Differential Seed Infection of Wheat Cultivars by *Stagonospora nodorum*

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ABSTRACT

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Seed of soft white winter wheat collected from New York regional cultivar trials in 1995 and 1996 were assayed on an agar medium selective for *Stagonospora nodorum*. Incidence of seed infection varied with production environment. Relative incidence of seed infection differed significantly among cultivars and was consistent across environments. The flag leaves and ears of 12 cultivars were inoculated quantitatively at flowering in a glasshouse. Cultivars did not differ significantly in disease on the flag leaves. Incidence of seed infection for all cultivars was above 60%, but was significantly lower in Delaware and Houser than in other cultivars. Results confirm that wheat cultivars differ in their relative susceptibility to seed infection by *S. nodorum*. Resistance in wheat to seed infection by *S. nodorum* may be a useful mechanism for reducing initial inoculum in areas where infected seed is considered the primary inoculum source for *Stagonospora nodorum* blotch.

Additional keywords: glume blotch, *Leptosphaeria nodorum*, *Phaeosphaeria nodorum*, *Septoria nodorum*, *Triticum aestivum*, varietal differences

Stagonospora nodorum blotch, incited by *Stagonospora nodorum* (Berk.) E. Castell. & Germano syn. *Septoria nodorum* (Berk.) Berk. in Berk. & Broome (teleomorph *Phaeosphaeria nodorum* (E. Müll.) Hedjar. syn. *Leptosphaeria nodorum* E. Müll.), is a yield-reducing disease of wheat worldwide (8). Foliar fungicides, crop rotation, and debris management contribute to disease management, but are often economically and agronomically unfeasible (9). No commercial wheat cultivar is immune to *Stagonospora nodorum*, but some cultivars are partially resistant to foliar infection (13). Seed-transmitted inoculum may initiate epidemics (1,10,12,15,18). Therefore, the reduction of seedborne inoculum presents another tactic for managing *Stagonospora nodorum* blotch. Wheat cultivar resistance to seed infection would provide a potential means for reducing seedborne inoculum.

Incidence of seed infection by *S. nodorum* is lower in Harus than in Geneva, two wheat cultivars grown extensively in New York in the 1990s (D. A. Shah and G. C. Bergstrom, unpublished). Plant architecture and foliar susceptibility to *S. nodorum*, factors that could affect seed infection incidence, are similar in Geneva and Harus. Our observations and those of oth-

ers (4,21,22) suggest that wheat cultivars differ in resistance to seed infection by *S. nodorum*. The objective of this research was to determine whether soft white winter wheat cultivars adapted to eastern North America vary in susceptibility to seed infection by *S. nodorum*.

MATERIALS AND METHODS

Field studies. Winter wheat seed were collected from cultivar performance trials at four sites in New York in 1995 and 1996. Sites were located near Lima, Waterloo, and Ithaca. The two Ithaca sites (Helfer and Ketola in 1995, Ketola and Snyder in 1996) were less than 5 km apart. Thirteen cultivars were available for this study in 1995, and 11 were available in 1996. These cultivars represented a cross section of soft white winter wheat adapted to New York. In both years, there were three plots of each cultivar at Lima and Waterloo, and two of each cultivar at the Ithaca sites. Six equidistant rows were sown at 150 kg ha⁻¹ in 1 × 12 m plots. After harvest, 200 seed per plot were assayed, without prior surface disinfestation, on a medium (11) selective for *S. nodorum*. Monthly rainfall data were obtained from the National Climatic Data Center's published records for the Avon, Waterloo, and Ithaca climatological stations. Ithaca and Waterloo sites were within 7 km of the weather stations. The Lima sites were approximately 16 km from the Avon station.

