

# Inheritance pattern and selection criteria for resistance to soybean cyst nematode races 3 and 9

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**Abstract** – The objective of this work was to determine soybean resistance inheritance to *Heterodera glycines* Ichinohe (soybean cyst nematode – SCN) races 3 and 9, as well as to evaluate the efficiency of direct and indirect selection in a soybean population of 112 recombinant inbred lines (RIL) derived from the resistant cultivar Hartwig. The experiment was conducted in a completely randomized design, in Londrina, PR, Brazil. The estimated narrow-sense heritabilities for resistance to races 3 and 9 were 80.67 and 77.97%. The genetic correlation coefficient ( $r_g = 0.17$ ;  $p \leq 0.01$ ) shows that some genetic components of resistance to these two races are inherited together. The greatest genetic gain by indirect selection was obtained to race 9, selecting to race 3 due to simpler inheritance of resistance to race 9 and not because these two races share common resistance genes. The resistance of cultivar Hartwig to races 3 and 9 is determined by 4 and 2 genes, respectively. One of these genes confers resistance to both races, explaining a fraction of the significant genetic correlation found between resistance to these SCN races. The inheritance pattern described indicates that selection for resistance to SCN must be performed for each race individually.

**Index terms:** *Glycine max*, *Heterodera glycines*, resistance, SCN, recombinant inbred lines.

## Padrão de herança e critérios de seleção para resistência às raças 3 e 9 do nematóide-de-cisto-da-soja

**Resumo** – O objetivo deste trabalho foi determinar a herança da resistência da soja às raças 3 e 9 de *Heterodera glycines* Ichinohe (nematóide-de-cisto-da-soja – NCS), e avaliar a eficiência da seleção direta e indireta em uma população de 112 linhagens recombinantes endogâmicas (RIL) derivadas da cultivar resistente Hartwig. O experimento foi conduzido em delineamento inteiramente casualizado, em Londrina, PR, Brasil. A herdabilidade em sentido restrito para resistência às raças 3 e 9 foi de 80,67 e 77,97%. O coeficiente de correlação genética ( $r_g = 0,17$ ;  $p \leq 0,01$ ) demonstrou que alguns componentes genéticos para resistência às duas raças são herdados conjuntamente. O maior ganho pela seleção indireta foi obtido na raça 9, selecionando-se para a raça 3, devido à herança mais simples na raça 9, e não pelo compartilhamento de genes comuns para resistência às duas raças. A resistência da cultivar Hartwig nas raças 3 e 9 é determinada por 4 e 2 genes, respectivamente. Um desses genes confere resistência a ambas as raças, o que explica parte da correlação genética entre a resistência a estas raças de NCS. O padrão de herança descrito indica que a seleção para resistência ao NCS deve ser realizada em cada raça individualmente.

**Termos para indexação:** *Glycine max*, *Heterodera glycines*, resistência, NCS, linhagens endogâmicas recombinantes.

### Introduction

Soybean cyst nematode (*Heterodera glycines* Ichinohe – SCN) is a pathogen that affects soybean production worldwide with losses that reached 9 million tons in 1998 (Wrather et al., 2001). Sources of resistance to SCN were reported first by Ross (Ross & Brim, 1957). Three recessive genes were reported as

responsible for resistance to race 1 in cultivar Peking (Caldwell et al., 1960). An additional dominant gene, that confers resistance to race 3 (*Rhg4*) and 4 (currently race 14) was found by Matson & Williams (1965) and Thomas et al. (1975), respectively.

Later, Hancock et al. (1987) reported one more recessive gene associated with resistance to “race X”. The resistance to race 3 in PI 88788 is conditioned by

one recessive and two dominant genes, and in cultivar Peking and PI 90763 by one dominant and two recessive genes (Rao-Arelli et al., 1992). Silva et al. (2007) found that the resistance to race 3 in cultivar Msoy 8001 is conferred by two recessive and epistatic genes, and resistance to race 14 in line S5995 is conferred by two recessive genes with duplicated action.

