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Mapping of loci involved in quantitatively inherited resistance to the potato cyst-nematode *Globodera rostochiensis* pathotype Ro1

C. M. Kreike[1], J. R. A. de Koning[1], J. H. Vinke[2], J. W. van Ooijen[3], C. Gebhardt[4], W. J. Stiekema[1]

[1] Department of Molecular Biology, DLO-Centre for Plant Breeding and Reproduction Research (CPRO-DLO) P.O. Box 16, 6700 AA Wageningen, The Netherlands
[2] Department of Arable and Fodder Crops
[3] Department of Population Biology
[4] Max-Planck-Institut für Züchtungsforschung, D-50829 Köln, Germany

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Abstract. We report the identification and mapping of two quantitative trait loci (QTLs) of *Solanum spegazzinii* BGRC, accession 8218-15, involved in resistance to the potato cyst-nematode *Globodera rostochiensis* pathotype Ro1, by means of restriction fragment length polymorphisms (RFLPs). For this purpose we crossed a susceptible diploid *S. tuberosum* with the resistant *S. spegazzinii*, and tested the F_1 population for resistance to the Ro1 pathotype. Since the F_1 segregated for the resistance, the *S. spegazzinii* parent was concluded to be heterozygous at the nematode resistance loci. For the mapping of the resistance loci we made use of RFLP markers segregating for *S. spegazzinii* alleles in the F_1. One hundred and seven RFLP markers were tested in combination with four different restriction enzymes; 29 of these displayed a heterozygous RFLP pattern within *S. spegazzinii* and were used for mapping. Analysis of variance (ANOVA) was applied to test the association of the RFLP patterns of these markers with nematode resistance. Two QTLs involved in disease resistance to *Globodera rostochiensis* pathotype Ro1 were identified and mapped to chromosomes 10 and 11 respectively.

Key words: *Solanum spegazzinii* – *Globodera rostochiensis* – Nematode resistance – QTL – RFLP

Introduction

Potato cyst nematodes belong to the major pests of potato. Two potato cyst nematode species are known, *Globodera rostochiensis* (Woll.) and *G. pallida* (Stone), and several pathotypes have been described for each species (Kort et al. 1977). Resistance to Ro1, the most important pathotype of *G. rostochiensis*, has been identified in several *Solanum* species (Ellenby 1952, 1954). A monogenic resistance locus (*H1*) present in *S. tuberosum* ssp *andigenum* (Toxopeus and Huijsman 1953) has been widely used in plant breeding and provides complete resistance to pathotypes Ro1 and Ro4. Recently, a gene-for-gene relationship between this resistance gene (*H1*) from *S. tuberosum* ssp *andigenum* and the avirulence gene of *G. rostochiensis* has been proven by Janssen et al. (1991). Another source of resistance to *G. rostochiensis* pathotype Ro1 is *S. spegazzinii*. *S. spegazzinii* is a diploid wild potato species which displays resistance to several *G. rostochiensis* and *G. pallida* pathotypes (Dellaert et al. 1988). Ross (1962) described two independent monogenic dominant resistance genes *Fa* and *Fb*, in the accession EBS510. The *Fa* gene is involved in resistance to *G. rostochiensis* pathotypes Ro1 and Ro2 while the *Fb* gene provides resistance to the Ro1 and Ro5 pathotypes and, in combination with minor genes, also to the Ro2, 3 and 4 pathotypes (Ross 1986).

With the use of molecular markers like RFLPs, linkage maps of the potato chromosomes have been constructed (Bonierbale et al. 1988; Gebhardt et al. 1989, 1991) that allow the genetic localisation of genes of interest. Concerning nematode resistance in potato, a resistance locus *Gro1* against *G. rostochiensis* pathotype Ro1 from *S. spegazzinii* has been mapped on chromosome 7 (Barone et al. 1990) and the *H1* resistance gene from *S. tuberosum* ssp *andigenum* on chromosome 5 (Gebhardt et al. 1993; Pineda et al. 1993).

