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# Soybean Stem Canker: An Emerging Disease Problem

Stem canker disease of soybean (*Glycine max* (L.) Merr.) is one of a group of diseases caused by fungi of the *Diaporthe/Phomopsis* complex. Stem canker is induced by *Diaporthe phaseolorum* (Cke. & Ell.) Sacc. var. *caulivora* Ath. & Cald. (*Dpc*), which is related to *D. phaseolorum* var. *sojae* Wehm. (*Dps*), the causal organism of pod and stem blight. Both *Dpc* and *Dps* have *Phomopsis* asexual states (Fig. 1). A *Phomopsis* not known to be connected with a sexual state also occurs on soybeans. This separate *Phomopsis* and *Dps* are important in seed decay. Although *Dpc* also reduces seed quality, it has become of major significance during the last 5 years because of its ability to kill the soybean plant well before harvest. Losses in the southeastern United States were estimated at $37 million in 1983, with many legal actions initiated because of alleged spread by seed to noninfested fields. We are directing this article primarily to an understanding of the southern soybean stem canker but will draw heavily on research conducted on the northern strains.

## History

Stem canker was originally described in Iowa in the late 1940s and the causal fungus identified as *D. phaseolorum* var. *batatatis*. Later, Athow and Caldwell (2) established the name *D. phaseolorum* var. *caulivora* for it. Symptoms (Fig. 2) begin as brick red lesions on the stems, usually at the nodes. As the disease develops, the lesion darkens, elongates, and becomes a sunken canker that often girdles the stem. Leaf symptoms appear as interveinal chlorosis and necrosis, followed by plant death with dead leaves retained. Multiple infections are common in southern stem canker but rare in northern. The disease was prevalent in the upper Midwest during the late 1940s and early 1950s but diminished in importance when the susceptible cultivars Hawkeye and Blackhawk were eliminated from production. Since then, stem canker has occurred sporadically but with little impact on yield. However, that the pathogen is still endemic in the upper Midwest can be deduced from the frequent reports of *Dpc* when seed are cultured.

During the past 10 years, significant stem canker damage to soybeans has been reported in the southern United States. The earliest observations in Mississippi in 1973 were followed by reports from Alabama in 1977, Tennessee in 1981, South Carolina and Georgia in 1982, Florida, Louisiana, and Arkansas in 1983, and Texas in 1984. In all these states, plantings of some cultivars were almost destroyed (seed yields $<100$ kg/ha), whereas other cultivars either were unaffected or were damaged at intermediate levels. In 1983, 80% of the fields in one Florida county (Escambia)—but $<2\%$ of the fields in adjacent counties—were affected. These reports suggest that major differences in severity are epidemiologically based and controlled by factors poorly identified at best. The recent report by Keeling (8) of six races of *Dpc* indicates its potential for further adaptation and spread into previously unaffected areas.

## Mycology

Within *D. phaseolorum*, four varieties, based in large part on host-parasite association, have been described. These are *phaseolorum* on lima beans, *batatatis* on sweet potato, and *caulivora* and *sojae* on soybean. Because these varieties differ very little in morphology, they are no longer accepted as valid taxonomic entities. Similarly, *D. phaseolorum, D. batatatis*, and *D. sojae*, originally described as separate species, are now considered to be the same.

Var. *caulivora* was differentiated from var. *sojae* by Athow and Caldwell (2) on the basis of the absence of an associated anamorph, occurrence of perithecia in caespitose clusters rather than singly, possession of shorter and more tapering perithecial beaks, and smaller asci and ascospores. Welch and Gilman (18) had