The use of resistant varieties is the safest and most economical method for an efficient control of this soybean pathogen. However, the high genetic variability exhibited by SCN (Riggs, 1961) and the restricted genetic base of soybean resistance make easy for this pathogen to break the resistance of available varieties (Dong et al., 1997). Therefore, development of new cultivars resistant to several populations of SCN is a constant need. The development of these varieties in breeding programs depends on the existence of soybean lines carrying the genes for resistance to SCN in high frequencies.

Phenotypic selection of resistant soybean genotypes to population of several SCN races is expensive and difficult to achieve, but it is a required step in genetic improvement. Indirect phenotypic selection is a useful method for selecting soybean genotypes carrying resistance genes to several SCN races. That is, to practice selection for one race of SCN and to evaluate the magnitude of correlated response to another race of the same pathogen to develop soybean lines with broad SCN resistance.

The objective of this work was to determine soybean resistance inheritance to SCN races 3 and 9, and to evaluate the efficiency of direct and indirect selection for resistance to these races using 112 recombinant inbred lines (RIL) derived from a cross between resistant cultivar Hartwig and susceptible line Y23.

## Material and Methods

A population of RIL segregating for resistance to SCN populations (races 3 and 9) was obtained from a single cross between line Y23 (susceptible) and cultivar Hartwig (resistant). Hartwig derives from the cross between Forrest x PI 437654 genotypes and appears to have retained most of the SCN resistance of PI 437654 genotypes (Vierling et al., 1996). The F<sub>2</sub> plants obtained from five F<sub>1</sub> plants were selfed until the F<sub>6</sub> generation by the single seed descent (SSD) method (Brim, 1966). The F<sub>6:7</sub> RIL were tested for resistance or susceptibility to SCN populations of races 3 and 9.

Two independent experiments to screen the RIL to SCN races 3 and 9 were conducted in a greenhouse, at Embrapa Soja, Londrina, PR, Brazil, in 2001, in completely randomized design comprising six replication and 112 treatments (RIL), in which RIL or genotypes were considered to be random. One plant growing in a 500 mL pot corresponded to one plot or replicate. Plants from each RIL, cultivar Hartwig, line Y23 and the soybean differential lines (cultivar Peking, cultivar Pickett, PI 90763 and PI 88788) as well as cultivar Lee 68 as susceptible control were included in each experiment.

Bioassays were carried out as reported by Concibido et al. (1994, 1996) and Schuster et al. (2001) with some modifications. Inoculum of both races were maintained on roots of cultivar Embrapa 20 grown in the greenhouse at 25–30°C. Populations of these races were considered to be near homogeneous as the original isolates were maintained isolated and reproduced for more than forty generations. Brown cysts and white females were retained on a 60 mesh sieve and disrupted in a tissue grinder to recover the SCN eggs. Seeds of each RIL were germinated in sand bed at 25°C and three-day-old seedlings were transferred to a 500-mL pot containing a mixture of sand and soil (1:2). After three days, each seedling (six from each RIL) was inoculated with 1,000 eggs of SCN, races 3 or 9. Plants were grown in the greenhouse at 25–30°C with a photoperiod of 16 hours of light and 8 hours of dark.

Thirty days after inoculation, plant roots of each RIL were washed with tap water and cysts were collected on 60 mesh sieves. In order to confirm the identity of the populations of SCN populations used, the female index (FI) for differential lines was calculated according to Riggs et al. (1988) as follows:  $FI = 100(\text{mean number of cysts and females present on each differential lines} / \text{mean number of cysts and females present on susceptible cv. Lee 68})$ .

The FI for each RIL was estimated in a similar way:  $FI = 100(\text{mean number of cysts and females present on each RIL} / \text{mean number of cysts and females present on susceptible line Y23})$ .

The susceptible line Y23 was used in the FI estimation for RIL to evaluate differences between the parents of the RIL. In order to classify the 112 RIL in relation to their response to SCN races 3 and 9, the method proposed by Schmitt & Shannon (1992) was used.

Genotypes were classified as follows: resistant (R) (FI<10), moderately resistant (MR) (FI≥10 and FI<30), moderately susceptible (MS) (FI≥30 and FI<60), and susceptible (S) (FI≥60).

The phenotype data were not transformed, even though in some cases they did not present a normal distribution, because this distribution is not expected when the trait is controlled by relatively few genes with moderate effects (Doerge & Churchill 1994; Mutschler et al., 1996). Furthermore, normalizing data through transformation might misrepresent differences among individuals by pulling skewed tails toward the center of the distribution (Mutschler et al., 1996).

