Effect of Previous Crop, Seedborne Inoculum, and Fungicides on Development of *Stagonospora* Blotch

E. A. Milus and D. B. Chalkley, Department of Plant Pathology, University of Arkansas, Fayetteville 72701

ABSTRACT

Milus, E. A., and Chalkley, D. B. 1997. Effect of previous crop, seedborne inoculum, and fungicides on development of *Stagonospora* blotch. *Plant Dis.* 81:1279-1283.

Stagonospora blotch of wheat has been difficult to control in the eastern United States. The objectives of this research were to evaluate the effects of field inoculum, seedborne inoculum, and seed treatment and foliar fungicides on *Stagonospora* blotch development and to develop more effective management strategies. In 1995 and 1996, similar experiments were established in "infested" and "clean" fields using two seed lots of Coker 9543 ("low" and "high" levels of seed infection) and six seed or foliar fungicide treatments. Planting in clean fields, planting seed with a low level of seedborne inoculum, treating seed with difenoconazole or triadimenol + thiram, and applying propiconazole or tebuconazole to the foliage all contributed toward reducing leaf infections by *Stagonospora nodorum*, severity of leaf and glume blotch, and incidence of *S. nodorum* in the harvested seed. Propiconazole alone was the least effective treatment. Planting in an infested field tended to negate the beneficial effects of low level of seed infection and fungicide seed treatments. Crop rotations and tillage that allow wheat debris to decompose before the next wheat crop along with difenoconazole or triadimenol seed treatment to reduce seedborne inoculum should be sufficient to avoid serious losses. In fields where wheat is grown every year, tillage and seed treatment would still be helpful, but a foliar fungicide at GS 8 may be necessary for adequate control. Applying an effective fungicide to seed appears to be a more efficient means of reducing seedborne inoculum than does producing seed with low levels of inoculum.

Stagonospora blotch caused by *Stagonospora nodorum* (Berk.) Castellani & E.G. Germano (= *Septoria nodorum* [Berk.] Berk. in Berk. & Broome, teleomorph *Phaeosphaeria nodorum* [E. Muller] Hedjaroude, = *Leptosphaeria nodorum* E. Muller) can cause serious yield and quality losses of wheat (*Triticum aestivum* L.) in the eastern United States. Adapted, highly resistant cultivars are not available (17). The epidemiology of *Stagonospora nodorum* blotch and the efficacy of various cultural practices have been investigated to develop more effective chemical and cultural control measures.

Possible sources of primary inoculum include infected seed and pycnidia and pseudothecia in debris of a previous wheat crop, volunteer wheat plants, and alternative hosts (5). In Indiana (17), initial symptoms usually appeared on the lower leaves in the early spring, and this was taken as an indication that primary infections occurred in the fall and the pathogen survived in symptomless leaves. The source

of primary inoculum in Indiana was unknown; however, infected seed or airborne ascospores were suspected, and pycnidiospores from crop debris were ruled out because of the common crop rotations. Disease development in Arkansas appeared to be similar (14), and nearly all infections up to Feekes growth stage (GS) 8 (flag leaf emergence) were symptomless (8).

The pathogen was found to be seedborne in a high percentage of wheat seed samples from North Carolina (2), Georgia (3), and New York (15), indicating that seed may be an important source of initial inoculum. Marked strains of the pathogen established in seed were later recovered from leaves and harvested seed, demonstrating that seedborne inoculum could cause disease (16). Luke et al. (9,11) found that both infected seed and infested debris in fields contributed to disease development in Florida and that seed treatment with benomyl or triadimenol reduced disease severity. Reducing seedborne inoculum was beneficial only in conjunction with crop rotation to reduce inoculum from the field. The relationship between incidence of seed infection and disease was nonlinear, and <10% seed infection was sufficient to cause severe epidemics (10,16).