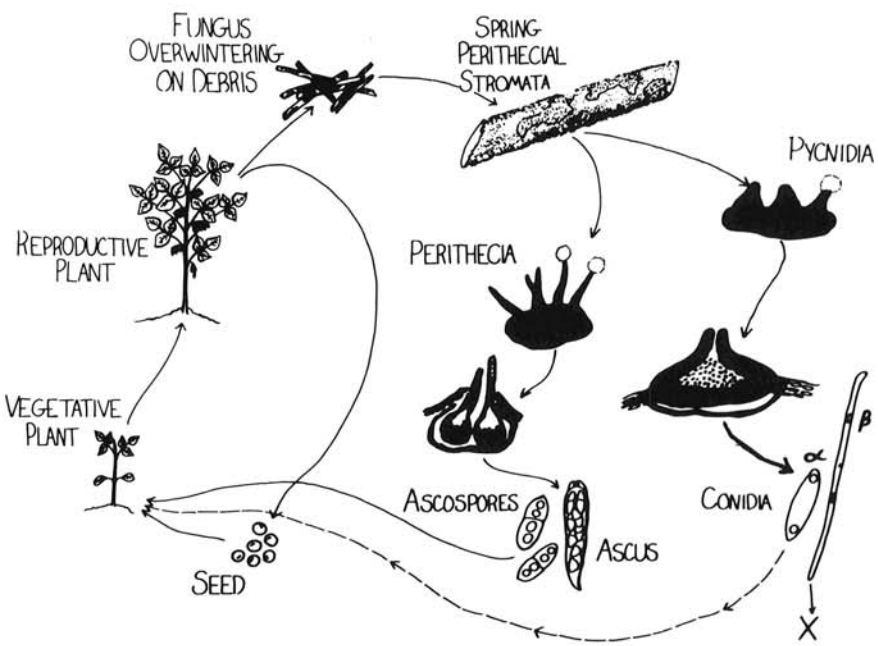


**Fig. 1. Disease cycle of *Diaporthe phaseolorum* var. *caulivora* causing stem canker of soybeans. The broken line indicates a spore of unknown epidemiologic importance, and the X indicates a spore with no known function.**

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previously found isolates from soybean cankers to differ from var. *sojae* in pathogenicity and symptoms, homothallism, caespitose perithecia, and absence of pycnidia. Hildebrand (5) noted that occurrence of perithecia of var. *caulivora* in caespitose clusters was not constant, and both he and Frosheiser (4) found pycnidia in isolates from soybean stem canker.

There appear to be insufficient grounds for maintenance of var. *caulivora* as a separate taxonomic entity. The strains associated with stem canker can, however, be consistently distinguished from others of *D. phaseolorum*. To accommodate this fact, Kulik (13) has advocated that the strains causing soybean stem canker be referred to as forma specialis *caulivora* to indicate association with a distinct disease condition. This recognizes that the distinction mainly involves physiological reaction, although this may be accompanied by slight morphological differences, especially in cultural characteristics in vitro (Fig. 3).

We have observed cultural and morphological differences between northern and southern biotypes. Overall colony surface appearance, stromata size, perithecial beak morphology, ascospore shape, conidiophore branching, production of alpha or beta conidia predominantly, and radial growth rate at 30 C are all criteria where stable discontinuities exist among isolates from different geographic origins. Our results indicate that observed differences, together with differing physiological capacity to induce stem canker, might justify separation of northern and southern races into separate forma speciales of *D. phaseolorum*.

## Races

Keeling (9) has compared *Dpc* isolates from across the United States for their pathogenicity on various soybean cultivars. He suggests there are six physiological races that can be differentiated by the cultivars Kingwa, Tracy-M, Arksoy, Centennial, S-100, and J77-339, with northern isolates classified as race 4, 5, or 6 and southern isolates as race 1, 2, or 3. Keeling, using the toothpick inoculation method, also observed that some northern cultivars were susceptible to southern isolates.

## Epidemiology

Long-distance movement of *Dpc* has been of particular concern because epidemics of the disease have occurred in the southeastern United States. There is ample evidence that the organism can be found on seed. Although seed from even the most severely affected southern fields typically does not exceed 5% detectable infestation, the northern stem canker organism has been reported at frequencies exceeding 20% of seed from infected plants (7). Numerous agronomists and plant pathologists working in the South have observed major differences in stem canker severity when comparing two seed lots of the same cultivar, planted side by side on the same day, on land with no history of stem canker. This indicates that seed are probably involved in disease spread.

The efficiency of spread by seed is a very controversial subject. Many papers indicate that all members of the *Diaporthe/Phomopsis* complex greatly reduce seed germination (11,15). Even with low percentages of infested seed and their reduced germinability, severe cases of stem canker have developed with seed as the only apparent inoculum source. This is supported by observations of clusters of diseased plants in these same fields and may indicate possible secondary disease cycles originating from primary inoculum introduced on seed. Soybean plants grown from the same seed lot planted at different locations on noninfested land develop levels of stem canker ranging from 0 to 100%. Although this observation supports the hypothesis that the seedborne fungus infects the plant through a secondary cycle and not by direct penetration from the cotyledon, no physical evidence of a secondary cycle originating from seed (i.e., pycnidia or perithecia formed on shed cotyledons) has been reported. Typically, neither pycnidia nor perithecia are observed on infected plants during the summer season, although Krausz and Fortnum (12) and Hildebrand (6) have observed perithecia during the growing season. Our observations indicate that perithecia usually develop during late winter (February–March) in the southeastern states, and that ascospore release typically begins during late April and continues into June (Fig. 4). These are the spores responsible for primary infections.