Phenotypic ( $\hat{\sigma}_p^2$ ) and genetic ( $\hat{\sigma}_g^2$ ) variances were estimated from the expectances of the mean squares:  $\hat{\sigma}_p^2 = GMS/r$ ;  $\hat{\sigma}_g^2 = (GMS - EMS)/r$  where GMS is the genetic mean square; EMS is the error mean square; and r is the number of replications. Narrow-sense heritability was estimated as  $h_n^2 = \hat{\sigma}_g^2 / \hat{\sigma}_p^2$ .

The genetic correlation between resistance to SCN, race 3 (R3) and race 9 (R9), was estimated as:

$$r_{g(R3,R9)} = \frac{C\hat{o}v_g(R3,R9)}{\sqrt{\hat{\sigma}_g^2(R3) \cdot \hat{\sigma}_g^2(R9)}}.$$

The correlation coefficient was tested using the t-value estimated as:

$$t = \frac{r_{g(R3,R9)}}{\sqrt{1 - r_{g(R3,R9)}^2}} \sqrt{n - 2},$$

with n - 2 degrees of freedom and

a level of significance of 1%, where n is the total number of RIL.

Selection gain to race j, by direct selection for race i, was estimated by the expression:  $SG_{j(i)} = (\bar{X}_{sj} - \bar{X}_{oj})h_j^2 = SD_{j(i)} \cdot h_j^2$ .  $SG_{j(i)}$  is the direct selection gain and  $SD_{j(i)}$  is the indirect selection differential, obtained for race j, as the difference between the mean value of the population without selection ( $\bar{X}_{oj}$ ) and the mean value of the RIL resistant to race j ( $\bar{X}_{sj}$ ), within the RIL resistant to race i.  $h_j^2$  is the heritability (narrow-sense) for race j. The percent SG was estimated as:  $SG\% = \frac{SG}{\bar{X}_0} \times 100$ . The expression  $SG = SD_{j(i)} \cdot h_j^2$  is the correlated response to race j, and SG will be, at most, equal to the direct gain in race i. The efficiency of indirect selection ( $SE_i$ ) estimated as  $SE_i = \frac{SG_{j(i)}}{SG_i} = \frac{SD_{j(i)}}{SD_i}$  was used as an indicator of the race i efficiency of genotypes resistant to both SCN races.

The number of genes controlling SCN resistance to races 3 and 9 was estimated using the chi-square test.

The Genes software (Cruz, 2001) was used for analyses of variance and estimation of the statistical parameters.

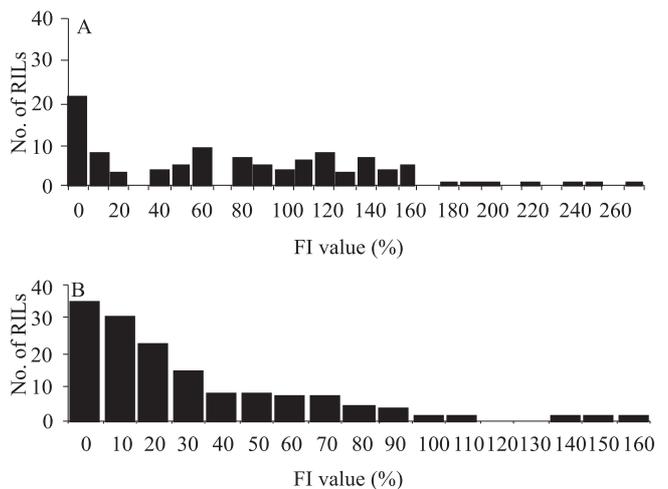
### Results and Discussion

Number of females present on cultivar Lee 68 and average FI for each differential line inoculated in the experiment is shown in Table 1. Differentials and susceptible control were used as monitor the SCN populations and their responses remained consistent while no race shifts occurred. The FI values distribution for each SCN race (Figure 1 A and B) shows that the resistance to SCN in soybean is a quantitative trait, as reported by other researchers (Mansur et al., 1993; Concibido et al., 1994; Webb et al., 1995; Yue et al., 2001).

