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Solanum mochiquense chromosome IX carries a novel late blight resistance gene *Rpi-moc1*

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Abstract Screening of a large number of different diploid *Solanum* accessions with endosperm balance number (EBN) 1 revealed segregation for strong resistance and sensitivity to *Phytophthora infestans* in accessions of *Solanum mochiquense*. Genetic analysis showed that resistance in *S. mochiquense* accession CGN18263 resides at the distal end of the long arm of chromosome IX, is linked to restriction fragment length polymorphism marker TG328 and is in the neighbourhood of the quantitative trait locus (QTL) *Ph-3* conferring resistance to *P. infestans* in tomato. This is the first genetic study of *S. mochiquense*, a wild diploid species originating from fog oases in the Peruvian coastal desert.

Introduction

The quest for plant resistance to potato late blight, caused by the oomycete *Phytophthora infestans*, began even before the biological cause of the Irish potato famine of 1848–1850 was discovered. Early hybridists reasoned that blighted potatoes would benefit from an infusion of "new blood" from South America (Stuart 1904). Some wild relatives of the potato are indeed resistant to potato late blight, but not all are readily crossable, and once these novel resistances were bred into varieties, new races arose that could overcome them. However, with the development of molecular genetics, these issues of crossability and durability are now easier to deal with. Novel resistance genes from wild relatives can be identified, cloned and transferred by means of genetic transformation. Genetic transformation is less time-consuming, avoids linkage drag and is applicable to a wider range of resistance sources than the traditional transfer method via crossing and repeated backcrossing. A further advantage of transformation is that it allows the efficient transfer of multiple strong *Rpi* (resistance to *P. infestans*) genes into a given cultivar. These "new cultivars" would consist of near-isogenic clonal lineages that are heterogeneous for *Rpi* gene composition, thereby mimicking the natural variation of *R* genes that exists in wild *Solanum* populations. This might avoid high selection pressures for virulence in the *P. infestans* population. The mutual protection of mixed *Rpi* genes may result in a natural and durable type of crop protection (Pink and Puddephat 1999; Jones 2001; Bergelson et al. 2001) that can reduce the need for costly fungicides. A prerequisite for this strategy is the screening, identification, mapping and cloning of many *Rpi* genes.

Gene banks store virus- and viroid-free wild germplasm from the *Solanum* gene pool that is freely available to the scientific community and breeders. As most diploid *Solanum* species are outbreeders represented by genetically variable populations in gene bank accessions, average resistance scores cannot reveal the genetic variation within these populations. Only fine screening of multiple individual plants within an accession can provide a firm basis for subsequent genetic analysis (Douches et al. 2001).

In the course of an extensive fine screening of diploid *Solanum* accessions, we encountered both strong *P. infestans* resistance and sensitivity in *Solanum mochiquense* Ochoa. Genetic analysis showed that the resistance in *S. mochiquense* accession CGN17731 resides at the distal end of the long arm of chromosome IX. We report here the results of the first genetic study of resistance to late blight in *S. mochiquense*, a Peruvian diploid species within *Solanum* (subsection Potato, series Tuberosa) with an endosperm balance number

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(EBN) of 1 [indicating that tetraploidized *S. mochiquense* may cross with diploidized *S. tuberosum* (EBN 2)].

Rpi genes from *S. mochiquense* and other wild *Solanum* species could contribute to *P. infestans* resistance in future potato crops. Wild germplasm mining may reveal the presence of many novel *R* genes needed for engineering durable resistance through manipulating *R* gene diversity (Niederhauser et al. 1996).

Materials and methods

Solanum mochiquense growing conditions

Seed of five *Solanum mochiquense* accessions (Table 1) was obtained from the Centre for Genetic Resources in Wageningen, the Netherlands (CGN). This seed was routinely treated with 1,000 ppm gibberellic acid (GA_3) and disinfected with 10% bleach before being placed on solid sterile MS (Murashige and Skoog 1962) medium (2% agarose) for germination. After approximately 3 weeks, germinated seedlings were transferred to a glasshouse with limited facilities for temperature control. Regular treatments with various fungicides and pesticides were necessary to control attack by thrips, aphids, spider mites, powdery mildew and early blight (*Alternaria solani*). About 4 weeks later, the plants started flowering. Plants were grown in 1- or 3-l pots and reached a height of about 50 cm with only limited trimming. Regular trimming of stolon growth was necessary to prevent invasion of neighbouring pots.