Based on their findings that 96% of the genetic diversity was within populations of *S. nodorum* and that alleles at RFLP loci were in gametic equilibrium, McDonald et al. (13) and Keller et al. (7) hypothesized that ascospores disseminated over long

distances were the primary source of inoculum. However, pseudothecia or ascospores of *S. nodorum* have never been found in Georgia or Florida (B. Cunfer, *personal communication*) or in New York (16).

Applying a foliar fungicide in the spring has been the usual means of controlling *Stagonospora* blotch, but the level of control has not been adequate in many cases. Reducing the amount of initial infection by crop rotation, planting seed with a low incidence of infection, or applying fungicides to the seed may slow disease development and reduce losses, but the relative efficacy of these practices is poorly understood. The objectives of this research were to evaluate the effects of field inoculum, seedborne inoculum, and seed treatment and foliar fungicides on *Stagonospora* blotch development and to develop more effective management strategies for *Stagonospora* blotch.

MATERIALS AND METHODS

Field plots. Field experiments were conducted during the 1995 and 1996 (year of harvest) growing seasons at the Southwest Research and Extension Center near Hope, AR. In each year, similar experiments were established in "clean" and "infested" fields that were approximately 400 m apart. The clean fields (different field each year) had not been planted to wheat for at least 4 years and likely had very low levels of the pathogen. The infested field was planted to wheat that had moderate (1995 experiment) or severe (1996 experiment) leaf and glume blotch the previous year and likely had high levels of the pathogen. Each field was a separate experiment, and no statistical comparisons could be made between the two fields.

Within each field the experimental design was a randomized complete block with two seed lots of cultivar Coker 9543 (moderately susceptible to *Stagonospora* blotch), six treatments, and three replications. The two seed lots were chosen to represent low and high levels of seedborne *S. nodorum* from a random sample of 30 certified seed lots that were assayed as described below. The same seed lots were used in both years. Seed lots with "low" and "high" levels of infection had 7 and 40% incidence of *S. nodorum*, respectively, in 1995 and 7 and 34% incidence, respectively, in 1996.

Treatments were: 1) nontreated; 2) seed treatment with triadimenol (Baytan 30F, 26

Corresponding author: E. A. Milus
E-mail: gmilus@comp.uark.edu

Current address of D. B. Chalkley: American Type Culture Collection, Rockville, MD 20852-1776.

Accepted for publication 29 July 1997.

Publication no. D-1997-0825-06R
© 1997 The American Phytopathological Society

g a.i./100 kg = 1.25 fl. oz./cwt) + thiram (Thiram 42S, 63 g a.i./100 kg = 2 fl. oz./cwt); 3) seed treatment with difenoconazole (Dividend 3F, 23 g a.i./100 kg = 1 fl. oz./cwt); 4) foliar application of propiconazole (Tilt 3.6E, 126 g a.i./ha = 4 fl. oz./acre) at GS 9 (flag leaf fully emerged); 5) seed treatment with difenoconazole followed by a foliar application of propiconazole at GS 9; and 6) seed treatment with difenoconazole followed by foliar applications of tebuconazole (Folicur 3.6F, 126 g a.i./ha = 4 fl. oz./acre) at GS 2 (beginning of tillering in the fall), 9, and 10.5 (flowering) in an attempt to create a disease-free situation.

Plots were planted on 3 November 1994 and 9 November 1995 using a plot drill equipped with a cone seeder. Individual plots were 3 m (14 rows) wide by 6 m long and surrounded by 3 m of oat to reduce spread of the pathogen among plots. Plant stand was measured 4 weeks after planting by counting the seedlings in six 1-m-long segments of row per plot for treatments 1–

3. Recommended fertility and weed control practices were applied uniformly across the fields as needed. Plots were harvested with a plot combine on 6 June 1995 and 24 June 1996. Grain moisture and test weight were measured with a GAC II grain analysis computer after passing the grain once through a seed cleaner. Yields were adjusted to 13% moisture.

