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Selection of virulence in *Meloidogyne chitwoodi* to resistance in the wild potato *Solanum fendleri*

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Abstract

The possibilities of selecting virulence from a virtually avirulent root-knot nematode population towards resistance in wild potato have been investigated. Single egg masses of *Meloidogyne chitwoodi*, which had occasionally been produced on roots of resistant *Solanum fendleri* gave rise to eight lines after one generation on tomato. Five lines were able to circumvent completely the resistance of *S. fendleri* 93-114-12 resulting in a susceptible response similar to that of the control potato cv Nicola. Subsequently, a resistance test with other resistant genotypes of *S. fendleri*, *S. bulbocastanum*, *S. stoloniferum* and *S. hougasii* revealed that the virulent lines were also able to break through the resistance in most other species, but clear differences were noticed between the virulent lines. The results suggest a simple inheritance of virulence in *M. chitwoodi* towards resistance in *S. fendleri*. However, more virulence factors are involved to explain the differences on the other *Solanum* species between the virulent lines. The implications of the ease to select virulence with respect to the practical use of resistance in potato breeding and growing are discussed.

Introduction

In several agricultural crops host plant resistance has proven to be a very effective and environmental friendly means of controlling root-knot nematodes, *Meloidogyne* spp., as an alternative to chemical nematicides. In the case of potato, resistance to *M. chitwoodi*, *M. fallax* and *M. hapla* is absent in currently used potato cultivars, but high levels of resistance to these nematode species have been identified in wild tuber-bearing *Solanum* spp., amongst which is *S. bulbocastanum* and *S. fendleri* (Brown et al., 1989; Brown et al., 1991; Janssen et al., 1996), and introgression into the cultivated potato gene pool is in progress (Brown et al., 1994; Janssen et al., 1997c).

Sometimes, resistance becomes ineffective when populations within a nematode species are able to overcome the plant resistance, i.e. being virulent. The occurrence of resistance/virulence interactions - sometimes leading to pathotype designations - has been described in several major agricultural crops, predominantly in the case of sedentary nematode species like *Globodera* spp. on potato (Kort et al., 1977), *Heterodera schachtii* on sugar beet (Müller, 1992), *Heterodera avenae* on cereals (Lasserre et al., 1996), and *Meloidogyne* species on tomato and other crops (e.g. Cook and Evans, 1987; Roberts, 1995).

Most information on virulence in *Meloidogyne* spp. is known with regard to the resistance in tomato based on the *Mi* gene, originally derived from *Lycopersicon peruvianum*. This resistance to the (sub-) tropical nematode species *M. incognita*, *M. arenaria* and *M. javanica* is based on a single dominant gene (Gilbert and McGuire, 1956) and has been frequently used to breed resistant tomato cultivars since 1950 (Roberts and Thomason, 1989). Virulent populations of *M. incognita*, *M. arenaria* and *M. javanica* towards the *Mi* resistance have been selected in the field after consecutive cultivation of resistant tomato cultivars (Riggs and Winstead, 1959; Sauer and Giles, 1959; Netscher, 1977), but resistance-breaking populations have also been found in areas which have not been pre-

viously exposed to resistance (Netscher, 1977; Prot, 1984; Roberts and Thomason, 1989; Berthou et al., 1989; Fargette and Braaksma, 1990). Selection experiments showed a progressive increase in the proportion of virulent nematodes after each successive generation (Netcher, 1977; Bost and Triantaphyllou, 1982; Triantaphyllou, 1987; Jarquin-Barberena et al., 1991; Castagnone-Sereno et al., 1994a), but the reproduction of naturally virulent populations on resistant cultivars remained superiour over that of the laboratory selected populations (Jarquin-Barberena et al., 1991; Castagnone-Sereno et al., 1994a). The relative slow adaptation towards increased virulence points towards the action of a complex genetic constitution (Triantaphyllou, 1987; Roberts and Thomason, 1989; Castagnone-Sereno et al., 1994b).

