Initiation of Septoria Nodorum Blotch Epidemics in Winter Wheat by Seedborne *Stagonospora nodorum*

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ABSTRACT

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To determine the potential contribution of seedborne *Stagonospora nodorum* to Septoria nodorum blotch epidemics, field plots that were isolated from other wheat plants or residues were sown to winter wheat with seed infection levels by *S. nodorum* of <1, 3, 10, 20, 30, and 40% in 1990-91, and 0.0, 0.5, 3, 11, 14, 19.5, 25.5, and 29% in 1991-92. In 1990-91, a season mildly conducive to Septoria nodorum blotch, even plots sown to seed with less than 1% infection by *S. nodorum* developed epidemics. Seed infection level had a significant effect on disease incidence and severity at the main shoot and three tillers growth stage ($P < 0.0001$), on the F-5 leaf position at first node stage ($P < 0.05$), on the F-1 leaf position at late milk stage ($P < 0.1$), and on the percentage of harvested seed infected by *S. nodorum* ($P < 0.05$). The above relationships of disease and seed infection level were nonlinear and asymptotic. In 1991-92, a season more conducive to Septoria nodorum blotch, epidemics were initi-

ated in plots with seed infection levels as low as 0.5%. Seed infection level had a significant effect on disease incidence at the two-leaves-unfolded stage ($P < 0.05$), but not later that season. The contribution of seedborne *S. nodorum* to epidemic initiation also was assessed, in 1990-91, by following two isolates (with DNA fingerprints distinguishable from each other and background isolates) of *S. nodorum* from infected seed through the crop canopy. Isolates with DNA fingerprints identical to those of the respective seed isolates were recovered from F-5 leaves and from harvested seed. These results showed that seedborne *S. nodorum* was at least partially responsible for initiation of Septoria nodorum blotch on the foliage. Moreover, the presence of the same isolates in the seed used for sowing and the seed harvested gave corroborative evidence that seed populations of *S. nodorum* could initiate epidemics of Septoria nodorum blotch in new locations and could provide for year-to-year perpetuation of these populations.

Additional keywords: *Leptosphaeria nodorum*, *Phaeosphaeria nodorum*, *Septoria nodorum*, *Triticum aestivum*.

Septoria nodorum blotch, a foliage, glume, and stem disease of wheat (*Triticum aestivum* L.), is caused by the fungus *Stagonospora nodorum* (Berk.) Castellani & E. G. Germano = *Septoria nodorum* (Berk.) Berk. (teleomorph: *Phaeosphaeria nodorum* (E. Müller) Hedjaroude = *Leptosphaeria nodorum* E. Müller). It occurs in most countries where wheat is grown (8–11,36,38,46), and is responsible for significant reductions in yield (1,2,9,22,28). Four possible sources of inoculum of *S. nodorum* have been reported: *1)* pycnidiospores arising from infected wheat debris (17,20,43,44); *2)* ascospores arising from infected wheat debris (13,25,34,35,41); *3)* pycnidiospores arising from infected alternative gramineous hosts (18,27,51); and *4)* infected seed (7,8,15,16,19,30,48).

The soft white winter wheat production region of New York is representative of many soft winter wheat production areas throughout eastern and central North America, and is an appropriate agroecosystem in which to assess the role of seedborne inoculum in the initiation of Septoria nodorum blotch epidemics. *Stagonospora nodorum* is the most prevalent foliar pathogen of winter wheat in New York (45). Yet, the physical and temporal isolation of wheat fields (provided by the lack of continuous wheat cropping and the presence of small, scattered wheat fields in long-term rotation with nonsuscept crops) diminishes the epidemiological role of pycnidiospores from wheat debris, an important inoculum source where wheat is cropped continuously. Pseudo-

thecia and ascospores of *Phaeosphaeria nodorum* have not been found in northeastern North American wheat fields, and even if they do occur, their epidemiological role would be diminished under the New York cropping conditions described above. *Stagonospora nodorum* has a wide host range (26), but the epidemiological significance of alternative hosts as sources of inoculum for wheat remains undetermined. Evidence from wheat disease surveys in New York appeared to rule out cultural practices and climatic variation as the determinants of variability in Septoria nodorum blotch severity among fields (45). Surveys in 1990 and 1991 showed that most winter wheat sown in New York has a potential seedborne source of inoculum of *S. nodorum* but the level of infection varied widely by the year in which the seed was produced and by the specific lot (48). Furthermore, seed-to-seedling transmission efficiencies of nearly 100% have been recorded in New York field plots sown with seed infected by *S. nodorum* (G. C. Bergstrom, *unpublished*).