During the second year, a volumetric spore sampler with a 1.5-ml collection vial (Burkhard Manufacturing Co., Hertfordshire, England) was installed in each field to detect airborne inoculum. The spore samplers were set on platforms so that the suction orifice was approximately 1 m above the soil surface and were covered by 1.2-m-square roofs to protect them from rain. Collection vials were changed once per week from 29 November to 19 March and then three times per week until 17 May. Vials were refrigerated for 1–35 days before being assayed. Vial contents were suspended in 1.0 or 1.5 ml volumes of sterile water, and two replicates of 10^{-2} and

10^{-3} dilutions were plated on SNAW medium (12). Conidia of *S. nodorum* were dilution plated as checks.

Disease measurements. Because previous research (14) demonstrated that symptoms were not a reliable measure of infection before flowering, leaves were assayed four times during the season to determine the effects of level of seed infection and treatment on the incidence of symptomless infection. Leaves were disinfested in 0.5% sodium hypochlorite + 0.1% Tween 20 for 2 min (primary leaves) or 5 min, and plated on modified Bannan's medium (6) containing 20 mg of paraquat, 200 mg of chloramphenicol, 200 mg of fentin hydroxide, and 5 g of agar in 1 L of deionized water. Plates were incubated in a growth chamber at 20°C and 12-h photoperiod for 8 days and then observed microscopically for pycnidia. A leaf was considered infected if one or more pycnidia characteristic of *S. nodorum* were found, and data were converted to percentage of leaves infected.

The growth stages, leaf positions, and treatments assayed were as follows: i) GS 2 (tillering), primary leaves for treatments 1–3; ii) GS 4 (stem elongation), flag-3 and flag-4 leaves on the largest culms for treatments 1–3 and 6; iii) GS 9 (flag leaves fully emerged), flag-2 and flag-3 leaves of largest culms for treatments 1–3 and 6; and iv) GS 10.5 (flowering), flag and flag-1 leaves from largest culms for all treatments. At GS 9, 50 of the largest culms were collected and discarded from plots of treatments 4 and 5 so as not to bias yields among the treatments. Fifty leaves of each position were assayed from each plot. Except for primary leaves that could be assayed whole, a 5-cm-long segment from near the base of each leaf was assayed.

Fifty of the largest culms were collected from each plot at GS 11.2 (soft dough). For each plot, leaf blotch severity (percent flag leaf area diseased) and glume blotch severity (percent glume area diseased) of each culm were rated and then averaged before statistical analyses. The severity percentages (and their ranges) recorded were 0, 2 (trace–4), 7 (5–10), 15 (11–20), 30 (21–40), 50 (41–60), 70 (61–80), 85 (81–90), 93 (91–96), and 98 (>96).

The incidence of harvested seeds infected with *S. nodorum* was determined 4 to 8 weeks after harvest. Fifty seeds per plot were plated on SNAW medium, incubated in a growth chamber at 20°C with a 12-h photoperiod, and examined for mycelium, fluorescence, and pycnidia typical of *S. nodorum*. The assay was done twice and the results averaged before statistical analysis.

Statistical analysis. Within each field and year, data were analyzed as a randomized complete block design with a factorial treatment structure consisting of two seed lots and three to six treatments depending on the growth stage. Protected least sig-

Table 1. Incidence (%) of *Stagonospora nodorum* infection on flag-4 leaves at Feekes GS 4 in infested and clean fields in 1995 and 1996

Main effect	1995			1996	
	Infested field ^x		Clean field ^y	Infested field ^y	Clean field ^y
	Low	High	Mean	Mean	Mean
Treatment					
Nontreated	12.0 b	48.3 a	24.2 a	21.7 a	8.3 a
Triadimenol + thiram	8.0 bc	5.3 bc	2.2 b	25.5 a	0.0 b
Difenoconazole	1.3 bc	5.0 bc	2.7 b	15.3 a	0.7 b
Difenoconazole/ tebuconazole 1X ^z	0.7 bc	0.0 c	0.7 b	10.7 a	0.0 b
Level of seed infection					
High	–	–	11.5 a	19.8 a	3.8 a
Low	–	–	3.3 b	16.8 a	0.7 b

^x Interaction means are presented because there was a significant ($P \leq 0.0001$) interaction between treatment and level of seed infection. Values within the pair of columns followed by the same letter are not significantly different (LSD, $P = 0.05$).