In the case of *Meloidogyne* resistance genes identified in the *Solanum* gene pool, only the identification of a virulent population of *M. chitwoodi* towards resistant *S. bulbocastanum* has been reported (Mojtahedi and Santo, 1994; Van der Beek et al., 1998). This limited information on the incidence of virulent populations is likely to be due to the fact that resistance genes have been identified only recently (Brown et al., 1996; Janssen et al., 1997b) and have not been introduced yet in cultivars and exposed to many *Meloidogyne* populations. More knowledge concerning the abilities of root-knot nematodes species to overcome resistance genes is desirable and will contribute to strategies for durable resistance management before the introduction and widespread use of *Meloidogyne*-resistant potato cultivars.

Even on resistant wild *Solanum* genotypes, sometimes one or two egg masses of *M. chitwoodi* are observed which could be either due to females that escaped the resistance reaction or due to virulent females. The aim of this research was to investigate the possibilities of selection for virulence in *M. chitwoodi* towards resistance in wild potato species from such egg masses and the specificity of this virulence towards other resistant *Solanum* sources. In this report we describe the selection of completely virulent nematode populations using resistant *S. fendleri*, carrying the resistance gene R_{mc2}. The results will be discussed in relation to an effective and durable use of resistance genes in the gene pool of potato.

Materials and methods

Plant material

The resistance in *S. fendleri* genotype 93-114-12 was used for selection experiments on virulence in *M. chitwoodi*. This genotype possesses resistance to various populations of *M. chitwoodi* as well as *M. fallax*, but not *M. hapla* (Janssen et al., 1996; 1997a). The resistance is based on a single dominant gene R_{mc2}, which is present in a homozygous form assuming disomic inheritance (Janssen et al., 1997b). Very occasionally a single egg mass had been observed on this genotype in previous experiments. Crossing of this genotype with susceptible *S. fendleri* 93-115-7 produced population M94-49, which did not segregate for resistance in earlier experiments.

Other genotypes used were *S. fendleri* M94-79-1 - originating from a different accession than 93-114-12 -, *S. hougasii* 93-71-6, *S. bulbocastanum* 93-60-2 and SB22, *S. stoloniferum* 93-STOL-3 and *S. tuberosum* ssp. *tuberosum* cv Nicola. Except for the latter, all genotypes have moderate to high levels of resistance to *M. chitwoodi* and *M. fallax*. More detailed information on origin and resistance levels is provided elsewhere (Janssen et al., 1996, 1997a).

Origin and maintainance of the nematode population

Soil containing parts of *M. chitwoodi* infected roots of black salsify (*Scorzonera hispanica* L.) was sampled from a field near Heide, The Netherlands, in 1993. Previous crops were maize (1992), carrot (1991) and sugarbeet (1990). Tomato plants (cv Nematex) were grown in the infested soil and after 10 weeks 40 females with egg masses were individually identified to the species level of *M. chitwoodi* by isozyme pattern of esterase and malate dehydrogenase (Esbenshade and Triantaphyllou, 1990). All egg masses of positively identified females were used as inoculum for the multiplication and maintainance of the population on tomato in a temperature-controlled greenhouse (22 ± 2 °C) and the population was further designated as CHE. Each three to four months, the procedure of isolating 30 to 40 females with egg masses, identification by isozyme pattern and inoculation of egg masses on young growing plants was repeated, using a rotation on potato (cv Nicola), wheat (cv Minaret) and tomato as host plant to avoid the risk of adaptation to a single multiplication crop. According to its reaction on alfalfa, carrot and SB22, the population is characterized to be race 1 of *M. chitwoodi*.

Table 1. Multiplication of single egg masses, isolated from resistant *S. fendleri* population M94-49, on tomato. Reported are number of egg masses (em) 10 weeks after inoculation and second-stage juveniles (juv) of *M. chitwoodi* after 4 weeks hatching of the egg masses

inoculum	code	# em	# juv	juv/em
em 1	CHE L-1-0	32	6.640	208
em 2	CHE L-2-0	0	–	–
em 3	CHE L-3-0	19	3.520	185
em 4	CHE L-4-0	35	10.160	290
em 5	CHE L-5-0	41	7.720	188
em 6	CHE L-6-0	21	6.400	305
em 7	CHE L-7-0	52	10.480	202
em 8	CHE L-8-0	25	12.800	512
em 9	CHE L-9-0	0	–	–
em 10	CHE L-10-0	19	4.800	253
em 11	CHE L-11-0	0	–	–
CHE (400 juv)*	CHE Co-1-0	52	11.645	224

* Inoculum of control was obtained from maintainance population of CHE.