The present study was undertaken to determine the potential contribution of seedborne *S. nodorum* to initiation of Septoria nodorum blotch epidemics in winter wheat.

MATERIALS AND METHODS

Field experiments. Field experiments were conducted over two winter wheat growing seasons: 1990-91 (year 1) and 1991-92 (year 2). The experimental design was a randomized complete block with four replications in year 1 and two replications in year 2. Plots were sown with seed of the cultivar Geneva, the most widely grown cultivar in New York and susceptible to *S. nodorum*. Seed used for planting year 1 experiments had been harvested

in 1990 from plots inoculated with either isolate SN052NY-89 of *S. nodorum* (seedlot 1) or isolate SN167NY-87 (seedlot 2) during the 3-wk period between growth stages 65 and 73 (52). Spikes and flag leaves were sprayed with conidial suspensions adjusted to a concentration of 10^5 to 10^6 spores per milliliter at a rate of 20–40 ml suspension per m^2. The percentages of seed from seedlots 1 and 2 infected by *S. nodorum* were 81 and 87%, respectively, as determined on oxgall medium (32). Seed were mixed with seed from lots with 3% incidence of infection by *S. nodorum* to give seed infection levels by *S. nodorum* of 10, 20, 30, or 40%. Control plots were sown with triadimenol-treated seed (Baytan 30F at 0.98 ml/kg). Therefore, in year 1, six levels of seed infection by *S. nodorum* were represented: <1 (triadimenol-treated seed, no infection detected in laboratory assays), 3, 10, 20, 30, and 40%. In year 2, seven levels of seed infection by *S. nodorum* (0.5, 3, 11, 14, 19.5, 25.5, and 29) were achieved by blending of seedlots with seed infection levels of 0.5 and 29%. Each block also contained two additional plots sown to triadimenol-treated seed with pretreatment infection levels by *S. nodorum* of 0.5 and 29% (no infection detected in laboratory assays after treatment).

Wheat was sown in the fall at 150 kg/ha at 20-cm row spacings at the Robert Musgrave Research Farm, Aurora, NY, after the harvest of oats in August of each year and preparation of a seedbed by moldboard tillage and harrowing. Plots measured 4 m × 8 m in 1991 and 2 m × 4 m in year 2. Plots were separated from each other by 4–5 m. Interplots in year 1 were fallow through the fall and winter, and were sown to oats (not susceptible to *S. nodorum*) in the spring. Interplots and borders in year 2 were sown to wheat treated with both triadimenol and guazatine (Panoctine 35 at 2.0 ml/kg). Propiconazole (Tilt 3.6E) was applied at 291 ml/ha to interplots and borders at growth stage 30 (52) in year 2.

Plant densities were measured in early spring. In year 1, two 30-cm segments within different rows of a plot were chosen arbitrarily. Plants within each 30-cm row segment were counted. Plant density (number of plants per m^2) was based on the average of the two readings. In year 2, only one 30-cm row segment per plot was measured.