During the spring and summer, flowering continued for 1–3 months. The flowers produced a considerable amount of pollen and 100–300 seeds per berry following hand-pollination with non-self pollen using a fine paintbrush. No berries were observed on any other flowers, indicating the absence of pollinating insects and an effective self-incompatibility. Berries were harvested approximately 6 weeks following pollination when cracks appeared on their outer skin. Flowers and berries did not develop a brittle abscission layer at any stage and, consequently, did not suffer from the early dropping observed in some other *Solanum* species, most notably in *S. bulbocastanum*. During spells of hot weather, when glasshouse temperatures exceeded 30°C for several hours per day, the fertility of the flowers was reduced. In the autumn and winter, the plants grew less vigorously and fewer flowers were produced, but tuber development was better. Tuber formation was sometimes disappointingly low and quite variable (0–50 tubers of 1–3 cm in a 3-l pot). The most likely explanation for this variability is the interplay of the watering, trimming and fertilization regime.

Table 1 Reaction to *Phytophthora infestans* inoculation of five *Solanum mochiquense* accessions

Accession[a]	Reference data		Fine screening[b]	
	Phenotype	Source	R	S
CGN17731[c]	Very sensitive	CGN	2	13
CGN18263[d]	Very resistant	CGN	6	5
CGN20587	Sensitive	CGN	0	6
CGN21360	Sensitive	CGN	0	12
GLKS2319	Sensitive	Ruíz de Galarreta et al. (1998)	2	4

[a]CGN, Centre for Genetic resources in the Netherlands (http://www.plant.wageningen-ur.nl); GLKS, Gross Lüsewitz, Konrad Schüler, Germany

[b]Number of plants showing resistant (R) or sensitive (S) phenotypes

[c]PI 283114; 79°2′W, 8°7′S (PI, Pullman Institute Plant Introduction USA)

[d]PI 338616; 78°45′W, 8°25′S

Phytophthora infestans strains, inoculation and pathotest scoring

Two *Phytophthora infestans* isolates (98.170.3 and Met+) were kindly provided by Dr. David Shaw, Bangor University, UK. These two British isolates (mating type A1) will break through most of the *S. demissum* resistances present in the standard 11 Black differentials (Malcolmson and Black 1966). We tested these isolates repeatedly on these differentials and found that 98.170.3 is race 1.3.4.10.11 and Met+ is race 1.3.4.7.8.10.11. Unknown resistances recognized by both isolates may be novel or identical to *R2*, *R5*, *R6* or *R9*.

The isolates were maintained at 4°C on cleared solid 20% V8 agar (2%) supplemented with 100 $\mu g\ ml^{-1}$ carbenicillin. For long-term storage (1–2 years) we kept small plugs of agar with mycelium in sterile water at 4°C. Fresh sporangia were produced in a weekly cycle on detached leaves of a suitable, sensitive plant. During the latency period, the detached leaves were kept in petri dishes together with wetted pieces of florist's foam. This produces a moderately humid microenvironment inside the petri dish. The petri dishes were placed in an incubator set at 18°C and a 18/8-h (light/dark) photoperiod. Mature, fresh sporangia were harvested by rinsing the leaves with sterile demineralized water in a shallow dish, followed by a 1- to 4-h incubation of sporangia in water at 10°C to induce zoospore release.

Fresh zoospores were used to infect detached leaves. Four drops of water containing approximately 100 zoospores each were inoculated on the adaxial side of *S. mochiquense* leaves as they are more water repellent on the abaxial side. Sporulation and leaf blackening started on sensitive leaves about 5 days post-inoculation. Resistance scores were taken after 7 days. Each pathotest was conducted using two leaves placed in a 9-cm petri dish that was wrapped with cling film to prevent excessive drying. When the two leaves did not show the same reaction, the pathotest was considered to have been unreliable. Pathotests for individual genotypes were repeated independently to obtain a reliable score.

