Expression of Nematode Resistance in Plant Introductions of *Arachis hypogaea*

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ABSTRACT

The peanut root-knot nematode (*Meloidogyne arenaria*, race 1) is a world-wide pest of peanut (*Arachis hypogaea* L.). Several moderately resistant genotypes have been identified in the cultivated peanut species. Our objective was to determine the expression of resistance for six of these genotypes. We examined four potential expressions of resistance—(a) fewer second-stage juveniles (J2) penetrate the roots, (b) fewer J2 establish functional feeding sites, (c) slower maturation, and (d) reduced fecundity (eggs per female). Seedlings of the susceptible cultivar Florunner and the resistant genotypes were inoculated with J2 of *M. arenaria*, and transplanted 3 d later to synchronize nematode development. Penetration was assessed at 3 and 10 d; development at 10 (or 12), 17, 22, and 27 d; and fecundity at 60 d after inoculation. The experiments were conducted in a greenhouse or growth chamber. The number of J2 within the roots was similar in resistant and susceptible peanut after 3 d; however, numbers were lower in two of the resistant genotypes than in Florunner after 10 d. A greater percentage of J2 failed to develop in all of the resistant genotypes (72 to 79%) than in Florunner (50%) after 17 d. Of the J2 that did begin to develop, the rate of maturation and fecundity was similar in resistant and susceptible genotypes. A lack of development indicates that the J2 failed to establish a feeding site. Therefore, the primary expression of resistance in the six peanut genotypes appears to be a reduction in the percentage of J2 that establish a functional feeding site. The decline in J2 after infection may be related to the failure to establish a feeding site.

Key Words: *Meloidogyne arenaria*, nematode resistance, resistance expression, root-knot nematode.

Meloidogyne arenaria (Neal) Chitwood, race 1 is a parasite of peanut (*Arachis hypogaea* L.) worldwide. In the southern U.S. (AL, FL, GA, SC, and TX), this nematode reduces peanut yield by 3 to 15% annually (Koenning *et al.*, 1999). Crop rotation and nematicides are the primary management tactics available to growers to reduce losses to this nematode. However, because of the extensive host range of *M. arenaria*, few marketable nonhost crops are available for rotation with peanut in the U.S. Nematicides reduce early season nematode densities; but frequently, densities at the end of the growing season are similar in nematicide-treated and untreated plots (Hagan and Hewlett, 1989; Culbreath *et al.*, 1992). Peanut cultivars with resistance to *M. arenaria* would be an effective and inexpensive alternative to nematicides. Furthermore, a highly resistant peanut cultivar would substantially suppress nematode populations throughout the growing season.

Resistant genotypes are characterized by lower nematode reproduction relative to a susceptible genotype. The mechanism by which the resistant plants suppress nematode reproduction varies depending upon crop species and resistance genes. In susceptible plants, second-stage juveniles (J2) penetrate roots in the zone of elongation, migrate to the root apex, then reverse direction and migrate up the root, eventually entering the vascular cylinder (Wyss *et al.*, 1992). The J2 then induces a cluster of giant-cells in the vascular tissue and begins to feed. As the juvenile acquires nutrition from the giant-cells, it swells and molts to the third- (J3) and then the fourth-stage (J4) juvenile. The J4 molts into a sedentary adult female or occasionally into a migratory male. Several researchers have investigated the effect of plant resistance on various life stages of *Meloidogyne* species. In resistant plants, fewer J2 may enter roots (Call *et al.*, 1996; Pedrosa *et al.*, 1996), perhaps because of insufficient chemical cues released from the roots, or the J2 may penetrate the roots but then egress a few days later (Herman *et al.*, 1991). After penetration, the J2 may fail to enter the vascular system (Potenza *et al.*, 1996); or they may enter the vascular system but are prevented from establishing a feeding site by a hypersensitive reaction (Kaplan and Davis, 1987). In some resistant plants, the J2 establishes a feeding site, but the rate of maturation or fecundity of females may be lower than in susceptible plants because of reduced food quality or quantity, or collapse of the giant-cells (Moura *et al.*, 1993; Windham and Williams, 1994; Creech *et al.*, 1995; Call *et al.*, 1996; Pedrosa *et al.*, 1996; Sydenham *et al.*, 1996).

