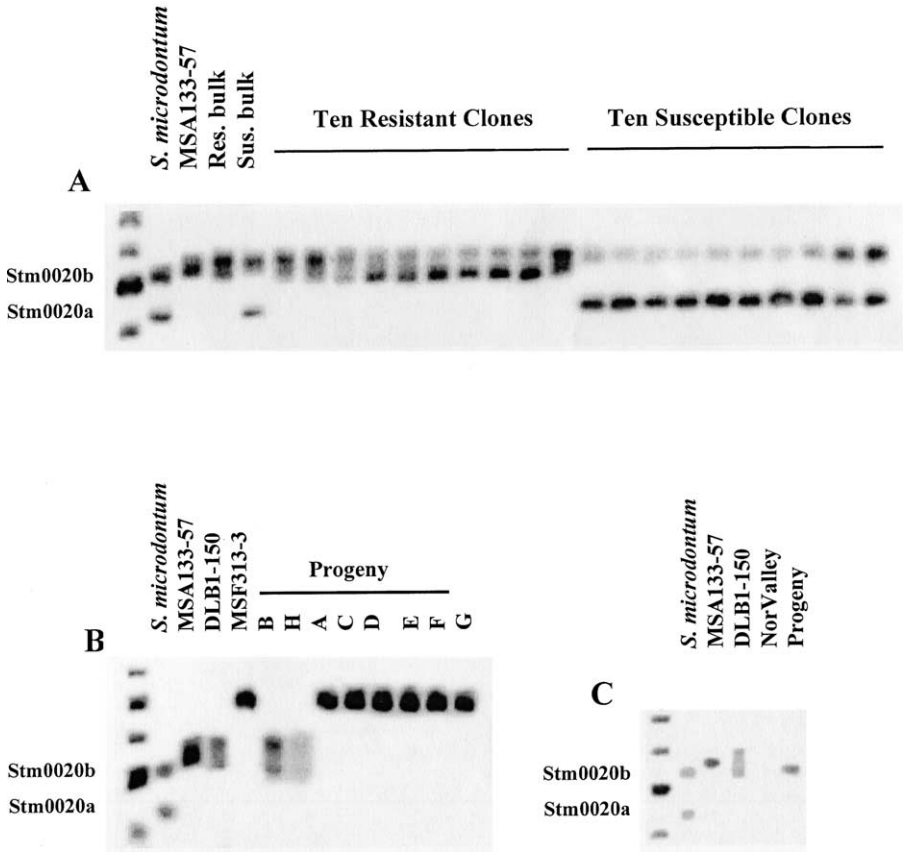

Fig. 2. Negative image of the simple sequence repeat marker Stm0020 fragments separated in 3% (w/v) Metaphor agarose gel run at 100 V for 4.5 h. A) *S. microdontum* (resistant parent), MSA133-57 (susceptible parent), bulks and ten resistant and susceptible progeny clones, respectively. Stm0020b linked with resistance and Stm0020a linked with susceptibility, both from *S. microdontum*. B and C) SSR marker Stm0020b followed through polyploidization. Parents of the diploid population, diploid resistant clone of the progeny (DLB1-150) and tetraploid parents with respective tetraploid progeny clones.

chromosome IV (Meyer et al., 1998; Pande et al., 2001) in the cultivar Stirling (Meyer et al., 1998). Different QTL associated with late blight resistance were identified in two clones of *S. microdontum* from the same accession. From one clone, a QTL was mapped to chromosome IV and the other clone a QTL was mapped to chromosome X (Sandbrink et al., 2000).

Marker Assisted Selection and QTL Introgression

The mapping population combined late blight resistance from *S. microdontum* with acceptable maturity and other desirable tuber qualities from the diploid breeding clone MSA133-57 (an interspecific hybrid). This *S. microdontum* clone used was the most distinct source of late blight resistance compared with cultivated potatoes on the basis of isozymes and microsatellite analysis (Bisognin and Douches 2002). The high levels of genetic diversity involved in the crosses resulted in a high proportion of markers segregating in the F_1. Most markers associated with late blight resistance were linked in coupling with the *S. microdontum* parent. The ST3334b allele from MSA133-57 was linked in repulsion with resistance reducing the mean RAUDPC of the progeny from 0.361 to 0.195 in 1999 and from 0.138 to 0.083 in 2000. The Stm0020b allele from *S. microdontum* was linked in coupling with resistance reducing the mean RAUDPC of the progeny from 0.402 to 0.156 in 1999 and from 0.156 to 0.070 in 2000. The 38 individuals of the progeny that showed the Stm0020b band and did not show the ST3334b band had a mean RAUDPC of 0.149 in 1999 and 0.066 in 2000. Conversely, the 30 individuals that did not show the Stm0020b band and showed the ST3334b band had a mean RAUDPC of 0.441 in 1999 and 0.169 in 2000. One of these SSR markers (Stm0020) mapped on the region of the highest LOD scores of the QTL and showed two bands in *S. microdontum*, one linked with resistance (Stm0020b) and one with susceptibility (Stm0020a) in the progeny (Fig. 2A). The marker Stm0020b is more suitable for marker assisted selection, since it is linked in coupling with resistance and showed to be more effective in reducing the mean RAUDPC of the individuals.

Among the late blight resistant clone of the progeny, the clone DLB1-150 was used for unilateral polyploidization to introgress the resistance in to cultivated potato. DLB1-150 was the 2*n*-pollen donor and the band linked with resistance could be followed through polyploidization (Fig. 2B). From eight progeny individuals of the cross with the late blight susceptible breeding clone MSF-313-3, two had the resistant band (Stm0020b marker) from DLB1-150. This Stm0020b marker could also be

followed in the progeny from the cross NorValley × DLB1-150 (Fig. 2C). The uniqueness of the Stm0020b band from *S. microdontum* should allow this SSR marker and the associated late blight resistance QTL to be monitored in other crosses with tetraploid germplasm. Therefore, Stm0020b can be used in a marker assisted selection program to introgress a new source of resistance to late blight from the wild species *S. microdontum* to cultivated potato and to combine with other previously mapped QTLs for late blight resistance (e.g., Stirling, *S. bulbocastanum, S. berthaultii, S. microdontum*) to improve the level and durability of resistance.

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