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Inheritance of Resistance to *Meloidoygne incognita* in Primitive Cotton Accessions from Mexico

J. L. Starr,[1] E. R. Moresco,[2] C. W. Smith,[3] R. L. Nichols,[4] P. A. Roberts,[5] P. Chee[6]

Abstract: Few sources of resistance to root-knot nematodes (*Meloidogyne incognita*) in upland cotton (*Gossypium hirsutum*) have been utilized to develop resistant cultivars, making this resistance vulnerable to virulence in the pathogen population. The objectives of this study were to determine the inheritance of resistance in five primitive accessions of *G. hirsutum* (TX1174, TX1440, TX2076, TX2079, and TX2107) and to determine allelic relations with the genes for resistance in the genotypes Clevewilt-6 (CW) and Wild Mexico Jack Jones (WMJJ). A half-diallel experimental design was used to create 28 populations from crosses among these seven sources of resistance and the susceptible cultivar DeltaPine 90 (DP90). Resistance to *M. incognita* was measured as eggs per g roots in the parents, F_1 and F_2 generations of each cross. The resistance in CW and WMJJ was inherited as recessive traits, as reported previously for CW, whereas the resistance in the TX accessions was inherited as a dominant trait. Chi square analysis of segregation of resistance in the F_2 was used to estimate the numbers of genes that conditioned resistance. Resistance in CW and WMJJ appeared to be a multigenic trait whereas the resistance in the TX accessions best fit either a one or two gene model. The TX accessions were screened with nine SSR markers linked to resistance loci in other cotton genotypes. The TX accessions lacked the allele amplified by SSR marker CR316 and linked to resistance in CW and other resistant genotypes derived from this source. Four of five TX genotypes lacked the amplification products from the marker BNL1231 that is also associated with the resistant allele on Chromosome 11 in WMJJ, CW, NemX, M120 RNR and Auburn 634 RNR. However, all five TX genotypes produced the same amplification products from three SSR markers linked to the resistant allele on Chromosome 14 in M120 RNR and M240 RNR. The TX accessions have unique resistance genes that are likely to be useful in efforts to develop resistant cotton cultivars with increased durability.

Key words: Allelic relationships, cotton, *Gossypium hirsutum*, host resistance, inheritance of resistance, *Meloidogyne incognita*, molecular markers, root-knot nematode.

Meloidogyne incognita is an important pathogen of upland cotton (*Gossypium hirsutum* L.) and is found in all cotton producing regions in the United States (Koenning et al., 2004; Starr et al., 2005). Damage thresholds for *M. incognita* have been estimated to be as low as 10 juveniles/500 cm^3 soil at planting (Starr et al., 1989). Management of this economically important pathogen relies primarily on the application of nematicides and crop rotation (Starr et al., 2007). However, one of the most widely used nematicides on cotton, aldicarb, is being withdrawn from the market. Use of genetic resistance would be a more cost effective and environmentally sound approach to suppression of nematode populations and to reduce yield losses. Despite the availability of several potentially useful sources of resistance since the early 1900s (Starr and Roberts, 2004; Gutiérrez et al. 2010), few high yielding cotton cultivars with resistance to root-knot nematodes have been developed. Studies using the root-knot resistant cultivars 'Acala NemX' (Ogallo et al., 1997), 'Paymaster H1560', and 'Stoneville LA887' (PI 547084; PVP 9100065; Jones, et al., 1991; Koenning et al., 2001) have demonstrated the potential for increased cotton yields in nematode-infested fields and effective suppression of the nematode population. In addition to the direct benefit of resistance to cotton in infested fields, the suppression of root-knot population densities reduces the potential for yield losses to subsequent susceptible crops (Ogallo et al. 1999).

