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Virulence in the beet cyst nematode *(Heterodera schachtii) versus* some alien genes for resistance in beet

Wouter LANGE *, Jochen MÜLLER ** and Theo S. M. DE BOCK *

* *DLO-Centre for Plant Breeding and Reproduction Research (CPRO-DLO), P.O. Box 16, 6700 AA Wageningen, The Netherlands, and*

** *Biologische Bundesanstalt für Land- und Forstwirtschaft, Institut für Nematologie und Wirbellierkunde, Toppheideweg 88, 4400 Münster, Germany.*

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Summary – Three populations of the beet cyst nematode (BCN), *Heterodera schachtii,* were used in resistance tests with various plant materials of the genus *Beta.* Population 129-SB had been selected for virulence to gene(s) for resistance obtained from chromosome pro-1 of *B. procumbens* and is considered to be a new pathotype of this nematode species. In addition, an unselected sib population (129-FR) and the wild-type population BCN-WA were used. The wild species *B. procumbens* and *B. patellaris* showed nearly complete resistance to all three nematode populations. Population 129-SB was able to produce cysts on the two breeding stocks of sugar beet *(B. vulgaris* subsp. *vulgaris),* with resistance from chromosome pro-1. However, the mean number of cysts per plant was significantly less than on the susceptible control cv. Regina. Nematode population 129-SB also was able to break the resistance from chromosome pat-1 of *B. patellaris,* when present as a monotelosomic addition. The monosomic addition carrying chromosome pro-7 of *B. procumbens* and the partially resistant accession BMH of the wild sea beet *(B. vulgaris* subsp. *maritima)* showed resistance in tests with all three nematode populations, but population BCN-WA produced significantly more cysts on the susceptible plants and on BMH than the other two populations. These differences indicate variation in the nematode populations for fitness and perhaps also for virulence. It is proposed to modify the nomenclatural system for the naming of the postulated genes for resistance to *H. schachtii* on the chromosomes pro-1, pat-1 and pro-7, and to name them *Hs1 pro-1*, $Hs^{pat\text{-}1}$, and *Hs2 pro-7,* respectively.

Résumé – ***Virulence du nématode à kystes de la betterave* (Heterodera schachtii) *vis-à-vis de certains gènes étrangers de résistance chez la betterave*** – Trois populations du nématodes à kystes de la betterave (BCN), *Heterodera schachtii,* sont utilisées dans des tests de résistance chez un matériel végétal varié du genre *Beta.* La population 129-SB a été sélectionnée pour sa virulence envers le ou les gènes de la résistance obtenue à partir du chromosome pro-1 de *B.procumbens;* elle est considérée comme représentant un nouveau pathotype de cette espèce de nématode. Une population « sib » (129-FR) et la population type sauvage BCN-WA ont été également utilisées. Les espèces sauvages *B. procumbens* et *B. patellaris* font montre d'une résistance presque complète aux trois populations. La population 129-SB est capable de former des kystes sur les deux lignées de betterave à sucre *(B. vulgaris* subsp. *vulgaris)* résistantes par leur chromosome pro-1. Cependant, le nombre de kystes y est significativement plus faible que pour le cv. Regina, sensible, utilisé comme témoin. La population 129-SB est également capable de briser la résistance provenant du chromosome pat-1 de *B. patellaris* si ce dernier est présent en tant qu'addition monotélosomique. L'addition monosomique portant le chromosome pro-7 de *B. procumbens* et l'accession, partiellement résistante, BMH de la betterave maritime sauvage *(B. vulgaris* subsp. *maritima)* sont résistantes aux trois populations du nématode, encore que la population BCN-WA produise un nombre significativement plus élevé de kystes sur les plantes sensibles et sur l'accession BMH que les deux autres populations. Ces différences révèlent chez ces populations une variabilité de leur adaptation et peut-être également de leur virulence. Il est proposé de modifier la nomenclature utilisée pour les gènes supposés de résistance à *H. schachtii* sur les chromosomes pro-1, pat-1 et pro-7, et de les nommer $Hs1^{pro\text{-}1}$, $Hs1^{pat\text{-}1}$ *et* $Hs2^{pro\text{-}7}$, *respectivement.*

Key-words : Nematodes, *Heterodera schachtii, Beta vulgaris, Beta procumbens, Beta patellaris,* resistance, virulence.

