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Genetics of soybean–*Heterodera glycines* system

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Genetics of resistance to soybean cyst nematode (SCN), *Heterodera glycines* Ichinohe is very complex. Crosses involving PI 437654, which is resistant to all races of cyst nematodes with other sources of resistance (Peking, PI 88788, and PI 90763) indicated that resistance to race 3 was controlled by four genes, two of which were dominant resistance genes and the other two were recessive resistance genes. For race 5, a four gene model with two recessive and two dominant resistance genes in epistasis has been proposed. For race 14, the results suggested a three gene model with one dominant and two recessive alleles. Several other plant introductions have been isolated which have different genes conditioning resistance. Most of the currently grown soybean varieties derived resistance from Peking and/or PI 88788. Resistance to SCN in these soybean varieties has broken down because of the emergence of several new races and populations of SCN. The use of PI 437654 or Hartwig and other plant introductions with different genes for resistance will broaden genetic diversity and stabilize yield.

Keywords: *Glycine max*; *Heterodera glycines*; inheritance; resistance; soybean; soybean cyst nematode.

Introduction

Soybean (*Glycine max* (L.) Merr.) is known as a 'Cinderella crop' because of its ascendence from a little known unimportant hay crop of the 1930s in the United States to a major crop of national and international importance. In 1985, soybean was rated as the leading agricultural export crop of the United States (Hapgood 1987). It provides approximately 60% of the world's vegetable protein and 30% of its vegetable oil (Fehr 1987). Initially, very few pests and diseases appeared to be important constraints for profitable soybean production in the United States. As the area under soybean increased, many pests and diseases emerged as important yield reducers. The soybean cyst nematode (SCN), *Heterodera glycines* Ichinohe, is one of such internationally important pests of soybean. First reported as *Heterodera schachtii* in 1915 in Japan (Hori 1915; Winstead *et al.* 1955), it was discovered in the United States in 1954. Since then it has been found in all major soybean-producing states of the United States and has become the most severe pest of soybeans in the mid-west, northern, southern and southeastern regions. In 1979, the yield losses caused by *H. glycines* were estimated at 1.52 million tons, worth $352 million, in the southern states (Brewer 1981). Recent crop loss estimates have shown that SCN is also the most yield-suppressing pest of soybean in the north-central region, which produces approximately 80% of the total soybeans in the United States (Doupnik 1993). The management of yield losses caused by nematodes is based on the use of resistant cultivars, crop rotations, cultural practices and nematicides. Cultivation of SCN-resistant cultivars is one of the most common methods of controlling the nematode and it is generally used as a sole means of nematode control. It is estimated that cultivation of the nematode-resistant cultivar 'Forrest' prevented crop losses worth $405 million between 1975 and 1980 (Bradley and Duffy 1982). More than 130 commercial cultivars with resistance to the nematode are presently available in the United States (Anand 1992a). Besides the United States, the nematode causes significant damage to soybean in Brazil, Canada, China, Colombia, Korea and Indonesia; however, the quantum of research work done and, consequently, the availability of information on the soybean–*H. glycines* system is much greater in the US than in any other country.

This paper elucidates the genetic diversity in *H. glycines* populations, sources of resistance in soybean and techniques commonly used to study the inheritance of resistance as these relate to genetics of the soybean–*H. glycines* system.

Genetic diversity

Variation in the pathogenic nature of *H. glycines* populations was first reported within 5 years of the commercial use of resistance (Ross 1962) and the extremely variable nature of the nematode genome was soon evident (Miller 1970). A group of nematologists and breeders together proposed the term 'race' to differentiate nematode populations based on their abilities to reproduce on a set of selected soybean host differentials. The race differentials were Peking, Pickett, PI (Plant Introduction) line 88788 and PI 90763. Cultivar 'Lee' was recommended as the standard susceptible for race identification tests. A scheme for the identification of races was suggested and four races were identified (Golden *et al.* 1970). Riggs *et al.* (1981)