**Table 1.** Soybean cyst nematode (SCN) races used in the study and their mean female index on host differentials<sup>(1)</sup>.

SCN	Host reaction of susceptible check (%)				
	Lee 68	Peking	Pikett 71	PI 90763	PI 88788
Race 3	100	3.94	0.88	1.13	4.4
	+	-	-	-	-
Race 9	100	12.47	11.51	5.07	5.07
	+	+	+	-	-

<sup>(1)</sup>Based on the SCN race classification scheme (Riggs et al., 1988), an individual is considered susceptible (+ reaction) if the number of females recovered on it is greater or equal to 10% of the number on the susceptible check; and resistant (- reaction) if the number of females recovered on it is less than 10% of the number on the susceptible check.



**Figure 1.** Distribution of FI values obtained in a RIL population of soybean inoculated with soybean cyst nematode, population race 3 (A) and 9 (B).

The normality of the data was rejected for the two races according to the Shapiro-Wilk test, and were  $W = 0.9428$  ( $p = 0.0000$ ) and  $W = 0.8269$  ( $p = 0.0000$ ) for race 3 and 9, respectively. Probably, only a few major SCN resistance genes were segregating in the RIL population analyzed. Few major genes were associated with resistance to SCN according to other groups (Caldwell et al., 1960; Matson & Williams, 1965; Hancock et al., 1987; Mansur et al., 1993). The mean FI of the parents, and the mean, minimal and maximal values for FI of the RIL population are shown in Table 2. Transgressive segregation was detected for the response to races 3 and 9. This type of segregation is interesting for plant breeding because it allows selection on descendant individuals with a number of trait loci superior to the parents (Ramalho et al., 2000).

Analysis of variance of the phenotypic data of the two SCN races, the corresponding means square as well

**Table 2.** Mean FI values of the parents and RIL. Mean, minimum and maximum FI values in the RIL population for response to populations of soybean cyst nematode races 3 and 9.

SCN	Hartwig mean	Y23 mean	RIL population		
			Mean	Minimum	Maximum
Race 3	4.60	39.33	88.58	0.00	278.5
Race 9	8.40	59.25	32.39	0.00	163.5

**Table 3.** ANOVA of the response of a soybean RIL population to races 3 and 9 of soybean cyst nematode<sup>(1)</sup>.

Estimation	Race 3	Race 9
GMS	25,491.88	5,485.93
EMS	4,927.62	1,208.48
F value	5.1667**	4.5395**
$\hat{\sigma}_g^2$	3,432.38	713.95
$\hat{\sigma}_p^2$	4,254.85	915.65
$\hat{\sigma}_e^2$	822.47	201.70
$h_n^2$	80.67	77.97
Mean	88.58	32.39

<sup>(1)</sup>GMS: genotype mean square; EMS: error mean square;  $\hat{\sigma}_g^2$  genetic variance;  $\hat{\sigma}_p^2$  phenotypic variance;  $\hat{\sigma}_e^2$  environmental variance;  $h_n^2$ (%): narrow-sense heritability. \*\*Significant at 1% of probability.

**Table 4.** Estimation of the genetic gain obtained by direct (SG, %) and indirect (GS<sub>i</sub>, %) selection and of the efficiency of indirect selection (SE<sub>i</sub>, %) for resistance to races 3 and 9 populations of soybean cyst nematode<sup>(1)</sup>.

Selection	Response	$\bar{X}_o$	$h_n^2$	No. of resistant RIL	$\bar{X}_s$	SD	SG	GS <sub>i</sub>	SE <sub>i</sub>
Race 9	Race 9	32.39	0.7797	51	8.76	-23.63	56.67	-	-
	Race 3	88.58	0.8067	14	86.28	-2.30	-	2.09	2.78
Race 3	Race 3	88.58	0.8067	23	5.93	-82.65	75.27	-	-
	Race 9	32.39	0.7797	14	22.98	-9.41	-	22.65	39.97

<sup>(1)</sup>( $\bar{X}_o$ ): FI mean value in the original population;  $h_n^2$ : narrow-sense heritability;  $\bar{X}_s$ : FI mean value in the selected RIL population; SD: selection differential; SG%: gain by direct selection; GS<sub>i</sub>%: gain by indirect selection; SE<sub>i</sub>%: efficiency of indirect selection.

as estimates of the different genetic parameters are shown in Table 3. Genetic variance for the resistance to SCN among the RIL was significant according to the F test. The significant genetic variance among the RIL for both SCN races indicates resistance genes are segregating in the population making phenotypic selection for resistance feasible to both population of races 3 and 9.

