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Cotton Resistance to the Root Knot–Fusarium Wilt Complex. II. Relation to Root-Knot Resistance and its Implications on Breeding for Resistance[1]

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ABSTRACT

The fusarium wilt fungus, *Fusarium oxysporum* Schlect f. *vasinfectum* [(Atk.) Synd. & Hans.], and the root-knot nematode (RKN) [*Meloidogyne incognita* (Kofoid & White) Chitwood] cause a destructive disease complex in cotton (*Gossypium hirsutum* L.). This study was conducted to determine the range of genetic resistance to RKN in a group of 18 cotton cultivars and breeding lines, to evaluate the relationship between genes for resistance to fusarium wilt disease (FW) and the genes for RKN resistance, and to ascertain the relative importance of the two types of resistance in controlling FW. Numbers of RKN eggs produced on the cottons ranged from <1000/plant on Auburn 623 RNR to >148 000/plant on 'Rowden'. One-eighth as many and 18 times more RKN eggs/plant were produced on Auburn 623 RNR and Rowden in 40 days, respectively, than were used to inoculate them. No commercial cultivar was resistant to RKN in this study. Two cotton cultivars expressed genetic resistance that provided moderate field resistance to FW independently of RKN resistance. Differences in resistance to FW in the field among cotton cultivars and breeding lines were determined more by RKN resistance than by genetic resistance to FW. Results indicate that cotton can be developed with genetic resistance to FW independently of RKN resistance and that this genetic resistance probably would provide moderate field resistance to FW. High field resistance to the RKN-FW complex depends on high RKN resistance, and cotton cultivars with high RKN resistance probably would have adequate field resistance to this complex even if they were genetically susceptible to FW.

Additional index words: ***Meloidogyne incognita*** **(Kofoid & White) Chitwood,** ***Gossypium hirsutum*** **L.,** ***Fusarium oxysporum*** **Schlect f.** ***vasinfectum*** **(Atk.) Synd & Hans., Screening for resistance.**

EXTENSIVE evidence has been reported in cotton (*Gossypium hirsutum* L.) that the root-knot nematode, [*Meloidogyne incognita* (Kofoid & White) Chitwood] (RKN), greatly increases incidence and severity of fusarium wilt disease (FW), caused by *Fusarium oxysporum* Schlect f. *vasinfectum* [(Atk.) Synd. & Hans.]. Researchers began intensive efforts to develop FW resistant cultivars soon after RKN were first reported to be associated with FW in cotton in 1892 (1). These efforts continued for several decades before the importance of RKN in FW development was fully recognized (20). As more was learned about the damaging effects of RKN, both alone and in combination with FW fungus, increasing emphasis was placed on breeding for RKN resistance.

The importance of RKN in the RKN-FW complex was shown by the reduction of FW to insignificant levels when RKN were controlled by soil fumigation (14, 18, 21), or by RKN-resistant cotton (7, 18). This also was shown when RKN and FW fungus, together, caused severe wilting compared with almost no wilting by the fungus alone (11, 13, and R. Shepherd, 1975, unpublished).

It is still not known to what degree FW resistance is determined by genes for FW resistance, by genes for RKN resistance, or by a combination of both genes, because of the large effects of RKN on incidence of FW. Root-knot nematodes probably facilitate FW invasion of the vascular system and increase susceptibility to FW by inducing root-galls, giant cells, damaged tissue and general debilitation of plants. Even if genetic systems for resistance to RKN and FW are independent, high RKN resistance alone may be sufficient to prevent RKN effects that permit FW to develop and, thus, provide adequate resistance to the RKN-FW complex. In this event, it may be possible to develop cottons with resistance to the RKN-FW complex by breeding solely for high RKN resistance.

The objectives of this study were: i) to determine the range of genetic resistance to RKN in a group of cotton cultivars and breeding lines; ii) to study the relationship between genes for FW resistance and the genes for RKN resistance; and iii) to ascertain the relative importance of the two types of resistance in controlling FW. This information is needed to develop more efficient methods of breeding cotton for resistance to the two pests, and for developing more effective pest management procedures for controlling them.

