

DIFFERENCES IN VIRULENCE BETWEEN SOME POTATO CYST NEMATODE POPULATIONS TO POTATO GENOTYPES WITH MONOGENIC RESISTANCE TO *GLOBODERA PALLIDA* FROM *SOLANUM MULTIDISSECTUM* OR *S. TUBEROSUM* SSP. *ANDIGENA* CPC 1673

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Potato cyst nematodes (PCN), *Globodera pallida* and *G. rostochiensis*, are major pests of the potato crop. Major gene resistance to *G. pallida* was first demonstrated by Dunnett (1962) in *Solanum multidissectum* PH 1366. This gene (H_2) confers nearly complete resistance to *G. pallida* populations with pathotype designation Pa1 (Kort *et al.*, 1977). A second major gene resistance to *G. pallida* was reported recently (Arntzen *et al.*, 1993). No or very few cysts of *G. pallida* populations D234 and D236 are formed on genotypes with this resistance gene. The gene presumably originates from *S. tuberosum* ssp. *andigena* CPC 1673, which is also the source of the widely used H_1 resistance gene to *G. rostochiensis* (Ellenby, 1952).

S. multidissectum and *S. tuberosum* ssp. *andigena* are related species. They both belong to the Series Tuberosa from the subsection Potatoes of *Solanum* and originate from the Andes-region in South-America (Hawkes *et al.*, 1979). Chavez (1984) suggested that *S. tuberosum* ssp. *andigena* may have developed amongst others from *S. multidissectum*. Hosaka (1986) found considerable resemblance between the chloroplast DNA of *S. tuberosum* ssp. *andigena* and *S. multidissectum*. Consequently, similar genes for resistance may occur in both species.

In this study, the possible similarity of the H_2 gene from *S. multidissectum* and the recently discovered gene for *G. pallida* resistance from *S. tuberosum* ssp. *andigena* CPC 1673 was investigated by comparison of the virulence level of some PCN populations to potato genotypes with monogenic resistance from these sources.

Materials and methods

Three potato genotypes were used. The genotypes were P55/7, with monogenic resistance to pathotype Pa1 of *G. pallida* from *S. multidissectum* PH 1366 (Dunnett, 1962), the cultivar Multa, with monogenic resistance to some populations of *G. pallida*, presumably derived from *S. tuberosum* ssp. *andigena* CPC 1673 (Arntzen *et al.*, 1993), and the susceptible standard cultivar Maritta. In addition to *S. multidissectum*, P55/7 also has *S. tuberosum* ssp. *andigena* CPC 1685 as an ancestor (A. Thompson, pers. comm.), but it did not inherit the

H_1 gene (Kort *et al.*, 1977), found in CPC 1685 (Huijsman, 1955). Stem cuttings of the genotypes were made. After three weeks, rooted cuttings were transplanted in 150 ml pots, filled with 75 g peat soil.

Pots were inoculated at planting with a pre-set volume of cysts (Vinke *et al.*, 1992) of one of six PCN populations (Table 1). The populations Duddingston and Glarryford, which have been designated pathotype Pa1, originate from Great Britain; Pa1 has not been recorded from the Netherlands. The populations D234 and D236 from the Netherlands are known to be avirulent to the CPC 1673 *G. pallida* resistance gene. Two standard populations were also included, *G. rostochiensis* Mierenbos A (avirulent to the H_1 gene) and *G. pallida* Rookmaker, which have been used in various resistance tests before (Arntzen & van Eeuwijk, 1992). The latter population is highly virulent to some genotypes with resistance derived from *S. vernei* (Arntzen & van Eeuwijk, 1992). The weight of cyst samples of each of the populations was determined, as this has been shown to represent the number of eggs in the samples (Arntzen *et al.*, 1994). Pots were inoculated with an average of 34 cysts and inoculated pots were placed in a greenhouse in a randomized block design, at an average day temperature of 20 °C, and were watered twice daily. There were fifteen replicates.