Cultivated beet *(Beta vulgaris* subsp. *vulgaris)* is a host plant for two species of cyst nematodes. *Heterodera schachtii* Schmidt, the white beet cyst nematode, is a wide-spread pest in most areas of sugar beet cultivation and can cause considerable losses in yield. The yellow beet cyst nematode *(Heterodera trifolii* Goffart f. sp. *beta)* occurs mainly on sandy soils, e.g. in the southern part of The Netherlands. Except for leguminous crops it has a similar host range as *H. schachtii* (Maas & Heijbroek, 1982; Steele *et al.,* 1983). High levels of resistance to the beet cyst nematodes have never been found in cultivated beet. In agricultural practice the nematode is controlled by the application of nematicides, the use of resistant cruciferous green-manuring crops and a wider crop rotation.

The growing of resistant cultivars is considered to be a valuable alternative or addition to these measures. Therefore, the possibilities to transfer genes for resistance from wild *Beta* species to cultivated beet have been studied in many research programmes, and in most of

them the three species of section *Procumbentes* (= *Patellares*) have been used as source of the genes for resistance. The research programmes have encountered crossing barriers, an extremely low frequency of the introgression of the alien genes into the genome of sugar beet, a low level of tolerance in the plant material towards the induced hypersensitivity reaction, and a reduced sexual transmission of the introgressed genes. Furthermore it appeared that in the three species of section *Procumbentes* there are altogether six chromosomes carrying genes for resistance (for references see reviews by Lange *et al.*, 1990; Van Geyt *et al.*, 1990; Nakamura *et al.*, 1991; Roberts, 1992), which could be confirmed with a molecular probe (Jung *et al.*, 1992). Molecular genetics technologies are also being used to isolate the alien genes for beet cyst nematode resistance and to transfer such genes to cultivated beet (Jung *et al.*, 1990, 1992; Salentijn *et al.*, 1992).

A second source for resistance has been found in the wild sea beet, *B. vulgaris* subsp. *maritima*. This resistance is thought to be polygenic and recessive inherited (Heijbroek, 1977). Mesken and Lekkerkerker (1988) selected in this resistant material, studied the offspring of crosses between such material and cultivated beet, and concluded that resistance is not complete. Despite it, quite high levels of resistance could be reached.

Resistance breeding in beet has concentrated on *H. schachtii*. Until recently, this nematode was considered to be a constant factor in the host-parasite interaction, because no differences in virulence or pathogenicity had been observed between nematode populations. Müller (1992) collected 146 populations of *H. schachtii* and studied their virulence. He found that the three wild *Beta* species of section *Procumbentes* generally remained free of cysts. On selected resistant monosomic additions, carrying chromosome pro-1 of *B. procumbens*, often a low but variable number of cysts was formed. Müller (1992) interpreted this observation as an indication for the occurrence of virulence genes in a low frequency in the nematode populations. The cysts were collected and tested again on a diploid resistant breeding stock, in which the gene(s) for resistance of chromosome pro-1 had been introgressed. This procedure was repeated six times and the level of virulence of several nematode populations was increased considerably. In the two most advanced nematode populations the average multiplication rate on the resistant beet stocks reached a level of 52 % of that of the susceptible control, which level could not be increased further through three additional nematode generations of selection (Müller, unpubl.). The selected nematode populations were considered to be a new pathotype of *H. schachtii*.

The present study was undertaken to determine if the selected virulence of *H. schachtii* would be specific for the beet material on which the selection had been carried out, or if the increased virulence extended to other sources of nematode resistance.