**Table 1.** Pretransformed arc sine scale for assessing severity of stem canker in soybean fields

| Score | Description (per 30-m row) |
|---|---|
| 0.0 | No disease |
| 0.2 | One plant dead or dying |
| 0.5 | Three plants dead or dying |
| 0.7 | Seven plants dead or dying |
| 1.0 | 10% of plants dead or dying |
| 1.3 | 15% of plants dead or dying |
| 1.5 | 20% of plants dead or dying |
| 2.0 | 35% of plants dead or dying |
| 2.4 | 45% of plants dead or dying |
| 2.5 | 50% of plants dead or dying |
| 2.7 | 60% of plants dead or dying |
| 3.0 | 65% of plants dead or dying |
| 3.4 | 75% of plants dead or dying |
| 3.8 | 85% of plants dead or dying |
| 4.0 | 90% of plants dead or dying |
| 4.5 | 95% of plants dead or dying |
| 4.8 | Two or three live plants |
| 5.0 | All plants dead |

Since ascospores are exuded in a sticky matrix, dispersal is primarily by splashing of raindrops and windborne rain. These periods of rain not only serve for dispersal but also supply the necessary moisture for infection. The conditions necessary for infection have not been defined. Other spread, on a field-to-field basis or even on a regional basis, points to movement on debris contained in field equipment.

Athow and Caldwell (2) found that practically all natural infection occurred through the leaves and that removal of the first six trifoliolate leaves prevented stem canker development. Results of spray trials have frequently indicated that to adequately control stem canker, fungicides should be applied between two-leaf and eight-leaf stages. These facts, and evidence from our tests that disease is reduced by delayed planting, indicate that spore release and plant infection typically occur early in the crop season and, further, that spore dispersal may cease later in the season owing to exhaustion of perithecia. Our data from 1984 were a notable exception to these guidelines. During the 1984 season at our location, perithecia did not mature until late July, infections typically occurred after $V_{10}$ (10-leaf stage), and only the most susceptible cultivars showed symptoms. These observations open the door for research on prediction of spore release for more accurate timing of spray applications, similar to timing systems used for apple scab disease.

An interesting problem arises from the fact that *Dpc* can be isolated from mature soybean plants that were asymptomatic throughout the growing season. The organism can be isolated both from resistant cultivars and from susceptible cultivars infected too late in the season to produce symptoms. In both cases, debris from these plants supports the development of perithecia the next season, severely compromising the usability of cultivar rotation or disease history for predicting stem canker severity.

Several workers have examined the possibility of alternative hosts for *Dpc*. Roy and Miller (14) found *D. phaseolorum* on cotton (*Gossypium hirsutum* L.) that produced stem cankers when inoculated to soybean. Hildebrand (6) was unsuccessful in establishing infections in several weed species common to his research location in Canada. Roy, however, tentatively identified as *D. phaseolorum* a fungus isolated from weeds that caused cankers when inoculated to soybeans (*unpublished*).

## Disease Evaluation and Losses

Several systems for field evaluation of stem canker severity have been utilized by scientists working independently. The only system we have found to provide a strong correlation between disease severity and yield is a pretransformed arc


**Fig. 2. (A) Stem cankers developing at leaf node on cv. Hutton during pod-fill stage. (B) Elongate canker developing on cv. Davis during pod-fill stage. (C) Leaf symptoms typical of stem canker and other diseases that reduce vascular flow.**

