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Genetics of Resistance to Reniform Nematode in Upland Cotton

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ABSTRACT

Reniform nematode (RN), (*Rotylenchulus reniformis* Linford and Oliveira) is an economically important pest of upland cotton (*Gossypium hirsutum* L.). Sources of resistance to RN have been identified, but inheritance of resistance has not been studied previously. Four cotton lines [La RN-910, Auburn (Aub) 612-RNR, M 019-RNR, and 'Deltapine 41' (Dp 41)] were selected as parents for a genetic study of resistance to RN reproduction. Deltapine 41 (susceptible) was crossed to each of the three lines previously reported as resistant. Means of P_1, P_2, F_1, F_2, BC_1P_1, and BC_1P_2 generations in the three crosses were analyzed by the generation mean analysis procedure to estimate gene effects. Seedlings grown in the greenhouse were individually inoculated at the first true leaf stage with 2000 RN juveniles. After an average growth period of 43 d in the winter and 32 d in the summer, the entire root system of each plant was harvested, and RN eggs were extracted and counted. Significant ($p < 0.05$) differences among generations and nondiscrete frequency distributions in segregating populations indicated that RN resistance in two of three crosses (La RN-910 × Dp 41 and Aub 612-RNR × Dp 41) was under genetic control and inherited in a quantitative manner. No pattern was observed for the significance of additive and dominance gene effects, but significant ($P < 0.05$) epistatic gene effects occurred in most cases. The occurrence of transgressive segregation for susceptibility and epistatic gene effects suggest that resistance to RN in these cotton lines was controlled by two or more pairs of genes. Advancing generations to F_4 or F_5, while maintaining genetic variability prior to selection, may improve selection efficiency by reducing nonadditive gene effects for RN resistance. High coefficients of variation and general lack of additive gene effects suggest that future genetic and breeding studies for RN resistance may be enhanced by reducing environmental effects and exploring other sources of resistance.

THE RENIFORM NEMATODE (RN) was first reported as a parasite on roots of upland cotton in Georgia (Smith, 1940) and Louisiana (Smith and Taylor, 1941). This nematode has become a pest of economic importance in all coastal cotton-producing states from South Carolina to Texas (Veech, 1984; Blasingame and Patel, 1987).

The RN reproduces abundantly on cotton, and causes an increase in the incidence of fusarium wilt disease [*Fusarium oxysporum* Schleft. f. *vasinfectum* (Atk.) Snyd. & Hans.], a reduction in yield, a delay in maturity, a reduction in plant and boll size, and in some years, a reduction in lint percentage (Jones et al., 1959). Damage to cotton is caused by the female that feeds in the pericycle of the root (Yik and Birchfield, 1981). They observed typical pericycle hypertrophy, enlarged nuclei and nucleoli, cell wall dissolutions, and granular cytoplasm in susceptible cotton plants. In resistant plants, they observed collapsed or killed endodermal and pericycle cells at feeding sites (hypersensitivity), or restricted syncytial development.

Utilization of resistant cultivars could be an effective and economical means of controlling RN. Highly resistant cultivars could eliminate the necessity of nematicide application and even permit rotation with susceptible cultivars and/or crops (Shepherd, 1982a; Williams et al., 1983). Although high yielding RN resistant cultivars are not presently available, sources of resistance are known. Yik and Birchfield (1984) found all entries of *G. longicalyx* Hutch. & Lee to be immune and representatives of *G. somalense* (Gurke) Hutch. and *G. stocksii* Mast. to approach immunity. Compared with 'Deltapine 16' they reported the following genotypes to be resistant or highly resistant: *G. barbadense* L. (race stock T. 110), *G. arboreum* L. (PI 417895, PI 417891, and CB 3839), *G. herbaceum* L. (PI 408775), *G. raimondii* Ulbr. (no. 9), and *G. hirsutum* (race stocks T. 874, T. 893, and T. 903). The upland breeding strain La RB 15702 was found to be moderately resistant.

Beasley (1985) confirmed the high level of resistance of *G. barbadense* race stock T. 110 and the *G. hirsutum* race stock T. 893 as well as the moderate level of resistance of La RB 15702. In addition, he identified new sources of RN resistance in upland breeding lines La 434-1031-4-4 (La RN-4-4), La 434-1031-810909 (La RN-909), La 434-1031-810910 (La RN-910), Auburn (Aub) 79-G-20, Aub 80-180, Aub 566-RNR, Aub 612-RNR, and Aub 634-RNR; and in the following *G. hirsutum* race stocks and/or day-neutral converted race stocks: TR 19, TR 26, TR 28, TR 75, TR 176, TR 487, and TR 495.