Table 2. Number of egg masses on *S. tuberosum* cv Nicola and *S. fendleri* 93-114-12 after 8 weeks inoculation with 400 juveniles. The nematode populations (except the control CHE Co-1) originate from single egg masses isolated from resistant *S. fendleri*

inoculum	93-114-12		'Nicola'		
	rep.1	rep. 2	rep.1	rep. 2	rep. 3
CHE L-1-1	24	36	60	70	35
CHE L-3-1	0	0	80	115	–
CHE L-4-1	0	0	42	80	55
CHE L-5-1	0	0	40	80	140
CHE L-6-1	80	100	45	55	50
CHE L-7-1	115	90	110	92	85
CHE L-8-1	77	105	40	65	90
CHE L-10-1	90	70	135	90	90
CHE Co-1-1	0	0	40	35	–

Isolation and multiplication of nematode lines

Seedlings of *S. fendleri* population M94-49 were tested on resistance to CHE in square plastic tubes of 240 ml, filled with moist silver sand and NPK fertilizer. Five replicates of 'Nicola' were included in the experiment as a susceptible control. Plants were inoculated with 400 juveniles and roots were analyzed individually 8 weeks later for the presence of egg masses after staining with Phloxine B (Dickson and Strubble, 1965).

Females and egg masses present on the *S. fendleri* seedlings were carefully isolated and females were tested on isozyme pattern. Egg masses were individually inoculated on spatially isolated tomato plants for multiplication. Also, a tomato plant was inoculated with 400 juveniles of the original population CHE to produce a control population. Egg masses were isolated from tomato roots after 10 weeks and used for hatching of juveniles in water. Juveniles were regularly collected during four weeks and stored at 4 °C until use for the resistance test with *S. fendleri* 93-114-12.

Resistance test with S. fendleri *93-114-12*

Nematode lines were tested on three replicates of 93-114-12 and four of 'Nicola'. Plants were grown in 750 cc stone pots filled with silver sand and NPK fertilizer and inoculated with 400 juveniles. Eight weeks after inoculation egg masses were counted on two and three replicates of 93-114-12 and 'Nicola' respectively, on roots stained with Phloxine B. The remaining replicate was used for inoculum preparation for a subsequent resistance test with various *Solanum* genotypes.

Resistance test with various Solanum *genotypes*

Egg masses were isolated from roots of 'Nicola' and 93-114-12, if present, and inoculum was prepared as described before. A resistance test was performed with all nematode lines which had produced sufficient inoculum. At least 10 females of each nematode line have been verified for the presence of *M. chitwoodi* specific band pattern using Mdh and Est which indicates that resistance breaking has not been the result of contamination with other *Meloidogyne* species. Genotypes of *S. fendleri*, *S. bulbocastanum*, *S. hougasii*, *S. stoloniferum* and *S. tuberosum* were tested in four replicates in a completely randomized block following procedures described above. Data were analysed with ANOVA using Genstat 5.1.

Results

The resistance test of *S. fendleri* population M94-49 revealed no clearly susceptible genotypes among 185 seedlings tested. However, 12 seedlings were found with one egg mass and one seedling with two egg masses. The five replicates of 'Nicola' showed an average number of 66 egg masses per root system.

Table 3. Mean square root number of egg masses on various *Solanum* genotypes of avirulent and virulent nematode lines on resistant *S. fendleri* 93-114-12