DNA analysis and mapping

For DNA extraction and gel-extraction, the appropriate Qiagen products and protocols were used, namely, the DNeasy Midi and -Mini kit, the DNeasy 96 Plant kit and the Qiaquick Gel Extraction kit (Qiagen, Valencia, Calif.). *Taq* polymerase and PCR buffers from Qiagen were used for most of the PCR reactions, except in the second amplification for amplified fragment length polymorphisms (AFLP) where AmpliTaq Gold (Applied Biosystems, Foster City, Calif.) was used. PCR products were cloned into pGEM-T Easy (Promega, Madison, Wis.), and sequences were obtained by automated sequencing using the ABI PRISM Big Dye Terminator Cycle Sequencing Ready Reaction kit (PE Applied Biosystems) according to the manufacturer's instructions. AFLP reactions were performed essentially as described by Thomas et al. (1995) and Vos et al. (1995) using *Pst*I + 2 and *Mse*I + 3 primers and a pre-amplification step. The AFLP reaction products were denatured and fractionated by electrophoresis on a 4.5% acrylamide/7.5 *M* urea/0.5× TBE buffer (45 m *M* Tris-borate, 1 m *M* EDTA) gel run at a 100 W for 2.5 h. After electrophoresis, the gels were transferred to Whatman 3 MM paper, dried without prior fixing and exposed to X-ray film (X-OMAT AR, Kodak) for 1–7 days.

Interesting AFLP bands were excised from the gel and rehydrated in TE (10 m *M* Tris-HCl, 1 m *M* EDTA, pH 8.0) buffer. Gel slices were transferred to 50 µl fresh TE buffer and crushed, and the debris spun in a microfuge at maximum speed for 1 min. To clone the AFLP fragments, we used 2 µl of the supernatant for PCR amplification, applying the same conditions as in the original AFLP reaction.

After finding a 1:1 segregating population, we followed a three-stage mapping strategy. (1) AFLP analysis was applied to 14 bulked segregants to determine the resistant (R) or sensitive (S) phenotype alongside the R and S parents. This resulted in the identification of several AFLP bands that were linked to the resistance. Polymorphic bands were cloned and sequenced. (2) AFLP sequences were used to design primer pairs that would amplify orthologous fragments in *Lycopersicon esculentum* and its wild relative *L. pennellii*. There is a high degree of sequence polymorphism between these two species that can be revealed by digesting the amplified fragments with four-base cutter restriction enzymes. Once the polymorphic markers between the species had been identified, they were tested in a set of *L. esculentum* (Le) lines with known *L. pennellii* (Lp) introgressions covering the whole tomato genome (Eshed and Zamir 1994). The polymorphic sequences enabled us to identify introgression lines with the Lp allele, thereby providing us with a rough map position of the novel *Rpi* gene. (3) To confirm this map position, we searched for mapped potato and tomato markers in the region using the web-based data of the Solanaceae Genomics Network (SGN; http://www.sgn.cornell.edu), Deutsches Ressourcenzentrum für Genomforschung (GABI; http://gabi.rzpd.de), The Institute for Genomic Research (TIGR; http://tigrblast.tigr.org) and the National Center for Biotechnology Information (NCBI; http://www.ncbi.nlm.nih.gov/mapview). Selected markers were sequenced in the R and S parents, and those that were polymorphic were mapped in our segregating population of 70 individuals.

Results

Genetic variation in gene bank accessions

Fine screening of five *S. mochiquense* accessions using detached leaf assays revealed phenotypic variation for resistance in three of them (Table 1). Two accessions appeared to harbour no resistance, neither in the tests reported here nor in earlier tests done by CGN. Resistant leaves were completely disease-free, while sensitive leaves turned black within 4 days in response to extensive mycelium growth inside the leaf and started sporulating 5–7 days after inoculation (Fig. 1).

Developing a mapping population segregating for *Rpi-moc1*

Six crosses were made between resistant and sensitive plants from three *S. mochiquense* accessions (Table 2). Segregation of resistance in A988 (CGN 18263) indicates the presence of a heterozygous *Rpi* gene (*Rpi-moc1*). The inter-accession cross A988×A966 was used as our mapping population. Two other plants from accession CGN 18263 (A989 and A982) transferred resistance to 100% of their progeny, possibly due to homozygosity of the same *Rpi* gene. A different *Rpi* gene may segregate in A995 (GLKS 2319). The small number of segregants from GLKS 2319 does not enable