**Fig. 3. Variation in cultural characteristics of *Dpc* grown on PDA.**

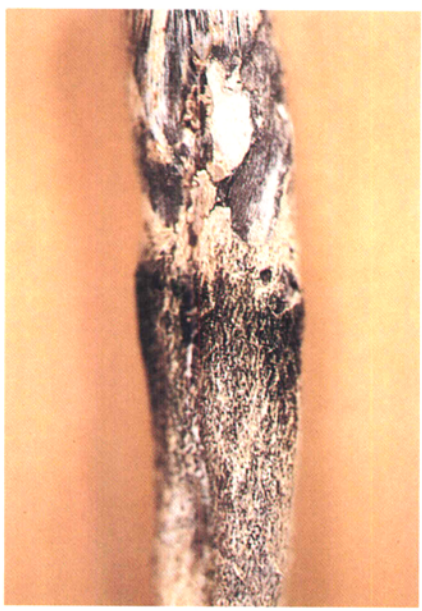

**Fig. 4. Dark perithecia forming in early spring on soybean crop debris.**

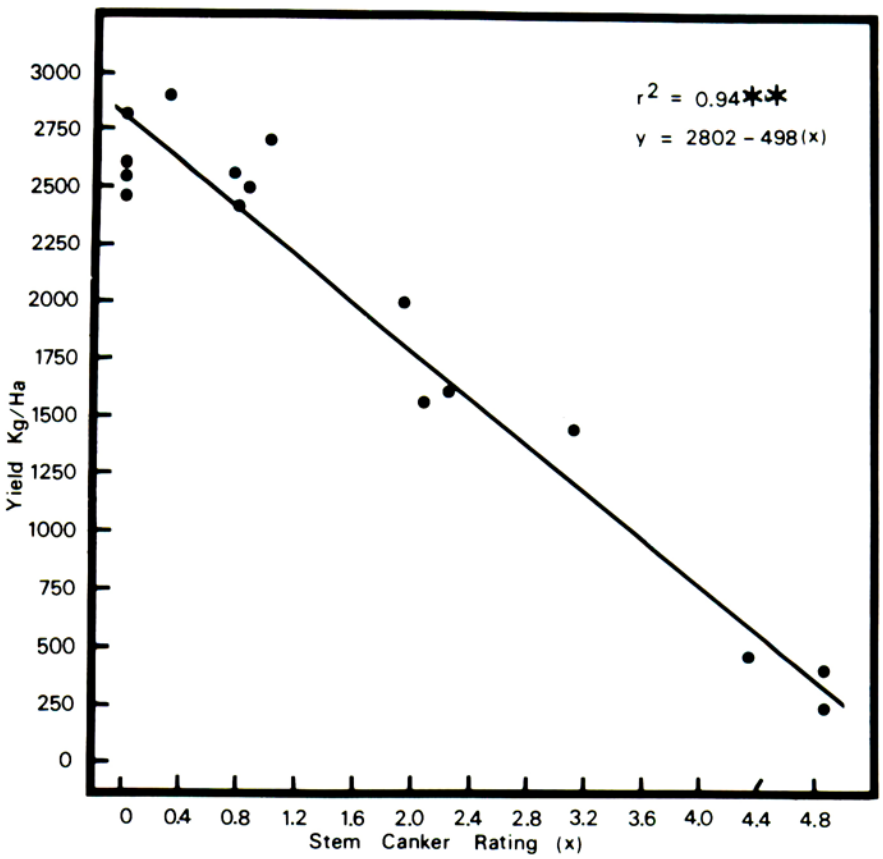


**Fig. 5. Relationship between soybean yield and stem canker severity according to the pretransformed arc sine rating system (Table 1).**

sine scale developed by our group (Table 1). This rating scale provides a strong linear relationship between disease and yield and is easily taught to the inexperienced evaluator. Data developed in 1983 spray trials relating average disease rating to average yield produced a linear regression line with an $r^2$ of 0.94, indicating that the rating scale had accounted for almost all of the yield variation (Fig. 5).

## Control

An understanding of the biology of *Dpc* has led to the development of tactics and recommendations for controlling stem canker in soybeans. These encompass a broad range, from sanitation, seed treatment and certification, rotation, time of planting, and cultivar selection to postemergence fungicides.

**Sanitation and clean seed.** Prevention of *Dpc* movement to noninfested fields was first directed at the mechanisms of long-distance dispersal of the organism. Any farm equipment that moves from infested to noninfested fields should be cleaned of all plant debris and soil. This recommendation must be coupled with knowledge of a field's disease condition the previous season and with the ability to recognize diseased plants occurring at frequencies of 0.1% or less in a field. The second means of movement is on seed. No seed harvested from fields infested with *Dpc* should be planted in noninfested fields. Seed sources often are unknown because seed from many fields are pooled when placed in commercial seed channels. Seedsmen should examine seed fields before harvest to determine the stem canker status. Research conducted by our group indicates that seed-treatment fungicides can greatly reduce but not eliminate stem canker. We found carboxin-thiram and carboxin-thiram-captan to be best for *Dpc*-infested seed. In our opinion, seed-treatment fungicides should be used 1) as assurance against high levels of stem canker inoculum entering a previously noninfested field, 2) as insurance only on seed thought to be noninfested, and 3) with the understanding that *Dpc*, if present, will not be totally eliminated.