Resistance to *M. incognita* in cotton has been studied in only a few genotypes. The resistance level varies from moderate in genotypes such as Clevewilt-6 (CW) (Miles, 1939) and Wild Mexico Jack Jones (WMJJ) (Jones et al., 1958), to near immunity in Auburn 623 RNR (which was developed from a cross between the former two genotypes) (Shepherd, 1979) and in lines developed from this source of resistance. Resistance in Auburn 623 RNR was reported to be a transgressive trait governed by one dominant and one additive gene (Shepherd, 1979), presumably one contributed by each of the parents. Subsequent studies on inheritance of resistance in genotypes derived from Auburn 623 RNR indicted that the resistance was conditioned by either two dominant genes (Zhou et al., 1999) or by one dominant and one additive gene (McPherson et al., 2004). Recent QTL analysis support these findings, and suggested that genes conferring resistance are located on Chromosome 11 and Chromosome 14 in lines derived from Auburn 623 RNR (Gutierrez et al., 2010; Shen et al. 2010). Further, the locus on Chromosome 11 appeared to be inherited from CW (Ynturi et al., 2006; Shen et al., 2006) whereas the locus on Chromosome 14 was inherited from WMJJ (Gutierrez et al., 2010; Shen et al., 2010). However, other studies suggested that the resistance in CW was either quantitatively inherited (Jones et al., 1958) or conditioned by a single recessive gene (Robinson, 1998; Zhou et al., 1999; Bezawada et al., 2003). The resistance in Acala NemX appears to be a single recessive gene (Wang et al., 2006a,b) but when this source of resistance was crossed with the susceptible *G. barbadense* Pima S-7, there was strong evidence of transgressive segregation, indicating that the susceptible parent was contributing gene(s)

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[1]Dept. Plant Pathology and Microbiology, Texas AgriLife Research, College Station, TX 77843-2132.
[2]Mato Grosso Cotton Institue, Cuiaba-MT, 78008-000, Brazil.
[3]Dept. Soil and Crop Sciences, Texas AgriLife Research, College Station, TX 77843-2474.
[4]Cotton Incorporated, 6399 Weston Parkway, Cary, North Carolina 27513.
[5]Dept. Nematology, University of California, Riverside, CA 92521-0415.
[6]Cotton Molecular Breeding Laboratory, University of Georgia, Tifton, GA 31793.
Email: j-starr@tamu.edu
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that enhanced the resistance observed in the progeny (Wang et al., 2008). A similar transgressive effect producing highly resistant F2 progeny was found in the intraspecific cross Acala NemX x WMJJ (Ulloa et al., 2010). There are no other reports that provide evidence regarding inheritance of resistance inWMJJ.

Although resistance to *M. incognita* has been deployed on a limited basis, there is evidence of virulence in some nematode populations to those sources of resistance. During the breeding efforts to develop the resistant cultivar Acala NemX, increased reproduction by a population of *M. incognita* isolated from fields repeatedly planted to genotypes carrying this source of resistance relative to nematode populations not repeatedly exposed to the resistance genes was observed (Ogallo et al., 1997). In two separate studies (Elliot et al., 1998; Zhou, et al., 2000) a few populations of *M. incognita* collected from cotton fields that had not been planted to resistant cotton cultivars were identified with apparent virulence on the resistance genotypes LA887, M315 RNR, and (or) Acala NemX. Collectively, these observations suggest that the resistance currently most widely used in breeding programs may lack durability. Thus, there is a need to identify additional sources of resistance that could be deployed in the event of widespread virulence against the currently used resistance.

In addition to these cotton accessions and cultivars known to be resistant to *M. incognita*, several other resistant cotton accessions have been identified (Shepherd, 1983; Robinson and Percival, 1997). No data are available regarding the inheritance of resistance in these genotypes. Our objectives were (i) to determine inheritance of resistance in five primitive cotton genotypes reported to be resistant to *M. incognita* by Robinson and Percival (1997) and later confirmed to be resistant by Faske and Starr (2009) and (ii) to test for possible allelic relationships among these genotypes and the resistance genes in CW and WMJJ.

Materials and Methods

The isolate of *M. incognita* (#98-1) that was used for all tests was a composite of 10 isolates collected from several cotton fields in west Texas and maintained on *Solanum lycopersicum* L. 'Rutgers' in a greenhouse. Species identification was confirmed by esterase and malate dehydrogenase isozyme phenotypes (Esbenshade and Triantaphyllou, 1990) and by species-specific PCR primers (Adams et al., 2006). Inoculum for all tests was eggs extracted from infected tomato using 0.6% NaOCl (Hussey and Barker, 1973).

The upland cotton genotypes used in this study were the susceptible cultivar DeltaPine 90 (DP90) and the resistant genotypes CW (PI165358), WMJJ (PI593649), and the resistant primitive accessions TX1174 (PI529971), TX1440 (PI438839), TX2076 (PI5011483), TX2079 (PI50186), and TX2107 (PI501514). Seed of all of the resistant genotypes were obtained from the USDA-ARS Crop Germplasm Research Unit in College Station, Texas (USDA National Plant Germplasm Collection, www.ars-grin.gov/cgi-bin/npgs/html/site_holding.pl?COT). Because the TX genotypes are photoperiodic, seeds of all genotypes were planted in hills at the USDA Cotton Winter Nursery at Tecoman, Colima, Mexico in the autumn of 2004 and 2005 and crosses made at that location. Likewise, seeds from the initial crosses were planted at that location to produce the F_2 generation.