Materials and methods

Plant materials and nematode populations

Plants were used of three wild *Beta* species with resistance to the beet cyst nematode (BCN), *H. schachtii*, viz. two of the species of section *Procumbentes* (= *Patellares*), *B. procumbens* Chr.Sm. ($2n = 18$) and *B. patellaris* Moq. ($2n = 36$), and the partially resistant wild sea beet, *B. vulgaris* L. subsp. *maritima* (L.) Arcang. (nomenclature according to Letschert, 1993). Seed of the *Procumbentes* species was obtained from the CPRO-DLO collection, and accession BMH of subsp. *maritima* was selected by Mesken and Lekkerkerker (1988) from PI 198758, which originated from a collection site near Le Pouliguen, France. Furthermore four resistant stocks originating from interspecific breeding programmes were used. Two were diploid ($2n = 18$) genotypes, viz. B883 (Heijbroek *et al.*, 1988), which is the same as the diploid Pro 1 used by Müller (1992), and AN1-65-2 (Lange & De Bock, unpubl.). Both stocks had been selected from resistant monosomic chromosome additions ($2n = 19$; Savitsky, 1975; Speckmann *et al.*, 1985) of beet, carrying chromosome pro-1 of *B. procumbens*. The remaining two were AN101 (Lange *et al.*, 1988; Van Geyt *et al.*, 1988) a resistant monosomic addition carrying chromosome pro-7 of *B. procumbens*, and AN5 (Speckmann *et al.*, 1985), a resistant monotelosomic addition carrying the long arm telosome of chromosome pat-1 of *B. patellaris*. Because of incomplete transmission of the extra chromosome or telosome carrying the gene(s) for resistance, AN101 and AN5 will segregate for plants with or without the alien chromosomal material, and the transmission rates were reported to be 15.5 % for AN101 (n = 3759) and 18.9 % (n = 15196) for AN5 (Lange *et al.*, 1990). The sugar beet variety, *B. vulgaris* L. subsp. *vulgaris* cv. Regina served as susceptible control.

Three nematode populations were used. Two of them (129-SB and 129-FR) originated from population 129, which was collected and described by Müller (1992). Population 129-SB had been multiplied six times on resistant diploid stocks, originating from B883. Population 129-FR went through the same number of multiplication cycles, but was grown only on susceptible fodder rape, *Brassica napus* L. cv. Velox. The third nematode population was called BCN-WA and consisted of the standard population of *H. schachtii* as used in the CPRO-DLO at Wageningen.

Experimental design

Tests for resistance were done as described by Toxopeus and Lubberts (1979). Young seedlings were transplanted into 36 ml PVC tubes filled with quartz sand, which was moistened with a nutrient solution. After ten days each plant was inoculated with a suspension containing about 300 pre-hatched juveniles of *H. schachtii*, by means of a veterinary inoculation gun. The experi-

ments were in an air-conditioned greenhouse, at about 22 °C. After four weeks, the root systems were carefully washed free of sand and the white females were counted under a stereoscopic microscope at × 10 magnification. Plants with less than ten females (further on called cysts) were considered to be resistant.

Two experiments were carried out, each with a randomised complete block design with three replicates. The maximum number of plants for each replicate was 24 or 32 in Experiment 1 or 2, respectively (Tables 1, 2), and for the two segregating accessions the total number of plants was increased by including extra plants (Table 3). Both experiments included all three nematode populations, the susceptible control cv. Regina, as well as the partially resistant *B. vulgaris* subsp. *maritima* accession BMH. The remaining resistant plant material was divided over the two experiments (Tables 1-3). The data were square root transformed before statistical analysis, but in the tables the original numbers are presented. The segregating families AN101 and AN5 could not be analysed statistically. The data of these families are presented in frequency distributions with the following classes of number of cysts per plant : 0-1, 2-4, 5-9, ... 197-225 (see Figure 1; note : the squares of the Figures 1, 2, ... 15 were used as the upper limits of the classes).