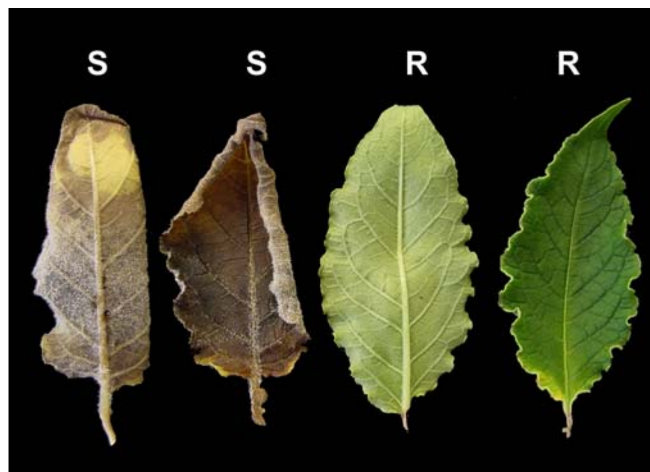

Fig. 1 *Solanum mochiquense* susceptible (*S*) and resistant (*R*) leaves, 7 days after inoculation with *Phytophthora infestans* zoospore suspension

Table 2 Crosses within *S. mochiquense* and late blight resistant (R) and sensitive (S) segregants in their progenies

Population parents[a]		Segregating progeny[b]	
Female	Male	R	S
A988, CGN18263, R	A966, CGN17731, S	51	45
A976, CGN17731, S	A995, GLKS2319, R	8	1
A989, CGN18263, R	A971, CGN17731, S	26	0
A982, CGN18263, R	A971, CGN17731, S	27	0
A996, GLKS2319, S	A993, GLKS2319, S	0	23
A983, CGN18263, S	A993, CGN17731, S	0	45

[a]Plant identifier number followed by its accession number and reaction to *P. infestans* inoculation: CGN Centre for Genetic resources in the Netherlands; GLKS Gross Lüsewitz, Konrad Schüler, Germany
[b]Number of plants showing resistant (R) or sensitive (S) phenotypes

distinction between a digenic 3:1 or a monogenic 1:1 segregation, but it suggests the presence of at least one major gene. Crosses between sensitive plants yielded 100% sensitive progenies, indicating the absence of recessive resistance genes or resistance suppressors.

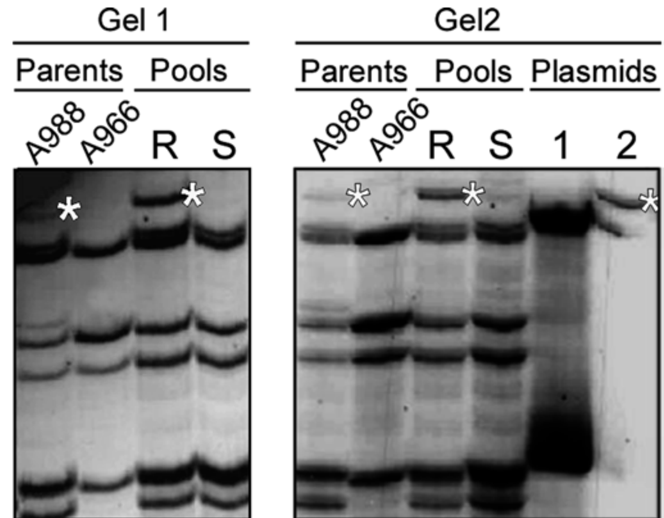

Fig. 2 AFLP on *S. mochiquense* for primer combination P2/M65 (+CT/+GGG). **Gel 1** In this partial image, an AFLP fragment (indicated by a *white star*) is polymorphic between the parents and the pools. This fragment is linked to *Rpi-moc1*. **Gel 2** The AFLP fragment was excised from the resistant parent and cloned. An AFLP reaction was performed using the plasmids carrying cloned fragments to select those with the correct size. Parents and pools were run alongside as size controls. *Plasmid 2* has the correct size and its sequence is homologous to that of EST E325111

AFLP, introgression lines and mapped marker conversions

A total of 32 AFLP primer combinations were used to obtain three polymorphic fragments linked to *Rpi-moc1*. Primer combination *Pst*I + CT/*Mse*I + GGG produced the largest fragment (410 bp) found only in the resistant pool and resistant parent (Fig. 2). The sequence of this fragment was similar to the singleton tomato expressed sequence tag (EST) SGN-E325111 (http://www.sgn.cornell.edu). Primers were designed on this sequence (Table 3) and tested on Le and Lp DNA. Digestion of the PCR product with *Alu*I revealed a polymorphism between these species. Analysis of Le/Lp introgression lines (Fig. 3) located this polymorphism in IL 9.3, indicating a map position at the distal end of the long arm of chromosome IX. Unfortunately, EST E325111 could not be mapped in the *S. mochiquense* population due to a lack of polymorphism within the amplified fragment.