**Postemergence chemicals.** Early efforts to control stem canker relied on applying

chemicals just before symptoms developed (3). Typically, these applications were during the early reproductive stages and were beneficial only when systemic fungicides were used. Efficacy was often enhanced by adjuvants, i.e., oil:surfactant blends, although results were erratic among fields and trials run in different years. In 1982, our research trials indicated that applications of benomyl were more beneficial during the vegetative period than during the reproductive period (Fig. 2). Further, A. Y. Chambers (*unpublished*) found that the nonsystemic (contact) fungicides were efficacious when applied during the early vegetative period. Data from our 1983 fungicide spray trials (Figs. 6 and 7) support our previous observations on spore release and infection during May and June. However, as we have pointed out, spore maturation and infection may occur later (as in 1984), resulting in little or no disease and obviating the need for fungicides. Evaluations of fungicides for control of stem canker on cultivars of various levels of susceptibility indicate that economic control cannot be achieved on highly susceptible cultivars. Cultivars of intermediate susceptibility respond to fungicide treatment with yield increases commensurate with disease control, whereas little or no benefit is obtained by treating resistant cultivars (Fig. 6). These data indicate that stem canker on cultivars of moderate susceptibility can be managed with foliar fungicides. Sprays should be applied during the early vegetative growth stages while spores are being actively produced. Sprays during this period can be banded (15–20 cm wide) over the small plants and thus are very economical. The treatment time could be refined if we had more detailed knowledge of infection parameters.

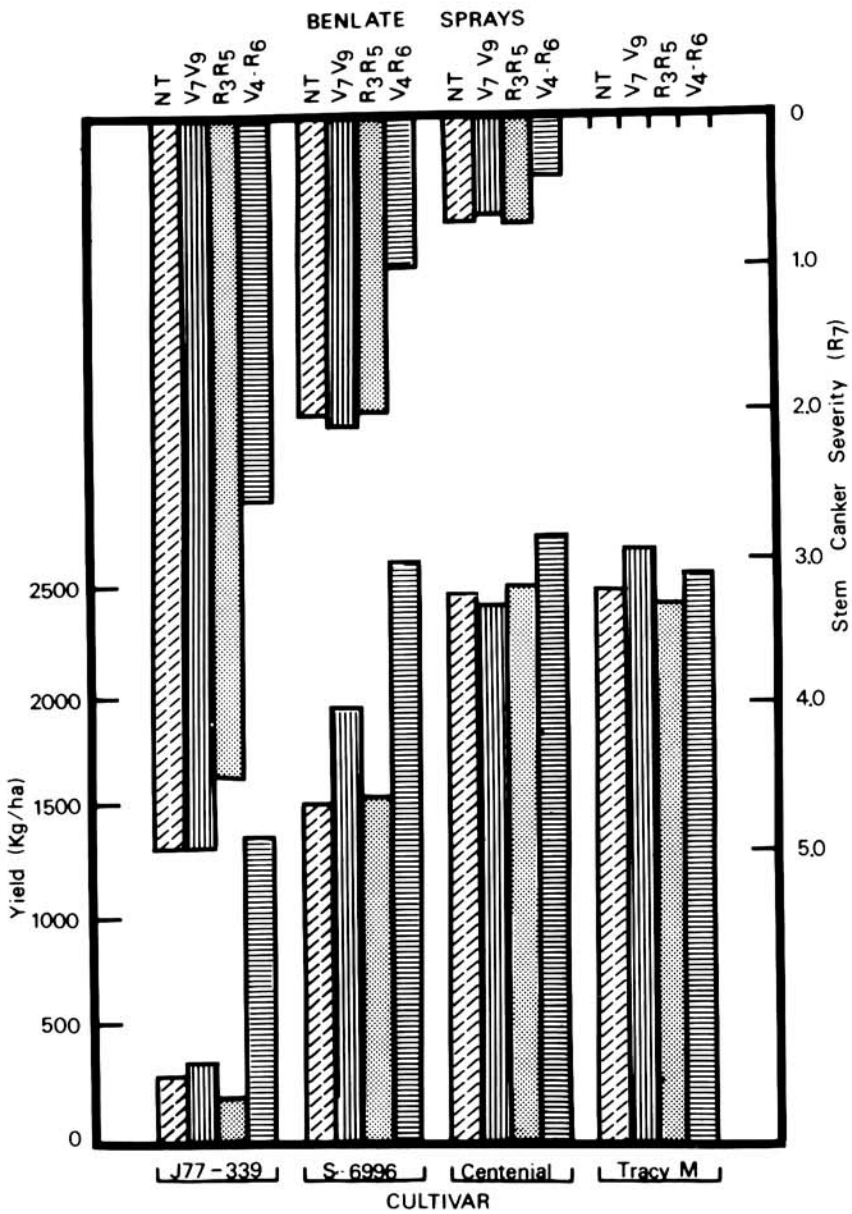


**Fig. 6. Yield response and stem canker severity of four soybean cultivars treated by four different schedules with Benlate 50WP (benomyl) at 0.56 kg/ha per application. NT = not treated; V = vegetative stage followed by number of leaves on main stem; R = reproductive stage followed by 3 (early pod set), 5 (pod fill), or 6 (filled pod). The $V_4$–$V_6$ schedule received five sprays, all others received two.**

