

The Incidence of *Septoria nodorum* in Wheat Seed

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ABSTRACT

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Wheat seed from six locations in Georgia was 0-59% infected with *Septoria nodorum*. Average incidence of infection from all samples was 10.7 and 9.4% for each of two index years. Shriveled and medium-sized seed had the highest percentage infection, but plump well-filled seed also had 8.1% infection during 2 yr. Differences in seed infection among the eight cultivars tested paralleled field ratings of earlier studies for susceptibility to glume blotch. Seed size

and infection by *S. nodorum* had no effect on seed germination. Seed infection was least in the mountain region of Georgia and greatest toward the southern part of the state. Foliar application of captafol and mancozeb fungicides reduced seed infection. Selective use of foliar and/or seed treatment fungicides and growing site may be a means for reducing seedborne *S. nodorum* in certified seed production.

Among the several sources of inoculum of *Septoria nodorum* (Berk.) Berk. (*Leptosphaeria nodorum* Müller), which causes glume blotch of wheat (*Triticum aestivum* L.), inoculum on straw residue has been considered most important (1, 12, 13, 15). However, several studies in Europe (1, 5) and South Africa (14) have shown that the pathogen also is carried on seed. Detection of seedborne *S. nodorum* is a major aspect of wheat seed health testing in Europe (4, 7, 8, 9). In North America, Machacek (10) reported the presence of *S. nodorum* in wheat seed in eastern Canada, but few studies have been reported in other regions of North America.

Septoria nodorum has caused losses estimated at 20% or more in the southeastern United States (11). The purposes of this study were to: (i) determine the level of *S. nodorum* in wheat seed at several locations in Georgia to evaluate the importance of this inoculum source, (ii) determine the association of seed infection with seed size, and (iii) assess possible control of seedborne infection by foliar application of fungicides. A preliminary report has been published (3).

MATERIALS AND METHODS

Seed survey.—Eight wheat cultivars widely grown in Georgia were selected for testing. These were: McNair 1813, Holley, Ga 1123, Coker 68-15, Arthur, Arthur 71, Oasis, and Abe. Samples of seed were collected in 1975 and 1976 at Calhoun (Mountain region), Experiment (Piedmont region), Midville, and Tifton (Coastal Plain region). In 1975, samples of the test cultivars also were collected at Plains (Coastal Plain), and in 1976 samples were collected at Athens (Piedmont). Ten samples from

Montana collected in 1975 also were tested for comparative purposes. The Montana samples were assayed only for the presence or absence of *S. nodorum*. Thirty seeds per sample were assayed initially, and if no infected seeds were found a second lot of 30 seeds was assayed.

Fungicide trials.—Field trials for fungicidal control of glume blotch were conducted at Experiment in 1975 and 1976. In 1975 Holley and McNair 1813, both highly susceptible to *S. nodorum*, received three foliar applications of mancozeb (a coordination product of zinc ion and manganese ethylenebisdithiocarbamate, Manzate 200, 1.45 kg a.i./hectare (ha) and captafol (Difolatan 4F, 1.21 kg a.i./ha). Two applications were made of benomyl (0.30 kg a.i./ha) + mancozeb (1.45 kg a.i./ha). In 1976 Holley, McNair 1813, Oasis, Arthur 71, and Holley L (a late-maturing selection from Holley) all were sprayed three times with captafol (1.21 kg a.i./ha) + mancozeb (1.45 kg a.i./ha). Fungicide applications began at boot stage (Feeke's scale value = 10.0) and subsequent applications were made every 10-14 days depending upon the frequency of rain.

Assay methods.—Preliminary studies revealed that because of a high incidence of saprobic fungi and other seedborne pathogens (e.g., *Fusarium* and *Helminthosporium* spp.) observation of coleoptile lesions alone was unreliable for confirmation of *S. nodorum* (5, 6, 9). In addition, these fungi overgrew *S. nodorum* when seeds were plated on potato-dextrose agar. Swellings caused by *S. nodorum* (5, 9) were observed occasionally on coleoptiles, but were not seen consistently enough to be a diagnostic criterion. The method chosen was a variation of the "freezing" method (9). Seeds were surface-sterilized for 10 min in 2% sodium hypochlorite, rinsed in sterile distilled water, and transferred to sterile petri dishes lined with filter paper. Filter pads were moistened with 2 ml of sterile distilled water containing oxytetracycline at 2 µg/ml to reduce bacterial growth.

The seeds were germinated at 18-20 C for 7 days under 15W cool-white fluorescent lamps (16 hr photoperiod, 2,700 lux), then frozen at -20 C for 24 hr. Observations of seedling lesions were made following an additional 7-10 days at 18-20 C in the light. Positive identification of *S. nodorum* was made by observation of pycnidia with oozing cirri ($\times 25$ magnification). Doubtful identifications were verified by staining pycnidia and conidia with lactophenol-cotton blue and observing conidia at $\times 250$.