In year 1, Septoria nodorum blotch incidence and severity were rated at growth stages 23, 31, 39, 77, and 83. In year 2, disease assessments were made at growth stages 12, 23, 61, 69, 75, 77, and 83. Ratings were made visually on 20 main tillers per plot, and were done separately for each leaf of these tillers for leaf positions not yet senescent. Disease incidence was defined as the percentage of plants showing symptoms of Septoria nodorum blotch; disease severity was defined as the percentage of leaf area necrotic. Each estimate was based on an average of over 20 leaves at the given leaf position. Disease severity assessments were made by the first author only, after training with the DISTRAIN computer program (developed by J. R. Tomerlin, USDA-ARS, Beltsville, MD). Characteristic lesions of Septoria nodorum blotch were not apparent before GS 30 in early May. To determine whether plants were infected, 20 plants per plot at growth stages 23 and 31 in year 1, and 10 plants per plot at GS 23 in year 2 were removed for laboratory analysis. Plants were air dried for 7 days at room temperature (20–24 C), then stored at 5 C. Chlorotic, necrotic, or lesion-bearing leaf or stem pieces from each of the plants were incubated in sterile moist chambers for 5 days under near-ultraviolet light (General Electric F40 BL fluorescent bulbs) with a 12-h photoperiod to induce development of pycnidia and pycnidiospores of *S. nodorum*. Leaf pieces were surface disinfected by washing in sterile distilled water to remove debris, followed by washing in 95% ethanol for 15 s, 1% sodium hypochlorite for 15 s, and rinsing in sterile distilled water. Plants were considered infected if pycnidia of *S. nodorum* were detected on one or more of the incubated leaf pieces.

At GS 77 and GS 83 in year 1, Septoria nodorum blotch severity ratings were confounded by the presence of powdery mildew (caused by *Blumeria graminis* (DC.) E. O. Speer f. sp. *tritici*), leaf rust (caused by *Puccinia recondita* Roberge ex Desmaz. f. sp. *tritici*), and Septoria tritici blotch (caused by *Septoria tritici*

Roberge in Desmaz.) on the flag leaf (F), and on leaves at positions below this leaf (i.e., F-1, F-2, etc.). Twenty main tillers per plot were removed for laboratory analysis. Incidence of foliar necrosis and the percentage of leaf area necrotic were recorded on the flag, F-1 and F-2 leaves at GS 77, and on the flag and F-1 leaves at GS 83. Ten lesions or necrotic areas were cut (1–1.5 cm lengths) from sampled leaves at each rated position for all plots. Leaf pieces were surface disinfected and incubated in moist chambers under near-ultraviolet light for the recovery of *S. nodorum* as described above. Mean severity of Septoria nodorum blotch at a given leaf position was calculated by multiplying the mean percentage of leaf area necrotic (as determined by visual ratings over 20 leaves at the position of interest) by the proportion of leaf pieces (out of 10) on which pycnidia of *S. nodorum* were produced.

Seed samples were stored at 5 C after harvest. Random samples of 200 seed per plot were assayed for infection by *S. nodorum* on SNAW medium (31) with Gelrite gellan gum substituted for agar (48).

Incidence and severity of Septoria nodorum blotch were found to be correlated with plant density at all growth stages. Plant density was not affected by the level of seed infection by *S. nodorum*. Analysis of covariance, with plant density as the covariate, was performed using SAS (42), to determine if the percentage of seed infected by *S. nodorum* affected incidence and severity of Septoria nodorum blotch. Analyses of covariance were done for all leaf positions and growth stages rated. A *P* value of 0.1 was chosen as the cutoff for significance. Significant effects were found only at four growth stages in year 1, and one growth stage in year 2. For these, the treatment means were first adjusted for plant density using the LSMEANS statement of SAS, and were then regressed against the percentage of seed infected by *S. nodorum* using the REG procedure of SAS (42). Linear, quadratic, and cubic models were tested. Exponential models were tested using the NONLIN procedure of SAS (42). Models that returned the highest R^2 values were considered the best descriptors of the relationship between disease and the percentage of seed infected by *S. nodorum*.

Restriction fragment length polymorphism analysis. Twenty three isolates of *S. nodorum* were obtained from seedlot 1 (harvested from a plot inoculated with isolate SN052NY-89) and another 17 from seedlot 2 (harvested from a plot inoculated with isolate SN167NY-87). Twelve isolates were obtained from seed harvested from plots adjacent to those that had been inoculated with either SN052NY-89 or SN167NY-87. These twelve isolates were assumed to represent the background population of *S. nodorum* already present in the field prior to the introduction of SN052NY-89 or SN167NY-87. All isolations were made by the transfer of hyphal tips from colonies of *S. nodorum* growing from infected seed on oxgall or Gelrite SNAW media. Transfers were made onto V8-juice agar (200 ml V8 juice, 3 g $CaCO_3$, 15 g agar per 800 ml of distilled water). Fourteen of the 17 isolates obtained from seedlot 2 were derived from single conidial transfers.