showed that the identification of physiological variants in the nematode population depended on the number of host differentials used and separated 38 nematode populations in 25 physiological groups based on their reaction on 13 differential hosts. However, no additional host differentials were included in the scheme of Golden *et al.* (1970). Using this scheme, the presence of a new race 5 in Japan (Inagaki 1979) and the United States (Young 1982) and races 6 and 7 in China were reported (Chen *et al.* 1988). Riggs and Schmitt (1988) expanded the original race identification scheme of Golden *et al.* (1970) to include all possible combinations of races with the same four host differentials and proposed 16 races of the nematode although several of these races have not been isolated. The genetic basis for the recognition of races is rather weak and races can at best be perceived as field populations with different frequencies of the genes that control parasitism. These gene frequencies are subject to change under the influence of selection due to growing resistant soybean. Anand (1992a) demonstrated that the race structure of SCN field populations can change and is greatly influenced by the soybean cultivars grown in that field. Despite several limitations, the scheme proposed by Riggs and Schmitt (1988) is the scheme most commonly used to characterize the soybean cyst nematode populations.

Procedures to ascertain resistance

Resistance to *H. glycines* is generally evaluated in a greenhouse. No standardized methodology and uniform guidelines have been developed to determine resistance in soybean genotypes to the nematode populations; however, a system of classification of resistance to races for advisory purposes was outlined by Schmitt and Shannon (1992). The race identification technique proposed by Golden *et al.* (1970) to identify races of the nematode is most commonly used to identify sources of resistance and study the inheritance of resistance. Seeds of the test genotypes are germinated in vermiculite and, after 3–4 days, uniform seedlings are transplanted into sterilized sand or soil in 7.6 cm diameter clay pots. Nematode eggs (1000–5000 per pot) collected from cysts of a specific population are added to the sand or soil 2–4 days after transplanting. The number of white female (cysts) on the roots and in sand or soil are extracted and counted. The cyst counts are converted to a cyst index by dividing the number of cysts on a test plant by the number of cysts on a standard susceptible cultivar 'Lee' × 100. Test plants with a cyst index of <10 are considered resistant.

Sources of resistance

Ross and Brim (1957) evaluated over 4000 soybean germplasm lines for resistance to *H. glycines* by means of a double-row planting method and found 'Ilsoy', 'Peking', PI 84751 and PI 90763 to be resistant to a field population (probably race 1) in North Carolina. Peking has been extensively used as a source of resistance in cultivar development. Epps and Hartwig (1972) found high levels of resistance in PI 88788, PI 89772, PI 87631-1, Cloud, Columbia, Peking, PI 84751 and PI 90763. Anand (1982) reported PI 416762 as an additional source of resistance to race 4. Among these, PI 88788 has been used very extensively as a source of resistance for race 4 and it is in the background of almost all the commercial soybean varieties released in the United States.

Anand and Gallo (1984) screened the entire soybean collection of approximately 10000 lines for resistance to purified homogeneous races 3, 4 and 5. Each test line was sown in nematode-infested soil in a greenhouse and cysts on the roots were counted. Forty-five soybean lines were found to be either resistant or moderately resistant to race 3, 13 were resistant or moderately resistant to race 4 and ten were resistant to race 5 (Anand *et al.* 1988). Additional sources of resistance to races 3, 5 and 14 have been identified (Young 1990). PI 437654 is the only line in the United States collection which is highly resistant or nearly immune to all known races of the nematode in the United States (Anand 1991).

Genetics of the soybean–cyst nematode system

Most studies on the genetics of the soybean–cyst nematode system deal primarily with the inheritance or resistance in soybean to the SCN populations and there has been hardly any investigation of inheritance of parasitism in SCN populations to soybean genotypes. If it is presumed that soybean and SCN co-evolved and genes for resistance and parasitism are complementary, the genetics of parasitism could be extrapolated from the knowledge about the inheritance of resistance (Triantaphyllou 1987). The inheritance of resistance to SCN in soybean was first studied by Caldwell *et al.* (1960) and resistance in soybean cultivar Peking to a field population (most probably race 1) of the nematode was found to be conditioned by three independent recessive genes, rhg_1, rhg_2 and rhg_3, PI 90763 and PI 84751 had the same genes for resistance as in Peking. Later, an additional dominant resistance gene (Rhg_4) was reported in Peking and found to be linked with a recessive allele (*i*) that controls seed coat colour (Matson and Williams 1965). Sugiyama and Katsum (1966) also reported a recessive resistance gene and its linkage with seed coat colour in Peking for resistance to SCN populations in Japan. Hartwig and Epps (1970) reported a recessive resistance gene in PI 90763 for SCN populations found in Virginia. These SCN populations were later categorized as race 2. Thomas *et al.* (1975) stated that the resistance in PI 88788 to race 4 was recessive to PI 90763 and moderate resistance in PI 90763 was recessive to Peking, a race

4-susceptible parent. Moderate resistance in PI 90763 was conditioned by one dominant and two recessive genes in crosses involving the susceptible cultivar 'Hill'. Hancock *et al.* (1987) reported a new race of SCN race 'X' (close to race 2) and found that a single recessive gene controlled resistance to this race in PI 90763 in crosses with PI 88788 and PI 209332.