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P = 0.05$).

^z By Feekes GS 4, tebuconazole had been applied one time as a foliar spray.

Table 2. Incidence (%) of *Stagonospora nodorum* infection on flag-3 leaves at Feekes GS 9 in infested and clean fields in 1995 and 1996

Main effect	1995			1996		
	Infested field ^x		Clean field ^y	Infested field ^y	Clean field ^x	
	Low	High	Mean	Mean	Low	High
Treatment						
Nontreated	37.3 b	81.3 a	32.3 a	70.2 ab	29.0 bc	84.7 a
Triadimenol + thiram	25.3 bc	26.0 bc	5.3 b	77.2 a	11.0 de	39.7 b
Difenoconazole	14.0 cd	18.7 bcd	5.0 b	55.3 b	27.3 bcd	15.3 cde
Difenoconazole/ tebuconazole 1X ^z	9.3 cd	2.7 d	0.7 b	52.0 b	16.0 cde	2.7 e
Level of seed infection						
High	–	–	15.5 a	69.4 a	–	–
Low	–	–	6.2 b	57.9 a	–	–

^x Interaction means are presented because there were significant ($P \leq 0.004$) interactions between treatment and level of seed infection. Values within each pair of columns followed by the same letter are not significantly different (LSD, $P = 0.05$).

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P = 0.05$).

^z By Feekes GS 9, tebuconazole had been applied one time as a foliar spray.

nificant difference tests at $P = 0.05$ were used to separate main effect and interaction means as appropriate.

RESULTS

Incidence of infection for primary leaves at GS 2 was low and variable in both years (data not shown). There were no significant differences among levels of seed infection or treatments.

Plant stands in 1995 were similar regardless of level of seed infection or seed treatment and averaged 46 and 44 plants/m in the infested and clean fields, respectively. In 1996, the seed lot with low level of infection had plant stands that were significantly ($P = 0.005$) less than the seed lot with high level of infection, but treatments were not significantly different. The seed lot with low infection averaged 37 and 29 plants/m and the seed lot with high infection averaged 46 and 34 plants/m in the clean and infested fields, respectively.

Of the two leaf positions sampled at GS 4, 9, and 10.5, the lower leaf position always had greater incidence of infection by *S. nodorum* and usually had more distinct differences among treatments and seed lots. Therefore, only data for the lower leaf position at each growth stage were reported. Actual incidence of infection was likely higher than estimated in this study because only a 5-cm-long portion near the base of each leaf was assayed.

Except for the infested field in 1996, the incidence of infection on flag-4 leaves at GS 4 was lower for the seed lot with low level of infection than for the seed lot with high level of infection (Table 1). There was a significant interaction ($P \leq 0.0001$) between level of seed infection and treatment for the infested field in 1995, due in large part to the higher incidence for two treatments in the seed lot with low infection compared with that in the seed lot with high infection. Compared to the nontreated check, triadimenol + thiram, difenoconazole, and difenoconazole followed by one application of tebuconazole in the fall significantly reduced the incidence of infection in the clean fields in both years and in the seed lot with high infection in the infested field in 1995.

Two significant interactions ($P \leq 0.004$) between levels of seed infection and treatments complicated interpretation of the data for incidence of infection at GS 9 (Table 2), but in general, a low level of seed infection and fungicides contributed toward a lower incidence of infection.