The development of Septoria nodorum blotch epidemics in field plots in year 1 was also studied by tracking the movement of the isolates SN052NY-89 and SN167NY-87 from infected seed through the crop canopy. Ten isolates of *S. nodorum* (from 9 different plots) were obtained from F-5 leaves at GS 31. Five of the isolates were from plots sown with seed from seedlot 1 and the other 5 from plots sown with seed from seedlot 2. Pycnidial development and sporulation were induced by incubating surface-disinfested leaf pieces under near-ultraviolet light (12-h photoperiod). Upon sporulation, isolations were made by transferring part of the cirrhus from a single pycnidium onto V8-juice agar. An additional six isolates were obtained from the harvested seed from one plot that had been sown to seed of seedlot 1 origin, and another three isolates obtained from harvested seed from one plot that had been sown to seed from seedlot 2. All isolations were made by the transfer of hyphal tips from colonies of *S. nodorum* growing from infected seed on oxgall or Gelrite SNAW media.

Fungal genomic DNA was isolated and digested with the restric-

tion enzyme *Eco*R1 as described previously (49). The digested DNA was hybridized with a combination of probes 6, 16, 45, 60, 74, and 153 (49).

RESULTS

Field experiments. Significant differences in Septoria nodorum blotch incidence and severity were found at GS 23 ($P < 0.0001$), the F-5 leaf position at GS 31 ($P < 0.05$), the F-1 leaf position at GS 77 ($P < 0.1$), and in the percentage of seed infected by *S. nodorum* at harvest ($P < 0.05$) in year 1 (Fig. 1), but only at GS 12 in year 2 ($P < 0.05$) (Fig. 2). In year 1, the relationships of disease and seed infection level were nonlinear and asymptotic. They were best described by equations of the form $y = A - Be^{(-cx)}$, where y represents incidence or severity of Septoria nodorum blotch and x the percentage of seed infected by *S. nodorum* at sowing. A, B, and c represent parameters determined by the fit of the equation to the data.

The severity of Septoria nodorum blotch was low in the upper canopy, even at late growth stages, although the disease was present in the lower canopy. Flag and F-1 leaves were completely free of disease at the completion of flag leaf emergence in year 1, with less than 0.5% of the F-2 leaf area exhibiting symptoms of Septoria nodorum blotch. In year 2, at the beginning of flowering, less than 0.5% of the F-2 leaf area exhibited symptoms of Septoria nodorum blotch, with the flag and F-1 leaves being completely free of disease. The severity of Septoria nodorum blotch on flag leaves averaged less than 0.5% at GS 77 in year 1, and did not increase beyond 3% by GS 83. Severe Septoria nodorum blotch did develop on the F-2 leaves by GS 77 and on the F-1 leaves by GS 83. Less than 50% of the F-1 leaf area was affected by Septoria nodorum blotch at GS 77, and averaged 14% on the flag leaves at GS 83 in year 2.

The percentage of seed infected by *S. nodorum* at harvest was negatively correlated with plant density in both year 1 and year 2 (Fig. 3), but could not be explained by disease severity in the upper canopy during the period of grain development.

Septoria nodorum blotch in plots sown with triadimenol-treated seed in year 2 did not develop until GS 77, when, on average, 31% of the F-1 leaf area was affected. However, this delay in the onset of disease did not result in a significant reduction of flag leaf symptoms or in the percentage of harvested seed infected by *S. nodorum*.