MATERIALS AND METHODS

Test for FW Resistance. Nine, 15, and 10 backcrossed (BC) RKN-resistant progenies of 'Auburn 56' (Aub 56), 'Coker 201' (COK 201), and 'Stoneville 213' (Stv 213), respectively, were field tested for FW resistance. Parent cultivars and Auburn 623 RNR, the nonrecurrent parent source of RKN resistance, also were included in the test. Progenies of Aub 56 and Cok 201 were BC_2F_4 and those of Stv 213 were BC_3F_4. They were developed by selection for RKN resistance but not for FW resistance in F_2 and F_3 following each BC. This test was conducted at the same field location and used the same procedures as described previously (19). The test was conducted at Tallassee, AL, on a Wickham sandy loam (a member of the fine-loamy, mixed Thermic Typic Hapludults), which was heavily infested with both RKN and FW fungus. The test had a randomized, complete block design with six replications. Each plot was a row 1 m wide × 9.14 m long, with plants spaced 70 to 100 mm apart. Plants were rated diseased with FW if they exhibited foliar and vascular discoloration in the first week of August. In October, stems of remaining plants were cut to expose vascular tissue and examined for vascular symptoms of FW. Data on percentages of plants exhibiting FW were analyzed using arcsine transformation procedures.

Tests for RKN Resistance. Eighteen cultivars and breeding lines were evaluated for resistance to RKN egg production in a series of three greenhouse tests. Cultivars were: Bayou, Coker 100 A (Cok 100), Coker 201, (Cok 201), Coker 310 (Cok 310), Delcot 277 (Del 277), Deltapine 16 (DPL 16), Deltapine Smoothleaf (DPL SL), Dixie King II (Dix K), Empire WR 61 (Emp 61), McNair 511 (McN 511), Model, Row-

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den, and Stoneville 213 (Stv 213). Breeding lines were: Auburn 623 RNR (Aub 623), Auburn 56 line (Aub 56 L), Auburn BR-1 (Aub BR-1), Clevewilt-6, and M-8. The RKN and FW resistance of Rowden and each of the breeding lines was described previously (19). Each test was arranged in a randomized complete block design with six replications. Twelve plants per cultivar or line constituted a replication.

Procedures used for assessing RKN egg production on test plants were reported previously (17). After planting in sand, newly emerged seedlings were transplanted into 193 × 178 mm pots filled with methyl bromide-fumigated soil into which approximately 8000 RKN eggs/pot had been previously deposited. Approximately 40 days after transplanting seedlings, soil was washed from plant roots, and RKN eggs were removed from roots and counted. To collect RKN eggs, infested roots were excised from stems and placed in a 0.47 L wide-mouth glass jar. The NaOCL solution (0.14 mol L^{-1}) was added at 25°C to these roots at the rate of approximately 35 L kg^{-1} root and the jar was sealed with a lid. Roots were shaken at 180 cycles/min for 4 min with a laboratory shaker to disperse eggs in egg masses attached to roots. The NaOCL solution with suspended eggs was poured into a 75 μm-mesh sieve nested over a 25 μm-mesh sieve. Eggs were collected on the 25 μm-mesh sieve, washed in flowing tap water for approximately 20 s and placed in a known volume of water in a sample bottle. To facilitate egg counting, samples were diluted 1:10 serially until they contained about 5×10^4 eggs/L. Eggs in three aliquots (each containing 10^{-3} L) per sample were counted under ×30 magnification with a binocular microscope, and counts were averaged. This average was used to calculate eggs per plant. Counts of eggs per plant were transformed to $\log_{10}$ for variance analysis.

Source of FW Data. Percentages of plants of 18 cotton cultivars and breeding lines grown in a greenhouse and exhibiting FW symptoms from vascular inoculation with spores of the fungus and from field tests of 18 cotton cultivars and lines grown in soils heavily infested with RKN and FW fungus were reported previously (19). These percentages were used to determine correlations of percentages of FW in a greenhouse and field with RKN reproduction.

RESULTS AND DISCUSSION

Test for FW Resistance. Mean percentages of plants with FW were significantly less in RKN resistant backcross-derived progenies grown in a RKN and FW infested nursery than in their RKN susceptible recurrent parent cultivars. The FW percentages were: 4% in 9 progenies of Aub 56 vs. 11% in Aub 56, 10% in 15 progenies of Cok 201 vs. 62% in Cok 201, and 12% in 10 progenies of Stv 213 vs. 72% in Stv 213. Thus, approximately one-third, one-sixth, and one-sixth as many plants developed FW symptoms, hereafter referred to as field FW, in the progenies as in the Aub 56, Cok 201, and Stv 213 recurrent parents, respectively. The progenies were developed by selecting only for RKN resistance. The lower wilting in the BC progenies probably resulted from their higher RKN resistance. This was suggested by BC progenies of Aub 56 and Cok 201 theoretically containing approximately 87% and BC progenies of Stv 213 approximately 93% of their recurrent parents' germplasm, which was FW susceptible.