Although RN-resistant lines have now been identified in upland cotton, the genetic basis of this resistance is unknown. Resistance to RN in soybean, *Glycine max* (L.) Merr., was reported by Harville et al. (1985) to be quantitative in nature and controlled by two pairs of genes with unequal effects. Resistance to RN in tomato, *Lycopersicon esculentum* Mill., (PI 375937) is controlled by at least one dominant gene, which may be closely linked to one or more genes for resistance to the sugar beet cyst nematode, *Heterodera schachtii* Schmidt, (Rebois et al., 1977).

The objective of the present study was to partition genotypic value for resistance to RN into additive, dominance, and epistatic gene effects. Knowledge of estimates and relative importance of these gene effects should be useful to cotton breeders in choosing breeding strategies and selection procedures for development of cotton cultivars with resistance to RN.

MATERIALS AND METHODS

Four cotton lines, La RN-910, Aub 612-RNR, M 019-RNR, and Dp 41, were selected as parents for this study, based on their observed reaction to the RN under field and greenhouse conditions (Beasley, 1985). The line La RN-910 is resistant to RN and root-knot, *Meloidogyne incognita* (Kofoid & White) Chitwood, nematodes (Jones et al., 1988); Aub 612-RNR and M 019-RNR, both provided by R.L.

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Shepherd and known to be resistant to root-knot nematode (Shepherd, 1982b, 1983), are also resistant to RN (Beasley, 1985); and Dp 41 is susceptible to RN (Beasley, 1985). Parental lines (P_1, P_2), F_1, F_2, and backcrosses of F_1 to P_1 (BC_1P_1) and P_2 (BC_1P_2) of each cross (La RN-910 × Dp 41, Aub 612-RNR × Dp 41, and M 019-RNR × Dp 41) were evaluated for RN reproduction in the greenhouse in 1986 and 1987 (Table 1).

The study consisted of two plantings of two blocks of each cross arranged in a randomized complete-block design. Each block consisted of 10 plants for each of the three nonsegregating generations (P_1, P_2, and F_1), 20 plants for each backcross generation (BC_1P_1 and BC_1P_2), and 40 plants for the F_2 generation. Each plant (experimental unit) was grown in an individual 1000-cm^3 plastic pot holding about 500 g of soil. Pots were half-buried in sand that was kept moist to reduce soil temperature fluctuations. A 50:50 mixture of Commerce silt loam soil (fine-silty, mixed, nonacid, thermic Aeric Fluvaquents) and river sand was steam sterilized and used for the 19 Dec. 1986 planting. Subsequent plantings were made in a 50:50 mixture of Olivier silt loam soil (fine-silty, mixed, thermic Aquic Fragiudalfs) and river sand that had been fumigated with methyl bromide. Two seed per pot were planted and, after emergence, plants were thinned to one seedling per pot. Soil from a cotton field at Perkins Road Agronomy Farm near Baton Rouge, LA, heavily infested with RN and not contaminated with root-knot nematodes, was screened by the modified LSU method (Beasley, 1985) prior to each inoculation date to collect inocula. At the first true leaf stage, each plant was inoculated with a 1-mL suspension containing a mixture of 2000 RN (juveniles, males, and infective females), along with a few other species of mostly nonplant-parasitic nematodes. One exception to this procedure was that plants in Planting 1 of Cross 1 were inoculated with 3000 to 3500 RN eggs. Inoculum was dispensed with a pipette into 5-cm deep depressions at two locations in the root zone. Dry soil was placed into the holes before and after nematode inoculum to avoid leaching of the inoculum.

Plants in the greenhouse were fertilized bi-weekly with 6.0, 2.6, and 5.0 mg plant $^{-1}$ of N, P, and K, respectively. Pesticides acephate (*O*, *S*-Dimethyl acetylphosphoramidothioate) and dicofol [4,4-Dichloro-α-trichloro-methylbenzhydrol] were used to protect plants from insects and mites. A period of 40 to 50 and 32 d for the first and second planting dates, respectively, was allowed for the infective females to infect the plants and to reproduce (Table 1). Extra time was allowed for plants inoculated with eggs over those inoculated with juveniles and for winter plantings over summer plantings. Air temperature ranges for the various plantings are given in Table 1.