Inoculum	*S. tuberosum*	*S. fendleri*		*S. hougasii*	*S. bulbocastanum*		*S. stoloniferum*
	'Nicola'	93-114-12	M94-79-1	93-71-6	93-60-2	SB22	93-STOL-3
CHE L-1-2$_{nic*}$	7.32	8.92	6.30	3.65	7.58	0.39	0.68
CHE L-1-2$_{114}$	7.69	7.08	3.99	3.33	5.08	0.39	0.25
CHE L-3-2$_{nic}$	8.98	0.25	0.00	0.00	0.00	0.00	0.00
CHE L-4-2$_{nic}$	8.46	0.00	0.00	0.00	0.00	0.00	0.00
CHE L-5-2$_{nic}$	9.57	0.00	0.00	0.00	0.00	0.00	0.00
CHE L-6-2$_{nic}$	8.59	7.79	5.91	3.45	5.71	1.11	0.00
CHE L-6-2$_{114}$	8.68	8.56	5.27	3.72	6.05	0.00	0.50
CHE L-7-2$_{nic}$	6.41	4.23	4.38	0.64	4.20	4.52	1.06
CHE L-7-2$_{114}$	10.21	8.16	5.52	1.70	7.03	6.97	0.97
CHE L-8-2$_{nic}$	8.84	8.58	6.96	3.99	5.84	0.39	0.25
CHE L-8-2$_{114}$	9.84	8.95	4.93	3.33	7.10	0.73	0.00
CHE L-10-2$_{nic}$	7.34	8.66	5.54	4.15	7.28	6.94	0.93
CHE L-10-2$_{114}$	7.76	9.50	5.39	1.96	6.36	5.78	0.60
CHE Co-1-2$_{nic}$	6.85	0.00	0.00	0.00	0.00	0.00	0.25
LSD ($P<0.05$) = 1.60							

* 'Nic' and '114' inoculum obtained from cv Nicola and *S. fendleri* 93-114-12 respectively.

Eleven of the 14 females on *S. fendleri* could be isolated and tested on Mdh pattern. All showed the *M. chitwoodi* specific band (data not shown).

Eight out of 11 egg masses reproduced well on tomato and gave rise to the nematode lines described in Table 1, which were used for the subsequent resistance test with 93-114-12 and 'Nicola' (Table 2). Five lines were able to totally overcome the resistance of *S. fendleri* 93-114-12 (hereafter called '114-virulent lines') and their numbers of egg masses were comparable to those on the susceptible 'Nicola'. The other three lines remained completely avirulent. On the replicates used for inoculum production a very similar virulence and avirulence pattern was observed for the nematode lines (data not shown).

Table 3 shows that in the following resistance test with the five *Solanum* genotypes no large significant differences were noticed between the lines derived from 'Nicola' and 93-114-12. The number of egg masses are presented as mean squares to obtain equality of variance for ANOVA purposes. The avirulent lines L-3, L-4 and L-5 showed the same reaction as the control population Co-1, i.e. all were successfully suppressed by the resistance of the different *Solanum* species. The 114-virulent lines all showed a similar virulent behaviour towards *S. fendleri* M94-79-1, although the mean number of egg masses was in general somewhat lower. All 114-virulent lines were also able to totally overcome the resistance of *S. bulbocastanum* 93-60-2, but suprisingly only two of the five lines were virulent on *S. bulbocastanum* SB22 as well. In the case of *S. hougasii* 93-71-6, four of five 114-virulent lines were able to overcome the resistance but average infection was not as high as in the other compatible combinations. The resistance of *S. stoloniferum* 93-STOL-3 remained effective against the 114-virulent populations.

Discussion

In this report we describe the selection of virulence in *M. chitwoodi* towards various resistant *Solanum* species from a virtually avirulent nematode population. A complete susceptible response of *S. fendleri* 93-114-12 was observed after only one selection of reproducing nematodes on resistant plants, indicating that virulence was already present in the avirulent population in a very low percentage. The ease to select for virulence leading to complete susceptibility and the similar behaviour of all virulent lines regarding the resistance of 93-114-12 suggests a simple inheritance of the virulence, possibly monogenic recessive as observed in many other resistance-breaking pathogens (Keen, 1990; Simms, 1996). The rapid increase of virulence in *M. chitwoodi* on resistant *S. fendleri* is in contrast to the slow progressive increase of virulence

in *M. incognita* on resistant tomato (Netcher, 1977; Bost and Triantaphyllou, 1982; Triantaphyllou, 1987; Jarquin-Barberena et al., 1991; Castagnone-Sereno et al., 1994a), which is thought to be more genetically complex (Triantaphyllou, 1987; Roberts and Thomason, 1989; Castagnone-Sereno et al., 1994b).