Restriction fragment length polymorphism analysis. Twenty of the 23 isolates of *S. nodorum* from seedlot 1 had the same DNA fingerprint as SN052NY-89 and one showed the same profile as SN167NY-87 (Table 1). Twelve of the 17 isolates of *S. nodorum* from seedlot 2 had the same DNA fingerprint as SN167NY-87 but none showed the same fingerprint as SN052NY-89. None
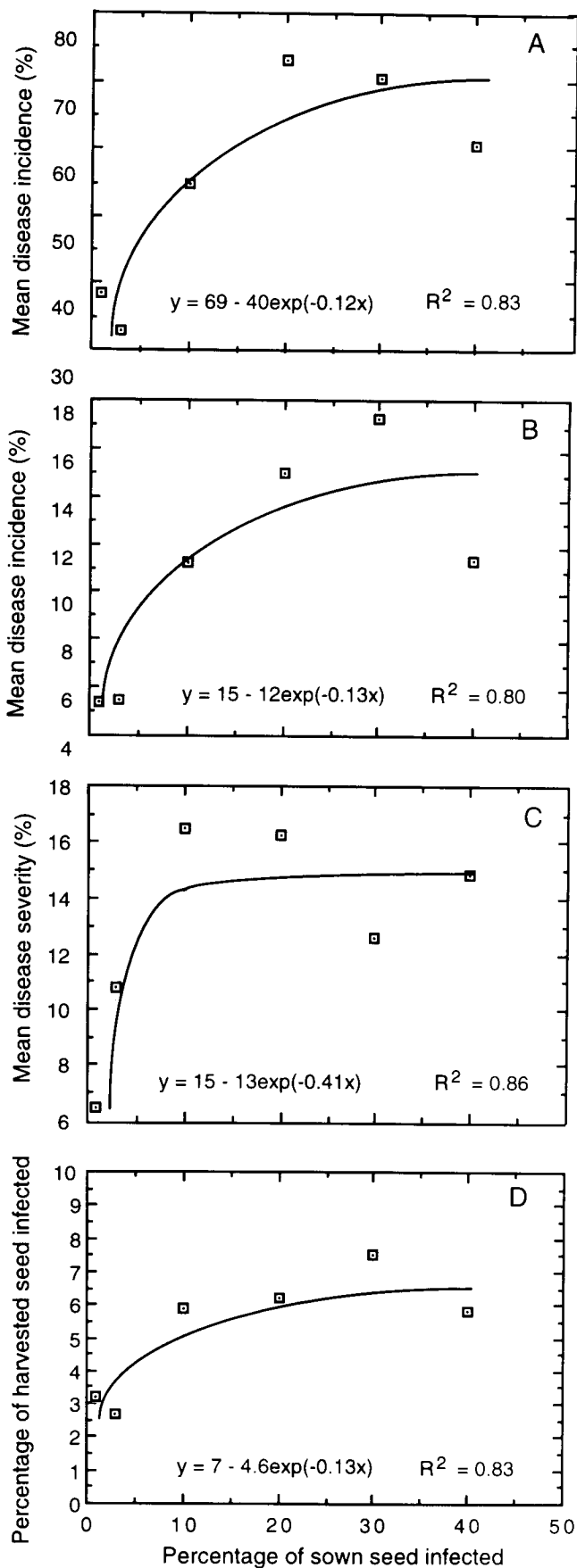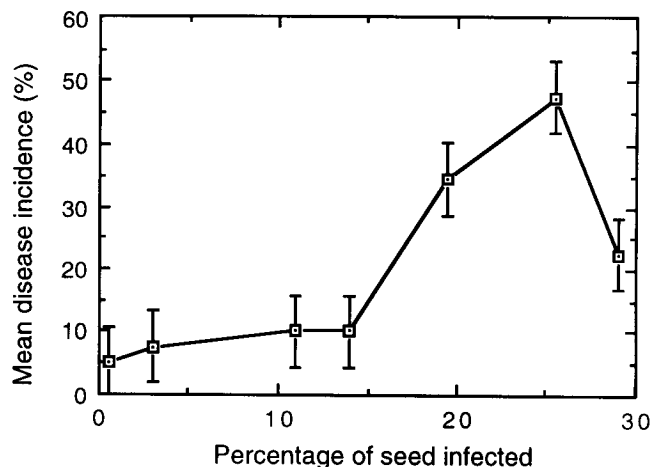
Fig. 1. Relationship between the incidence or severity of *Septoria nodorum* blotch and the percentage of seed infected by *Stagonospora nodorum* at sowing in winter wheat plots in 1990–91. **A**, GS 23; **B**, F-5 leaf position at GS 31; **C**, F-1 leaf position at GS 77; **D**, harvested seed. Each point represents the mean of eight plots.