Tests for RKN Resistance. Root-knot nematode eggs produced on the 18 cotton entries tested ranged from < 1000 RKN eggs/plant on Aub 623 to > 148 000 on Rowden (Table 1). Five different levels of RKN resistance were exhibited. Aub 623 had the highest level of resistance, with only about one-eighth as many RKN eggs produced on it as were used to inoculate the cotton. In comparison, about 18 times more eggs were produced on highly susceptible Rowden than were used to inoculate it. All widely grown cultivars screened in this study and in numerous previous studies (R.L. Shepherd, 1978, unpublished) have ranged from susceptible to highly susceptible to RKN reproduction.

Root-knot nematode resistance in cotton appears related to the nematode's failure to induce production of galls and giant cells in roots that would enable the nematode to mature and reproduce. An increased rate of terpenoid aldehyde production has been reported as a probable mechanism of RKN resistance in cotton (23).

Relationship of Field and Greenhouse Wilting to RKN Resistance. In a previous study (19) variation in resistance to FW in a greenhouse (hereafter referred to as greenhouse FW) accounted for 55, 38, and 29% of the variation in field FW in three series of field tests, respectively. Using data from the same tests, 71, 53, and 48% of the variation in field FW, respectively, was accounted for by variation in RKN resistance in this study (Table 2). Thus, variation in RKN resistance accounted for more variation in field resistance to FW among test cotton entries than was accounted for by variation in resistance to greenhouse FW. Field re-

Table 1. Mean numbers of root-knot nematode (RKN) eggs produced per plant on 18 cottons in three greenhouse tests.

Cultivar or breeding line	1000 RKN eggs/plant†
	no.
Auburn 623 RNR	1a*
Clevewilt-6	12b
Auburn BR-1	26bc
Bayou	29c
McNair 511	56d
Model	58d
Coker 100A	73de
Auburn 56 Line	74def
Stoneville 213	96efg
Deltapine 16	105efg
Empire WR61	122fg
Coker 310	127fg
Coker 201	130fg
Deltapine Smoothleaf	131fg
Dixie King II	135fg
M-8	141g
Delcot 277	144g
Rowden	148g

* Means within a column followed by the same letter were not significantly different at the 0.05 probability level, according to Duncan's Multiple Range Test.

† $\log_{10}$ of eggs/plant counts were used for the analysis of variance.

Table 2. Coefficients of determination (R^2), intercepts (a), and regression coefficients (b) for the regression of fusarium wilt disease (FW) percentages from four series of tests on numbers of root-knot nematode eggs per plant.†

Test series					
Designation	Tests	Entries	R^2	a	b
	no.				
Field 1	5	10	0.71	21.50	0.47**
Field 2	3	18	0.53	13.04	0.39**
Field 3	3–9	15	0.48	23.89	0.29**
Greenhouse	3	18	0.03	49.31	0.05 NS

** Significant at the 0.01 level.

† FW percentages were determined previously (19).