High levels of resistance to *M. arenaria* have been identified in wild *Arachis* spp. (Baltensperger *et al.*, 1986; Nelson *et al.*, 1989; Holbrook and Noe, 1990) and in cultivated *A. hypogaea* introgressed with resistance genes from wild species. Two advanced populations of *A. hypogaea* have been developed, one with genes introgressed from *A. batizocoi* Krapov. & W.C. Gregory × (*A. cardinasii* Krapov. & W. C. Gregory × *A. diogoi* Hoehne) and a second one from *A. cardinasii* (Nelson *et al.*, 1989; Stalker *et al.*, 1995; Starr *et al.*, 1995). High levels of nematode resistance in *A. cardinasii* are conditioned by two dominant genes (Garcia *et al.*, 1996) and are expressed as a hypersensitive-like reaction with few J2 showing signs of development (Nelson *et al.*, 1990). The genetics of nematode resistance in *A. batizocoi* and in *A. diogoi* is not known; however, the genes involved are different from those identified in *A. cardinasii* (Garcia *et al.*, 1996). Moreover, unlike *A. cardinasii*, there is no indication of a hypersensitive reaction in *A. batizocoi*. In the latter species, fewer J2 develop within the roots and,

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when development occurs, the rate of maturation is slower than in *A. hypogaea* (Nelson *et al.*, 1990).

Moderate levels of native resistance (not introgressed from wild species) exist in cultivated *A. hypogaea* (Holbrook and Noe, 1992; Holbrook *et al.*, 1996; Holbrook *et al.*, 1998; Holbrook *et al.*, 2000). Approximately 8% of plant introductions in the U.S. Peanut Germplasm Collection are estimated to have some level of resistance to *M. arenaria* (Holbrook *et al.*, 2000). Although the genetics of this resistance is unknown, some of the accessions in the collection likely contain different genes for resistance because they originate from distinct morphological and geographical clusters (Holbrook *et al.*, 2000). Expression of different mechanisms of resistance may indicate the presence of different resistance genes. The incentives for identifying different resistance genes are that combining these genes in the same genotype may improve the level and durability of the resistance.

The objective of this study was to determine whether the expression of resistance to *M. arenaria* is similar for six moderately resistant *A. hypogaea* genotypes (Table 1). We examined four potential expressions of resistance—(a) reduced penetration, (b) fewer J2 establish functional feeding sites, (c) slower maturation, and (d) reduced fecundity (eggs per female).

Table 1. Reproduction of *Meloidogyne arenaria* on susceptible (cv. Florunner) and six moderately resistant genotypes of *Arachis hypogaea* from various geographical locations.

Accession PI[a]	Genotype[b]	Origin[c]	Egg mass index[d]	Eggs
				no./g fresh root
565448	Florunner	U.S.	4.0	15496
295748	130F	Greece	2.2	2678
259639	186B	Cuba	2.3	1124
365100	10B	Swaziland	2.2	3850
512247	122cc	China	1.8	1689
295747	557cc	China	1.8	1052
196762	5cc	Greece	1.7	897

[a]U. S. Plant Inventory Number.

[b]Georgia accession numbers.

[c]Country from which accession was deposited into the U.S. Peanut Germplasm Collection.

[d]Index is based on a 0-5 scale where 0 = no egg masses; 1 = 1 to 2; 2 = 3 to 10; 3 = 11 to 30; 4 = 31 to 100; and 5 = more than 100 egg masses per root system. Nematode reproduction and egg mass indices on Florunner and the resistant genotypes were determined in a previous study (Holbrook *et al.*, 2000).

Materials and Methods

General Methods. Resistant and susceptible peanuts (Table 1) were grown in pots containing a loamy sand (85% sand, 11% silt, 4% clay; pH 5.3). Prior to planting, the soil was steam heated at 132 C for 6 hr and mixed with 2.2 and 0.4 g/kg dolomite lime and fertilizer (5-10-15 NPK), respectively. The plants were inoculated with nematodes 10-14 d after planting. Unless otherwise noted, the plants were maintained in a greenhouse at 20 to 35 C and watered as needed.