Entire root systems of individual plants were harvested to estimate nematode egg production. Soil was removed by soaking the root ball in water and carefully washing the roots free of soil. Each root system was blotted with a paper towel, weighed, and then placed in a 125-mL flask containing 100 mL of a 3.5 × $10^{-2}M$ sodium hypochlorite solution for 10 min to free the eggs from egg masses. Roots in each of 20 flasks were agitated as a group for 4 min on an electric shaker to disperse eggs. Eggs were separated from the root debris by pouring the suspension through nested 105 and 26 μm sieves. Eggs on the 26 μm sieve were then washed with tap water to remove residual sodium hypochlorite and backwashed into a beaker that was standardized to a volume of 50 mL. Nematode eggs in a 10-mL aliquot from each beaker were counted in a graduated plastic petri dish at 15× with a stereo microscope, and the counts corrected for 50 mL. Data for each plant were expressed as numbers of eggs per root system (EPRS) and eggs per gram of root (EPGR).

Analyses of variance were calculated for each cross at each planting date and combined over dates for EPRS and EPGR. Generations were considered as fixed effects, and replications and planting dates were considered as random effects. Genotypic values of EPRS and EPGR were partitioned into additive, dominance, and epistatic gene effects by generation mean analysis, following Gamble's (1962) method. Trigenic and higher interactions were assumed to be negligible. In the analysis of the data, population means for each experiment were calculated from individual plant data. The variances of the population means were used to estimate the variances of the estimated parameters. Estimates of these parameters provide an indication of the relative importance of the various types of gene effects included in the total genetic variation of a plant characteristic.

Table 1. Inoculation date, days after inoculation, and air temperature regime of crosses by planting dates (PD).

Cross and planting date	Inoculation date	Days after inoculation	Air temperature	
			minimum	maximum
		d	°C	
Cross 1 (La RN-910 × Deltapine 41)				
PD 1†	19 Dec. 1986	50	21§	31§
PD 2‡	20 May 1987	32	24	39
Cross 2 (Auburn 612-RNR × Deltapine 41)				
PD 1‡	27 Feb. 1987	41	21	34
PD 2‡	20 May 1987	32	24	39
Cross 3 (M 019-RNR × Deltapine 41)				
PD 1‡	6 Nov. 1986	40	20	27
PD 2‡	14 July 1987	32	25	42

† Inoculated with eggs.
‡ Inoculated with juveniles.
§ Mean during nematode development.

RESULTS

Parental and Generation Differences

In Cross 1 (La RN-910 × Dp 41), mean RN EPRS and EPGR produced on La RN-910 averaged 58 and 40%, respectively, of those produced on Dp 41, the susceptible parent (Table 2). Except at Planting 1 (low

Table 2. Generation means for reniform nematode eggs per root system and eggs per gram of root by crosses and planting dates (PD).†

Cross and generation	Eggs per root system		Eggs per gram of root	
	PD1	PD2	PD1	PD2
	no. (÷ 1000)			
Cross 1 [La RN-910 (P_1) × Deltapine 41 (P_2)]				
P_1	0.18c‡	10.83c	0.06d‡	1.27d
P_2	0.25c	24.34a	0.14cd	3.41a
F_1	0.62ab	16.91b	0.22bc	2.04c
F_2	0.76a	18.98b	0.30ab	2.78ab
BC_1P_1	0.33bc	15.65b	0.18bcd	2.30bc
BC_1P_2	0.73a	16.50b	0.35a	2.19bc
Cross 2 [Auburn 612-RNR (P_1) × Deltapine 41 (P_2)]				
P_1	2.96b	5.97b	0.50b	0.75b
P_2	4.33a	10.29a	0.76a	1.50a
F_1	2.86b	12.10a	0.43b	1.46a
F_2	2.74b	10.42a	0.40b	1.41a
BC_1P_1	2.99b	9.60a	0.52b	1.50a
BC_1P_2	2.86b	12.00a	0.54b	1.71a
Cross 3 [MO19-RNR (P_1) × Deltapine 41 (P_2)]				
P_1	11.00bc	3.37c	2.24b	0.72c
P_2	12.72abc	3.42c	3.21a	0.79bc
F_1	12.00abc	5.57abc	2.36b	0.94abc
F_2	13.02ab	7.34a	2.98ab	1.19a
BC_1P_1	10.56c	4.95bc	2.28b	0.89abc
BC_1P_2	14.18a	6.14ab	2.91ab	1.11ab

† Planting dates defined in Table 1.
‡ Means within a column of each cross with a letter in common do not differ at $P = 0.05$ according to LSD.

population), differences between these parents were significant ($P < 0.01$). These results confirm findings by Beasley (1985). Differences among generations (G) occurred for each trait at each planting date (PD) but not in the combined analysis. The interaction of PD × G was important for both traits.