Crosses were made among the eight parental lines in all combinations but without reciprocals and resulted in the development of 28 populations. For each population, the two parents (n = 10 to15 plants), the F_1 (n = 10 to 15 plants) and the F_2 generation (n = 60 to 75 plants) were screened for resistance, which was defined as inhibition of nematode reproduction relative to reproduction on a standard susceptible cotton genotype (Roberts, 2002). If both parental lines were resistant to *M. incognita* then DP90 was included in the test as the susceptible control. Each plant was inoculated with 10,000 eggs at ca. 2 wk after emergence and received an additional second inoculation of 4,000 eggs 4 wk after the first inoculation. Nematode reproduction was measured as eggs per g roots with eggs extracted from inoculated plants using NaOCl (Hussey and Barker, 1973) at ca. 8 wk after the first inoculation. F_1 populations were classified resistant if the mean number of eggs per g roots was significantly ($P < 0.05$) lower than that of the susceptible control. Individuals in the F_2 populations were classified as resistant or susceptible by comparison to the means of the resistant parent(s) and the susceptible control.

Mean numbers of eggs per g roots for parents, F_1, and the susceptible control were compared by analysis of variance using the SPSS 16.0 statistical program (SPSS Inc., Chicago, IL) with mean separations by Tukey's Honestly Significant Difference when appropriate. Segregation ratios of resistant and susceptible individuals in F_2 populations were fitted to different genetic models using Chi-square analysis.

The resistant TX genotypes were tested for allelic similarity, which may indicate sharing of a common resistance gene, by using SSR markers tightly linked to the resistance genes on Chromosome 11 (BNL1231, CIR069, CIR196, CIR316, GHACC1-CAPS, SCARP4M12) and on Chromosome 14 (CGR CGR5668, CIR381, UGT0045) in Acala NemX, CW, M120 RNR, M240 RNR, and WMJJ (Shen et al., 2006; Wang and Roberts, 2006; Wang et al., 2006b; Shen et al., 2010). DNA extraction, PCR amplifications and gel electrophoresis were performed using previously published protocols (Wang and Roberts, 2006; Shen et al., 2010).

Results

Reproduction of *M. incognita* in all 28 tests was higher on the susceptible DP90 (mean from all tests of 7,261

eggs/g roots, $P < 0.05$) than on any of the resistant parental genotypes (Fig. 1). Among the resistant cotton genotypes, nematode reproduction varied with TX1174 and TX2076 supporting less reproduction ($P < 0.05$) than the other genotypes. Reproduction of *M. incognita* on CW, WMJJ, TX1440, TX2079, and TX2109 was similar.

Mean nematode reproduction in the F_1 generation from the crosses DP90 x CW and DP90 x WMJJ were similar to or greater than reproduction on DP90 (Fig. 2). The mean nematode reproduction in the F_1 generation of all TX genotypes x DP90 was lower ($P < 0.05$) than reproduction on DP90 except for TX1440 x DP90, for which the mean of the F_1 generation was statistically similar ($P > 0.05$) to that on DP90.

Nematode reproduction was variable within the F_2 populations, ranging from 0 to > 27,000 eggs/g roots on individual plants. At least a few highly susceptible F_2 individuals (> 4,000 eggs/g roots) were observed in the progeny of all crosses for which were either DP90 or CW was a parent (Table 1). When WMJJ was crossed with the TX genotypes, susceptible F_2 individuals were observed when the other parent was TX1440 or TX 2076 but not when the other parent was TX1174, TX2079, or TX2107. Relatively few highly susceptible F_2 individuals were observed in progeny from crosses between the different TX genotypes, with highly susceptible individuals (> 4,000 eggs/g roots) being observed only in progeny from TX1440 x TX2107 and TX 2079 x TX2107.

Chi-square analysis of the segregation patterns among F_2 individuals for each cross of a resistant parent with the susceptible DP90 was used to estimate numbers of genes conditioning the resistance phenotype. Because of the near continuous variation in phenotype among the F_2 from each cross, each population was divided into a resistant and susceptible class at a point where a substantial difference in nematode reproduction was observed and that was within one standard deviation of the mean of the resistant parent. For most of the F_2 populations there were two possible ways to divide the population into resistant and susceptible individuals and each was tested separately.