To confirm the map position of *Rpi-moc1* on chromosome IX, we sequenced the potato RFLP marker CP110 (Rickert et al. 2003) in both the resistant and the sensitive parents of our mapping population. A detected single nucleotide polymorphism (SNP) was converted into a *Dde*I-dCAPS (cleaved amplified polymorphic sequence) marker (Table 3) using DCAPS FINDER 2.0 (http://helixwustledu/dcaps/dcaps.html), a programme that screens mismatch primers for polymorphic restriction enzyme recognition sites (Neff et al. 2002). Screening of

Table 3 Primers for PCR amplification of the markers used in this study (Le: *Lycopersicon esculentum*, Lp: *L. pennellii*)

Primer name[a]	Sequence (5′−3′)	Polymorphism[b]
E325111-f1	GTGATCGCGGTTGGAGTAAT	*Alu*I in Le/Lp
E325111-r2	CTCTAACTTCTCTATCAAAGTCCTCA	
CP110-r	TAGTAAGAGTACATGGAGAC	*Dde*I in A988/A966
CP110-f2	GATAACATCTTGGGTTACGTTGTC	
Sw5m237-f	TTAAGTCATCCCATGCCATAGAAT	*Eco*RI in A988/A966
Sw5m237-r	GGGAGATACGGTTGACGTTC	
T156-F2	AAGGCAGGAACAAGATCAGG	*Rsa*I in A988/A966
T156-R2t	TTGACAGCAGCTGGAATTG	
TG328-F	AATTAAATGGAGGGGGTATC	*Alu*I in A988/A966
TG328-R	GTAGTATTCTAGTTAAACTACC	

[a]E325111 was found by homology to an excised AFLP band. CP110, Sw5, T156 and TG328 were selected from published chromosome IX maps of potato and tomato
[b]Polymorphisms between Le and Lp or the resistant (A988) and sensitive (A966) parents of the mapping population are revealed by digestion of the amplified products with the restriction enzyme indicated

Fig. 3 Identification of chromosome IX as the carrier of the *Lycopersicon esculentum* (*Le*) and its wild relative *L. pennellii* (*Lp*) allele of E325111. The polymorphism between Le and Lp is revealed by restriction digestion with *Alu*I. *Lanes 1–12* Pools of introgression lines, each representing introgressions in single chromosomes. The complete set of 12 tomato chromosomes covering the Le chromosomes (from *1 to 12*) was used. *c* Control PCR without DNA, *M* 1-kb size marker (GibcoBRL, Gaithersburg, Md.)

the mapping population with CP110 revealed 12 recombinants among 67 segregants (18%), thereby providing further evidence for the linkage of *Rpi-moc1* to chromosome IX.

We next analysed *Sw-5*, a nucleotide-binding site-leucine-rich repeat (NBS-LRR) gene for tospovirus resistance from *Lycopersicon peruvianum* (Brommonschenkel et al. 2000; Spassova et al. 2001) that mapped to a location 20–40 cM distal to CP110. We converted an SNP in the middle of the gene into an *Eco*RI-dCAPS (Table 3). This dCAPS marker was visualized by silver staining on 8% polyacrylamide gel because the cleaved band was very weak, presumably due to the amplification of multiple *Sw-5* homologues (Fig. 4). The *Sw-5* marker identified nine recombinants in 68 segregants (14%) and one double recombinant. Comparative mapping data positions CP110 on the centromeric side and *Sw-5* on the telomeric side of *Rpi-moc1* (Fig. 5). This map position allowed us to select further markers.

An *Rsa*I CAPS marker based on the sequence of conserved orthologue set (COS) marker T156 was analysed in the mapping population. T156 identified five recombinants in 68 segregants (7%) that were shared with those detected with CP110 (Fig. 5). The RFLP marker TG328, 7 cM distal to T156, was converted into a CAPS marker. Digestion of the PCR products with *Alu*I revealed a polymorphism linked to *Rpi-moc1*. Analysis of TG328 in our mapping population detected no recombinants in 68 plants. We concluded that *Rpi-moc1* co-segregates with TG328, flanked by T156 and *Sw-5* on chromosome IX (Fig. 5).