**Cultivar reaction.** Differential reaction of soybean cultivars to stem canker infection was reported by several investigators in the United States and Canada during the 1950s. Concurrent with the reports of stem canker outbreaks in the Southeast was the reaction of cultivars expressed as differences in yield and/or percentage of dead plants among cultivars in infested plots. Backman et al (3) reported a complete range of cultivar reaction, from no disease on Tracy-M to severe infection on Hutton and RA 800; most cultivars had an intermediate reaction. Weaver et al (17) conducted extensive field experiments evaluating cultivars for disease development and seed yield and confirmed the disease resistance of Tracy-M (Fig. 8) but found that other cultivars with higher levels of stem canker, such as Braxton, Ransom, Davis, Wright, and Deltapine 105, had yields equal to or greater than those of Tracy-M (Fig. 9). Under conditions of plant stress induced by drought or nematodes, however, moderately resistant cultivars could suffer high levels of disease and severe losses. Conversely, under very low stress conditions combined with late infection (as occurred throughout the southeastern United States in 1984), even moderately susceptible cultivars could escape damage.

Keeling (8) further documented resistance of Tracy-M by inoculating seedlings in the greenhouse with toothpicks containing *Dpc*. He also inoculated CNS (Clemson Nonshattering) as a suspected source of resistance and Peking as a suspected source of susceptibility, because these cultivars tended to appear in the pedigrees of resistant and susceptible types, respectively. CNS proved to be as resistant as Tracy-M to greenhouse inoculation, and Peking was rated as moderately resistant. Thus, the source of susceptibility remains in doubt. Recently, however, Kilen et al (10) found that resistance in Tracy-M was conditioned by two major dominant genes.

On the basis of the results of these studies and observations by workers in many states, and for the purpose of recommending cultivars to farmers, cultivars are generally divided into four major groups according to relative stem canker resistance (Table 2). Our research on cultivars not adapted to areas where the stem canker organism is currently endemic indicates that certain tropical cultivars (Jupiter R and Santa Rosa R) are highly susceptible. Thus, the disease possibly could spread into tropical soybean-growing areas. Furthermore, certain northern cultivars (e.g., Elf and Gnome) with parentage lines from

southern cultivars (to incorporate the $dt_1$ gene for determinate growth habit) were also susceptible.

**Crop rotation.** Benefits of crop rotation to control stem canker have not been demonstrated in the Southeast, probably because of the relatively recent nature of the problem and the long-term nature of rotation studies. Because the fungus overwinters on crop debris, it would be logical to assume that rotation with a nonhost crop would lead to decomposition of the debris and would have some value in controlling the disease. Evidence by Roy and Miller (14) that stem canker inoculum can come from other host crops such as cotton may limit the value of rotation as a control measure. Currently, rotation to a nonhost crop such as corn or grain sorghum is recommended in Alabama and Mississippi for at least 2 years after conditions of severe disease infestation.

**Planting date.** We have demonstrated benefits of delayed planting, particularly for mid- to full-season moderately susceptible and susceptible cultivars. In 1982 delayed planting benefited even moderately resistant cultivars, but in 1983 dry weather limited yields of late-planted plots to the extent that only the most susceptible cultivars showed a yield increase. Disease levels in these experiments were less than 50% infected plants even on susceptible cultivars. Additional research on planting date is needed under higher levels of disease pressure to adequately assess the effect of late planting. Delayed planting appears to affect yield loss to stem canker in two ways. Plants are able to avoid the initial release of inoculum from crop debris that generally occurs from May to mid-June, so fewer plants are infected. In those that do become infected, the shorter vegetative and reproductive period induced by late planting allows less time for the disease to