An extensive research project, supported by special grants from the US Department of Agriculture, was initiated at the Delta Center, University of Missouri at Portageville, Missouri to understand the inheritance of resistance in various resistant germplasm lines and to determine the genetic relationships for resistance among them. Homogeneous populations of each race were developed for study and the greenhouse technique for inoculation was improved (Rao-Arelli and Anand 1987). For resistance to race 3, Peking, PI 90763, PI 438389B, PI 404166 and PI 404198A had genes in common. However, large genetic differences were observed in other resistant lines. F_2 populations from the cross PI 88788 × PI 438496B segregated into a 15:1 resistant:susceptible ratio, suggesting a two gene difference with each parent having one major gene for resistance at a different locus. Crosses between Peking × PI 88788, and PI 88788 × PI 90763 segregated very close to 13:3 resistant:susceptible in F_2 generations, indicating dominant recessive epistasis (Rao-Arelli and Anand 1988).

Additional genes for resistance to SCN race 3 were identified in Peking; one dominant and two recessive genes conditioned resistance in a cross Peking (resistant) × Essex (susceptible) (Rao-Arelli *et al.* 1992). The dominant gene in Peking was designated Rhg_4 (Matson and Williams 1965). Resistance in PI 88788 was governed by two dominant and one recessive genes and one of the genes is homologous to Rhg_4 and the second was proposed to be designated to Rhg_5 (Rao-Arelli *et al.* 1992). Resistance in the near-immune soybean line PI 437654 was controlled by one dominant and two recessive genes and both PI 437654 and Peking had genes in common for race 3 resistance (Myers and Anand 1991). The cross PI 437654(R) × PI 88788(R) segregated 13:3 (R):(S) indicating the presence of an additional gene for race 3 resistance.

Genetics of resistance to SCN race 5 have been investigated in Peking, PI 90763, PI 438489B, PI 404166, PI 89772 and PI 437654. Two recessive genes condition resistance in Peking and one gene governs resistance in PI 438489B (Anand and Rao-Arelli 1987, 1989). Resistance in PI 90763 is conditioned by one dominant and two recessive genes while in PI 424595 it is controlled by three recessive genes involving Essex as a susceptible parent (Anand 1994). The genotypes PI 90763 and PI 424595 share the two recessive genes but differ in two additional genes (Anand 1994). Resistance in PI 437654(R) × Essex(S) is controlled by four genes with two recessive and two dominant genes in duplicate dominant epistasis (Myers and Anand 1991). Crosses between resistant parents, Peking, with PI 90763, PI 404166 and PI 84751 have shown that

Table 1. Genes for resistance to *H. glycines* in soybean

Resistant parent	Susceptible parent	Gene symbols of resistant parent[a]	Gene action	Reference
Race 1				
Peking	Lee	rhg_1,rhg_2,rhg_3,Rhg_4	One dominant and three recessive	Caldwell *et al.* (1960) Matson and Williams (1965)
Race 3				
PI 437654	Essex	Ra_1Ra_1,ra_2ra_2,ra_3,ra_3	One dominant and two recessive	Myers and Anand (1991)
PI 90763	Essex	Rrr	One dominant and two recessive	Rao-Arelli *et al.* (1992)
Peking	Essex	Rrr	One dominant and two recessive	Rao-Arelli *et al.* (1992)
PI 88788	Essex	RRr	Two dominant and one recessive	Rao-Arelli *et al.* (1992)
Race 5				
Peking	PI 88788	–	Two recessive	Anand and Rao-Arelli (1989)
PI 90763	Forrest	–	Two recessive	Anand and Rao-Arelli (1989)
PI 438489B	PI 88788	–	One recessive	Anand and Rao-Arelli (1989)
PI 437654	Essex	rb_1rb_1,rb_2rb_2,Rb_3Rb_3,Rb_4Rb_4	Two recessive and two dominant	Myers and Anand (1991)
PI 90763	Essex	r_ar_a,r_br_b,R_cR_c	Two recessive and one dominant	Anand (1994)
PI 424595	Essex	r_ar_a,r_br_b,r_dr_d	Three recessive	Anand (1994)
Race 14				
PI 88788	Peking		Three recessive	Thomas *et al.* (1975)
PI 437654	Essex	Rc_1Rc_1,rc_2rc_2,rc_3rc_3	One dominant and two recessive	Myers and Anand (1991)
PI 437654	PI 90763		One dominant	Myers and Anand (1991)
PI 437654	Peking		One recessive	Myers and Anand (1991)

