RESEARCH NOTES

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Expression of Resistance to *Meloidogyne arenaria* in *Arachis batizocoi* and *A. cardenasii*¹

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Through most of the southern United States, peanut is attacked by the root-knot nematodes *Meloidogyne arenaria* Neal (Chitwood) and *M. hapla* Chitwood. Ninety percent of the peanut fields in North Carolina are infested with *M. arenaria* and (or) *M. hapla* (11). *Meloidogyne arenaria* is predominant in the more southern portions of the United States, occurring in 41% of the peanut fields in Alabama (5) and 30% of those in Texas (14). Although accurate estimates of losses resulting from *M. arenaria* on peanut are lacking, losses in excess of 30% of the yield potential have been observed in heavily infested fields (Starr, unpubl.).

Despite the relative importance of M. arenaria as a pathogen of peanut, no cultivar resistant to this nematode is available. nor has any source of resistance been identified in the thousands of genotypes of A. hypogaea that have been examined (4,6,7). Resistance to M. arenaria has been identified in other Arachis species, but they are genetically incompatible with A. hypogaea (1). We recently identified A. batizocoi Krap & Greg nom. nud. and A. cardenasii Krap & Greg nom. nud. as species that are both resistant to M. arenaria and compatible with A. hypogaea (8,9). This study further examined the interaction of M. arenaria with A. batizocoi and A. cardenasii to determine when and how resistance is expressed.

Penetration of roots and postinfection development of *M. arenaria* race 1 on two detected in the roots of *A. batizocoi* and *A. cardenasii* than in *A. hypogaea* at 18 and 30 days after inoculation (Table 1).

Development of M. arenaria on A. hypogaea was consistent with that expected for a susceptible host (12); advanced, swollen [2 associated with well-developed giant cells were observed at 7 days after inoculation. At 18 days after inoculation, 29% of the population were adult females and 0.5% had begun egg production (Fig. 1). By 30 resistant genotypes (A. batizocoi K-9484 and A. cardenasii GKP-10017) were compared with that on a susceptible A. hypogaea cultivar, Tamnut 74, in a controlled environment at 28 C with a 13-hour day (218 μ E m⁻²s⁻¹). Seed of all lines were germinated on moist paper for 72 hours at 28 C then transplanted singly into 470-cm³ plastic cups filled with a 1:1:1 (v:v:v) mixture of steam-pasteurized sand : peat : vermiculite. Each seedling was inoculated 7 days later by pipetting a suspension of 2,500 second-stage juveniles (J2) into depressions in the soil around the base of each seedling. Inoculum was obtained by the method of Vrain (13).

Three plants of each genotype were arbitrarily selected for evaluation at 3, 7, 12, 18, 24, and 30 days after inoculation. Penetration and postinfection development of *M. arenaria* were determined by microscopic examination of cleared and stained roots (2,12). Data on fresh root weights, numbers of nematodes per gram of root, and stage of nematode development were subjected to analysis of variance by the SAS GLM procedure (10). The experiment was repeated once and the data combined prior to analysis.

No significant difference in numbers of

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 TABLE 1. Meloidogyne arenaria in roots of three

 Arachis species.

Days after inoculation	Nematodes/g fresh root weight		
	A. hypogaea	A. batizocoi	A. cardenasii
7	225 a	210 a	213 a
18	58 a	30 Ь	14 b
30	47 a	25 Ь	7 Ь

Values are means of two experiments, each with three replications. Means within a row followed by the same letter are not significantly different (P = 0.05).

J2 per gram of root of the three Arachis spp. was observed at 3 or 7 days after inoculation. Fewer nematodes (P = 0.05) were days after inoculation, 34% of the population were adult females with eggs. Development of M. arenaria on A. batizocoi was slower than it was on A. hypogaea, with 10% of the population in the advanced J2 stage at 7 days after inoculation, compared with 21% on A. hypogaea. Adult females comprised 2% of the population 18 days after inoculation, and by 30 days after inoculation only 10% of the population had developed into adult females. No females with eggs were observed on A. batizocoi. Giant cells associated with nematodes on A. batizocoi were smaller than those on A. hypogaea.

Little development of *M. arenaria* occurred on *A. cardenasii*; less than 5% of the population developed to the advanced J2 stage (Fig. 1). Only a single nematode was observed to have developed beyond the advanced J2 stage. No giant cell complexes were observed; instead, most nematodes at 7 days after inoculation and beyond were associated with necrotic host cells.

These data confirm and extend our earlier report (8,9) of the resistance to *M. ar*enaria in *A. batizocoi* and *A. cardenasii*. Further, the data provide evidence that the mechanisms of resistance to *M. arenaria* differ in these two species. Because of the almost complete lack of development of the nematodes and the numerous necrotic host cells observed at 7 days after inoculation, the resistance of *A. cardenasii* appears to be of the "hypersensitive response" type. It is thus likely to be governed



FIG. 1. Development of *Meloidogyne arenaria* on *Arachis hypogaea, A. batizocoi,* and *A. cardenasii.* A). At 7 days after inoculation. B). At 18 days. C). At 30 days.

by relatively few genes and may be relatively easy to manipulate in a breeding program (3). In contrast, the resistance of *A. batizocoi* is of the "rate-reducing" type, possibly conditioned by a larger number of genes, and thus may be more difficult to manipulate in a breeding program.

Both A. batizocoi and A. cardenasii can be crossed with A. hypogaea to produce fertile, interspecific hybrids (Simpson, unpubl.). One breeding line (TP-135) with resistance to M. arenaria has been developed from such an interspecific hybrid (8,9). Thus, development of peanut cultivars with high levels of resistance to M. arenaria may now be possible; however, additional work is needed to determine the genetic mechanisms of resistance and to determine if this resistance will be effective against most populations of the pathogen.

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