In Cross 2 (Aub 612-RNR × Dp 41), mean RN EPRS and EPGR produced on Aub 612-RNR averaged 63 and 58%, respectively, of those produced on Dp 41 (Table 2). At each planting, the parents were different for both traits. These results also confirm previous findings of resistance by Beasley (1985). Generations of the cross differed significantly ($P < 0.05$) for each trait at each PD but not in the combined analysis. The PD × G interaction was important for each trait.

Mean RN EPRS and EPGR produced on M 019-RNR averaged 93 and 81%, respectively, of those produced on Dp 41 (Table 2). Except at PD 1, where the M 019-RNR and Dp 41 parents differed in EPGR, the two parents did not differ in RN reproduction. These results partially confirm findings by Beasley (1985), who tested M 019-RNR against Dp 41 and day-neutral converted Texas Race Stock 19 and found it to be similar to La RN-910 and Aub 612-RNR in RN resistance. Generations differed for each trait at each PD but not in the combined analysis though interactions of PD × G were unimportant. (Table 2).

Estimates of Gene Effects

In Cross 1, estimates of genetic components of genotypic values indicated that additive (*a*) gene effects reduced EPRS and EPGR at PD 1 (Table 3). Since coefficients of variation were very high and parents did not differ significantly ($P > 0.05$) at PD 1, the level of confidence in these results is low. Additive effects were not detected in PD 2. Dominance (*d*) effects were found for both EPRS and EPGR and PD 2, and they were consistently negative for both traits. Comparisons of F_1 and backcross generation means with the midparent value show partial dominance for resistance, the desired direction for a hybrid cotton breeding program. Estimates of pooled additive × additive (*aa*) gene effects were negative and significant ($P < 0.05$) for EPRS at both plantings and EPGR at PD 2. These estimates were in the desirable direction. Estimates of pooled additive × dominance (*ad*) gene effects for both traits were negative ($P < 0.10$) at PD 1 but positive and significant ($P < 0.01$) at PD 2. The *ad* effects were in the undesirable direction and would be difficult to manipulate in a cotton breeding program. Estimates of pooled dominance × dominance (*dd*) gene effects for both traits were generally positive but nonsignificant.

In Cross 2, estimates of pooled additive gene effects generally were in the resistant (negative) direction and pooled dominance effects in the susceptible (positive) direction, but both were either nonsignificant ($P > 0.10$) or significant at the $P < 0.10$ level (Table 3). The dominance effects were in the undesirable direction to be useful in a hybrid cotton breeding program. Pooled *aa* effects for both traits were in the positive direction but significant ($P < 0.05$) only for EPGR at PD 1. Pooled *ad* gene effects were consistently nonsignificant ($P > 0.10$). Estimates of pooled *dd* gene effects were generally negative for both traits but significant ($P < 0.05$) only for EPGR at PD 2. Directions of the *d*, *aa*, and *dd* gene effects are generally opposite to those observed for these effects in Crosses 1 and 3.

In Cross 3, parents differed significantly ($P < 0.05$) for mean numbers of EPGR only at PD 1 (Table 2). Estimates of genetic components, therefore, are relevant only for EPGR at PD 1 (Table 3). Additive and dominance gene effects significantly ($P < 0.10$) re-

Table 3. Estimates of genetic components of generation means and their standard errors for number of reniform nematode eggs per root system and eggs per gram of root by crosses and planting dates (PD).