In a sample of the resistant plants of AN101 and AN5 the presence of the extra chromosome or telosome was checked by chromosome counting. Root tips were pretreated in aqueous 8-hydroxyquinoline (0.002 Mol, 5h, at 6 °C), fixed in acetic ethanol (1:3), hydrolised in 1 N hydrochloric acid (7 min, at 60 °C), squashed in 45 % aqueous acetic acid, and stained with 1 % aqueous crystal violet.

Results

In total fifteen cysts were formed on 423 plants of *B. procumbens* and *B. patellaris* (Table 1), with no more than two cysts per plant, confirming the high resistance of these two *Beta* species to all three populations of *H. schachtii* used. Because of the extremely low numbers of cysts the results on the two species of section *Procumbentes* were omitted from statistical analysis.

The selection of *B. vulgaris* subsp. *maritima* accession BMH and the sugar beet cv. Regina were tested twice (Tables 1, 2) and the two experiments produced consistent differences in the number of cysts formed. Population BCN-WA produced most cysts, and accession

Table 1. Mean numbers of cysts of *Heterodera schachtii* and numbers of plants of four *Beta* accessions tested in Experiment 1 with three nematode populations (129-SB, 129-FR and BCN-WA).

Accession	*Number of cysts/plant*			*Number of plants*		
	129-SB	129-FR	BCN-WA	129-SB	129-FR	BCN-WA
B. procumbens	0.09	0.03	0.00	70	72	71
B. patellaris	0.04	0.01	0.04	70	69	71
B. vulgaris subsp. *maritima* acc. BMH	4.3 *d* *	2.6 *d*	44.1 *c*	69	70	66
cv. Regina	79.1 *b*	70.8 *b*	114.0 *a*	56	68	66

* different letters indicate significant difference ($P < 0.05$).

Table 2. Mean numbers of cysts of *Heterodera schachtii* and numbers of plants of four *Beta* accessions tested in Experiment 2 with three nematode populations (129-SB, 129-FR and BCN-WA).

Accession	*Number of cysts/plant*			*Number of plants*		
	129-SB	129-FR	BCN-WA	129-SB	129-FR	BCN-WA
B883	27.8 *c* *	3.1 *e*	0.4 *f*	95	96	95
AN1-65-2	29.8 *c*	6.9 *d*	3.2 *de*	93	96	94
B. vulgaris subsp. *maritima* acc. BMH	5.9 *de*	2.7 *e*	23.1 *c*	94	95	96
cv. Regina	57.7 *b*	65.0 *b*	84.9 *a*	94	92	91

* different letters indicate significant difference ($P < 0.05$).

BMH was a much poorer host than the susceptible cv. Regina. The differences between the two populations 129 and population BCN-WA were stastistically significant ($P < 0.05$) and BCN-WA produced proportionally more cysts on accession BMH. Finally it was observed that on the plants of accession BMH, and especially with population BCN-WA, many of the cysts were markedly smaller than usual.

In Table 2 the results with the two diploid accessions, B883 and AN1-65-2, both carrying the gene(s) for nematode resistance originating from chromosome pro-1 of *B. procumbens*, have been summarized. Both accessions showed various degrees of cyst formation after inoculation with the three nematode populations. Despite the stastistically significant differences between two of the accessions for two of the three populations, the overall pattern of nematode reproduction shows much similarity. Population BCN-WA on average produced fewest cysts, however, the number of cysts appeared to be greater than that produced on the original wild species (Table 1). The greatest numbers of cysts on B883 and AN1-65-2 (about 50% of that on cv. Regina) were produced by population 129-SB, indicating that the resistance had been partly overcome. There were large and significant differences between the numbers of newly formed cysts by the nematode populations 129-SB and 129-FR, on both B883 and AN1-65-2, where the differences between 129-FR and BCN-WA were small and only significant for B883.

