Segregation for Stem Canker resistance in Southern Soybean crosses

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Abstract

Stem Canker caused by *Diaporthe phaseolorum* (Cooke & Ellis) Sacc. f. sp. *meridionalis* Morgan-Jones (Dpm) can cause significant yield losses in soybean [*Glycine max* (L.) Merr.] in the southern USA. Some cultivars are resistant. The parental donor of resistance is unknown for many cultivars, therefore it is not known which genes are the most common in the elite germplasm base. Progeny from 44 resistant X resistant soybean genetic populations were evaluated for their reaction to the fungus causing stem canker disease. Susceptible progeny were identified in only 8 populations. The frequency of susceptible plants in those was low. This indicates that few loci are represented in the current elite breeding gene pool. An implication from the study is that breeders can delay testing for stem canker resistance until very late phases of development if both parents are resistant.

Introduction

Stem canker, caused by *Diaporthe phaseolorum* (Cooke & Ellis) Sacc. f. sp. *meridionalis* Morgan-Jones (Smith and Backman, 1989) can cause significant soybean crop losses in the southern USA (Sciumbato, 1993). Plants are usually infected early in the season, but symptoms do not appear for several weeks. Symptoms in susceptible plants are external dark colored lesions on stems, followed by an interveinal chlorosis and necrosis of leaves. Yield reductions due to stem canker can be very high depending on susceptibility of cultivars and time of infection (Backman et al., 1985).

Many cultivars presently available to growers are resistant to stem canker (White et al., 1998) and use of resistant cultivars is an effective means of controlling the disease. Four genes conferring resistance, *Rdc1-4*, have been described (Bowers et al., 1993; Kilen and Hartwig, 1995). The parental donor of resistance is unknown for many cultivars, therefore it is not known which of these genes, if any, are the most common in the elite germplasm base. Knowledge of allelic

relationships among genes in the breeding pool would aid breeders in designing mating schemes and subsequent selection protocol. This information might also reveal level of genetic diversity in elite soybean germplasm for stem canker resistance. This in turn would either ease or raise concerns about genetic vulnerability for this trait.

The objective of this research was to evaluate segregation for stem canker resistance within soybean populations derived from two resistant parents.

Materials and Methods

A total of 30 lines were used in the study (Table 1). All lines were known to be resistant based on data from previous studies (White et al, 1998; Tyler, 1998). Based on previous evaluations, inheritance of resistance in these lines was determined to be monogenic (unpublished data, 1998). Crosses were made in the field in 1993, 1994, 1995, and 1996 at Stoneville, MS to generate 44 genetic populations for the allelism studies. F1 plants were grown in a greenhouse the following winter, and each population was advanced from F2 to F5 generation by single seed descent. A variable number of F6 plants from each cross were evaluated for reaction to stem canker. Seed from these crosses and seed of susceptible checks 'Hartwig' (Anand, 1993) and 'S59-60'; and resistant check 'Hutcheson', were planted in the field. Forty d after emergence plants were inoculated using the toothpick inoculation technique (Keeling, 1982). The stem canker fungus used to colonize the toothpicks was isolated from soybean plant debris collected on a farm near Hattiesburg, MS in 1986, designated isolate 86-26. Plants were evaluated for their reaction to stem canker from 67 to 84 d after inoculation when symptoms of susceptibility were fully expressed in the susceptible checks. Plants were evaluated by noting the presence or absence of external lesions at the toothpick puncture point. Dead plants or plants with external lesions of any size were rated as susceptible (Bowers et al., 1993), whereas plants lacking external lesions were rated as resistant.

Results and Discussion

In each year response of susceptible and resistant checks indicated that the inoculation procedure was effective (data not shown). Evidence of segregation in the populations would indicate that parents carry different genes for resistance. In all documented genetic studies stem canker has been conditioned by a single dominant gene (Bowers et al., 1993; Kilen and Hartwig, 1995; Tyler, 1996). In a cross segregating for two dominant unlinked genes the expected ratio of resistant to susceptible lines would approach 3:1 as increasing generations of selfing reduce dominance masking. Among the 44 populations only 8 showed segregation for stem canker reaction (Table 1). This indicates that for 36 combinations, resistance is governed at a single locus. For many of those parents resistance is known or presumed to be derived from 'York'. This resistance is carried by Hutcheson and 'DP415', two frequently used parents.