Cross and gene effect	Eggs per root system		Eggs per gram of root	
	PD 1‡	PD2‡	PD 1	PD 2
	no. (÷ 1000)			
Cross 1 (La RN-910 × Deltapine 41)§				
m	0.87 ± 0.08**	20.33 ± 1.06**	0.36 ± 0.03**	2.89 ± 0.16**
a	−0.37 ± 0.14*	−0.85 ± 1.80	−0.16 ± 0.05**	0.10 ± 0.27
d	−0.55 ± 0.44	−12.29 ± 5.58*	−0.05 ± 0.16	−2.44 ± 0.84**
aa	−0.96 ± 0.40*	−11.61 ± 5.12*	−0.18 ± 0.15	−2.14 ± 0.77**
ad	−0.35 ± 0.18†	5.91 ± 2.20**	−0.12 ± 0.07†	1.17 ± 0.33**
dd	0.59 ± 0.73	16.29 ± 9.19†	−0.22 ± 0.27	1.91 ± 1.38
Cross 2 (Auburn 612-RNR × Deltapine 41)				
m	2.11 ± 0.24**	7.68 ± 0.71**	0.29 ± 0.04**	1.10 ± 0.11**
a	−0.04 ± 0.41	−2.40 ± 1.23†	−0.04 ± 0.08	−0.20 ± 0.19
d	0.06 ± 1.26	5.32 ± 3.78	0.32 ± 0.23	1.09 ± 0.57†
aa	0.90 ± 1.15	1.35 ± 3.47	0.54 ± 0.21*	0.75 ± 0.53
ad	0.74 ± 0.50	−0.24 ± 1.50	0.09 ± 0.09	0.17 ± 0.23
dd	0.18 ± 2.10	−4.08 ± 6.26	−0.58 ± 0.39	−1.99 ± 0.95*
Cross 3 (M 019-RNR × Deltapine 41)				
m	11.31 ± 0.60**	6.34 ± 0.57**	3.04 ± 0.19**	0.99 ± 0.09**
a	−3.61 ± 1.00**	−1.21 ± 0.98	−0.61 ± 0.32†	−0.22 ± 0.16
d	−2.25 ± 3.12	−4.91 ± 3.02	−1.91 ± 1.01†	−0.54 ± 0.50
aa	−2.59 ± 2.86	−7.11 ± 2.77*	−1.54 ± 0.93†	−0.73 ± 0.46
ad	−2.86 ± 1.25*	−1.16 ± 1.21	−0.13 ± 0.40	−0.18 ± 0.20
dd	0.77 ± 5.15	2.75 ± 5.00	1.34 ± 1.67	0.08 ± 0.83

†,*,** Significantly different from zero at the 0.10, 0.05, and 0.01 probability levels, respectively, according to *t*-test.
‡ Planting dates defined in Table 1.
§ Inoculated with eggs in PD 1 only.

duced EPGR below midparent value. Pooled *aa* gene effects were negative and significant ($P < 0.10$); other epistatic effects were nonsignificant ($P > 0.10$).

DISCUSSION

Because of the nature of the materials used in this study, it is likely that assumptions of absence of multiple alleles, absence of lethal genes, and constant viability of genotypes were met. Estimates of *a* and *d* effects are biased by epistatic effects and linkage disequilibrium (Hayman, 1960), and linkage disequilibrium is likely to occur in quantitative traits controlled by large numbers of genes (Comstock and Robinson, 1952).

The assumption of absence of genotype × environment (G × E) interaction was not realized in the material under study. Reniform nematode reproduction was poorer in the winter and summer plantings than in the spring and fall, and the generations of Crosses 1 and 2 were differentially affected by dates (Table 2). The bias caused by G × E in the estimates of gene effects is of unknown magnitude and direction and may not be the same for each parameter. There were six generations and five parameters in the model, so predicting the extent and nature of bias would be difficult. Since each experiment was divided into two blocks and grown at two planting dates, the bias due to G × E in estimating gene effects may have been reduced. However, it is realized that number of environments was minimal.

Since the susceptible parent, Dp 41, was common in all three crosses, differences in the direction of gene effects would be due to the genetics of the resistant parents. In general, the direction of *d* and *aa* effects in the cross involving La RN-910 was negative and that of *dd* effect was positive, whereas, direction of these effects was opposite in the cross involving Aub 612-RNR. Differences in direction of these gene effects suggest that La RN-910 has a different genetic mechanism for resistance to RN than Aub 612-RNR, and their combination through breeding may lead to increased resistance to RN. The M 019-RNR line did not show enough resistance to RN under our conditions to be considered a good source of resistance.

Significant epistatic gene effects in most cases and evidence of transgressive segregation for susceptibility based on frequency distribution of individual plant data by generations suggest that resistance to RN in La RN-910 and Aub 612-RNR is controlled by two or more pairs of genes.

Advancing generations to F_4 or F_5, while maintaining genetic variability prior to selection, may improve selection efficiency for RN resistance by increasing homozygosity and thereby reducing nonadditive gene effects. However, the general lack of additive effects implies difficulty in breeding for resistance to RN using germplasm lines involved in this study. Fortunately, other sources of resistance to RN are known and their use in breeding and genetic studies should be explored.

Future genetic and breeding studies on RN resistance would be enhanced by reducing environmental influence. Though we were consistent in inoculation levels of juveniles, there may have been variation in number of infectious females that invaded roots. The collection and use of RN juveniles from freshly hatched eggs or the direct use of soil that is uniformly infested with RN as inocula perhaps would be superior to the soil-extracted-juvenile technique used in this study. The latter technique may have caused injury and stimulated diseases of RN infective females. Also, the use of growth chambers or limiting greenhouse studies to the less harsh environments of spring and fall, may reduce environmental effects. Higher inoculation levels should reduce the frequency of plants that escape infection and improve the identification of resistant plants and progenies.

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