For the introgression of monogenic traits into *S. tuberosum*, screening and selection for the phenotype

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Correspondence to: W. J. Stiekema

can be straightforward, but for quantitative traits, marker-based selection with RFLPs is advantageous. In this paper we describe the inheritance of the quantitative resistance to the *G. rostochiensis* Ro1 pathotype from *S. spegazzinii* BGRC accession 8218-15. The aim of this research is to map the loci involved in this resistance by means of RFLPs. Markers close to the resistance loci can subsequently be used for marker-based selection. To gain further insight into the nature of the detected resistance loci we also analyzed the interaction between the genes and their individual contribution to the resistance.

Materials and methods

Plant material

S. spegazzinii BGRC accession 8218-15 was used as a pollen donor in a cross with the diploid susceptible *S. tuberosum* SH 78-88-1320. Ninety-six F_1 plants were tested for resistance to *G. rostochiensis* pathotype Ro1 as described below. The *S. spegazzinii* parent appeared to be heterozygous for the resistance loci because resistance to pathotype Ro1 was segregating in the F_1 progeny. Mapping of the resistance genes with RFLPs was, therefore, performed in this F_1 population.

RFLP analyses

Thirty-one genomic tomato clones (Tanksley et al. 1987) and 76 genomic *S. spegazzinii* clones, indicated by TG and Ssp, respectively, were used in combination with four restriction enzymes (*Hin*dIII, *Eco*RI, *Eco*RV, *Dra*I) to detect heterozygous RFLP patterns within *S. spegazzinii*. Segregating RFLP patterns in the F_1 progeny were required to identify associations with the resistance loci that were also segregating in the F_1 population. The chromosomal location of the TG clones in potato has been determined previously (Bonierbale et al. 1988; Gebhardt et al. 1991) and the 31 TG clones we used were evenly distributed over the potato chromosomes. The 76 Ssp clones were a random sample of a genomic *Pst*I library from *S. spegazzinii* 8218-15, and are currently being mapped on the potato chromosomes in another population as well (C. M. Kreike, manuscript in preparation).

Leaf material was harvested during propagation of the plants in the greenhouse and stored at −80 °C. DNA isolation and digestion were carried out according to Dellaporta et al. (1983). Southern blotting and hybridisation were performed with the non-radioactive hybridisation and detection techniques described previously (Kreike et al. 1990).

Three consecutive RFLP screenings were carried out. (1) Initially a random sample of 57 F_1 plants was taken. (2) If the association between marker-genotype and nematode resistance was significant at the 10% level then an additional set of 19 plants was screened. (3) If association at the 5% significance level was found, all F_1 plants were screened with that RFLP marker. Equal segregation of the RFLP alleles in the F_1 population was determined with the chi-square goodness-of-fit test.

Resistance test

Five tubers of each F_1 individual were tested for resistance to *G. rostochiensis* pathotype Ro1 in a randomized block experiment. The parents and a susceptible standard (*S. tuberosum* cv Maritta) were included in the test. Cysts from the "Mierenbos A" population of *G. rostochiensis* pathotype Ro1 (Arntzen and van Eeuwijk 1993) were used as an inoculum. Sprouted tubers (one per replicate) were planted in separate clay pots, 10 cm in diameter (295 ml), which were filled with loam sand. The pots were each inoculated with 30 cysts enclosed in a nylon net, allowing the hatched larvae to pass and invade the roots. Growing conditions, soil temperature and soil moisture control were as described by van der Wal and Vinke (1982). After 4 months of plant growth the water supply was gradually stopped. One month later, when the soil was air-dry, the cysts were recovered by flotation. The nylon inoculation net was retrieved so that only newly-formed cysts were collected and counted.

Statistical analysis

A normalizing transformation $[^{10}\log(x+1)]$ was performed on the counts of the newly-formed cysts per replicate. These transformed data of the resistance test were investigated with analysis of variance (ANOVA) for a randomized block design. From this ANOVA the heritability of the genotypic mean values for Ro1 resistance could be calculated with formula (1):

$$h^2 = \frac{\sigma_g^2}{\sigma_g^2 + \frac{\sigma_e^2}{5}} \quad (1)$$

h^2 = heritability, σ_g^2 = genetic variance, σ_e^2 = environmental variance.