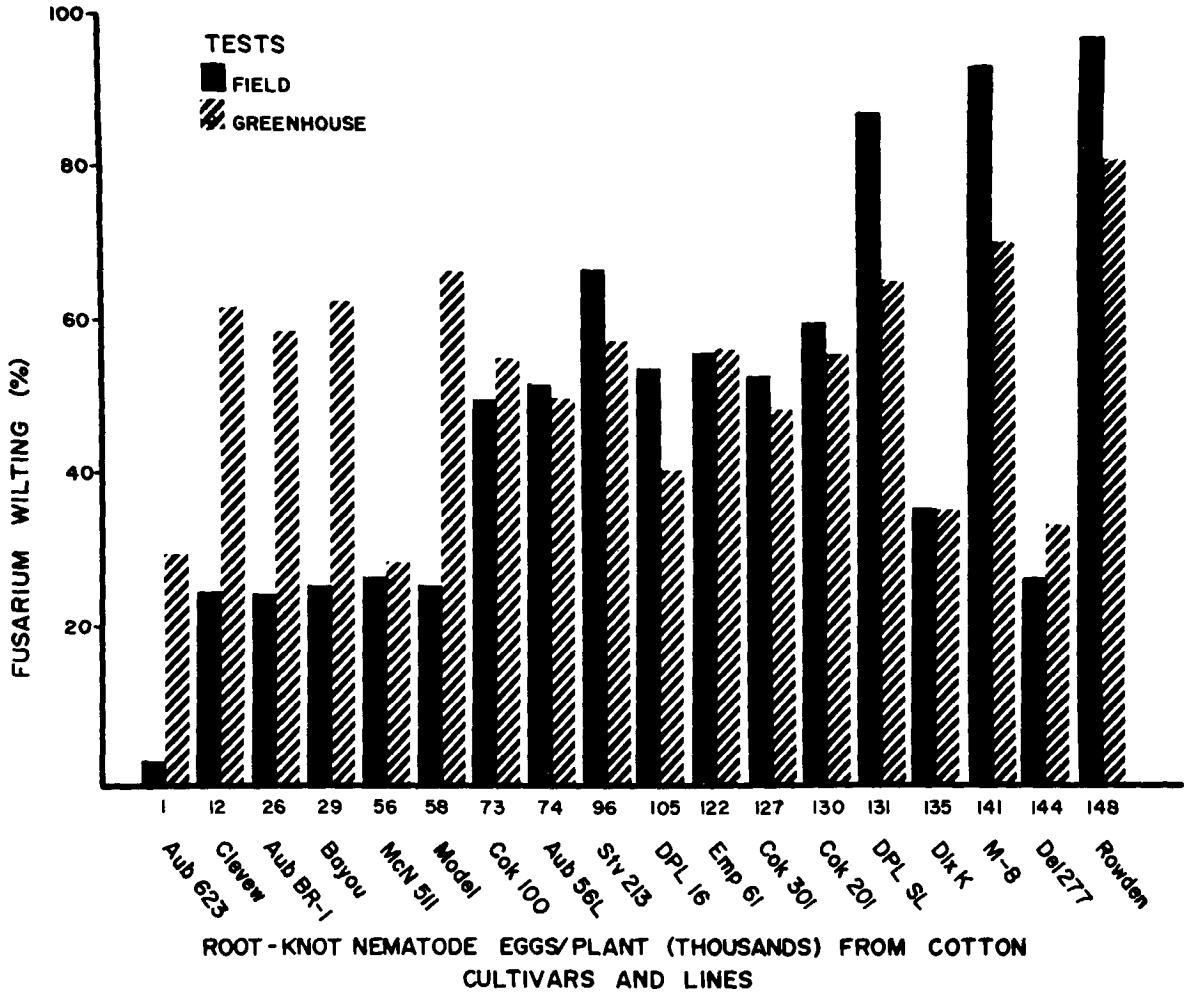

Fig. 1. Relationship of fusarium wilt disease (%) of 18 cottons grown in greenhouse and field tests to numbers of root-knot nematode eggs produced per plant in a greenhouse.

sistance to FW apparently was determined more by RKN resistance than by vascular resistance to FW. An example of the magnitude of change in field resistance to FW among cotton entries tested is shown by the regression of wilting percentages in Field Test Series 2 on RKN egg numbers ($Y = 13.04 + 0.39X$) (Table 2). This indicates that field FW decreased 1% for each decrease of 390 RKN egg number. These results agree with previous reports that RKN resistance was more important than FW resistance in controlling the RKN-FW disease complex of cotton (7, 16).

The relationship of incidence of FW in greenhouse and field grown plants to RKN reproduction is shown in Fig. 1. The FW percentages were obtained from data on vascular inoculation in the series of three greenhouse tests and from Field Test Series no. 2 in a previous study (19).

As shown in Fig. 1, cotton entries with highest RKN resistance in this study generally had highest field resistance to FW. These cotton entries varied widely in vascular resistance to FW, as indicated by their variation in greenhouse FW. They also had higher greenhouse FW than field FW. Field resistance to FW of Clevew, Aub BR-1, Bayou, and Model resulted mainly from their RKN resistance. This was indicated by their high incidence of greenhouse FW (low vascular resistance to FW) while being among the most RKN resistant of cotton entries tested. Field resistance to FW in Aub 623 and McN 511 probably resulted from both RKN resistance and vascular resistance to FW. The higher RKN resistance of Aub 623 provided it with much higher field resistance to FW than in McN 511.