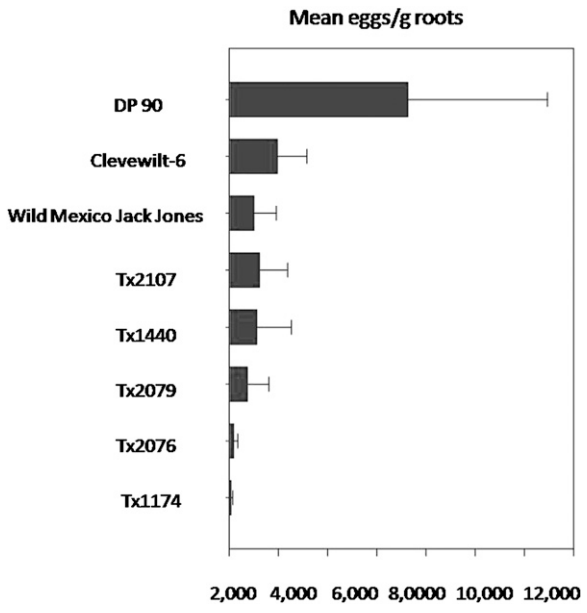

FIG. 1. Reproduction of *Meloidogyne incognita* on several cotton genotypes used as parental lines in the half-diallel test. Each value is the mean of seven separate tests with a minimum of 10 individual/test. Bars = 1 SE.

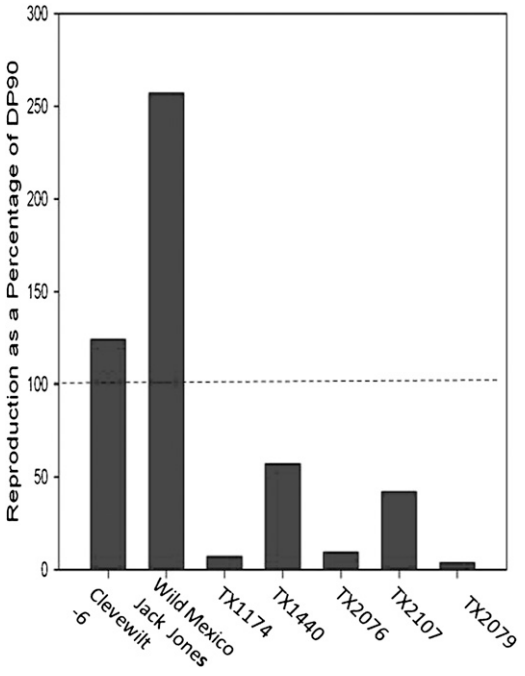

FIG. 2. Reproduction of *Meloidogyne incognita,* as a percentage of the susceptible genotype DP90, in the F_1 generation generated from crosses of seven resistant cotton genotypes with the susceptible genotype DP90.

For the crosses of DP90 x CW or DP90 x WMJJ, where the mean number of eggs per g roots of the F_1 generation plants was equal to or greater than that of DP90, it was concluded that resistance was inherited as a recessive trait. The observed segregation patterns in these two F_2 populations did not fit ($\chi^2 > 3.86$, $P < 0.05$) either a one or two recessive gene model (Table 2). Because the mean reproduction on the F_1 individuals for each of the TX genotypes, except for TX1440, was less than the reproduction on DP90, resistance was assumed to be inherited as a dominant trait. Although the mean level of reproduction in the F_1 generation for TX1440 was not significantly different from reproduction on DP90, it was numerically less than that on DP90 and therefore resistance in TX1440 was hypothesized to be dominantly inherited. Depending on which of the possible segregation patterns that were tested, inheritance of resistance in each of the TX genotypes fit either a one dominant gene (3R:1S) or a one dominant gene/one recessive gene (13R:3S) model (Table 2). Only for DP90 x TX2107 did one of the selected segregation ratios fit a two dominant gene model.

The TX genotypes were compared to other resistant cotton genotypes using a total of nine DNA markers

TABLE 1. Reproduction of *Meloidogyne incognita* on the susceptible cotton genotype DP90 and F_1 and F_2 generation individuals derived from crosses among several cotton genotypes in a half-diallel test.