'RA452' carries resistance apparently derived from 'Williams', as its other parent 'Essex' is highly susceptible to stem canker.

The response of progeny derived from 'Dixie 478' X Hutcheson suggest that Dixie 478 carries a different gene from that donated by York. There is however an inconsistency in that the gene carried by Dixie 478 is allelic with that in DP3478, but there was no segregation in the Hutcheson x DP3478 cross. There is a similar discrepancy with Manokin. There was no segregation in the Hutcheson x Manokin cross or the RA452 x Hutcheson cross suggesting that those lines carried the same gene. However in the RA452 x Manokin cross there was segregation. This disagreement is not easily explained. Segregation within a parent could account for the occurrence of susceptible progeny, however resistant to susceptible ratios should be approaching 1:1 at F6 in a cross segregating for one dominant gene. The ratios in the above mentioned crosses showed a poor fit to a 1:1 ratio. This apparent discrepancy could occur if one of the resistant parents was segregating for two or more genes. Another explanation is that in certain backgrounds the resistance gene is not expressed. All lines that lack a major resistance gene do not show the same level of disease development and perhaps similarly all lines having a major gene do not express the same level of resistance.

The large number of crosses not segregating for stem canker resistance indicate heavy representation at one locus. The crosses selected for the study were not necessarily intended to represent the full range of genetic diversity available in the Southern elite gene pool. There has been no report of race development in *D. phaseolorum,* (Tyler, 1996; G.L. Sciumbato, personal communication, 1999) i.e., all resistant lines have shown resistance to all isolates tested. Results of this study suggest that the Southern elite gene pool could be genetically vulnerable if pathogenic races develop.

Another implication from the study is that breeders can delay testing for stem canker resistance until very late phases of development if both parents are resistant because of the rarity of segregating crosses and low frequency of susceptible types within segregating crosses.

References

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TABLE 1. Response of F6 plants from 44 soybean crosses to stem canker.

PEDIGREE	NUMBER OF PLANTS	
	RESISTANT	SUSCEPTIBLE
Avery X DP3589	44	0
D91-4619 X DP3588	45	0
D91-4619 X V91-3036	29	0
DELSOY 5500 X D91-4619	24	0
DELSOY 5500 X DP3588	22	4
DIXIE 478 X DP3478	26	0
DIXIE 478 X DT94-3866	27	0
DP3588 X LA88-25723	11	0
DP3588 X S46-44	28	0
DP415 X HS89-3261	54	4
DT94-1803 X V91-3036	30	0
DT94-2155 X V91-3036	24	0

H5088 X S57-11	14	2
H5088 X DP3588	42	0
H5088 X DP3589	16	0
HBK 49 X DP3589	66	0
HBK 49 X Hutcheson	10	0
HBK 49 X Manokin	28	0
Holladay X DP3589	42	0
Hutcheson X DIXIE 478	22	2
Hutcheson X DP3478	12	0
Hutcheson X DP3588	29	0
Hutcheson X DP3589	49	0
Hutcheson X H5088	22	0
Hutcheson X Manokin	34	0
Hutcheson X PI371612	83	0
Hutcheson X S46-44	27	0
KY91-1352 X DP3588	27	0
Manokin X DP3589	88	0
N90-516 X DIXIE 478	21	4
N90-516 X DP3588	40	0
N90-516 X H5088	21	0
N90-516 X UARK-5798	21	0
PI371612 X DP3478	20	13
PI371612 X Manokin	28	4
PI398469 X DP3589	11	0
RA452 X DP3478	22	0
RA452 X DP3589	85	0
RA452 X DT94-3863	24	0
RA452 X DT94-3866	28	0
RA452 X Hutcheson	43	0

RA452 X Manokin	31	5
V90-1012 X DP3588	48	0
V91-2935 X D91-4619	24	0