Estimated narrow-sense heritability was 80.67% for race 3 and 77.97% for race 9 (Table 3). Even though the heritability estimates depend on the genetic material and the method used (Falconer & Mackay, 1996), there is good agreement between the heritability indexes verified in the literature (Hancock et al., 1987; Mansur et al., 1995; Schuster et al., 2001; Yue et al., 2001) and the ones obtained in this study. High heritability indexes found indicate that a few major genes are segregating in the population and the practice of phenotypic selection for resistance would be efficient for both races of SCN. Genetic correlation for resistance to both races ( $r_g = 0.17$ ) was statistically significant ( $p \leq 0.01$ ), showing that some genetic factors associated with resistance to both SCN races are jointly inherited.

In order to efficiently use indirect selection in a soybean breeding program for SCN, the proportion of resistant lines with resistance to population of one or both races must be evaluated. According to the Schmitt & Shannon (1992) method, each RIL was classified as resistant (R and MR) if the female index (FI) to either or both population was  $< 30$ , and susceptible (MS and S) if FI was  $\geq 30$ . The greatest estimates of GS%, GS<sub>i</sub>% and SE<sub>i</sub>% were obtained when selection was practiced for race 3. Selection for race 9 showed a GS of 57%, but this did not mean a genetic gain for resistance to race 3 (Table 4).

Only one gene was necessary for a RIL to be classified as MR to race 9, whereas two were needed for a RIL to be classified as MR to race 3 (Table 5). The genes necessary for MR to race 3 are epistatic, that is, only plants with the two genes are MR to SCN. Because the

RIL is constituted by homozygous lines, it is impossible to know whether the genes are dominant or recessive (Schuster and Cruz, 2004).

Table 5 also shows an analysis of inheritance of resistance to one of the SCN race when RIL was resistant to the other. The tests were quite consistent and indicated that genes necessary to make soybean lines MR to race 3 and race 9 are different, which can be verified in a combined analysis of these two races. Out of 112 RIL analyzed, 14 were R+MR for race 3 and race 9 simultaneously. It is exactly the expected segregation of three independent genes ( $\chi^2 = 0.0$ ;  $p = 100\%$ ) that is, one gene for race 9 and two genes for race 3.

Results indicates that the genes needed to confer MR to races 3 and 9 of SCN in the cultivar Hartwig were not common. Only one gene was necessary for MR to race 9, and it is independent if the RIL were or not MR to race 3. Similarly, two genes were necessary for MR to race 3, and it is independent if the RIL were or not MR to race 9.

This explains why a larger proportion of RIL MR to race 9 was obtained when direct selection was performed for race 3 and not in the opposite case. Therefore, it was not possible to obtain genetic gain for resistance to race 3 by selecting for resistance to the race 9 (Table 4). Since only one gene was necessary for the RIL to be MR to the race 9, it is expected that half of the RIL population will be resistant to race 9, either when the RIL is selected as MR to the race 3 or when no selection is practiced at all. Similarly, it is expected that only one fourth of the RIL population will be resistant to race 3, either for the RIL selected as MR to race 9 or when selection is not conducted.

The results also explain why a larger coincidence is obtained among the susceptible plants than among the resistant ones (Table 5). These results are expected

when one gene is necessary to give the RIL MR to race 9 and two epistatic genes are necessary to give MR to race 3. To be MR for both races, a soybean line needs to have three genes. If a line lost the gene for race 9 and one gene for race 3, it will be susceptible for both races.