Meloidogyne arenaria race 1 (Gibbs isolate), originating from a peanut field in Tifton, GA, was cultured alternately on tomato (*Lycopersicon esculentum* Mill. cv. Rutgers) and peanut to reduce potential contamination from *M. incognita* (a parasite of tomato but not peanut). Eggs were collected from roots of tomato using NaOCl (Hussey and Barker, 1973) and placed in hatching dishes to obtain J2 for the experiments. The hatching dishes were composed of two layers of tissue paper supported by a screen and placed in a dish containing enough water to form a thin layer over the tissue. The J2 that hatched on the tissue and migrated into the water were collected daily. Suspensions of J2, collected over a 4-d period, were bubble-aerated at room temperature until inoculation. The J2 were added to two opposing holes made within 2 cm of the plant stem. The number of J2 added to each pot varied among the experiments.

Penetration. Peanut seedlings in 200-cm^3 pots were inoculated with 916 ± 12 ($\bar{x}$ ± SE) J2 per pot. After 3 d, the plants were uprooted, rinsed free of adhering soil, and either transplanted into 700-cm^3 pots containing uninfested soil or reserved for nematode extraction. Three and 10 d after inoculation, the roots were weighed, cut into *ca.* 3 cm-long pieces, stained with an acid fuchsin, lactoglycerol solution (Bridge *et al.*, 1982), and homogenized in a blender for 30 sec. Stained nematodes were separated from the root debris using nested 75- and 25-µm-pore sieves and then counted. Two trials were performed with six replicate plants per genotype per day for each trial, except PI 365100 which was replicated only three times on day 10 in Trial 1. Numbers were transformed by square root, and a two-way analysis of variance (ANOVA) was used to determine the effect of genotype and trial on the number of nematodes in the roots 3 and 10 d after inoculation. Differences among genotypes were determined with Fisher's LSD test ($P \leq 0.05$). Nematode numbers are presented as the total per root system.

Development. Nematode development in resistant and susceptible genotypes was compared in two separate experiments. In the first experiment, the proportion of swollen juveniles was determined. Pots containing 200 cm^3 of soil and a peanut seedling were inoculated with 980 ± 1 J2. To synchronize nematode development, the plants were uprooted 3 d after inoculation, rinsed free of soil, and transplanted into pots containing 700 cm^3 of uninoculated soil. Seven plants of each genotype were uprooted 10 and 17 d after inoculation, and the roots stained with acid fuchsin as previously described. The roots from day 10 were homogenized in a blender for 30 sec and the stained nematodes were separated from root debris with nested 250-, 75-, and 25-µm-pore sieves. Because of the difference in nematode size and fragility between day 10 and 17, roots from day 17 were homogenized for 15 sec and the stained nematodes were separated from root debris with nested 850-, 150-, and 25-µm sieves. Using an inverted microscope, the stained nematodes were classified as "developing" or "undeveloped" if they were larger than or similar in size to the infective J2, respectively. The experiment was performed twice. A two-way ANOVA was used to determine the effect of genotype and experimental trial on the proportion (transformed by arcsine) of develop-

ing juveniles. Data from day 10 and 17 were analyzed separately. Fisher's LSD test ($P \leq 0.05$) was used to separate differences among the genotypes. The resistant genotype PI 196762 was not included in this experiment due to poor germination.

In the second experiment, the rate of nematode maturation was determined. Pots containing 200 cm^3 soil and a peanut seedling were inoculated with 2101 ± 79 J2. Plants were uprooted 3 d after inoculation, rinsed free of soil, and transplanted into pots containing 700 cm^3 of uninoculated soil. The plants were maintained in a growth chamber at 28-30 C and 12 hr of light. At 12, 17, 22, and 27 d after inoculation, three plants per genotype per day were sampled. Roots were weighed, cleared with NaOCl (Byrd *et al.*, 1983), cut into *ca.* 2-cm pieces, and stained with a acid fuchsin, lactoglycol solution (Bridge *et al.*, 1982). The stained roots were weighed again and a small weighed subsample (*ca.* 0.5 g) was taken. Using a dissecting microscope, the nematodes in the subsample were dissected out of the root to determine the stage of development. If less than 30 nematodes were found, a second or third subsample was taken and the nematodes dissected out of the root. The nematode stages identified were vermiform J2, swollen J2, third- and fourth-stage juvenile (J3-4), males coiled within the fourth-stage cuticle, and adult females. Third- and fourth-stage juveniles were grouped together because these stages could not be easily distinguished. There were three replicate pots for each sampling day and genotype, and the experiment was performed twice. A two-way ANOVA was used to determine the effect of genotype and trial on the proportion (transformed by arcsine) of developing individuals that became adult females on each sampling day. Regression analysis was used to determine rate of maturation for each genotype. The maturation rate is equal to the slope of the proportion females plotted against sampling day and included only those individuals that had begun to develop (i.e., swell). A t-test was used to determine whether the rate of maturation in the resistant genotypes was different from that in the susceptible Florunner.