Glasshouse studies. Seed of the cultivars AC Ron, NYBatavia, Caledonia, Cayuga, Delaware, Diana, Geneva, Harus, Houser, Karena, and Pioneer 2737W were germinated on moist blotting paper in glass

petri plates. Seedlings with disease symptoms or visibly colonized by fungi were discarded. Germinated seedlings were vernalized by incubating the petri plates at 4.4°C for 7 to 8 weeks. Blotters were re-moistened with sterile distilled water as needed. Vernalized seedlings were transplanted into a soil mix in 15-cm-diameter pots and were spaced evenly on a glasshouse bench at six seedlings per pot. Three pots were planted for each cultivar. A single pot was a replicate arranged in a completely randomized design. Plants were grown under natural glasshouse lighting supplemented with high pressure sodium lamps (400W) to give a 16-h photoperiod. Ambient glasshouse temperature varied from 15 to 29°C.

Isolate SN038NY-89 of *S. nodorum* was grown on V8 juice agar (200 ml of V8 juice, 3 g of CaCO₃, 15 g of agar per 800 ml of distilled water) under near ultraviolet light (General Electric F40 BL fluorescent bulbs) for 10 to 12 days at room temperature (22 to 24°C). Plates were flooded with sterile distilled water, and spores were dislodged by rubbing the agar surface gently with a glass rod. After approximately 5 min, the agar surface was rubbed a second time with the rod. The resulting spore suspension was decanted into a flask. Spore concentration was measured with the aid of a hemacytometer and adjusted with water to 10⁶ spores per ml. The surfactant Tween 20 (polyoxyethylene sorbitan monolaurate) was added at 1 drop per 250 ml of spore suspension.

Flag leaves and ears were spray-inoculated with the spore suspensions when the plants in individual pots reached the flowering stage. Cultivars were inoculated at the same phenological stage, although not necessarily at the same time. Flag leaves were rubbed gently before inoculation to enhance droplet retention. Leaves and ears were sprayed until just before runoff. Inoculated plants were allowed to dry for approximately 20 to 30 min. The plants were then put in a mist chamber for 48 h, after which they were returned to the glasshouse bench.

Tillers emerging after inoculation were removed by periodic trimming. Seven days after inoculation, the number of lesions per 10 cm² was counted on the flag leaves of five to eight individual tillers per pot by holding a transparent grid with 1 cm² divisions against the leaf surface. Lesions were counted per 5 cm² if the leaves were small. Fifteen days after inoculation, the percent leaf area chlorotic or necrotic was deter-

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mined on flag leaves. Assessments were made after practicing with the DISTRAIN program (20).

Mature seed were hand-harvested. Two hundred seed per replicate were plated onto *Stagonospora nodorum* agar for wheat (SNAW) medium (11) to assay for infection by *S. nodorum*. Less than 200 seed were available for nine replicates. The minimum number of seed assayed per replicate was 150.

We performed the experiment twice. Clark's Cream was added in the second experiment because with this cultivar we had observed high foliar susceptibility but low seed infection incidence by *S. nodorum* in the field (D. A. Shah and G. C. Bergstrom, unpublished).

Data analysis. Data were analyzed using SAS Proc Mixed (SAS Institute, Carey, NC). The Mixed procedure allows both fixed and random effects in the same model. Proc Mixed provides restricted maximum likelihood estimates of variance parameters, which are preferable to traditional ANOVA estimates (17).

For the regional trials, cultivar was a fixed effect; location, year, and their respective interactions with cultivar were random effects. In the glasshouse studies, cultivars were inoculated at the same phenological stage (beginning of flowering), but not necessarily at the same chronological age. It is possible that inoculation date influenced the outcome of the host-pathogen interaction because glasshouse conditions varied daily. Data from glasshouse experiments one and two were combined into a single dataset, and inoculation date was included in the model as a random effect.

Mixed linear models assume that the residual errors are normally distributed (and hence have constant variance). Data transformations to meet these assumptions were approached empirically. For the glasshouse data, a square root transformation stabilized the variances of lesion counts made 7 days after inoculation, and the arcsine square root transformation was used for the seed infection data. No transformations were needed for other datasets.

Data from regional trials were also analyzed nonparametrically. Cultivar means

for incidence of seed infection by *S. nodorum* were ranked within each location from lowest to highest. Kendall's coefficient of concordance (W), which measures the agreement in the ranks among locations, was calculated. If there is perfect agreement in the ranks of the cultivars for incidence of seed infection among the different locations, then W is 1.0. The test and statistics are described in detail (3).