The results for AN101, carrying chromosome pro-7 of *B. procumbens*, and AN5, carrying the long arm telosome of chromosome pat-1 of *B. patellaris*, are presented in Table 3 and Figure 1, together with the data on cv. Regina. Clear segregations in resistant (less than ten cysts/plant) and susceptible individuals were found in AN101 when challenged with each of the three nematode populations. The frequencies of resistant plants were 16.1 % for 129-SB, 12.0 % for 129-FR and 14.3 % for BCN-WA, with an overall mean of 14.1 %, and the number of chromosomes in the resistant plants that were checked was as expected, $2n = 19$. The average numbers of cysts on the resistant plants were low, but not as low as on *B. procumbens* (Table 1), the origin of the extra chromosome carrying the gene(s) for resistance. The susceptible plants showed a mean number of cysts which was similar to that of cv. Regina or slightly lower, and the distribution of the susceptible plants of AN101 over the classes of number of cysts per plant was rather similar to that of cv. Regina (Fig. 1).

The results of the tests with AN5 and the nematode populations 129-SB were different. No segregation could be observed, although plants with the alien telosome, carrying the gene(s) for resistance, must have been present (all plants of AN5 came from the same plant population). The frequency distributions of AN5 and cv. Regina, when infested with 129-SB (Fig. 1) and the mean values for cysts per plant (45.3 and 57.7, respectively; Table 3) demonstrate a certain difference between these accessions. The results for AN5 and the nematode populations 129-FR and BCN-WA showed more or less the same pattern as the three tests with AN101 (Table 3; Fig. 1). The two groups of plants segregated for resistance. The proportions of resistant plants were 17.2 % and 18.2 %, respectively, with a total mean of 17.7 %, and the number of chromosomes of the resistant plants that were checked was $2n = 18$+telo. The average numbers of cysts on the resistant plants again were higher than those on the donor species *B. patellaris* (Table 1), and the averages of the number of cysts on the susceptible plants were slightly below those on cv. Regina.

Table 3. Mean numbers of cysts of *Heterodera schachtii* and numbers of plants of resistant and susceptible fractions in offspring of two mono(telo)somic additions in *Beta vulgaris* (AN101 and AN5) and in cv. Regina, tested with three nematode populations (129-SB, 129-FR and BCN-WA).

Plant material	*Number of cysts/plant*			*Number of plants*		
	129-SB	129-FR	BCN-WA	129-SB	129-FR	BCN-WA
EXPERIMENT 1						
AN101 resistant *	1.4	0.7	0.5	31	24	27
susceptible	68.3	70.3	102.7	162	176	162
cv. Regina **	79.1	70.8	114.0	56	68	66
EXPERIMENT 2						
AN5 resistant *		3.6	1.8	0	25	27
susceptible	45.3	59.8	75.0	143	120	121
cv. Regina **	57.7	65.0	84.9	94	92	91

* plants with less than ten cysts; ** data also presented in Tables 1 or 2.