Fecundity. Peanut seedlings planted in 10-cm square pots (700 cm^3) were inoculated with 1019 J2 and 8190 eggs in Trials 1 and 2, respectively. After 60 d, the plants were uprooted, rinsed free of adhering soil, and the egg masses stained with phloxine B (Holbrook *et al.*, 1983). The first seven to 11 individual egg masses encountered were collected from each root system and placed in a 10-cm^3 glass vial containing 6.5 mL of water. To dissolve the gelatinous matrix that surrounds the eggs, 1.6 mL of a 5.25% NaOCl solution was added to the vial. The vials were sealed with a rubber stopper and placed on a vortex mixer for three to four 30-sec intervals. The freed eggs were collected on a 25-µm-pore sieve, rinsed with tap water, and counted. The average number of eggs per egg mass was determined for each plant. There were three to five replicate plants per genotype for each trial. Differences in fecundity were determined using ANOVA and Fisher's LSD test on square-root-transformed data.

Results and Discussion

Penetration. Three days after inoculation, the number of J2 within the roots of the moderately resistant genotypes was similar to the number in Florunner (Fig. 1). There was an interaction between peanut genotype and experimental trial on day 3 ($P = 0.008$). When the trials were analyzed separately for day 3, a greater number of J2 were within the roots of PI 259639 in Trial 2 than were within the roots of PI 365100, PI 196762, and PI 295747 ($P \leq 0.05$); however, such differences were not apparent in Trial 1, and in neither trial were there differences between Florunner and the resistant genotypes. These results indicate similar penetration of resistant and susceptible roots by J2 and are consistent with most studies that have examined root penetration as a mechanism of nematode resistance (Herman *et al.*, 1991; Moura *et al.*, 1993; Windham and Williams, 1994; Creech *et al.*, 1995; Potenza *et al.*, 1996; Sydenham *et al.*, 1996). The few studies that have demonstrated differences in root penetration between resistant and susceptible genotypes could not eliminate early nematode egression from the resistant genotype as a possible cause (Call *et al.*, 1996; Pedrosa *et al.*, 1996).

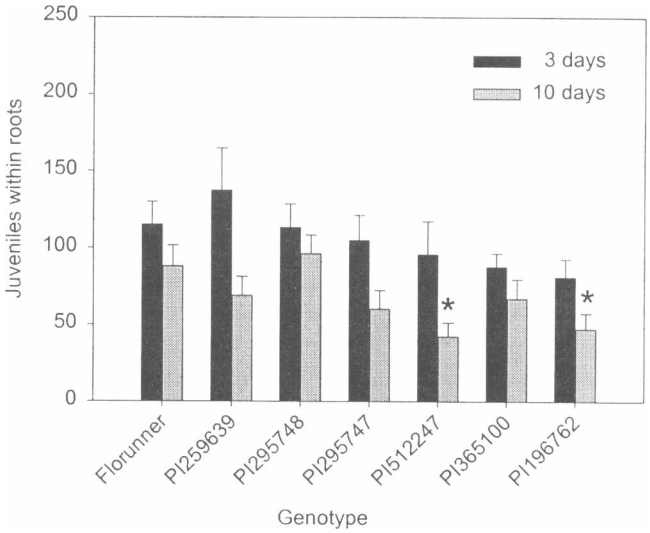

Fig. 1. Numbers of *Meloidogyne arenaria* juveniles per root system in susceptible (cv. Floruner) and resistant genotypes of *Arachis hypogaea* 3 and 10 d after inoculation of second-stage juveniles. Bars with an asterisk are different from Florunner ($P \leq 0.05$) on that sampling day.