Incidence of infection on flag-1 leaves at GS 10.5 was numerically higher in infested fields than in clean fields (Table 3). A low level of seed infection significantly decreased incidence in clean fields but not in infested fields. Compared to the nontreated check, a difenoconazole seed treatment followed by two foliar applications of tebuconazole and difenoconazole seed treatment and a foliar application of propiconazole

reduced the incidence of infection in all comparisons and in all but one comparison, respectively. Seed treatments alone were effective in only two comparisons. Propiconazole alone was not different from the nontreated check in any of the comparisons.

In 1995, there was a trend for less leaf and glume blotch the clean field than in the infested field, and significantly less disease developed in plots grown from seed with a low level of seed infection than in plots grown from seed with a high level of infection (Table 4). Seed treatments provided as good or better control than a propiconazole foliar application in all comparisons and, in clean fields, were no different from a difenoconazole seed treatment followed

by three foliar applications of tebuconazole. Except for leaf blotch in the infested field, the level of control provided by difenoconazole seed treatment followed by a foliar application of propiconazole was not different from that provided by difenoconazole seed treatment followed by three foliar applications of tebuconazole. In 1996, very little leaf and glume blotch developed because of drought conditions during the spring, and there were no significant differences among levels of seed infection or treatments (data not shown).

Yields among levels of seed infection or treatments were not significantly different in 1995 or 1996 (data not shown). Compared to the nontreated checks in 1995, test weight was increased by all treatments in

Table 3. Incidence (%) of *Stagonospora nodorum* infection on flag-1 leaves at Feekes GS 10.5 in infested and clean fields in 1995 and 1996

Main effect	1995		1996		
	Infested field ^x		Clean field ^y	Infested Field ^y	Clean field ^y
	Low	High	Mean	Mean	Mean
Treatment					
Nontreated	80.0 ab	92.0 a	71.8 a	42.7 a	17.0 a
Triadimenol + thiram	75.3 abc	67.3 abc	38.3 b	35.7 a	9.7 ab
Difenoconazole	72.3 abc	49.3 cd	26.3 bc	32.5 a	6.0 ab
Propiconazole	66.7 abc	83.3 ab	63.0 a	29.2 a	8.3 ab
Difenoconazole/ propiconazole	21.0 e	64.0 bc	12.3 c	27.7 a	0.7 b
Difenoconazole/ tebuconazole 2X ^z	34.7 de	13.3 e	14.7 c	7.3 b	2.3 b
Level of seed infection					
High	—	—	46.4 a	31.6 a	12.9 a
Low	—	—	29.1 b	26.7 a	1.8 b

^x Interaction means are presented because there was a significant ($P = 0.01$) interaction between treatment and level of seed infection. Values within the pair of columns followed by the same letter are not significantly different (LSD, $P = 0.05$).

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P = 0.05$).

^z By Feekes GS 10.5, tebuconazole had been applied two times as a foliar spray.

Table 4. Leaf blotch severity on flag leaves and glume blotch severity on heads at Feekes GS 11.2 in 1995

Main effect	Leaf blotch, % ^w			Glume blotch, % ^w	
	Infested field ^x		Clean field ^y	Infested field ^y	Clean field ^y
	Mean	Low	High	Mean	Mean
Treatment					
Nontreated	73.3 a	11.3 b	33.3 a	17.0 a	15.0 a
Triadimenol + thiram	55.0 b	5.0 bc	4.7 bc	7.8 b	3.2 c
Difenoconazole	55.0 b	2.7 bc	11.0 b	8.3 b	3.5 c
Propiconazole	42.7 b	6.3 bc	8.0 bc	15.5 a	8.2 b
Difenoconazole/ propiconazole	25.0 c	1.7 c	5.0 bc	6.0 bc	3.3 c
Difenoconazole/ tebuconazole 3X ^z	8.0 d	1.3 c	1.7 c	2.2 c	2.3 c
Level of seed infection					
High	41.8 a	—	—	10.9 a	7.7 a
Low	38.3 b	—	—	8.0 a	4.1 b

^w Percentage of leaf or glume area diseased based on visual estimates from 50 main culms per plot that were averaged before statistical analysis.