RESULTS

Field studies. The incidence of seed infection by *S. nodorum* varied from less than 1% to greater than 50% in 1995 and 1996, depending on the trial's location (Table 1). Mean incidence of seed infection varied among the cultivars studied (Table 1). Incidence of seed infection by *S. nodorum* differed significantly among cultivars when data were analyzed as a mixed linear model ($P = 0.01$). Year of production accounted for less than 1% of the random variation in seed infection (Table 2). Seed production environment accounted for most of the random variation in seed infection. Year and location interactions with cultivar accounted for less than 10% each of the random variation in seed infection (Table 2). Pairwise comparisons between cultivars were made using data combined over both years since year of production was not a significant explanatory variable (Table 3). Caledonia, Cayuga, and Geneva had significantly higher incidences of seed infection than the other cultivars.

Kendall's coefficient of concordance, which measures the consistency in rank across locations, was significant in both years ($W_{1995} = 0.839$, $W_{1996} = 0.835$; $P < 0.001$). Thus, cultivars for which incidence of seed infection was low at one location

also exhibited low seed infection at other locations.

Incidence of seed infection averaged over all sampled plots was 7.8% in 1995 and 21.5% in 1996. Differences in seed infection between years and among locations may be explained, in part, by differential rainfall. In general, lower levels of seed infection occurred in locations that received less rain during stem elongation and grain development stages. Rainfall during April through June was two to four times higher in 1996 than in 1995. Among the Ithaca sites, seed infection was higher in both years at Ketola. Increased seed infection at Ketola may have been due to a microclimate more favorable for inoculum production or to enhanced spore dispersal resulting from brief, localized thundershowers that are common in the area.

Glasshouse studies. There were no significant differences among the cultivars in the number of lesions per cm^2 7 days after inoculation ($P = 0.56$) or in flag leaf disease 15 days after inoculation ($P = 0.076$). Date of inoculation accounted for 33 and 75% of the random variation in number of lesions per cm^2 7 days after inoculation and in flag leaf disease 15 days after inoculation, respectively. All cultivars developed glume blotch, but symptoms could not be differentiated visually.

Incidence of seed infection was above 60% for all cultivars but varied significantly among cultivars ($P = 0.0001$). Most differences among cultivars in seed infection were less than 10% (Table 4). Statistical significance was attributable to the lower incidences of seed infection in Delaware and Houser. Inoculation date did not account for any of the random variation in seed infection incidence.

Table 2. Covariance parameter estimates for incidence of seed infection by *Stagonospora nodorum* among wheat cultivars grown in eight different field environments

Random effect parameter	Estimate	Standard error	Proportion of total random variance (%)
Year	0.00004	0.0078	0.2
Environment	0.0192	0.0113	69.4
Year $\times$ cultivar	0.0024	0.0008	8.8
Environment $\times$ cultivar	0.0022	0.0012	7.9
Residual	0.0038	0.0004	13.7

Table 1. Incidence of winter wheat seed infection (%) by *Stagonospora nodorum* in eight different field environments

Environment	Cultivar												
	Houser	Delaware	Annett	Chelsea	Pioneer 2737W	AC Ron	NY Batavia	Karena	Harus	Caledonia	Diana	Arkona	Geneva Cayuga
Helfer-1995	1.0	0.8	2.8	5.3	6.8	6.0	8.3	9.0	10.0	10.3	20.8	18.5	15.8
Ketola-1995	11.5	11.5	19.5	19.0	29.5	30.8	34.5	36.8	42.3	53.5	50.0	39.5	56.3
Lima-1995	0.0	0.3	0.2	0.3	0.5	0.5	0.5	1.3	1.2	0.5	0.3	2.5	0.7
Waterloo-1995	0.3	0.7	1.0	1.3	1.0	1.2	1.3	2.0	1.7	2.2	1.8	4.8	3.2
Ketola-1996	22.5	...	...	24.3	31.5	25.3	44.3	24.0	23.3	57.5	26.0	...	69.0
Lima-1996	5.2	...	...	9.8	12.5	13.4	10.0	20.2	15.3	37.8	25.3	...	36.2
Snyder-1996	9.0	...	...	21.0	23.5	16.3	32.0	22.5	27.8	46.8	26.8	...	66.0
Waterloo-1996	0.7	...	...	0.5	2.5	1.7	5.8	6.8	3.2	15.5	6.5	...	10.2
Mean-1995	2.6	2.8	4.8	5.4	7.7	7.9	9.1	10.2	11.3	13.6	14.8	15.1	15.6
Mean-1996	8.1	...	...	12.2	15.5	12.8	20.0	17.4	15.8	36.9	20.1	...	41.8