Fig. 2. Relationship between the incidence of Septoria nodorum blotch at growth stage 12 and the percentage of seed infected by *Stagonospora nodorum* at sowing in winter wheat plots in 1991–92. Each point represents the mean of two plots. Standard error bars are shown.

of the isolates of *S. nodorum* sampled from seed harvested from plots adjacent to those that had been inoculated with either SN052NY-89 or SN167NY-87 had a fingerprint identical to either of these two isolates (Table 1). Isolates of *S. nodorum* with DNA fingerprints identical to those of SN052NY-89 and SN167NY-87 were recovered from the F-5 leaves at GS 31 and also from seed harvested from the field plots at the end of year 1 (Table 1).

DISCUSSION

Seedborne *Stagonospora nodorum* has been suggested frequently as an inoculum source for Septoria nodorum blotch (7,8,15,16,19,30,48). However, there are few reports in which foliar epidemics of Septoria nodorum blotch have been linked to seed infection by *S. nodorum*. In field studies of Septoria nodorum blotch, one major problem was relating the development of a foliar epidemic to a seedborne source of initial inoculum when there were other possible sources in the same area. In Florida, severity of Septoria nodorum blotch on the spikes, flag, and F-1 leaves during flowering and grain filling, and the percentage of seed infected at harvest, were correlated with the percentage of seed infected by *S. nodorum* at sowing (29). However, the possibility of outside inoculum sources could not be eliminated. Field experiments in Germany suggested that infected seed were the main source of primary inoculum of *S. nodorum*. Infected seed were sown in plots containing a point source of crop residue infected by *S. nodorum*. Septoria nodorum blotch was observed throughout the field, suggesting that infected seedlings, rather than the crop residue, were the main source of inoculum (39), but it is possible that disease was initiated by airborne ascospores of *Phaeosphaeria nodorum*, an important consideration in that ascospores of *P.*

Fig. 3. Relationship between the percentage of harvested seed infected by *Stagonospora nodorum* and plant density in plots of winter wheat in 1990–91 and 1991–92. Each point represents a single plot. Two hundred seed per plot were assayed on *S. nodorum* agar for wheat (SNAW) medium with Gelrite gellan gum substituted for the agar (48).

nodorum from wheat stubble were trapped in high quantities in autumn and spring in other experiments in Germany (35).

We studied Septoria nodorum blotch epidemics during two years in New York in plots isolated from other wheat plants or residue and sown to wheat seed with various levels of infection by *S. nodorum*. Significant relationships between disease at early wheat developmental stages and seed infection level suggested that seedborne inoculum of *S. nodorum* contributed to Septoria nodorum blotch epidemics in both years. Additionally, we tracked the movement of two isolates from infected seed through the crop canopy. These two isolates could be distinguished from one another and other isolates from the same seedlots by their DNA fingerprints. As a small number of isolates was sampled, we refrain from making quantitative assessments on the transmission of particular isolates of *S. nodorum* through the crop canopy. Isolates identical to SN052NY-89 and SN167NY-87 were recovered from the canopy during crop development and from the seed harvested at the end of the season. Isolates identical to SN167NY-87 were recovered at higher frequencies than those showing DNA fingerprints of SN052NY-89. Isolate SN167NY-87 produced more pycnidia and cirrhi than isolate SN052NY-89 in culture, suggesting that SN167NY-87 was the more aggressive isolate and perhaps providing an explanation for its apparently greater contribution to infection in the upper canopy. These results showed that seedborne *S. nodorum* was at least partially responsible for initiation of Septoria nodorum blotch on the foliage. Moreover, the presence of the same isolates in the seed used for sowing and the seed harvested gave corroborative evidence that seed populations of *S. nodorum* could initiate epidemics of Septoria nodorum blotch in new locations and could provide for year-to-year perpetuation of populations. McDonald et al (33) considered the finding of many different multilocus genotypes (based on DNA fingerprinting) and few widely distributed clones in a field population of *S. nodorum* to constitute evidence that sexual ascospores were the primary source of initial inoculum for a Septoria nodorum blotch epidemic. We suggest that their results might also be explained by a genotypically diverse seed population of *S. nodorum* with subsequent clonal reproduction in the field by production of pycnidiospores.