To make a RIL resistant ( $FI < 10$ ) to races 9 and 3, two and four genes are needed respectively. That is, one additional gene for race 9 and two additional genes for race 3 were needed in order to pass from MR to R (Table 5). This is a clear evidence of additivity of the gene action for SCN resistance. At the same time, these genes are also epistatic, because all of the resistance genes may be present to confer SCN resistance

Although only six RIL were resistant to race 3 ( $FI < 10$ ), it seems fairly probable that only one additional gene is needed to obtain resistance to race 9 if the RIL is resistant to race 3. That is, one of the two needed genes to make a RIL resistant to race 9 it also conferred resistance to race 3. Three additional genes are necessary for a RIL to be resistant to race 3 if it is resistant to race 9. That is, one of the four genes needed to make a RIL resistant to race 3 is involved in resistance to race 9.

A combined analysis of these two races can also be verified. Out of 112 RIL analyzed, 3 RIL were R ( $FI < 10$ ) for race 3 and race 9 simultaneously. It is adjusted to a ratio of 1:31, that in a RIL population means a segregation of five independent genes ( $\chi^2 = 0.0653$ ;  $p = 79.82\%$ ). Although resistance to race 3 is conferred by four genes, and resistance to race 9 is conferred by two genes, the combined resistance to these two races need five genes (and not six), because one gene is common for resistance to race 3 and race 9.

The ratio among the R ( $FI < 10$ ) and MR ( $FI \geq 10$  and  $FI < 30$ ) RIL also demonstrates that the difference

**Table 5.** Individual and combined inheritance resistance to soybean cyst nematode populations (races 3 and 9) in a soybean RIL population obtained from the cross between the resistant cultivar Hartwig and susceptible line Y23.

FI<30 (R+MR)	R+MR	S	Hypothesis <sup>(1)</sup>	$\chi^2$	P (%)
Race 9	51	61	1:1	0.8928	34.47
Race 3	23	89	1:3	1.1904	27.52
R+MR to race 3, segregation for race 9	14	9	1:1	1.0869	29.71
R+MR to race 9, segregation for race 3	14	37	1:3	0.1633	68.60
FI<10 (R)	R	S	Hypothesis <sup>(1)</sup>	$\chi^2$	P (%)
Race 9	20	92	1:3	3.0476	8.08
Race 3	6	106	1:15	0.1523	69.63
R to race 3, segregation for race 9	3	3	1:1	0.0000	100.00
R to race 9, segregation for race 3	3	17	1:7	0.1142	73.53
Only R and MR group	R	MR <sup>(2)</sup>	Hypothesis <sup>(1)</sup>	$\chi^2$	P (%)
Race 9	20	31	1:1	2.3725	12.35
Race 3	6	17	1:3	0.0144	90.42

<sup>(1)</sup>In RIL, 1:1, segregation of one gene; 1:3, segregation of two epistatic genes; 1:7, segregation of three epistatic genes; 1:15, segregation of four epistatic genes. <sup>(2)</sup> $FI \geq 10$  and  $FI < 30$ .

between R and MR is due to one gene for race 9 and two genes for race 3 (Table 5).

For resistance to race 9 ( $FI < 10$ ), the hypothesis of three genes also could be accepted ( $p = 8.65$ ). However, in this case, no other combined hypothesis could be accepted. It is also demonstrated that MR to race 9 is conferred by one gene, and the difference between MR and R is one additional gene, that is, the hypothesis of two genes for resistance to race 9 ( $FI < 10$ ) is the most probable, according to the combining analysis.

The presence of a common gene explains part of the significant genetic correlation for resistance of populations to both races of SCN. Figure 2 schematically postulates the distribution of genes involved in the resistance (R) or moderate resistance (MR) to races 3 and 9 of SCN. The postulation is that those genes must be considered in the following order: genes 1 and 2 are epistatic and confer moderate resistance to race 3. In the presence of these two genes, genes 3 and 4 confer complete resistance to race 3. Genes 3 and 4 are additive and epistatic to genes 1 and 2. That means that the four genes are needed to obtain complete resistance ( $FI < 10$ ) to race 3. Similarly, gene 5 confers moderate resistance (MR) to race 9 and in its presence with gene 4, acting in an additive and epistatic way, it confers complete resistance to race 9.

Additional experiments are needed to identify these genes. It can be hypothesized that gene 4 is *rhg1*, that is involved with resistance to many races of SCN. The hypothesis is that genes 1, 2 and 3 are race specific to race 3, and gene 5 is race specific to race 9. This

approach will be used to other races, in order to understand the relationship with resistance genes to different SCN races, and to identify molecular markers to perform marker assisted selection specific for SCN races.