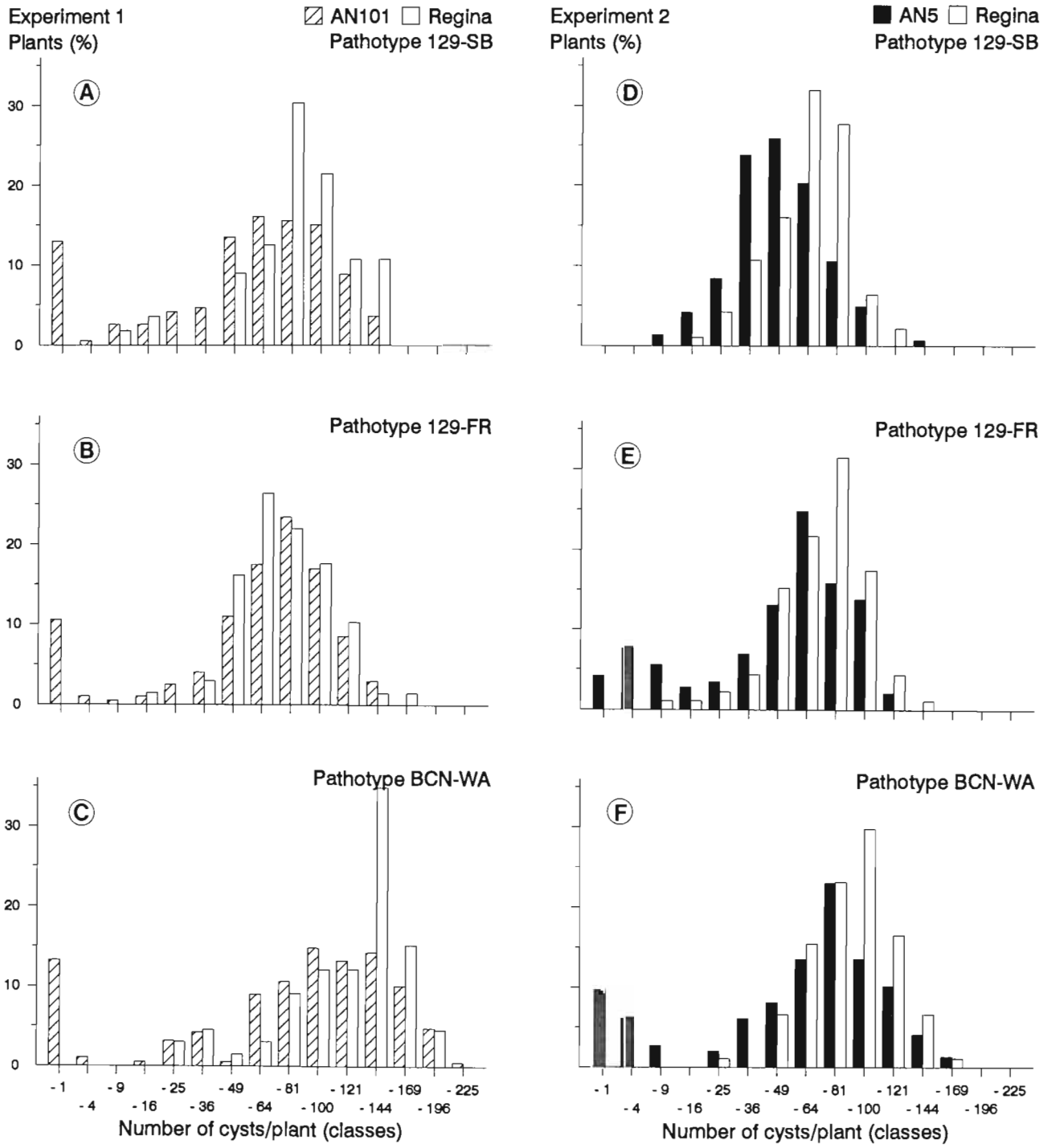

Fig. 1. Frequency diagrammes of plants of monosomic addition AN101 (Experiment 1 : A, B, C), monotelosomic addition AN5 (Experiment 2 : D, E, F) and cv. Regina (A-F), tested with three populations of *Heterodera schachtii :* 129-SB (A, D), 129-FR (B, E) and BCN-WA (C, F). The plants are grouped in classes according to the number of cysts per plant (only the upper limits of the classes are given).

Discussion

Müller (1992) selected a pathotype of *H. schachtii* virulent against the resistance obtained through a monosomic addition of chromosome pro-1 of *B. procumbens* in diploid *B. vulgaris,* which resistance has been used extensively in breeding programmes in Europe and the USA (Savitsky, 1975; Heijbroek *et al.,* 1988; Lange *et al.,* 1990; Nakamura *et al.,* 1991). In the present study, the host-parasite relation of this new pathotype (nematode population 129-SB) to various sources/genes for resistance was compared to that of two other unselected nematode populations. Population 129-SB produced many cysts on the resistant diploid genotype B883 that had been used to select for virulence, and the mean number of cysts per plant was about half of that of the susceptible control variety. Population 129-SB reached the same level of reproduction on AN1-65-2, a resistant diploid accession that was developed in CPRO-DLO at Wageningen, as on B883. The original plant material of the Wageningen programme was different from that of B883, but the alien chromosomes in the monosomic additions of the two programmes were identified to be of the same type (Van Geyt *et al.,* 1988). The equal performances of population 129-SB on both diploid stocks suggest that the same gene(s) for resistance have been transferred.