Cotton entries with intermediate levels of RKN resistance had relatively high field and greenhouse FW percentages and the smallest differences between them.

Cotton entries with lowest RKN resistance had higher field FW than greenhouse FW. They had the highest susceptibility to field FW of cotton entries tested except Del 277 and Dix K (Fig. 1). The combination of moderate field resistance to FW and high RKN susceptibility in Del 277 and Dix K was rarely observed in previous tests of numerous cotton accessions (R.L. Shepherd, 1979, unpublished). This combination indicates that genes for FW resistance in Dix K and Del 277 were genetically independent of those for RKN resistance. Independence of these genes was also indicated by the nonsignificant correlation between wilting percentages resulting from vascular inoculation in a previous study (19) and numbers of RKN eggs/plant in this study ($r = 0.17$, NS).

The results in the present study agree with a pre-

vious report that field resistance to FW may result from one or more possible mechanisms of resistance, including prevascular and vascular mechanisms (22). Field resistance also may result from mechanisms in roots such as root-knot resistance. prevascular mechanisms of resistance may contribute to field resistance to FW, but no cotton in this study exhibited field resistance to FW that could be attributed to such mechanisms. Existence of prevascular mechanisms that act independently of either vascular mechanisms, RKN resistance, or both, was not indicated in this study. Existence of prevascular mechanisms would have been indicated if any cotton studied had exhibited even moderate field resistance to FW combined with both vascular susceptibility to FW and RKN susceptibility (Fig. 1).

Effects of RKN on Incidence of FW. Root-knot nematodes in the field probably facilitated FW invasion of the vascular system of RKN-susceptible cotton plants. Root-knot nematodes probably further increased susceptibility of these cotton plants to FW by inducing root galls, giant cells, and damaged tissue. These abnormal cells are produced in response to feeding by the nematode. Many substances including free amino acids, amides, nucleic acids, N, P, and auxins (probably including indoleacetic acid), significantly increased in the galls and giant cells compared with normal xylem cells, (15, 24). This may explain the findings of many researchers that galls, and particularly giant cells, are a highly favorable substrate for robust fungal growth, compared with contents of normal xylem cells. Normal xylem cells support only weak, slow fungal growth (12).

Garrett (5) reported that vascular-wilt fungi must have a source of energy for developing a threshold level of inoculum potential before it can overpower a plant's innate defenses and spread systemically. Galls and giant cells in susceptible cotton may provide the fungus with much of the energy it needs for this purpose. With this assumption, the more galls, giant cells, and damaged tissue that are induced by RKN in cotton roots, the more energy the fungus would have for invading the plant, overcoming its innate defenses, and causing symptoms of FW. Conversely, with greater RKN resistance, numbers of galls and giant cells and the incidence of wilting will be lower. Root-knot nematodes produce a general debilitating effect on cotton that also may aid the fungus in overcoming the cotton's innate resistance. These RKN effects could explain the high correlation between field resistance to FW and RKN resistance reported herein.

Potential Use of RKN Resistance to Control FW. The potential of resistant cotton to eliminate RKN in the soil, and, consequently, to prevent them from predisposing cotton to FW, was indicated in this study. This was shown by many times fewer RKN eggs/plant being recovered from RKN-resistant Aub 623 than were used to inoculate it (Table 1). This potential also was indicated in a previous study (18) in which RKN were reduced to extremely low numbers in soil where RKN-resistant cotton was grown each year. In addition, the resistant cotton controlled RKN and FW in susceptible cotton grown subsequently on the same soil because of far fewer RKN eggs released into the soil by RKN-resistant plants. Other workers (7, 13, 14) also have reported that FW was reduced to low levels by controlling or eliminating RKN.

Evidence that genes for RKN resistance provide resistance to FW was obtained from RKN-resistant progenies of Aub 56, Cok 201, and Stv. 213. They exhibited high field resistance to FW although they were selected solely for RKN resistance by backcrossing and theoretically contained 87 to 93% of the FW-susceptible germplasm of their parent cultivars. Further evidence that RKN resistance also provided FW resistance was provided by cotton entries with highest RKN resistance. They exhibited lower field FW than greenhouse FW. In comparison, except for Del 277 and Dix K, cotton entries with lowest RKN resistance exhibited higher field FW than greenhouse FW (Fig. 1).