The association of marker genotypes with resistance was assessed with one-way ANOVAs based on the averages over the five replicates of the log-transformed cyst counts. A significance level of 5% was employed. The magnitude of the marker-associated phenotypic effect is described by the coefficient of determination (R^2), which represents the fraction of the total variance accounted for by the marker genotypes.

The interaction between the loci involved in Ro1 resistance was resolved by constructing a two-way ANOVA. The RFLP patterns of two markers were combined so that four marker-genotype classes were obtained. The mean number of cysts was calculated for each class.

Results

RFLP analysis

Two different sets of markers, TG and Ssp, were employed to detect RFLP patterns that segregated for *S. spegazzinii* alleles in the F_1 population. This screening yielded 29 suitable clone-restriction enzyme combinations (Table 1). None of the four markers known to map on chromosome 8 showed segregation for *S. spegazzinii* alleles. The chromosomal location of the 29 clones is given in Table 1, column 1, while one Ssp markers could not be mapped on the chromosomes. The localisation of the Ssp markers on the chromosomes was done using another population as well (C. M. Kreike, manuscript in preparation). Most loci followed a 1:1 segregation pattern but seven out of the 29 clones (24%) showed a distorted segregation (Table 1, column 2). Only 27% of the markers that were used to find heterozygous *S. spegazzinii* alleles were informative, whereas 40% were heterozygous for *S. tuberosum* alleles.

Table 1. Segregation ratios of RFLP markers heterozygous for *S. spegazzinii* alleles and the association of these markers with resistance against *G. rostochiensis* pathotype Ro1

Marker	Chromosome	Ratio	*P*
Ssp 55	1	30:44	
TG34	2	25:28	
TG 134	3	17:32*	
Ssp 80	3	32:27	
TG 123	4	37:32	
Ssp 27	4	47:40	(0.053)
TG 23	5	30:11*	
TG 115	6	37:37	
TG 118	6	20:53*	
TG 143	7	33:48	
Ssp 51	7	39:31	
Ssp 57	7	25:19	
Ssp 132	7	27:26	
TG 8	9	38:28	
TG 9	9	36:19*	
TG 63	10	53:31*	0.014
Ssp 64	10	49:42	
Ssp 75	11	43:50	0.012
TG 30	11	29:40	0.041
TG 28	12	19:31	
Ssp 83	12	36:14*	
Ssp 67	12	26:25	
Ssp 126	12	25:23	
Ssp 129	12	24:18	
Ssp 37		33:36	
Ssp 112		37:30	
Ssp 124		29:22	
Ssp 72		33:21	
Ssp 45		15:36*	

* Significant distortion of the segregation ratio 1:1 ($P < 0.05$)
P = Probability level for association with disease resistance, only shown if $P < 0.05$

Statistical analysis

The number of newly-formed cysts in the F_1 population ranged from zero to almost 500. One cyst and 323 cysts were formed respectively on the resistant *S. spegazzinii* parent and the susceptible *S. tuberosum* parent while on the susceptible standard cultivar, Maritta, approximately 1300 cysts were counted. The frequency distribution of the average of the log-transformed cyst count per F_1 genotype is given in Fig. 1. Kurtosis and skewness were 0.006 and -0.093, respectively, which is not indicative of a significant deviation from a normal distribution. This means that the Ro1 resistance studied is quantitatively inherited. The genetic variance was very significant ($P < 0.001$, Table 2) and emphasised the segregation of resistance loci in the F_1. The heritability of the genotypic mean values for Ro1 resistance was estimated to be 0.63.