Cross	Eggs/ g roots		
	DP90	F_1 generation	F_2 generation
DP90 x CW	10,462 ± 4,186	13,001 ± 10,100 NS	0 to 20,933
DP90 x WMJJ	3,576 ± 1,921	9,196 ± 4,346 *	16 to 10,055
DP90 x TX1440	3,774 ± 2,887	2,773 ± 2,286 NS	0 to 7,445
DP90 x TX1174	6,525 ± 7,457	457 ± 443 **	0 to 11,810
DP90 x TX2076	12,172 ± 13,007	1,618 ± 1,440 *	0 to 23,856
DP90 x TX2079	2,949 ± 2,509	106 ± 284 **	0 to 4,529
DP90 x T2107	4,337 ± 3,777	1,827 ± 2936 *	0 to 13,944
CW x WMJJ	7,565 ± 6,433	3,458 ± 2,165 *	0 to 24,319
CW x TX1440	10,306 ± 6.692	253 ± 337 **	0 to 27,456
CW x TX1174	14,355 ± 14,257	40 ± 2 **	0 to 12,014
CW x TX2076	1,464 ± 886	12 ± 9 **	0 to 6,890
CW x TX2079	14,649 ± 12,489	888 ± 1,289 **	0 to 7,214
CW x TX2107	6,582 ± 7,083	687 ± 750 *	0 to 4,368
WMJJ x TX1440	6,528 ± 4074	176 ± 422 **	0 to 9,370
WMJJ x TX1174	3,179 ± 2,723	258 ± 257 *	0 to 1,307
WMJJ x TX2076	5,862 ± 5,036	2,526 ± 3,060 NS	0 to 16,190
WMJJ x TX2079	4,571 ± 2,571	81 ± 46 **	16 to 1,579
WMJJ x TX2107	2,313 ± 1,358	186 ± 282 **	0 to 1,785
TX1440 x TX1174	5,433 ± 3,871	100 ± 170 **	0 to 700
TX1440 x TX2076	4,382 ± 2,971	142 ± 88 **	0 to 1,677
TX1440 x T2079	4,491 ± 5,094	92 ± 141**	0 to 198
TX1440 x TX2107	7,731 ± 5,780	2,030 ± 1,344 *	0 to 5,707
TX1174 x TX2076	3,319 ± 3,490	28 ± 29 **	0 to 322
TX1174 x TX2079	1,930 ± 1,005	5 ± 4 **	0 to 38
TX1174 x TX2107	1,156 ± 2296	20 ± 17 **	0 to 217
TX2076 x TX2079	1,256 ± 1,254	182 ± 172 *	0 to 1,446
TX2076 x TX2107	2,119 ± 768	46 ± 62 **	0 to 1,698
TX2079 x TX2107	5,699 ± 4,159	2,336 ± 1,036 *	0 to 4,950

Values for DP90 and F_1 generations are means ± one standard error whereas the values for F_2 are the range for all individuals. *, ** indicate significant differences from the susceptible DP90 at $P = 0.05$ and 0.01, respectively. NS = nonsignificant.

linked to resistance genes previously identified on Chromosome 11 and Chromosome 14. All TX genotypes tested lacked the PCR fragments amplified by the SSR marker CIR316 linked to the resistant allele on Chromosome 11 in Acala NemX, CW, M120 RNR and M240 RNR (Fig. 3A). Similarly, four of five TX genotypes lacked the PCR amplification products from the marker BNL1231 that is also associated with the resistant allele on Chromosome 11 in WMJJ, CW, NemX, M120 RNR and Auburn 634 RNR (Fig. 3B). However, all five TX genotypes carry the same PCR amplification productions from three SSR markers associated with the resistant allele on Chromosome 14 in M120 RNR and M240 RNR (Fig. 4).