After 6 weeks, when the new cysts were well developed, rootballs were removed from the pots and the number of newly formed cysts visible on the surface of the rootball was counted. This number is sufficiently representative of the total number of newly formed cysts (Forrest & Holliday, 1979). A score (1-5) was given for the size of the root system. The observed numbers of newly formed cysts were transformed to logarithms with base 10 of the numbers of cysts plus 1, to obtain homogeneity of variance. Correlations between data, which did not show a normal distribution, were calculated using Spearman's rank correlation coefficient r_s .

Results and discussion

The mean weight of samples of cysts for inoculum varied little between PCN populations (Table 1). The

Table 1. Number of cysts on three potato genotypes, P55/7, with monogenic resistance to *Globodera pallida* from *Solanum multidissectum*, Multa, with monogenic resistance to *G. pallida* from *S. tuberosum* ssp. *andigena* CPC 1673, and the susceptible standard cultivar Maritta, with six PCN populations. For each PCN population separately, means carrying different letters, are significantly different from each other (LSR; $P < 0.05$). Species, place of origin and weight of inoculum samples of the populations are also given.

| Populations | Species | Place of origin | Weight of inoculum (mg) | Potato genotypes | | |
|-------------|-------------------|-----------------|-------------------------|------------------|-------|---------|
| | | | | P55/7 | Multa | Maritta |
| Duddingston | <i>G. pallida</i> | Duddingston, GB | 1.38 | 1 a | 53 b | 168 c |
| Glarryford | <i>G. pallida</i> | Glarryford, GB | 1.42 | 2 a | 64 b | 275 c |
| D234 | <i>G. pallida</i> | Smilde, NL | 1.42 | 31 b | 0 a | 208 c |
| D236 | <i>G. pallida</i> | Anlo, NL | 1.35 | 50 b | 0 a | 191 c |
| Rookmaker | <i>G. pallida</i> | Valthe, NL | 1.53 | 7 a | 73 b | 156 c |
| Mierenbos A | <i>G. rost.</i> | Wageningen, NL | 1.49 | 94 a | 166 b | 246 c |

root score was not significantly correlated ($r_s = 0.05$) with the log transformed number of newly formed cysts visible. On the pathotype Pa1 differential P55/7, very few cysts were formed with the *G. pallida* populations Duddingston and Glarryford (Table 1), which is in accordance with their classification as pathotype Pa1. No cysts were found with *G. pallida* populations D234 and D236 on Multa, confirming earlier observations (Arntzen *et al.*, 1993). A considerable number of cysts was found with D234 and D236 on P55/7, and with Duddingston and Glarryford on Multa (Table 1). These results show clearly that the major genes for resistance to *G. pallida* from *S. multidissectum* and from *S. tuberosum* ssp. *andigena* CPC 1673 are different.

Significantly fewer cysts were produced on Multa than on Maritta with all populations, confirming in part earlier results (Arntzen *et al.*, 1993). This low level resistance of Multa presumably has a separate genetic basis from the monogenic resistance to D234 and D236 (Arntzen *et al.*, 1993). Significantly fewer cysts were also produced on P55/7 by all populations, as compared to Maritta. Kort (1974), Stone *et al.* (1979) and Ross (1986) reported already considerably less cysts on P55/7, compared to susceptible standard cultivars, with various PCN populations. The very low number of cysts on P55/7 by the Rookmaker population is of potential interest, as it has a relatively high virulence level to several genotypes with resistance derived from *S. vernei* (Arntzen & van Eeuwijk, 1992). It would be interesting if the resistance to Rookmaker is conferred by the H_2 gene. However, previous studies have suggested that the P55/7 ancestors *S. multidissectum* and *S. tuberosum* ssp. *andigena* CPC 1685, apart from the H_2 and the H_1 gene respectively, contained additional PCN resistance (Dunnett, 1962; Trudgill & Parrott, 1969).

The results, presented here, clearly show that the major genes conferring *G. pallida* resistance from *S. multidissectum* and *S. tuberosum* ssp. *andigena* CPC 1673 are different. A new virulence group within *G. pallida* might

be distinguished (Arntzen & van Eeuwijk, 1992). The results demonstrate clear differences in virulence for the resistance gene between *G. pallida* populations.

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