DISCUSSION

Soft white winter wheat cultivars in this study differed significantly in susceptibility to seed infection by *S. nodorum*. Our results and those of Cunfer (4), Weber et al. (22), and Vanco (21) suggest that it might be fruitful to survey a broader array of wheat cultivars and lines for resistance to seed infection by *S. nodorum* in order to identify the best sources of resistance for wheat improvement. Genotypes surveyed should include lines with previously identified resistance to foliar and glume infection.

Crop architecture is a potentially important factor affecting splash dispersal of pycnidiospores and hence inoculum availability on the spikes. With the exception of Clark's Cream (has a long peduncle that causes ears to be extended further above the leaf canopy), the cultivars we studied did not differ markedly in architectural traits such as leaf angle, internode length, or peduncle length (data not shown). Crop architecture was removed as a factor in the glasshouse inoculation studies. Even after direct application of spores to the ears, relatively lower incidences of seed infection

were found in the cultivars Delaware and Houser, which was consistent with the assays of field-infected seed.

Organ-specific resistance (6) may be operational at the foliar and seed levels. In wheat, there is evidence of genetic independence of foliar and glume resistances to *Stagonospora nodorum* blotch (2,5). There is also evidence that common genes are involved in foliar and glume resistance (7). In wheat glumes inoculated with *Fusarium graminearum*, there is a greater and earlier accumulation of pathogenesis-related proteins in resistant compared with susceptible cultivars (14). The heritability and genetic nature of partial resistance to seed infection in wheat cultivars should be investigated.

Our studies were conducted with a single virulent isolate of *S. nodorum*. Future studies, especially on wheat genotypes expressing higher levels of resistance to seed infection, should address the possibility of significant isolate by cultivar interactions. Limited physiologic specialization of *S. nodorum* for virulence on leaves of wheat cultivars has been demonstrated, but field resistance to infection in wheat is

generally observed to be nonspecific, and aggressiveness is considered to be the principal attribute of pathogen populations (16).

Stagonospora nodorum blotch is an undermanaged disease in some wheat production systems where adapted wheat cultivars are susceptible and producers don't routinely apply foliar fungicides (19). In some of these production systems, such as in New York, epidemics may be initiated primarily by seedborne inoculum (19). Seedborne inoculum might be reduced significantly by the integrated use of wheat cultivars with resistance to seed infection, aggressive disease management in seed production fields, seed health testing for *S. nodorum*, and seed treatment with efficacious fungicides. Wheat cultivars with partial resistance to seed infection by *S. nodorum* could play an important role in the integrated management of *Stagonospora nodorum* blotch in regions where epidemics are initiated by seedborne inoculum.

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Table 3. Pairwise differences in least square means for seed infection (%) by *Stagonospora nodorum* for winter wheat cultivars grown in eight different field environments