DISCUSSION

These data confirm and expand on previous reports (Robinson and Percival, 1997; Faske and Starr, 2009) that the five primitive TX cotton accessions tested are resistant to *M. incognita*. Further, these data support the hypothesis that these five primitive cottons have resistance genes that are different from those present in CW, WMJJ and the numerous resistant genotypes (the Auburn and M series lines) that have been developed from these two sources of resistance. These data also confirm previous reports (Jones, 1958; Zhang et al., 1999, Bezawada et al., 2003) that resistance in CW is inherited as a recessive trait. Our observation that the resistance in WMJJ is a recessive trait is new. Although Acala NemX was not included in these tests, the resistance in that genotype is also reported to be inherited as a recessive trait (Wang et al., 2006a,b). Although resistance in each of these three genotypes (CW, Acala NemX, and WMJJ) is inherited as a recessive trait, the available data from studies with molecular markers linked to each resistance locus and with resistance segregation from combinations of crosses indicate that whereas the genes in CW and Acala NemX may be the same or in close proximity Chromosome 11 the gene from WMJJ is distinct (Bezawada et al., 2003; Shen et al., 2006, Wang and Roberts, 2006, Wang et al. 2006 ab; Niu et al., 2007; Gutiérrez et al., 2010; Roberts and Ulloa, 2010; Ulloa et al., 2010). That the resistance in each of the TX genotypes is inherited as a dominant trait is strong

TABLE 2. Inheritance of resistance in progenies of several cotton lines resistant to *Meloidogyne incognita* when crossed with the nematode-susceptible genotype DP90.

		F_2 Observed		χ^2 value for expected ratio			
Cross	F_1	R	S	1:3	1:15	3:13	7:9
DP90 x CW	S	41	19	60.1*	394*	96.8*	14.7*
DP90 x WMJJ	S	51	14	99.1*	578*	152*	31.8*
		42	23	54.4*	377*	89.5*	11.5*
				3:1	1:15	13:3	
DP90 x TX140	R	45	9	2.58	8.85*	0.328	
DP90 x TX1174	R	66	9	6.76*	4.23*	1.41	
		49	16	0.005	37.4*	9.67*	
DP90 x TX2076	R	60	9	5.26*	5.43*	1.47	
		58	11	3.02	11.1*	21.7*	
DP90 x TX2079	R	45	14	0.051	30.8*	0.960	
		35	24	7.73*	119*	18.62*	
DP90 x TX2107	R	56	14	5.75*	0.178	5.75*	
		50	10	0.171	11.11*	0.171	

* indicates the observed ratio of differs ($P < 0.05$) from the expected value based on χ^2 analysis.

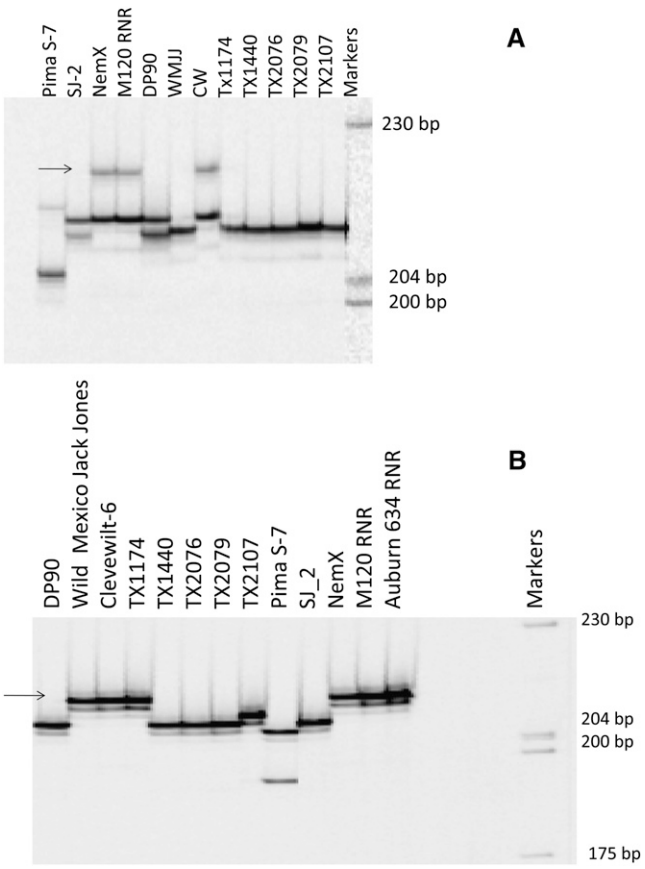

FIG. 3. PCR amplification products from genomic DNA from several *Meloidogyne incognita*-resistant and susceptible cotton genotypes using the primers for SSR markers A) CIR316, and B) BNL1231 that are linked to a resistance allele on Chromosome 11. Arrows indicate alleles associated with resistance in some resistant genotypes.

evidence that the TX genotypes possess one or more unique resistance genes.