The low levels of cyst production of the nematode populations 129-FR (the non-selected sib population of 129-SB) and BCN-WA on B883 and AN1-65-2 clearly show the resistance of these breeding stocks to the unselected nematode populations. Population 129-FR produced somewhat more cysts than BCN-WA, which is interpreted as being the result of the presence, in a low frequency, of the genes for virulence. AN1-65-2 was slightly less resistant to populations 129-FR and BCN-WA than B883. This difference was unexpected and might be explained to be the result of differences in the genetical background of the plant material.

Earlier studies (Van Geyt *et al.,* 1988; Lange *et al.,* 1988) have shown that the extra chromosome of *B. procumbens* in the monosomic addition AN101 is other than chromosome pro-1 (the source of the gene(s) for resistance transferred into B883 and AN1-65-2). The results of the present study confirm that the alien chromosome in AN101 (chromosome pro-7) also carries one or more genes for resistance to *H. schachtii,* effective against all three nematode populations. This leads to the conclusion that chromosome pro-7 carries at least one gene for resistance that is different from that derived from chromosome pro-1 and transferred into B883 and AN-1-65-2.

The extra telosome in AN5 originated from *B. patellaris*. On the basis of isozyme patterns and the resistance to *H. schachtii* this chromosome is considered to be at least partly homologous (or homoeologous) with chromosome pro-1 (Lange *et al.,* 1990). After inoculation with populations 129-FR and BCN-WA the resistance showed up nicely, resulting in segregation for this trait in the plant material. Contrastingly, the inoculation with population 129-SB did not result in segregating plant material, indicating that the gene(s) for resistance can be overcome by the virulence of the new pathotype. This result supported the suggestion that chromosome pro-1 and the chromosome from *B. patellaris* (pat-1) carry the same gene and might be homologous. It also was observed that after inoculation with population 129-SB the mean number of cysts per plant in AN5 was less than in the susceptible control variety. This might be the result of a lower level of reproduction on the about 18 % of the plants of AN5 which carried the alien telosome.

Both Jung *et al.* (1992) and Salentijn *et al.* (1992) used resistant chromosome fragment additions to select molecular markers that are linked to the gene(s) for resistance. The markers were used in hybridisation experiments with DNA from resistant plants of various accessions. Small repetitive DNA probes were selected, which did not hybridise to *B. vulgaris*, but hybridised to all stocks with resistance from section *Procumbentes*. Thus the existence of at least six chromosomes carrying gene(s) for resistance was confirmed. Salentijn *et al.* (1992) also selected two low copy probes and produced evidence for the presence of homology between the chromosomes pro-1 and pat-1 and the lack of such homology between the chromosomes pro-1 and pro-7. They also indicated the existence of structural differences or incomplete homology between chromosomes pro-1 and pat-1, which corroborated the earlier observations of Speckmann *et al.* (1985) and De Jong *et al.* (1986) that the postulated genes on the chromosomes pro-1 and pat-1 are located on the short arm and the long arm of the chromosomes, respectively.

Savitsky (1975) proposed to use the symbol *N* for the postulated dominant gene which had been transferred from chromosome pro-1 of *B. procumbens* into sugar beet. In the light of the results of the studies discussed above, it is proposed to modify the nomenclatural system for the naming of genes for resistance to the beet cyst nematode, *H. schachtii*. The gene symbol *N* stands for nematode resistance, which is considered to be too general for the present situation. As there are more nematode species known to be parasitic on beet (e.g. *H. trifolii* f. sp. *beta* and also species of *Meloidogyne*), the nomenclatural system should be able to cope with different genes for resistance. Therefore it is proposed to use the symbol *Hs* for genes for resistance to *H. schachtii*. The different reactions to pathotypes will be shown by different numbers, whereas the origin of the alien genes is given in an additional superscript. The gene in the material of Savitsky (1975, 1978; see also Yu, 1984 *a*), in B883 and in AN1-65-2, is called $Hs1^{pro-1}$. The one from *B. patellaris,* in accession AN5, which has a similar reaction to the new pathotype of *H. schachtii,* thus is called $Hs1^{pat-1}$. And the third gene, which occurs in

AN101, originates from *B. procumbens*, and gives rise to a different reaction to the new pathotype, is called $Hs2^{pro-7}$.