For all genotypes, there were fewer nematodes within the roots on day 10 than there were on day 3 (Fig. 1). On day 10, the trends in number of juveniles per root system for the different peanut genotypes were similar for Trial 1 and 2 (i.e., no interaction between genotype and trial). The percentage decrease tended to be greater for PI 512247 (56%), PI 259639 (50%), PI 295747 (42%), and PI 196762 (41%) than it was for Florunner (23%), PI 365100 (23%), and PI 295748 (15%). Ten days after inoculation, there were fewer juveniles in the roots of PI 512247 and PI 196762 than were in the roots of Florunner ($P \leq 0.05$). The lower numbers of juveniles observed in roots of PI 512247 and PI 196762 than in roots of Florunner may be the result of greater egression from these resistant genotypes. Greater egression of J2 from roots of resistant than from roots of susceptible genotypes has been demonstrated in several plant species (Reynolds *et al.*, 1970; Herman *et al.*, 1991). However, we did not conclusively demonstrate that J2 egressed from roots and, therefore, we cannot eliminate the pos-

sibility that the decline in number of juveniles within roots between days 3 and 10 was due to death and decay of J2 within the roots.

Development. In the first experiment, the percentage of J2 that had begun to develop in the moderately resistant genotypes was similar to Florunner 10 d after inoculation but was less than Florunner 17 d after inoculation (Fig. 2). At 17 d, 50% of the J2 were undeveloped in Florunner whereas 70% or more were undeveloped in the resistant genotypes. A lack of development indicates that the J2 failed to established a functional feeding site in the vascular tissue. The reason for this failure is unknown. No hypersensitive reaction was observed that would have prevented formation of a functional feeding site. Furthermore, we did not determine whether the undeveloped J2 were alive or dead, or whether they were within the vascular or cortical tissue. Potenza *et al.* (1996) showed that in a resistant alfalfa cultivar, J2 did not move from the root apex into the vascular system as they did in the susceptible cultivar during the 72 hr following inoculation. Moreover, few of these misplaced J2 could be found in either the root apex or vascular tissue after 7 d; apparently, they had migrated out of the root.

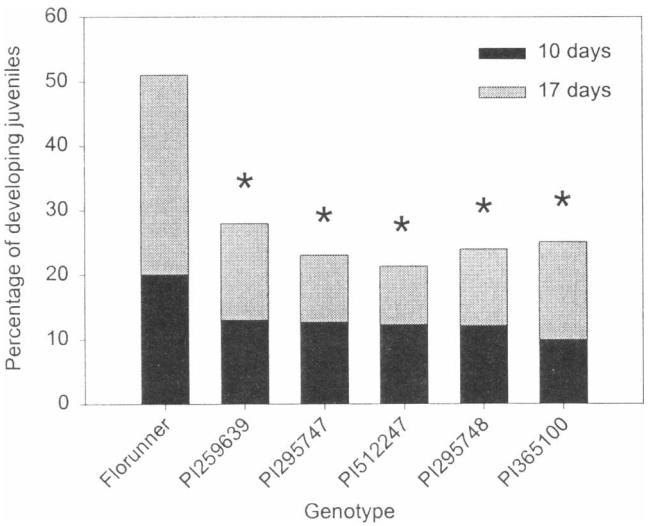

Fig. 2. Percentage *Meloidogyne arenaria* juveniles that had begun to develop (i.e., swell) in susceptible (cv. Florunner) and resistant genotypes of *Arachis hypogaea* 10 and 17 d after inoculation of second-stage juveniles. Bars with an asterisk are different from Florunner ($P \leq 0.05$).

The high percentage of J2 that failed to develop even in the susceptible Florunner may have been due to root damage caused by transplanting nematode-infected seedlings. Peanut seedlings were transplanted 3 d after inoculation into uninoculated soil to prevent continued penetration of juveniles and to synchronize development; however, this procedure resulted in considerable root damage. When plants were uprooted 7 to 14 d after transplanting, limp and decaying roots were observed. It is likely that some nematodes had infected sections of roots that later were damaged during transplanting.