One-way ANOVAs were carried out to find associations between the 29 RFLP markers which showed segregation for the *S. spegazzinii* alleles and the nematode resistance of the F_1 progeny. *P*-values < 0.05, with respect to the association of the RFLP markers with Ro1 resistance, are indicated in Table 1, column 4. The only three RFLP markers that fulfil this requirement are TG 63 on chromosome 10 and Ssp 75 and TG 30 on chromosome 11. Ssp 75 and TG 30 were linked by 10 cM (Fig. 2). The map position of Ssp 75 was also confirmed by placing it within the framework of the potato RFLP map of Gebhardt et al. (1991). The marker Ssp 27 on chromosome 4 is on the verge of significance ($P = 0.053$). Table 3(a, b) shows the

Table 2. Analysis of variance (ANOVA) of the five replications after transformation [$^{10}\log(x+1)$] of the original data of the resistance test

Source	*df*	SS	MS	*P*
Between replication	4	5.37	1.34	0.002
Between F_1 plants	95	80.00	0.84	<.001
Residual	351	107.50	0.31	

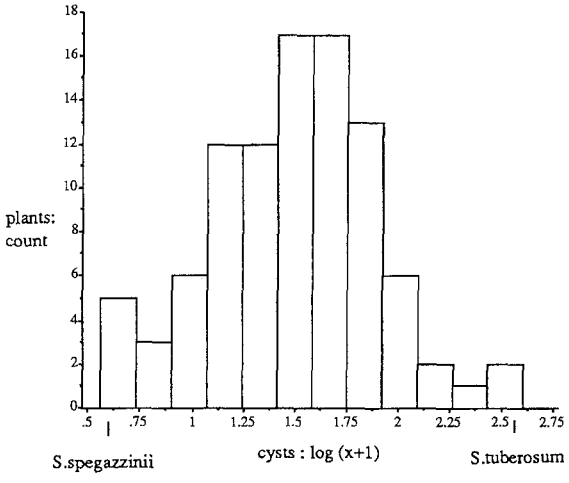

Fig. 1. Frequency distribution of the mean number of cysts formed on the 96 F_1 plants

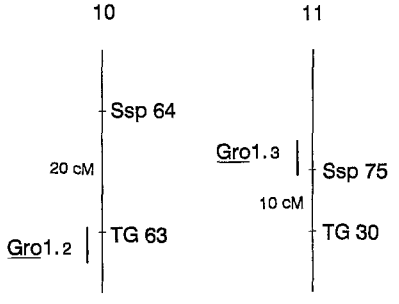

Fig. 2. Localization of the resistance loci *Gro1.2* and *Gro1.3* on chromosome 10 and 11, respectively

Table 3a. One-way ANOVA for association of the RFLP markers TG 63 with disease resistance against *G. rostochiensis* pathotype Ro1. Below the ANOVA the mean number of cysts calculated for each marker-genotype class are presented. MGC = marker-genotype classes
TG 63

Source	*df*	SS	MS	F value	*P*
Between MGC	1	1.1	1.1	6.34	0.014
Within MGC	82	14.21	0.17		
Total	83	15.31			

MGC	Number of plants	Mean	Std. error
1	53	1.56	0.06
2	31	1.32	0.07

Table 3b. One-way ANOVA for association of the RFLP markers Ssp 75 with disease resistance against *G. rostochiensis* pathotype Ro1. Below the ANOVA the mean number of cysts calculated for each marker-genotype class are presented. MGC = marker-genotypes classes
Ssp 75

Source	*df*	SS	MS	F value	*P*
Between MGC	1	1.06	1.06	6.56	0.012
Within MGC	91	14.72	0.16		
Total	92	15.78			

MGC	Number of plants	Mean	Std. error
1	43	1.60	0.06
2	50	1.39	0.06

ANOVA results for the markers TG 63 and Ssp 75; the latter has a higher significance level for being associated with an Ro1 resistance locus on chromosome 11 than does TG 30. These data indicate that loci are present on chromosomes 10 and 11 which are involved in quantitative resistance to *G. rostochiensis* pathotype Ro1.

Since the ANOVA assumes a normal distribution of the data, and this assumption might be violated with the present data, we verified the ANOVA results by performing a non-parametric test (Mann-Whitney U-test) on the 29 RFLP markers. Again the markers TG 63, Ssp 75 and TG 30 were the only ones to be significantly associated ($P < 0.05$) with Ro1 resistance (data not shown). These results are again a strong indication that loci are present on chromosomes 10 and 11 which are involved in quantitative resistance to *G. rostochiensis* pathotype Ro1.