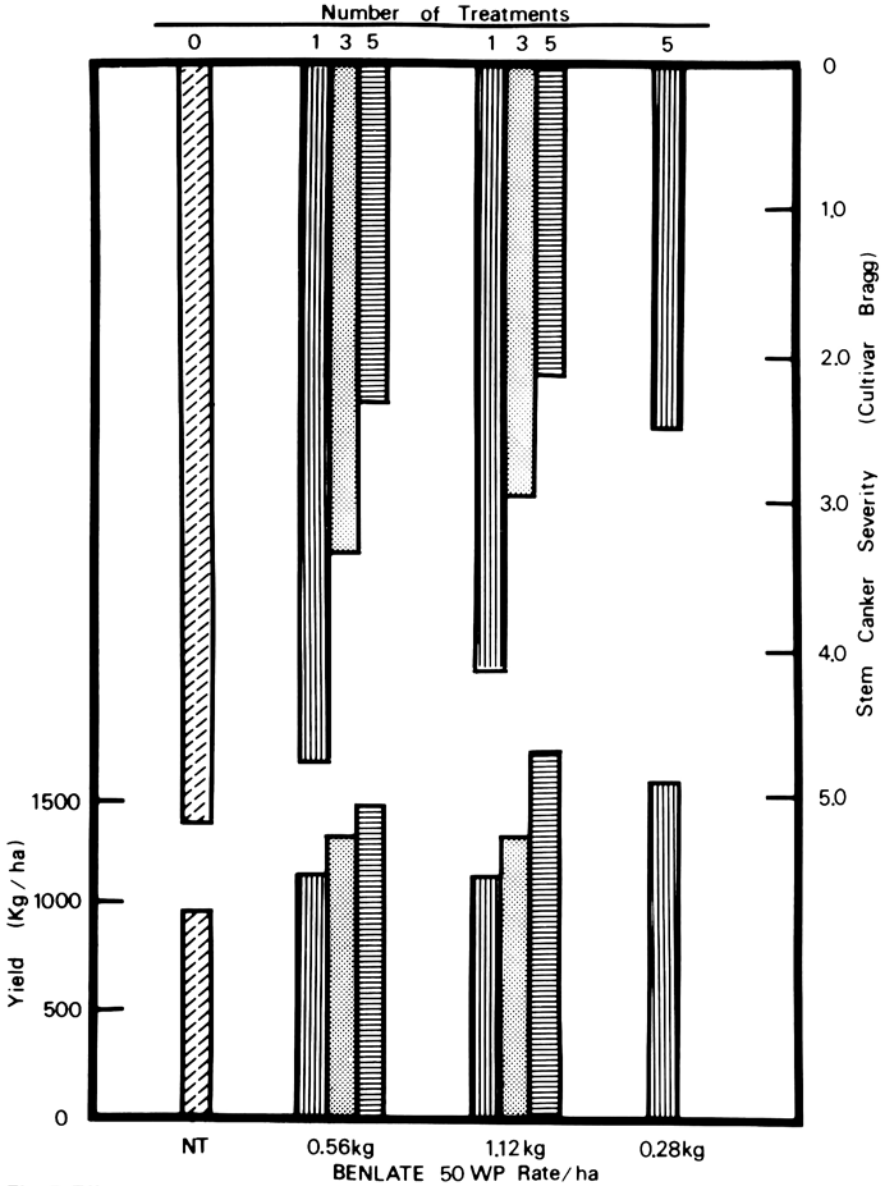


**Fig. 7. Effects of Benlate 50WP (benomyl) application rate and number of applications on disease severity and yield of cv. Bragg. 0 = no treatment (NT), 1 = $V_4$ only, 3 = $V_4 + V_7 + V_{10}$, and 5 = $V_4 + V_7 + V_{10} + R_3 + R_5$.**

**Table 2.** Relative resistance of selected soybean cultivars to southern isolates of *Diaporthe phaseolorum* var. *caulivora*, causal organism of stem canker