The transmission efficiency of *S. nodorum* from seeds to seedlings depends on such factors as soil moisture and soil temperature (3,4). Incidence of infected seedlings at 3 wk after sowing in year 2 (Fig. 2) indicated efficient seed-to-seedling transmission of *S. nodorum* under local soil conditions. In our experiments, the number of infected plants was, however, increased substantially by horizontal plant-to-plant inoculum dispersal prior to pseudostem elongation in both year 1 and year 2. Secondary inoculum
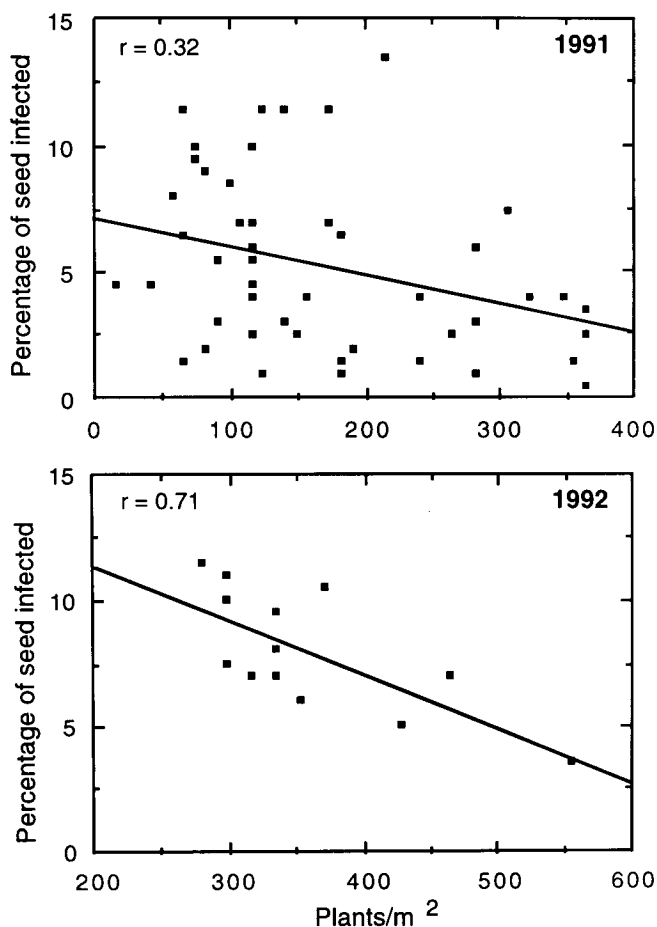
TABLE 1. DNA fingerprints of isolates of *Stagonospora nodorum* sampled from winter wheat field plots in 1990–91

Isolate used for seed inoculation[a]	Origin of recovered isolate	No. isolates with profile[b]		Total[c]
		SN052NY-89	SN167NY-87	
	Background seed[d]	0	0	12
SN052NY-89	Seedlot 1[e]	20	1	23
SN167NY-87	Seedlot 2	0	12	17
SN052NY-89	F-5 leaf	1	0	5
SN167NY-87	F-5 leaf	0	5	5
SN052NY-89	Harvested seed	2	0	6
SN167NY-87	Harvested seed	0	3	3

[a] Plots were sown to seed inoculated in 1990 with either SN052NY-89 or SN167NY-87 during grain filling.
[b] Digested DNA was hybridized with a combination of probes 6, 16, 45, 60, 74, and 153 (49).
[c] Total number of isolates screened.
[d] Seed harvested in 1990 from plots adjacent to those inoculated with either SN052NY-89 or SN167NY-87.
[e] Isolates were sampled from the seed used for sowing, F-5 leaves and the seed harvested from experimental plots representing three different stages of the epidemic.

production and dispersal could have occurred in the fall. Mature pycnidia of *S. nodorum* were observed on coleoptiles in field plots 6 wk after planting in subsequent experiments (G. C. Bergstrom, *unpublished*).