Further evidence for the unique nature of the resistance in the TX genotypes is the observation that they all lack the allele for the three SSR markers (CIR316, CIR069, and SCARP4M12) tightly linked to the resistance gene on Chromosome 11. Further, the marker BNL1231, which is also linked to the resistance on Chromosome 11, was polymorphic among the TX genotypes. Although the absence of the alleles linked to resistance in other genotypes could be due to various recombination events, their absence is consistent with the resistance being due to different resistance genes located elsewhere in the genome.

Additional support for the unique nature of the resistance in the TX genotypes is the observation that when any of the five were crossed with the resistant CW, at least some of the F_2 generation individuals were highly susceptible. All the progeny from these crosses would be resistant if both parents carried identical genes for resistance. However, when WMJJ was crossed with each of the TX genotypes, susceptible individuals were observed in the F_2 generation only for two of the five TX genotypes (TX1440 and TX2076). The other three TX genotypes were not clearly distinct from WMJJ by this comparison. Thus by these criteria, the TX genotypes were distinct from CW but TX1174, TX 2079, and TX2107 were not clearly distinct from WMJJ.

Among the TX genotypes, TX1174 and TX2076 consistently exhibited the highest level of resistance, whereas the resistance of the other three genotypes was more moderate and similar to that of CW and WMJJ. When the segregation of resistance in the F_2 generations from the crosses among these five TX genotypes is considered, susceptible individuals were observed in only two of 10 populations, suggesting that these genotypes likely share one or more alleles with respect to the resistance genes. That no polymorphisms were observed among WMJJ and the TX genotypes for the SSR markers linked to the resistance gene on Chromosome 14 support this hypothesis. This is perhaps not surprising because the five TX genotypes and WMJJ were all collected from the same geographical region in Mexico, between 18° 03' N to 20° 23' N and 83° 22' W to 101° 36' W.

The estimates of the numbers of genes that condition resistance in the genotypes examined were equivocal. A one or two gene model fit the segregation patterns of each of the TX genotypes. The segregation ratio for CW did not fit a one gene model as proposed by others (Zhou et al., 1999; Bezawada et al.,2003) but was consistent with multiple recessive genes as first proposed by Jones (1958). In all populations tested there was evidence of transgressive segregation as there were individuals in the F_2 generation of each population that had greater resistance than either parent, consistent with previous reports (Shepherd, 1979; Wang et al., 2008; Ulloa et al, 2010).

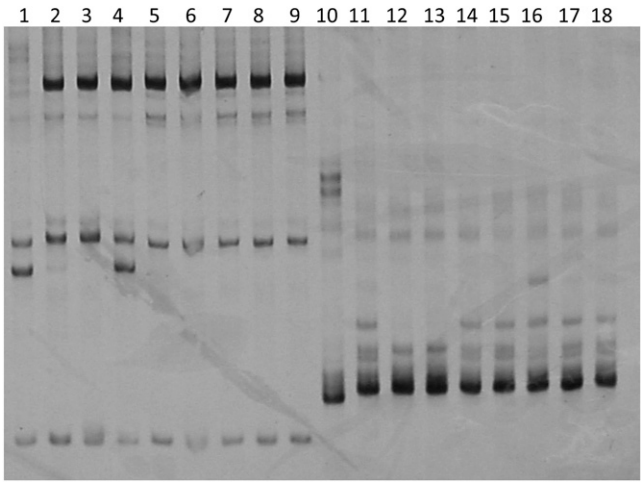

FIG. 4. PCR amplification products from genomic DNA from several *Meloidogyne incognita*-resistant and susceptible cotton genotypes using the primers for SSR markers CIR381 LN 1-9) and CGRH5668 (Ln 10-18) that are linked to a resistance allele on Chromosome 14. LN 1 & 10 = susceptible Pima S-6, LN 2 & 11 = M120 RNR, LN 3 & 12 = M240 RNR, LN 4 & 13 = susceptible Coker 201, LN 5 & 14 = TX1174, LN 6 & 15 = TX1440, LN 7 & 16 = TX2076, LN 8 & 17 = TX2079, LN 9 & 18 = TX2107.

In summary, resistance in the Texas genotypes appears to be dominant, and conditioned by a few genes that are probably different from the resistance genes currently used in most breeding programs. Because this resistance appears to be conditioned by one or two gene it should be relatively easy to introgress into modern cotton cultivars. Thus, these several sources of apparently novel resistance can serve to broaden the base of resistance available to cotton breeding programs and should contribute to durability of the resistance phenotype.

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