^x Interaction means are presented because there was a significant ($P = 0.017$) interaction between treatment and level of seed infection. Values within the pair of columns followed by the same letter are not significantly different (LSD, $P = 0.05$).

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P = 0.05$).

^z By Feekes GS 11.2, tebuconazole had been applied three times as a foliar spray.

the infested field and by all treatments except propiconazole alone in the clean field (Table 5). In 1996, none of the treatments improved test weight. Levels of seed infection did not affect test weight in either year or field (data not shown).

In clean fields but not in infested fields, the incidence of *S. nodorum* in harvested seed was significantly lower in the seed lot that had a low level of infection than in the seed lot that had a high level of infection (Table 6). Also, incidence tended to be lower in clean fields than in corresponding infested fields. Compared to the nontreated check, seed treatment with difenoconazole followed by three foliar applications of tebuconazole was the only treatment that reduced incidence in all years and fields. Difenoconazole followed by a foliar application of propiconazole was not different from difenoconazole followed by three foliar applications of tebuconazole. Propiconazole alone did not reduce incidence in any year or field. Other treatments gave variable results.

S. nodorum was not detected in any of the spore samples from the infested or clean fields. *S. nodorum* pycnidiospores plated as checks always grew into distinctive colonies, and common saprophytic fungi were identified from the samples.

DISCUSSION

Planting in clean fields, planting seed with a low level of seedborne inoculum, treating seed with difenoconazole or tri-

adimenol + thiram, and applying propiconazole or tebuconazole to the foliage all contributed toward reducing leaf infections by *S. nodorum*, severity of leaf and glume blotch, and incidence of *S. nodorum* in the harvested seed. Propiconazole alone was the least effective treatment. Planting in an infested field tended to negate the beneficial effects of low level of seed infection and fungicide seed treatments as was reported by Luke et al. (9,11).

Even though no statistical comparisons were possible between infested and clean fields, greater values for incidence of infection and disease severity in many comparisons indicate that crop debris in the infested fields contributed inoculum to epidemics. Crop debris in the infested field appeared to provide abundant inoculum by GS 4 in 1996 but not 1995. Inoculum from crop debris may have been more important in 1996 because dry soil conditions from June through October prevented incorporation of the crop debris, and most of the crop debris was still on the soil surface. Also, 1996 was the second year for planting Coker 9543 in this field, and it is possible that pathogen strains with preference for this cultivar had increased as was reported for *Septoria tritici* by Ahmed et al. (1).

The absence of *S. nodorum* in samples from spore samplers designed to collect airborne ascospores and the large differences between clean and infested fields only 400 m apart indicate that airborne ascospores were not abundant. The recov-

ery of saprophytic fungi from spore samplers and growth of *S. nodorum* from pycnidiospores used as checks indicate that the pathogen would have been detected by the spore samplers if it was present. Therefore, rain-splashed pycnidiospores appear to be the likely form of inoculum from crop debris. This is contrary to the conclusions of McDonald et al. (13) and Keller et al. (7), who based their conclusions on high percentages of genetic variation within populations and on the existence of gametic equilibrium and the lack of clonal lineages in *S. nodorum* populations.

Seedborne inoculum appeared to be important for initiating epidemics. A low level of seedborne inoculum and the seed treatments used in this study were effective for reducing the incidence of leaf infection at GS 4 except in the infested field in 1996 when inoculum from crop debris apparently overwhelmed any beneficial effects of low seed infection or seed treatment. The potential for seedborne inoculum appears to be high in Arkansas. Of the 30 certified seed lots of Coker 9543 assayed in this study, all were infected with *S. nodorum*, and infection levels ranged from 7 to 40%. Of the seed lots produced during this study, all were infected. A combination of clean field, low level of seed infection, difenoconazole seed treatment, three applications of tebuconazole, and less than optimum conditions for disease still allowed some seed to become infected.