The levels of cyst formation in the two wild species of section *Procumbentes* were the lowest of all resistant plant materials tested, which is in concordance with earlier studies (e.g. Hijner, 1951; Yu, 1984 *b;* Müller, 1992). This means that the resistances in B883, AN1-65-2, AN101 and AN5 in fact are incomplete as compared to the original species. The high level of resistance in *B. procumbens* might be explained by the combined action of at least the postulated genes $Hs1^{pro-1}$ and $Hs2^{pro-7}$. The high level of resistance in *B. patellaris* justifies the expectation that other genes for resistance than $Hs1^{pat-1}$ might occur in this species. However, the testing of more than a hundred unidentified monosomic additions, each carrying an extra chromosome of *B. patellaris*, has not resulted in finding such genes (Lange *et al.*, 1990). From the close relationship between *B. procumbens* and *B. webbiana* Moq. (Wagner *et al.*, 1989; Reamon-Ramos & Wricke, 1992) it can be expected that the genes for nematode resistance on chromosomes web-1 and web-7 are homologous to *Hs1* and *Hs2*, respectively. All these (postulated) genes behave as dominant heritable factors, when transferred into the cultivated species. They might be assigned to the group of major genes, which are supposed to have a positively hostile effect, resulting from recognition processes (Trudgill, 1992).

Further variation seems to be possible. Speckmann *et al.* (1985) reported the finding of a monosomic addition from *B. procumbens* (accession AN14), in which the extra chromosome was identified as pro-7 (Van Geyt *et al.*, 1988), and which exhibited partial resistance to a wild type population of *H. schachtii*. In *B. webbiana* a third chromosome (web-8) was identified carrying gene(s) for incomplete resistance (Jung & Wricke, 1987; Reamon-Ramos & Wricke, 1992). However, it is unclear yet if these genes should be considered as major genes.

The results with the selected plant material of accession BMH of *B. vulgaris* subsp. *maritima* (Tables 1, 2) demonstrate the (partial) resistance of this stock. The reproduction rates of the nematode populations 129-SB and 129-FR were of the same levels as reported by Mesken and Lekkerkerker (1988; pers. comm.) and population BCN-WA gave rise to more cysts per plant. Many of the cysts on accession BMH were smaller than those on cv. Regina. The latter phenomenon had already been observed by Mesken and Lekkerkerker (pers. comm.), who consider it to be an important factor of the resistance mechanism. The same authors also observed that the resistance of BMH has a recessive and polygenic inheritance. This leads to the proposition to assign the resistance of BMH to the type which is supposed to involve reduced susceptibility (Trudgill, 1992).

In the susceptible and partial resistant host-parasite situations tested, population BCN-WA produced more cyst per plant than the two populations 129. The observed differences were new and unexpected, and might be attributed to differences in fitness of the nematodes, and for accession BMH perhaps also to differences in virulence between population BCN-WA and the other two nematode populations.

The new pathotype of *H. schachtii* is shown to have a virulence that can break only certain specific genes for resistance, but not all. The occurrence of such virulence is rare (Müller, 1992) and the breaking of resistance does not result in complete susceptibility, as observed by Müller (1992) and confirmed in the present study. This means that the alien genes for resistance are still very valuable for breeding sugar beet with resistance to the beet cyst nematode. In analogy with similar host-parasite interactions, such as between potato and the potato cyst nematodes, it would seem appropriate to combine at least two different sources of resistance in the new varieties, and to apply resistance management systems.

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