## Conclusions

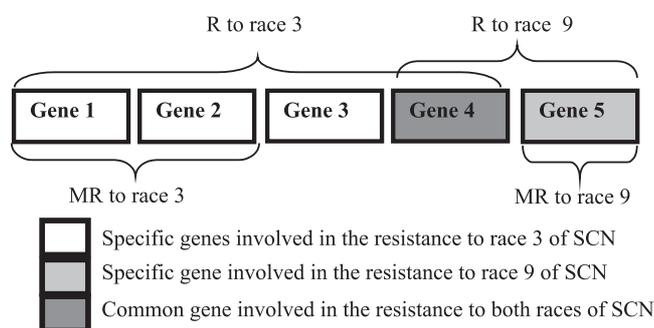
1. The resistance to SCN race 3 in the cultivar Hartwig is conferred by four genes: a pair of epistatic genes for MR ( $FI < 30$ ) RIL and an additional pair of additive and epistatic genes for R ( $FI < 10$ ) RIL.
2. The resistance to race 9 in the cultivar Hartwig is controlled by two genes, one of them is enough to make the RIL moderate resistant (MR) ( $FI < 30$ ) and the other one is needed for resistance (R) ( $FI < 10$ ); the second gene is one in the second pair of genes involved in the resistance to race 3.
3. The greatest genetic gain is obtained when selection is practiced on race 3 due to the simpler inheritance pattern of resistance to race 9 and not because both races have a common resistant gene.
4. To obtain genetic improvement of soybean for high resistance levels to races 3 and 9 of SCN, selection must be performed individually for each race.

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## References

- BRIM, C.A. A modified pedigree method of selection in soybeans. **Crop Science**, v.6, p.220, 1966.
- CALDWELL, B.E.; BRIM, C.A.; ROSS, J.P. Inheritance of resistance of soybeans to cyst nematode, *Heterodera glycines*. **Agronomy Journal**, v.52, p.635-636, 1960.
- CONCIBIDO, V.C.; DENNY, R.L.; BOUTIN, S.R.; HAUTEA, R.; ORF, J.H.; TOUNG, N.D. DNA marker analyses of loci underlying resistance to soybean cyst nematode (*Heterodera glycines* Ichinohe). **Crop Science**, v.34, p.240-246, 1994.
- CONCIBIDO, V.C.; DENNY, R.L.; LANGE, D.A.; ORF, J.H.; YOUNG, N.D. RFLP mapping and marker-assisted selection of soybean cyst nematode resistance in PI 209332. **Crop Science**, v.36, p.1643-1650, 1996.
- CRUZ, C.D. **Programa GENES**: aplicativo computacional em genética e estatística. Viçosa: UFV, 2001. 648p. Disponível em: <http://www.ufv.br/dbg/genes/genes.htm>. Acesso em: 24 set. 2007.



**Figure 2.** Schematic representation of the genes involved in the resistance to races 3 and 9 of soybean cyst nematode. Although the genes are represented in a block, they are not linked. R: resistant; MR: moderately resistant.