Based on incidence of seed infection and patterns of disease development, seedborne inoculum was considered epidemiologically important in North Carolina (2), Georgia (3,4), Florida (9–11), and New York (15,16). Shaner and Buechley (17) concluded that, compared to moderate epidemics, severe epidemics have a slower rate of increase but begin at an earlier stage of wheat growth and therefore have a longer time to develop. Seedborne inoculum infects seedlings shortly after emergence (5), thereby giving the longest possible time for epidemic development.

The high incidence of infection in nontreated plots by GS 9 helps to explain why foliar fungicides alone are not very effective controls for *Stagonospora* blotch. Foliar fungicides are applied at GS 8 or later, and much of the epidemic has already occurred by this time.

The cause for differences in plant stand between the two seed lots in 1996 is unknown. Both seed lots were stored under the same conditions and had 94% germination rates approximately 2 months before planting. Germination tests conducted after the stand differences were found showed that the low and high seed lots had germination rates of 74 and 95%, respectively. There was no evidence of insect damage.

Yield measurements in 1995 were confounded by scattered patches of barley yellow dwarf, deer feeding during the spring, and bird feeding before harvest. In

Table 5. Test weight (kg/m³) of grain harvested from infested and clean fields in 1995 and 1996

Treatment	1995		1996	
	Infested field ^y	Clean field ^y	Infested field ^y	Clean field ^y
Nontreated	697 c	716 b	697 a	689 ab
Triadimenol + thiram	715 b	730 a	697 a	685 b
Difenoconazole	714 b	729 a	697 a	694 ab
Propiconazole	716 b	716 b	700 a	688 ab
Difenoconazole/propiconazole	725 b	730 a	694 a	694 ab
Difenoconazole/tebuconazole 3x ^z	739 b	736 a	700 a	697 a

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P=0.05$).

^z By harvest, tebuconazole had been applied three times as a foliar spray.

Table 6. Incidence (%) of *Stagonospora nodorum* in seed harvested from infested and clean fields in 1995 and 1996

Main effect	1995		1996	
	Infested field ^y	Clean field ^y	Infested field ^y	Clean field ^y
Treatment				
Nontreated	48.8 a	28.2 a	7.8 a	5.5 a
Triadimenol + thiram	23.8 b	10.7 b	7.2 a	4.3 ab
Difenoconazole	25.5 b	8.7 b	6.0 ab	2.0 bc
Propiconazole	39.3 a	24.0 a	4.5 ab	5.8 a
Difenoconazole/propiconazole	17.7 bc	5.5 b	8.8 a	1.7 bc
Difenoconazole/tebuconazole 3X ^z	7.7 c	2.7 b	2.2 b	0.3 c
Level of seed infection				
High	29.9 a	17.7 a	6.6 a	4.2 a
Low	24.4 a	8.8 b	5.6 a	2.4 b

^y Means for treatment or level of seed infection within a column followed by the same letter are not significantly different (LSD, $P=0.05$).

^z By harvest, tebuconazole had been applied three times as a foliar spray.

1996, confounding factors were not a problem, but differences in yield were not expected because there were no differences in disease among levels of seed infection or treatments. Test weights in 1996 were low because wet weather delayed harvest.

Several recommendations for controlling *Stagonospora* blotch can be made based on the results of this research. Crop rotations and tillage that allow wheat debris to decompose before the next wheat crop along with difenoconazole or triadimenol seed treatment to reduce seedborne inoculum should be sufficient to avoid serious losses. In fields where wheat is grown every year, tillage and seed treatment would still be helpful, but a foliar fungicide at GS 8 may be necessary for adequate control. Planting a different cultivar each year may avoid selecting and increasing strains of the pathogen with a preference for a certain cultivar. Given the difficulty of producing seed with low levels of seedborne inoculum and the lack of seed testing for infection, applying an effective fungicide to seed appears to be a more efficient means of reducing seedborne inoculum.