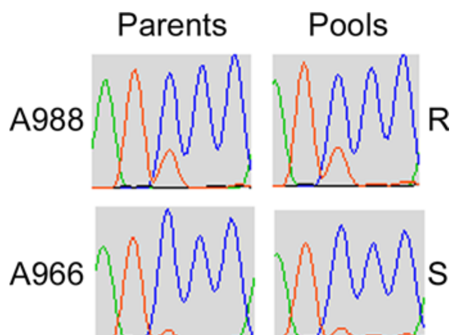

Fig. 4 Sequence trace obtained from *Sw-5* PCR products. The sequence of *Sw-5* reveals a single nucleotide polymorphism between the parents and pools of the *Rpi-moc1* mapping population, indicating linkage between *Sw-5* and *Rpi-moc1*. Note that the heterozygous peaks have a 3:1 ratio, probably due to co-amplification of an *Sw-5* homologue. R Resistance, S sensitive

Discussion

Many *Rpi* genes have been identified and named. In this article we follow the system of van der Vossen et al. (2003) for naming new *Rpi* genes. This system seems to be the best choice because several series of *Rpi* genes from different *Solanum* species can be incorporated easily.

Of the 11 genes described from the hexaploid *S. demissum* (Black et al. 1953; Malcolmson and Black 1966), five have been mapped: *R1* on chromosome V (Leonards-Schippers et al. 1992) in a cluster with several *R* genes conferring resistance to diverse pathogens (Grube et al. 2000); *R3*, *R6* and *R7* in a cluster on chromosome XI (El-Kharbotly et al. 1994) and *R2* on chromosome IV (Li et al. 1998). Other *Rpi* genes from diploid wild species that have already been mapped are R_{ber} from *S. berthaultii* on chromosome X (Ewing et al. 2000), *RB* (Naess et al. 2000), R_{ABPT} (Park et al. 2003) and *Rpi-blb2* (Van der Vossen et al. 2004) from *S. bulbocastanum* on chromosomes VIII, IV and VI, respectively, and *Rpi1* from *S. pinnatisectum* on chromosome VII (Kuhl et al. 2001).

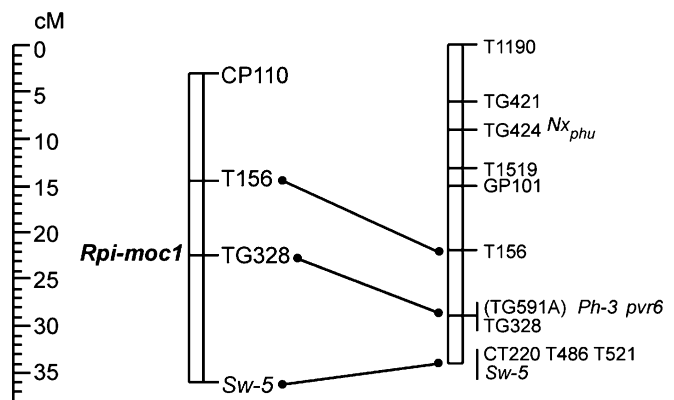

Fig. 5 Map of *S. mochiquense* chromosome IX near *Rpi-moc1*. On the *right*, the corresponding region in a cross of Le×Lp (00, NCBI MAPVIEWER) is shown. *Ph-3* is a *P. infestans* partial resistance gene (Chunwongse et al. 2002), *Sw-5* is an NB-LRR tospovirus resistance gene (Brommonschenkel et al. 2000), Nx_{phu} confers resistance to potato virus X (PVX, Tommiska et al. 1998), *pvr6* is a recessive virus resistance gene from pepper (Caranta et al. 1996). The location of *Ph-3* and *pvr6* is inferred from its linkage with TG591A. TG591A co-segregates with TG328 in another tomato map (Le×Lp 92, NCBI MAPVIEWER). A QTL for resistance to *Clavibacter michiganense* was associated with TG424 (Sandbrink et al. 1995)

The first *Rpi* gene that was cloned and sequenced was *R1* (Ballvora et al. 2002), soon followed by *RB* (Song et al. 2003; van der Vossen et al. 2003) and Rpi-blb2 (Van der Vossen et al. 2004). All three belong to the class of *R* genes with a coiled coil domain, nucleotide binding site and leucine rich repeats (CC-NB-LRR). Fine mapping of the *R3* locus revealed that this race-specific resistance is controlled by two tightly linked genes that are likely to be *I2* homologues, so *R3* also belongs to the CC-NB-LRR class of *R* genes (Ori et al. 1997; Huang et al. 2004). NB-LRR genes are involved in the recognition of avirulence factors associated with specific strains of pathogens as diverse as aphids, nematodes, viruses and fungi. Several *Rpi* genes are found in clusters of various *R* genes that sometimes co-localize with *R*-gene homologues of unknown function. This clustering is more pronounced when the *R* genes and homologues on tomato, red pepper and potato are combined on a comprehensive *Solanum* map (Grube et al. 2000).