Not all nematode lines which had been multiplied from one reproducing female on resistant *S. fendleri* were able to circumvent the resistance. If - in a simple case of one recessive gene responsible for virulence - few heterozygous nematodes were able to reproduce exceptionally on resistant plants, a significant proportion of homozygous recessive genotypes would be expected after one generation through sexual mating or meiotic parthenogenesis. Taking into account the almost complete absence of reproducing egg masses in both subsequent resistance tests, these lines are unlikely to possess any virulence genes as present in the virulent lines. The original reproducing females may be considered as genetically avirulent and apparantly have accidently escaped the resistant response. Also tomato cultivars with resistance to *Meloidogyne* spp. based on the *Mi* gene are not immune and incidental egg mass formation can be observed without a clear indication of genetical virulence in these cases (Roberts and Thomason, 1989).

The virulence which has been selected towards resistance from 93-114-12 was found not only to be effective against the resistance from another *S. fendleri* accession, but also against resistances in various other wild *Solanum* species, which are only distinctly related to each other (Hawkes, 1990). This would suggest a broad action of the virulence. However, clear and significant differences were expressed between the 114-virulent lines on the different resistant sources. These results can better be explained by the action of more virulence genes which behaviour is covered by the presence of one or two major genes, responsible for the compatible reaction on *S. fendleri*. Remarkably, there were also pronounced differences between the two genotypes of *S. bulbocastanum* indicating not only the presence of different virulence factors in the nematode but also different resistance factors within this *Solanum* species. The existence of several virulence factors has also been noticed within *M. hapla*, but virulence patterns were already shown without (apparent) selection (Janssen et al., 1997a; Van der Beek et al., 1998). With *M. incognita*, it was demonstrated that naturally virulent populations on *Mi*-resistance were able to break through other resistance genes present in the *L. peruvianum* complex (Roberts et al., 1990; Veremis and Roberts, 1996), but the more artificially selected *Mi* resistance-breaking populations could not circumvent these other resistance genes (Roberts et al., 1990).

The resistance present in *S. stoloniferum* remained effective against all virulent lines. While the resistance in *S. fendleri*, *S. hougasii* and *S. bulbocastanum* is proven or suggested to be based on single dominant resistance genes (Brown et al., 1996; Janssen et al., 1997b; Rouppe van der Voort et al., 1998), the resistance in *S. stoloniferum* is based on several resistance genes (Janssen et al., 1997b). Also the resistance mechanism in the latter seems to be different. In preliminary histological studies it was observed that the growth of nematodes is strongly but not completely inhibited in the roots of *S. stoloniferum*. In contrast, in roots of resistant *S. fendleri*, *S. hougasii* and *S. bulbocastanum* nematodes were not able to develop at all, presumably due to a hypersensitive reaction (G.J.W. Janssen, unpublished results).

The observed occurrence of virulence and the ease of selection has implications for the practical use of future *Meloidogyne* resistant potato cultivars. Although the incidence of virulence in natural field populations is likely to be small at present due to lack of a selective advantage of these populations, an intensive use of the same source of resistance will quickly lead to predominance of virulent populations and the ineffectiveness of resistance on those fields. Examples with the exploitation of other resistance genes against nematodes have shown that selection for virulence is indeed to be expected, when the virulence is already present in the natural genetic pool of the nematode species (Cook and Evans, 1987; Bakker et al., 1993) as seems to be the case for the virulence on *S. fendleri* 93-114-12. On the other hand, the dispersion of virulence will be a relative slow process compared to foliar fungi due to the soil relatedness. Despite frequent observations of virulent populations towards resistance genes against *Meloidogyne* spp., resistant cultivars have proven their value and still remain effective in large areas, like for example the root-knot nematode resistant *Prunus* rootstock cv Nemaguard and the numerous tomato cultivars with *Mi* resistance (Cook and Evans, 1987; Roberts, 1995; Esmenjaud et al., 1996). The threat of selecting virulence should therefore not prohibit the introduction and use of *Meloidogyne* resistance into commercial potato cultivars, but stimulate the cautious introduction of multiple resistant sources within the framework of an integrated pest management.

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