In the second experiment, there were no adult females or males 12 d after inoculation. Starting from day 17, the percentage of females increased over time in all genotypes except PI 295747 where the percentage of females increased from 43 to 58 % from day 17 to day 22, but declined to 32% on day 27 (Table 2). These erratic results are probably due to sampling error caused by the small sample size (n = 3) on day 27. Genotypes PI 295748 and PI 259639 had a lower percentage of adult females than Florunner ($P \leq 0.05$) on day 17; however the percentage of females was similar to Florunner on days 22 and 27. Genotype PI 295747 had a lower percentage of females than did Florunner on days 17 and 27, but not on day 22. The rate of maturation to adult females did not differ among genotypes (Table 2) or experimental trials. In calculating this rate, we used only those individuals that showed signs of development to avoid confounding the maturation rate with failure of the J2 to initiate a functional feeding site.

Table 2. The percentage of developing *Meloidogyne arenaria* that reached the adult female stage 17 to 27 d after inoculation and the rate of maturation in nematode susceptible (cv. Florunner) and resistant genotypes of *Arachis hypogaea*.

Genotype	Females[a] 17 d	22 d	27 d	Rate of maturation slope ± SE[b]	P value	R^2
	---------- % ----------					
Florunner	26 ± 6	46 ± 9	64 ± 11	0.0433 ± 0.0067	0.0001	0.69
PI 365100	34 ± 4	44 ± 9	68 ± 8	0.0434 ± 0.0060	0.0001	0.73
PI 512247	34 ± 8	58 ± 4	64 ± 9	0.0466 ± 0.0059	0.0001	0.77
PI 295748	20 ± 8*	45 ± 9	54 ± 7	0.0393 ± 0.0069	0.0001	0.63
PI 259639	13 ± 5*	42 ± 8	43 ± 3	0.0340 ± 0.0059	0.0001	0.64
PI 295747	43 ± 6*	58 ± 4	32 ± 4*	0.0304 ± 0.0082	0.002	0.42
PI 196762	26 ± 6	41 ± 9	55 ± 5	0.0379 ± 0.0068	0.0001	0.64

[a]The percentage of females was calculated from the total number of individuals that had begun to develop (i.e., swell) within the roots. Undeveloped second-stage juveniles and males were not included in the total. Percentages are the means of two trials (n = 6), except day 27 which is the mean from trial 2 (n = 3). An asterisk next to a percentage indicates a difference from Florunner on that sampling day ($P \leq 0.05$).

[b] Slopes were calculated from the regression of percentage females against days after inoculation (12, 17, 22, and 27 d).

Nematode eggs were first observed 22 d after inoculation in both experimental trials. In Trial 1, females were producing eggs in all genotypes by day 22 with approximately 10% of the females containing egg masses (data not shown). In Trial 2, eggs were not found in PI 295748, PI 512247, and PI 365100 on day 22; however, females were producing eggs in all genotypes by day 27 with approximately 10% of the females containing egg masses.

Males, because they are free living, did not carry over from one sampling period to the next and were difficult to enumerate. Nevertheless, there appeared to be no effect of peanut genotype on the percentage of the population that developed into males. The percentage of males coiled within the J4 cuticle varied between 3 and 17% and was not consistently high in any one genotype

(data not shown). Moreover, a shift in the sex ratio towards males would have resulted in an overall lower percentage of females in one of the resistant genotypes, a reaction we were unable to document.

Fecundity. Fecundity of females was similar in all peanut genotypes. The number of eggs per egg mass ranged from 213 to 356 for the resistant genotypes and was 284 ± 46 for Florunner (data not shown).

In summary, we were unable to detect any differences in expression of resistance in the six peanut genotypes examined. In all genotypes, resistance was expressed as a reduction in the percentage of juveniles that initiated a functional feeding site within the vascular tissue. We observed a greater postinfectional decline of juveniles in roots of some of the resistant genotypes compared to Florunner. However, nematode death or migration out of roots may be related to the inability of the juvenile to establish a feeding site.

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