Rpi-moc1 also maps within a cluster of resistance genes (Fig. 5). It is close to the QTL *Ph-3* near TG591A from *Lycopersicon pimpinellifolium* (Chunwongse et al. 2002) that confers 71% variance of resistance to *P. infestans* in a cross with *L. esculentum*. It is more loosely clustered with *Sw-5*, a CC-NB-LRR gene for tospovirus resistance from *L. peruvianum* (Brommonschenkel et al. 2000), and Nx_{phu}, a gene for hypersensitive resistance to PVX linked to TG424 (Tommiska et al. 1998). This loose cluster also carries a QTL from *L. peruvianum* contributing to *Clavibacter michiganensis* (bacterial canker) resistance in tomato (Sandbrink et al. 1995) and *pvr6*, a recessive, *pvr2*-dependent pepper veinal mottle virus resistance from *Capsicum annuum*. *Pvr6* maps 10–20 cM from TG57 (Caranta et al. 1996) and possibly a similar distance from TG591A (Pflieger et al. 1999).

Solanum mochiquense has been tested for *Phytophthora infestans* resistance in a few other studies. Remarkably, no resistance was detected by Van Soest et al. (1984) and Ruíz de Galarreta et al. (1998) in the same accessions. Both studies only tested an average resistance based on 20–24 plants, and paid no attention to low proportions of highly resistant plants. On the other hand, Perez et al. (2000) found 11 susceptible, 25 intermediate, and 12 resistant plants in a fine screening of one CIP accession. This underlines the importance of fine screening. *S. mochiquense* may also be a source for other disease resistances: Ruíz de Galarreta et al. (1998) observed resistance to potato leaf roll virus (PLRV) in GLKS 2319, and the CGN database mentions blackleg (*Erwinia carotavora*) resistance in CGN18263.

Several researchers have pointed out that Mexico is a rich source of *Rpi* gene diversity (Van Soest et al. 1984). This is best illustrated with the examples of *S. demissum*, *S. bulbocastanum*, *S. pinnatisectum* and *Solanum brachistotrichum* (W.D. Smilde and J.D.G. Jones, unpublished data). However, the central Andes region of Bolivia and the north of Argentina is also a source of *Rpi* genes, most notably in *S. berthaultii* and *S. microdontum* but also in *S. okadae* and *S. neorossii* (G. Brigneti and J.D.G. Jones, unpublished data). In this light, it was unexpected to find resistance originating in the Peruvian foothills of the Andes where the infection pressure of *Phytophthora infestans* populations is believed to be low. This prompted us to investigate resistance in *S. chancayense* (CGN18036 and CGN18356), a close relative of *S. mochiquense* from the same region. This species was only recently introduced into the gene bank system and its response to *P. infestans* was unknown. Although we did not find monogenic resistance to late blight, plants from these accessions were not typically sensitive to the pathogen, and some degree of resistance was observed. Our pathogen tests are not designed to detect quantitative resistance, but the possibility remains that *S. chancayense* may harbour resistance QTLs against *P. infestans*.

Among botanists, the foothills of the Andes are famous for their 'fog oases' in an otherwise stony desert landscape (Dillon 1997). The desert climate is irregularly interrupted by El Niño events that bring heavy rain to the dry areas. The fog oases or *lomas* formations harbour unusual plant communities consisting of numerous endemic species. *S. mochiquense* and *S. chancayense* belong to this unique vegetation. Our discovery of important resistance in a few *lomas* near Lima and Trujillo makes it worthwhile to collect more plant material from other fog oases. Also, it would be interesting to observe natural infection by *P. infestans* in the *lomas* vegetation and to study plant-pathogen co-evolution in a natural metapopulation, defined by fog islands. *Lomas* are found in 80 distinct locations along 3,500 km of South American coastline, from Peru into Chile. These *lomas* may become the Galapagos of the *Rpi* evolutionist, if not the source of a diverse set of *Rpi* genes. These novel genes may enable us to implement a strategy for durable late blight resistance based on a potato *Rpi* gene polyculture.

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