| Resistant | | Moderately resistant | | Moderately susceptible | | Susceptible | |
|---|---|---|---|---|---|---|---|
| Maturity group[a] | Cultivar | Maturity group | Cultivar | Maturity group | Cultivar | Maturity group | Cultivar |
| I | Blackhawk | V | Terra Vig 505 | II | Gnome | V | AP 55 |
| | Hardin | | Deltapine 105 | III | Elf | | A 5539 |
| | Hodgson 78 | | Deltapine 345 | | Sprite | | RA 502 |
| II | Century | | Wilstar 550 | IV | Pixie | VI | RA 604 |
| | Corsoy 79 | | Shiloh | V | Bedford | | Brysoy 9 |
| | Hawkeye | VI | Centennial | | Forrest | | Bradley |
| III | Cumberland | | Davis | | Essex | VII | Coker 237 |
| | Williams 82 | | RA 680 | | A 5474 | | Bragg |
| V | Bay | | Coker 156 | VI | Jeff | | McNair 770 |
| VI | Tracy-M | VII | Wright | | Lee 74 | | Wilstar 790 |
| VII | Braxton | | Ransom | | S69-96 | | RA 701 |
| VIII | Dowling | | Coker 317 | | Deltapine 506 | VIII | Coker 338 |
| | | | GaSoy 17 | VII | Gregg | | Hutton |
| | | VIII | Coker 368 | | Gordon | | RA 801 |
| | | | Coker 488 | VIII | Foster | IX | Jupiter R |
| | | | Cobb | | Kirby | | Santa Rosa R |

[a] Reactions of cultivars in maturity groups I, II, III, IV, and IX are based on greenhouse inoculation tests; all others are based on field tests.

**Fig. 8. Severely infected J77-339 soybeans (foreground) contrast strongly with resistant cultivar Tracy-M. Both cultivars are at filled-pod stage ($R_6$).**


**Fig.9. Resistant cv. Braxton and susceptible cv. Hutton at pod-fill stage.**

develop to the point where the plants die before pod fill.

**Tillage.** Because *Dpc* overwinters on crop debris, effects of tillage depend largely on whether the previous year's crop was infected or inoculum is introduced from some other source. Tyler et al (16) reported significantly more stem canker in no-till plots than in four other tillage regimes and speculated that the lower incidence of brown spot (*Septoria glycines* Hemmi) in no-till plots caused plants to retain their lower leaves, providing an entry site for *Dpc*; whether inoculum was present at planting was not known.

## Breeding for Resistance

When stem canker became prevalent in the Midwest in the 1950s, severity declined with the elimination of susceptible cultivars, so that continued breeding efforts to control the disease were not necessary (1). Many cultivars listed in Table 2 as susceptible or moderately susceptible have been among the most popular cultivars in their respective maturity groups. What effect eliminating these cultivars from production will have on future incidence of stem canker is not known. The current seriousness of the problem, however, has led many breeders in southern states to begin screening for resistance. With the adaptation of the toothpick inoculation technique by Keeling (8), breeders can effectively screen large numbers of lines in the seedling stage against known isolates of the pathogen. A major problem facing breeders in the future is the apparent association between stem canker susceptibility and cyst nematode resistance. Of the cultivars listed as resistant or moderately resistant in Table 2, only Centennial and Coker 368 also have resistance to both cyst and root-knot nematodes. This observation led Keeling (8) to suspect Peking, a source of cyst nematode resistance, as the source of stem canker susceptibility. Though Peking proved to be moderately resistant to toothpick inoculation, its field resistance (and that of other nonadapted northern and tropical cultivars) to southern isolates is difficult to determine because its extremely early maturity in southern latitudes causes early conversion of the plant to the reproductive stage, not allowing adequate time for disease progress.

Thus there are two major goals in breeding for stem canker resistance. First, to screen all breeding lines in the greenhouse or field to avoid the release of susceptible types; and second, to develop types that have combined resistance to stem canker and the major nematode species. This second objective may be accomplished by backcrossing stem canker resistance into lines with multiple nematode resistance. Backcrossing (or modified pedigree methods involving backcrossing) appears to be the method of choice because seedlings are easily screened in the greenhouse and because genes for resistance are qualitatively inherited (10). Backcrossing would also be the fastest way to introduce resistance genes into a wide range of agronomically acceptable genotypes. Evidence that physiological races of the pathogen may exist would present complications for plant breeders and make the utilization of a single source of resistance, such as Tracy-M, inadvisable.

## Conclusions

Soybean stem canker in the South has emerged from an obscure disease to a disease of major economic concern in a period of 10 years. Current research indicates that the aggressive strains of *D. phaseolorum* var. *caulivora* found in the southern United States are different from their northern counterparts, but a definitive study on their taxonomic status

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has not been done. The severity of stem canker in the South has already forced farmers to abandon some cultivars and to alter several aspects of their cultural practices. Unfortunately, there are not enough well-adapted cultivars to deal with the problem solely by this method. Such practices as early-season fungicide applications may be necessary while the selection of stem canker-resistant cultivars is being broadened. The races that have already developed are jeopardizing the long-term status of resistant cultivars. Further, we do not yet know the northern limit of adaptability of the southern strains, which could threaten the soybean production areas of the midwestern United States.

## Acknowledgments

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