The segregation of RFLP markers showing heterozygosity of the susceptible parent *S. tuberosum* SH 78-88-1320 was also scored in the F_1 population. Ssp 75, which was associated with nematode resistance in *S. spegazzinii*, also segregated for *S. tuberosum* alleles. Forty-three RFLP markers were examined with ANOVA for associations with Ro1 resistance as described above (data not shown). None of the 43 RFLP markers were significantly associated with Ro1 resistance; therefore, no influence on the resistance from the susceptible parent *S. tuberosum* could be detected.

Table 4. Two-way ANOVA table of the markers TG 63 and Ssp 75. Below the ANOVA the mean number of cysts calculated and the number of plants (in brackets) for each marker-genotype class are given

Source	*df*	SS	MS	F value	*P*
TG 63 (A)	1	1.21	1.21	7.76	0.007
Ssp 75 (B)	1	1.34	1.34	8.60	0.004
Interaction	1	0.40	0.40	2.60	0.111
Error	79	12.29	0.16		

		Ssp 75		
		1	2	Total
TG 63	1	1.65 (21)	1.50 (32)	1.56 (53)
	2	1.52 (16)	1.07 (14)	1.31 (30)
	Total	1.59 (37)	1.37 (46)	1.47 (83)

The coefficient of determination (R^2) calculated for TG 63 and Ssp 75 was 7% for both. This means that 14% of the total variation and 22% of the genetic variation can be explained by these two markers.

To determine the interaction between the two RFLP loci that were associated with resistance to *G. rostochiensis* pathotype Ro1, a two-way ANOVA was performed (Table 4). No interaction was found between markers TG 63 and Ssp 75 indicating that both loci were additive.

Discussion

Genetic diversity

Ross (1962) was the first to describe the inheritance of the resistance of *S. spegazzinii* EBS510 to *G. rostochiensis*. He reported high numbers of resistant progeny after crossing *S. spegazzinii* with a susceptible *S. tuberosum* and postulated that two independant dominant genes were responsible for the resistance. Momeni et al. (1969) used another genotype of *S. spegazzinii* and detected only one dominant gene involved in nematode resistance. Also Barone et al. (1990) found a clear 1:1 segregation in an F_1 population, indicating monogenic inheritance of this resistance trait. The present study, however, employed yet another source of *S. spegazzinii* which does not reveal a

simple qualitative inheritance of the resistance to pathotype Ro1, but rather a quantitative inheritance. These data show that the species *S. spegazzinii* is very diverse with respect to nematode resistance, as was already noted by Jones and Pawelska (1963).

Furthermore, the loci involved in the quantitative Ro1 resistance described here, are located on different chromosomes to the locus mapped by Barone et al. (1990), who located the monogenic resistance *Gro1* on chromosome 7. They are also different from the resistance locus *H1* from *S. tuberosum* ssp *andigena*. This locus mapped to chromosome 5 (Gebhardt et al. 1993; Pineda et al. 1993). Quantitative resistance to *G. rostochiensis* pathotype Ro1 has only been previously observed in *S. vernei* (Plaisted et al. 1962). We describe here for the first time quantitatively-inherited resistance to *G. rostochiensis* pathotype Ro1 from *S. spegazzinii*. We were able to observe and map this quantitative resistance since no monogenic resistance locus was present in our *S. spegazzinii* parent that could conceal the expression of minor, quantitative loci.

RFLP analysis

The level of heterozygosity found with RFLP analysis in *S. spegazzinii* was lower than the level of heterozygosity found in *S. tuberosum*. Both species are self-incompatible so the difference could be due to the fact that *S. tuberosum* has been crossed with several wild species for improvement of the crop which has resulted in a highly polymorphic species (Gebhardt et al. 1989), whereas the *S. spegazzinii* accession used here has apparently a much smaller gene pool and, therefore, a lower level of heterozygosity.

Distorted segregation ratios were detected for 24% of the markers that were segregating for *S. spegazzinii* alleles. Skewed segregation has been reported previously and seemed to be associated with distinct parts of the chromosomes (Bonierbale et al. 1988. Gebhardt et al. 1991). In the present experiment *S. spegazzinii* was used as a male parent. The segregation of alleles from *S. tuberosum*, which was used as a female parent, was skewed in only 18% of the markers used (data not shown). We assume that the higher distortion in *S. spegazzinii* could be caused by selective elimination of male gametes (Rick 1969).