ACKNOWLEDGMENTS

We thank the Arkansas Wheat Promotion Board for financial support, Ed Gbor for statistical ad-

vice, and Terry Kirkpatrick and J. D. Barham for assistance with field plots.

LITERATURE CITED

- Ahmed, H. U., Mundt, C. C., Hoffer, M. E., and Coakley, S. M. 1996. Selective influence of wheat cultivars on pathogenicity of *Mycosphaerella graminicola* (anamorph *Septoria tritici*). *Phytopathology* 86:454-458.
- Babadoost, M., and Hebert, T. T. 1984. Incidence of *Septoria nodorum* in wheat seed and its effects on plant growth and yield. *Plant Dis.* 68:125-129.
- Cunfer, B. M. 1978. The incidence of *Septoria nodorum* in wheat seed. *Phytopathology* 68:832-835.
- Cunfer, B. M. 1983. Epidemiology and control of seed-borne *Septoria nodorum* on wheat. *Seed Sci. Technol.* 11:707-718.
- Eyal, Z. 1981. Integrated control of *Septoria* diseases of wheat. *Plant Dis.* 65:763-768.
- Eyal, Z., Scharen, A. L., Prescott, J. M., and van Ginkle, M. 1987. The *Septoria* Diseases of Wheat: Concepts and Methods of Disease Management. CIMMYT, Mexico, DF.
- Keller, S. M., McDermott, J. M., Pettway, R. E., Wolfe, M. S., and McDonald, B. A. 1997. Gene flow and sexual reproduction in the wheat glume blotch pathogen *Phaeosphaeria nodorum* (anamorph *Stagonospora nodorum*). *Phytopathology* 87:353-358.
- Large, E. C. 1954. Growth stages in cereals. *Plant Pathol.* 3:128-129.
- Luke, H. H., Barnett, R. D., and Pfahler, P. L. 1985. Influence of soil infestation, seed infection, and seed treatment on *Septoria nodorum* blotch of wheat. *Plant Dis.* 69:74-76.
- Luke, H. H., Barnett, R. D., and Pfahler, P. L. 1986. Development of *Septoria nodorum* blotch on wheat from infected and treated seed. *Plant Dis.* 70:252-254.
- Luke, H. H., Pfahler, P. L., and Barnett, R. D. 1983. Control of *Septoria nodorum* on wheat with crop rotation and seed treatment. *Plant Dis.* 67:949-951.
- Manandhar, J. B., and Cunfer, B. M. 1991. An improved selective medium for the assay of *Septoria nodorum* from wheat seed. *Phytopathology* 81:771-773.
- McDonald, B. A., Miles, J., Nelson, L. R., and Pettway, R. E. 1994. Genetic variability in nuclear DNA in field populations of *Stagonospora nodorum*. *Phytopathology* 84:250-255.
- Milus, E. A., Penix, S. E., and Gbur, E. E. 1993. Progress of *Septoria nodorum* infection on susceptible and moderately resistant wheat cultivars. *Ark. Farm Res.* 43(1):14-15.
- Shah, D., and Bergstrom, G. C. 1993. Assessment of seedborne *Stagonospora nodorum* in New York soft white winter wheat. *Plant Dis.* 77:468-471.
- Shah, D., Bergstrom, G. C., and Ueng, P. P. 1995. Initiation of *Septoria nodorum* blotch epidemics in winter wheat by seedborne *Stagonospora nodorum*. *Phytopathology* 85:452-457.
- Shaner, G., and Buechley, G. 1995. Epidemiology of leaf blotch of soft red winter wheat caused by *Septoria tritici* and *Stagonospora nodorum*. *Plant Dis.* 79:928-938.