	AC Ron	Annett	Arkona	Caledonia	Cayuga	Chelsea	Delaware	Diana	Geneva	Harus	Houser	Karena	NY Batavia	Pioneer 2737W
AC Ron	...	0.6	9.4	16.1** ^a	36.6***	1.2	2.9	8.3	21.2**	4.1	5.4	3.7	5.5	1.8
Annett	0.6	...	10.0	16.7*	37.2**	0.6	2.3	8.9	21.8*	4.7	4.8	4.4	6.1	2.4
Arkona	9.4	10.0	...	6.7	27.2**	10.6	12.3	1.1	11.8	5.3	14.8*	5.6	3.9	7.6
Caledonia	16.1*	16.7*	6.7	...	20.5*	17.3*	19.0*	7.8	5.1	12.0*	21.6**	12.4*	10.6	14.3*
Cayuga	36.6***	37.2**	27.2**	20.5*	...	37.8***	39.5***	28.3**	15.4*	32.5**	42.1***	32.9**	31.1**	34.8***
Chelsea	1.2	0.6	10.6	17.3*	37.8***	...	1.7	9.5	22.4**	5.4	4.2	5.0	6.7	3.0
Delaware	2.9	2.3	12.3	19.0*	39.5***	1.7	...	11.1	24.1**	7.0	2.6	6.6	8.4	4.7
Diana	8.3	8.9	1.1	7.8	28.3**	9.5	11.1	...	12.9*	4.2	13.7*	4.5	2.8	6.5
Geneva	21.2**	21.8*	11.8	5.1	15.4*	22.4**	24.1**	12.9*	...	17.1*	26.7**	17.5*	15.7*	19.4*
Harus	4.1	4.7	5.3	12.0*	32.5**	5.4	7.0	4.2	17.1*	...	9.6	0.4	1.4	2.3
Houser	5.4	4.8	14.8*	21.6**	42.1***	4.2	2.6	13.7*	26.7**	9.6	...	9.1	10.9	7.2
Karena	3.7	4.4	5.6	12.4*	32.9**	5.0	6.6	4.5	17.5*	0.4	9.1	...	1.7	2.0
NY Batavia	5.5	6.1	3.9	10.6	31.1**	6.7	8.4	2.8	15.7*	1.4	10.9	1.7	...	3.7
Pioneer 2737W	1.8	2.4	7.6	14.3*	34.8***	3.0	4.7	6.5	19.4*	2.3	7.2	2.0	3.7	...

^a Significantly different at $P = 0.1^*$, 0.01^{**} , and 0.001^{***} .

Table 4. Pairwise differences in least square means for seed infection (%) for winter wheat cultivars inoculated with *Stagonospora nodorum* in the glasshouse

	AC Ron	Caledonia	Cayuga	Clark's Cream	Delaware	Diana	Geneva	Harus	Houser	Karena	NY Batavia	Pioneer 2737W
AC Ron	...	1.14	1.43*	0.77	3.45** ^a	0.05	1.66*	0.72	7.17***	0.05	0.50	0.40
Caledonia	1.14	...	0.02	0.04	8.40***	0.70	0.05	0.05	13.63***	1.64	0.13	0.19
Cayuga	1.43*	0.02	...	0.10	9.13***	0.93	0.01	0.12	14.53***	1.98*	0.24	0.32
Clark's Cream	0.77	0.04	0.10	...	7.37**	0.42	0.17	0.00	12.34***	1.19	0.03	0.06
Delaware	3.45**	8.40***	9.13***	7.37**	...	4.35**	9.67***	7.21***	0.71	2.72*	6.48***	6.13***
Diana	0.05	0.70	0.93	0.42	4.35**	...	1.12	0.38	8.42***	0.20	0.22	0.16
Geneva	1.66*	0.05	0.01	0.17	9.67***	1.12	...	0.20	15.18***	2.25*	0.34	0.43
Harus	0.72	0.05	0.12	0.00	7.21***	0.38	0.20	...	12.15***	1.13	0.02	0.05
Houser	7.17***	13.63***	14.53***	12.34***	0.71	8.42***	15.18***	12.15***	...	6.11***	11.22***	10.77***
Karena	0.05	1.64	1.98*	1.19	2.72*	0.20	2.25*	1.13	6.11***	...	0.84	0.71
NY Batavia	0.50	0.13	0.24	0.03	6.48***	0.22	0.34	0.02	11.22***	0.84	...	0.01
Pioneer 2737W	0.40	0.19	0.32	0.06	6.13***	0.16	0.43	0.05	10.77***	0.71	0.01	...

^a Significantly different at $P = 0.1^*$, 0.01^{**} , and 0.001^{***} based on arcsine square root transformed incidence of seed infection.

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