Statistical analysis

The small number of RFLP markers displaying polymorphism in the F_1 population did not allow the use of interval mapping (Lander and Botstein 1989), since no contiguous RFLP linkage map could be constructed. However, it is also possible to test single RFLP markers for association with quantitative traits by means of analysis of variance (e.g., Keim et al. 1990). We applied this method to examine the 29 segregating RFLP markers and were able to detect three markers, TG 63, Ssp 75 and TG 30, that were significantly associated with the resistance trait. A significance level of 5% for the association of an RFLP marker with Ro1 resistance was obtained both after ANOVA and after the Mann-Whitney U-test (non-parametric test). We realise that this may lead to a high overall error rate. Therefore, these significant associations must be seen as strong indications of the presence of quantitative resistance loci. The fact that both linked markers on chromosome 11 are significantly associated with nematode resistance, strengthens the point that a QTL is located on chromosome 11. The three significant markers identified two QTLs for resistance to *G. rostochiensis* pathotype Ro1. We propose to name these loci on chromosome 10 and 11 as *Gro1.2* and *Gro1.3*, respectively, analogous to the monogenic resistance locus *Gro1* on chromosome 7 against *G. rostochiensis* pathotype Ro1 (Batone et al. 1990).

Only 22% of the genetic variation can be explained with the two loci we found. There are two possible explanations for this. Firstly, although the RFLPs are linked to loci involved in nematode resistance, the distance between marker and locus can still be large. Cross-overs between markers and resistance locus will reduce the association found with ANOVA. Secondly, there may be other loci involved in Ro1 resistance that could not be detected with the experimentation employed. A larger F_1 population and more segregating markers are needed to detect these QTLs. It is possible that the RFLP marker Ssp 27, which was on the verge of significance for association with Ro1 resistance, is linked to a third locus but the distance between marker and locus may be too large to detect a significant association. Additional markers, segregating for *S. spegazzinii* alleles, are needed to investigate whether a third locus on chromosome 4 is present, but because of the lack of heterozygosity in *S. spegazzinii* such markers are hard to obtain. A backcross population, with a higher level of heterozygosity will be more informative to find additional resistance loci and markers closer to the *Gro1.2* and *Gro1.3* loci.

The potato genome is homoeologous to the tomato genome and the numbering of the chromosomes is similar (Bonierbale et al. 1988; Gebhardt et al. 1991). Interestingly, in tomato, resistance loci to *Fusarium oxysporum* (I2) and *Stemphylum sp.* have also been mapped to chromosome 11 (Sarfatti et al. 1989; Behare et al. 1991, respectively). The *Fusarium oxysporum I2* locus in tomato is mapped on the long arm of chromosome 11 (Segal et al. 1992). The *Gro1.3* resistance locus has also been mapped on this arm. Interestingly, the monogenic *Gro1* resistance locus (Barone et al. 1990) from *S. spegazzinii* is closely located to yet another *Fusarium* resistance locus, *I1*, (Sarfatti et al. 1991) on

chromosome 7 (Leonards-Schippers et al. 1992). It is striking that specific resistance to different pathogens have been mapped to the same chromosome regions. Maybe the organisation of the resistance genes found on the specific chromosome regions in plants is similar to the human MHC complex (Dangl 1992). Analogous to the MHC complex these chromosome regions could have hotspots for recombination and therefore allow the creation of new resistance genes with different specificities.

This article has described the identification and localisation of quantitative resistance loci to *G. rostochiensis* pathotype Ro1 by means of RFLPs. It also shows that the identified loci account for 22% of the genetic variation and that they have an additive gene action. To explain all the genetic variation a closer linkage of RFLPs to the identified Ro1 resistance loci is needed. Putative additional loci will be detected by screening a backcross population of a resistance F_1 plant and the susceptible *S. tuberosum* parent with RFLP markers.

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