[a]The gene symbols for different studies do not correspond with each other.

genes in Peking compared to other parents were located at different loci. Lack of segregation in crosses PI 90763 × PI 404166, Peking × PI 438489B and PI 90763 × 438489B indicates that resistance in these parents is controlled by alleles at the same loci. There is at least one resistance gene that is different in Peking, PI 90763 and PI 404166 (Anand and Rao-Arelli 1987).

Germplasm resistant to race 4 (Anand *et al.* 1988) is now considered resistant to race 14 based on the new race classification proposed by Riggs and Schmitt (1988). Because of the complex nature of this race, it has not been studied extensively (Thomas *et al.* 1975; Myers *et al.* 1989). A cross of PI 437654(R) × PI 90763(S) revealed the presence of a dominant gene for resistance whereas, PI 437654(R) × PI 88788(R) showed a recessive gene. The cross PI 437654(R) × Essex(S) demonstrated the presence of one dominant and two recessive genes for resistance to race 14 (Myers and Anand 1991). There are some indications of the existence of more than two alleles for resistance at the same loci. Thomas *et al.* (1970) postulated that three alleles at one locus would explain the resistance in PI 88788 to SCN race 4 being recessive to the moderate resistance of PI 90763. Segregation in the cross PI 90763 × Peking for reaction to race 5 is indicative of the presence of multiple alleles for one of the existing loci in each parent with non-allelic interaction (Anand and Rao-Arelli 1989).

Resistance to SCN in soybeans is a recessive trait; susceptibility has incomplete dominance over resistance in PI 90763. Peking and PI 437654 for SCN race 3 (Rao-Arelli *et al.* 1989) and over-dominance of susceptibility over resistance in PI 88788 (Myers *et al.* 1989). More genes probably condition resistance to SCN than have been described (Luedders 1989). Development and use of standardized methodologies for identification and classification of resistance is crucial. The use of homogeneous nematode populations (not field populations) for identification of genes for resistance is one of the most important requirements. The development and use of newer methodologies to produce inbred populations and the production of hybrids between them should prove useful for identification of gene effects in diverse backgrounds. A technique developed by Dropkin and Halbrendt (1986) to conduct these studies deserves the attention of soybean breeders and nematologists.

Enormous infraspecific variation in the SCN genome has caused a great deal of concern to breeders as new races and populations of SCN continue to develop making resistant cultivars less effective. It is, however, apparent from the inheritance studies that such sources of resistance as Peking, PI 90763 and PI 88788 have many genes for resistance since diverse SCN populations have low reproduction on these genotypes. PI 437654 may have even more genes since it rarely allows any cyst development. In fact, discovery of PI 437654 as a source of resistance to all the known races of SCN is one of the most significant developments in the management options to limit the losses caused by the nematode. PI 437654 along with the cultivar Hartwig, which derived resistance from Forrest and PI 437654 (Anand 1992b) are being widely used in breeding programmes across the US and new cultivars with resistance derived from these sources should be capable of resisting development of all the races. Currently, the management of losses caused by SCN is based mainly on the cultivation of SCN-resistant soybean varieties. However, resistance is not durable because of infraspecific genetic diversity in SCN populations. A system approach to reduce SCN damage through a combination of a variety of tactics including natural environment modifications, predators and parasites, cultivation of blends of SCN-resistant and -susceptible cultivars (Anand *et al.* 1995) and, when necessary, chemical pesticides is required.

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