- DOERGE, R.W.; CHURCHILL, G.A. Issues in genetics mapping of quantitative trait loci. In: LOWER, R. (Ed.). **ASHS/CSSA Joint Plant Breeding Symposium on Analysis of Molecular Marker Data**. Corvallis: Oregon State University, 1994. p.27-32.
- DONG, K.; BAKER, K.R.; OPPERMAN, C.H. Genetics of soybean-*Heterodera glycines* interactions. **Journal Nematology**, v.29, p.509-522, 1997.
- FALCONER, D.S.; MACKAY, T.F.C. **Introduction to quantitative genetics**. 4<sup>th</sup> ed. Harlow: Longman Press, 1996. 480p.
- HANCOCK, J.A.; HANCOCK, F.G.; CAVINESS, C.E.; RIGGS, R.D. Genetics of resistance in soybean to Race X of soybean cyst nematode. **Crop Science**, v.27, p.704-707, 1987.
- MANSUR, L.M.; CARRIQUIRY, A.L.; RAO-ARELLI, A.P. Generation mean analysis of resistance to Race 3 of soybean cyst nematode. **Crop Science**, v.33, p.1249-1253, 1993.
- MATSON, A.L.; WILLIAMS, L.F. Evidence of a fourth gene for resistance to the soybean cyst nematode. **Crop Science**, v.5, p.477, 1965.
- MUTSCHLER, M.A.; DOERGE, R.W.; LIU, S.C.; KUAI, J.P.; LIEDL, B.E.; SHAPIRO, J.A. QTL analysis of pest resistance in the wild tomato *Lycopersicon pennellii*: QTLs controlling acylsugar level and composition. **Theoretical and Applied Genetics**, v.92, p.709-718, 1996.
- RAMALHO, M.A.P.; SANTOS, J.B.; PINTO, C.A.B.P. **Genética na agropecuária**. Lavras: Editora UFLA, 2000. 472p.
- RAO-ARELLI, A.P.; ANAND, S.C.; WRATHER, J.A. Soybean resistance to soybean cyst nematode race 3 is conditioned by additional dominant gene. **Crop Science**, v.32, p.862-864, 1992.
- RIGGS, J.P. Physiological strains of *Heterodera glycines*. **Plant Disease Reporter**, v.46, p.766-769, 1961.
- RIGGS, R.D.; SCHMIDT, D.P.; NOEL, G.R. Variability in race tests with *Heterodera glycines*. **Journal Nematology**, v.20, p.565-572, 1988.
- ROSS, J.; BRIM, C.A. Resistance of soybean to the soybean-cyst nematode as determined by a double-row method. **Plant Disease Reporter**, v.41, p.923-924, 1957.
- SCHMITT, D.P.; SHANNON, G. Differentiating soybean responses to *Heterodera glycines* races. **Crop Science**, v.32, p.275-277, 1992.
- SCHUSTER, I.; ABDELNOOR, R.V.; MARIN, S.R.R.; CARVALHO, V.P.; KIIHL, R.A.S.; SILVA, J.F.V.; SEDIYAMA, C.S.; BARROS, E.G.; MOREIRA, M.A. Identification of a new major QTL associated with resistance to soybean cyst nematode (*Heterodera glycines*). **Theoretical and Applied Genetics**, v.102, p.91-96, 2001.
- SCHUSTER, I.; CRUZ, C.D. **Estatística genômica aplicada a populações derivadas de cruzamentos controlados**. Viçosa: UFV, 2004. 568p.
- SILVA, M.F.; SCHUSTER, I.; SILVA, J.F.V.; FERREIRA, A.; BARROS, E.G.; MOREIRA, M.A. Validation of microsatellite markers for assisted selection of soybean resistance to cyst nematode races 3 and 14. **Pesquisa Agropecuária Brasileira**, v.42, p.1143-1150, 2007.
- THOMAS, J.D.; CAVINESS, C.E.; RIGGS, R.D.; HARTWIG, E.E. Inheritance of reaction to race 4 of soybean cyst nematode. **Crop Science**, v.15, p.208-210, 1975.
- VIERLING, R.A.; FAGHIHI, J.; FERRIS, V.R.; FERRIS, J.M. Association of RFLP markers with loci conferring broad-based resistance to the soybean cyst nematode (*Heterodera glycines*). **Theoretical and Applied Genetics**, v.92, p.83-86, 1996.
- WEBB, D.M.; BALTAZAR, B.M.; RAO-ARELLI, A.P.; SCHUPP, J.; CLAYTON, K.; KEIM, P.; BEAVIS, W.D. Genetic mapping of soybean cyst nematode race-3 resistance loci in the soybean PI 437654. **Theoretical and Applied Genetics**, v.91, p.574-581, 1995.
- WRATHER, J.A.; ANDERSON, T.R.; ARSYAD, D.M.; TAN, Y.; PLOPER, J.; PORTA-PUGLIA, L.D.; RAM, H.H.; YORINORI, J.T. Soybean disease loss estimates for the top 10 soybean-producing countries in 1998. **Canadian Journal of Plant Pathology**, v.23, p.115-121, 2001.
- YUE, P.; ARELLI, P.R.; SLEPER, D.A. Molecular characterization of resistance to *Heterodera glycines* in soybean PI 438489B. **Theoretical and Applied Genetics**, v.102, p.921-928, 2001.

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