Prior research by Brennan et al (5) on the horizontal dispersal of pycnidiospores of *S. nodorum* indicated that an initial low percentage of diseased plants provided sufficient inoculum for epidemic development. Under simulated conditions, pycnidiospores of *S. nodorum* were deposited mainly within 50 cm of a target by rain splash. With a windspeed of 3 m/s, the splash zone increased to 2 m downwind, with some pycnidiospores being deposited as far as 4 m downwind (6). Griffiths and Ao (14) calculated that an epidemic could develop if as little as 0.016% of the seeds in a field gave rise to seedlings infected by *S. nodorum*. Our results suggest that their estimate may be accurate under conditions highly conducive to disease.

Weather conditions have been found to drive the development of Septoria nodorum blotch epidemics (12,50). Environment did become the predominant factor in determining the rate of increase of Septoria nodorum blotch epidemics in the upper canopy in our experiments. A decrease in the amount of initial inoculum present in the form of infected seed was sufficient to delay the onset of epidemics as well as to reduce the level of disease on upper leaves and harvested seed during the first growing season. Atypically dry conditions decreased the rate of development of Septoria nodorum blotch that season. In the second growing season, more conducive to the development of Septoria nodorum blotch, the rate of disease increase was high enough to offset an initial delay in the onset of epidemics.

Stagonospora nodorum occurred in harvested seed even following growing seasons in which Septoria nodorum blotch severity was low (48, these results). It was especially striking that *S. nodorum* was recovered from seed harvested in year 1, produced under conditions of infrequent rainfall for pycnidiospore dispersal. The absence of mature pycnidia of *S. nodorum* on the flag leaves during the grain formation period excluded them as the source of pycnidiospores for ear infection. Therefore, pycnidiospores that initiated ear (and seed) infections most likely arose from leaves lower in the canopy. Kelaniyangoda and Frauenstein (24) and Käsbohrer and Hoffmann (23) found that the F-2 and F-3 leaf positions contributed most of the inoculum for ear infection.

Plant densities varied among plots in both year 1 and year 2. In year 1, Septoria nodorum blotch severity was positively correlated with plant density in the range of 50–370 per m² but was negatively correlated with plant density in year 2 in the range of 270–560 per m² (*data not shown*). In both year 1 and year 2, lower percentages of seed infected by *S. nodorum* were harvested from plots with higher plant densities. This agreed with data reported by Orth and Grybauskas (37). Higher plant densities provide a microenvironment more conducive to the development of Septoria nodorum blotch at a given level in the crop canopy (47) but may inhibit the dispersal of pycnidiospores to the ears.

Infection of seed by *S. nodorum* ensures not only the overseasoning but also the distribution of the pathogen wherever the seed is sown. The control of seedborne *S. nodorum* to levels of 0.05% or less infected seed will require an integration of cultural and chemical methods. Control strategies in seed production fields should be aimed at preventing ear infection by *S. nodorum* and will require assessments of the F-2 and F-3 leaf positions for Septoria nodorum blotch throughout grain development. In grain production fields, Septoria nodorum blotch may be reduced by sowing seed that has been certified to contain a low (but, as yet, undetermined) percentage of infection by *S. nodorum* and that has been further reduced by treatment by fungicides effective against *S. nodorum*. Cultivar resistance, rotation with nonsuscept crops, and debris management, as well as foliar applications of fungicides, when warranted, will further reduce the level of Septoria nodorum blotch.

Infected seed as a source of initial inoculum of *S. nodorum* has not been integrated into any of the existing models that simulate Septoria nodorum blotch epidemics (12,21,40). Modeling the early stages of Septoria nodorum blotch development from in-

fected seed would help in identifying important epidemiological aspects of the disease. Models could be used to direct future research on the epidemiology and control of seedborne *S. nodorum*.

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