Another indication that genes for RKN resistance confer resistance to FW was provided by Aub BR-1. This cotton was developed by intensive selection for FW resistance in a field heavily infested with RKN and the FW fungus (8). It did not exhibit high vascular resistance to FW in the greenhouse, as expected, but it did exhibit higher RKN resistance than most of the cotton entries tested (Fig. 1).

Other nematodes, such as *Rotylenchulus reniformis* Linford & Oliveira, have been reported to predispose cotton to FW. However, all cotton cultivars and breeding lines known to be even moderately resistant to FW in combination with RKN have had equal or greater FW resistance when exposed to other nematodes.

Mechanisms whereby Genes for RKN Resistance may Provide Resistance to FW. Results of the present and previous studies suggest several ways that genes for RKN resistance may confer resistance to FW in FW susceptible cotton. Products of genes for RKN resistance in root exudates in the rhizosphere and/or inside plants may differentially induce growth of microorganisms that inhibit FW. Bell (2) reported that roots of susceptible cotton exuded more of certain amino acids into the rhizosphere, and possibly into xylem vessels, than did resistant cotton plants. This may profoundly affect rate of host colonization and wilting severity. Bird (3) suggested that cotton has a genetic potential to alter its natural symbiotic microflora unfavorable to pathogens. Nematode injury may trigger production of substances in the plant conditioned by RKN-resistance genes that provide resistance to FW. This resistance my last while effects of nematode injury persist, which coincides with the time that susceptible cotton apparently is most vulnerable to invasion by the FW fungus. In addition, some mechanisms of RKN resistance may be the same ones that provide resistance to FW. Production of phytoalexins, indoleacetic acid, and ethylene have been reported to be associated with plant injury by RKN (4, 23, 24) and colonization, or pathogenesis, by FW (6, 9, 10). Some or possibly all defensive responses to RKN injury by RKN resistant cotton could also provide resistance to FW since both pests apparently induce several similar plant responses. The RKN-resistance genes may provide resistance against FW by preventing RKN from inducing physical and/or physiological changes in roots that make the cotton susceptible to FW. Predisposition of cotton to FW has been shown by cotton plants that were highly susceptible to FW in the presence of RKN but highly resistant to FW in the absence of the nematode. The abnormal

cells induced in roots by RKN, as discussed above, probably provide the energy required by the fungus to develop the level of inoculum potential necessary for pathogenesis. Therefore, prevention of RKN effects and/or reduction in RKN numbers by RKN resistance in cotton may slow fungal development of inoculum potential to the extent that innate host defenses can block its systemic advance, even in the cotton plants that are genetically susceptible to FW.

Accumulated evidence presented from this study and previous reports indicate that genes for RKN resistance confer significant resistance to FW in cotton. However, additional research is needed to further elucidate the resistance mechanisms involved in the association of FW resistance with RKN resistance.

Implications on Breeding for Resistance. Results of this study indicate that cotton cultivars and breeding lines developed by screening for resistance to FW in fields infested with RKN and FW fungus may either contain moderate RKN resistance, as in Clevew, Aub BR-1, Bayou, and Model; moderate resistance to FW as in Dix K and Del 277; or some degree of resistance to both pests, as in Aub 623 and McN 511. Type and degree of resistance will depend on genotypes present in the initial breeding populations and the level of selection pressure applied (Fig. 1). Auburn BR-1, for example, was developed by intensive field selection for resistance to FW. It is susceptible to vascular wilting but is moderately resistant to RKN, which apparently resulted from correlated response to field selection for FW resistance.

Results indicate that genes in Del 277 and Dix K for resistance to FW exist independently of those for RKN resistance. However, there is no evidence that genetic resistance to FW in either prevascular or vascular phases would provide any more than the moderate levels of field resistance to FW without RKN resistance also being present.

Results of this study further indicate that plants with genes for high levels of RKN resistance would resist RKN, and in addition benefit subsequently grown susceptible crops by depressing RKN populations to low levels in the soil. In areas where RKN levels are high, genetically RKN resistant plants probably would possess higher field resistance to FW than plants with the genes for FW resistance only.

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