Three lots of 50 randomly selected seeds were examined from each sample. Seeds in each sample were divided into three arbitrary categories: shriveled, slightly shriveled (medium-sized), and plump (well-filled), to assess the possible influence of infection on seed size. Seed germination was recorded to compare the relation of seed size and *S. nodorum* infection to germination.

Twenty-five randomly-selected coleoptiles and pericarps with suspected *S. nodorum* pycnidia were used to prepare conidial suspensions; these were inoculated onto Holley seedlings to verify the identity and pathogenicity of the isolates.

RESULTS

Seed survey.—Sixty-eight seed samples from six locations in Georgia were examined from the 1975 and 1976 crop. Pycnidia of *S. nodorum* developed most abundantly on the coleoptile, particularly the base. Pycnidia sometimes formed in the pericarp and in the filter paper surrounding the seedling. Severely infected seeds were recognized by a mass of grayish mycelium and bright pink tendrils of conidia which oozed from the pycnidia. Although the seeds were surface-sterilized, fungal contamination was common on germinating seedlings.

When conidia from suspected pycnidia of *S. nodorum* were inoculated onto Holley leaves, lesions appeared 10-14 days after inoculation from 24 of 25 seed samples.

Effect of location and cultivar.—McNair 1813 had the highest seed infection in four of nine location \times years tests, and this cultivar had the second highest incidence of seed infection in four other tests (Table 1). Holley ranked second overall, and along with Coker 68-15 and Ga 1123, was intermediate in percentage infected seed.

Arthur and its related cultivars, Arthur 71, Oasis, and Abe, had the lowest percentage of infected seed at all locations. Highest infection for these cultivars exceeded 16.7% only once (Table 1). Seed infection was 1% or less in 8 of 32 location-years for these cultivars and averaged 6.4% for all 33 location-years.

In general percentage infection by *S. nodorum* was greatest in seed from the southern, and lowest in seed from the northern, part of the state. Percentage infection was lowest at Calhoun, the northernmost location (Appalachian mountain region). The highest percentage infection, averaged over all cultivars, was at Experiment (central Georgia-Piedmont) and Tifton (southern Georgia-Coastal Plain). *Septoria nodorum* was not detected in any samples from Montana.

Association of seed size and *Septoria nodorum* infection with germination.—When data were summarized for both years, germination ranged from 83-86% regardless of seed size and regardless of seed infection (Table 2). *Septoria nodorum* infection was highest in medium-sized seeds, being nearly twice as high as in plump seeds (16.6% vs. 8.5%, Table 2). In 1976, infection of shriveled and medium-sized seed was the same (Table 2). Plump seed had the lowest infection both years. The composite seed infection for all samples was 10.7% and 9.4% in 1975 and 1976, respectively. Aside

TABLE 1. Percentage *Septoria nodorum* seed infection of eight wheat cultivars from various locations in Georgia

Cultivar	Calhoun ^a		Athens	Experiment		Midville		Plains	Tifton	
	1975	1976	1976	1975	1976	1975	1976	1975	1975	1976
Holley	5.0 ^b	6.7	6.7	39.0	14.7	12.0	1.3	17.0	27.0	13.0
McNair 1813	...	13.5	6.0	48.3	23.3	16.0	2.0	23.0	59.0	24.0
Ga 1123	2.0	0	4.0	15.0	15.3	6.7	0	...	10.0	13.5
Coker 68-15	2.0	4.7	4.7	13.0	32.7	19.0	4.0	15.5
Arthur	1.0	4.7	...	7.0	15.3	12.0	...	2.0	7.7	3.5
Arthur 71	1.0	0.7	4.7	1.0	11.3	4.0	0	0	8.0	13.5
Oasis	...	2.7	7.3	4.0	16.7	5.0	0	3.0	7.0	10.0
Abe	2.0	3.3	6.0	8.0	...	8.5	30.0

^aReading across from left to right, locations are listed from north to south in the State of Georgia, USA.

^bPercentage based on 150 seed assayed.

TABLE 2. Incidence of *Septoria nodorum* infection of wheat seed according to seed size

Seed size	1975			1976		
	Seed observed (no.)	Germinated seed (%)	Infected seed (%)	Seed observed (no.)	Germinated seed (%)	Infected seed (%)
Shriveled	735	85.5	12.8	2,411	84.9	10.2
Medium	590	87.3	16.6	2,890	86.0	10.4
Plump	2,349	79.3	8.5	2,840	87.0	7.7

from the size differences, there were no discernible symptoms of infection on any seeds.

Effect of years.—Weather records were obtained for the Experiment, Georgia, location only. The spring of 1975 was mild and wet (70 cm of rain between 1 February and 1 June). This period represents the period of winter dormancy to crop maturity. In contrast, the spring of 1976 was dry. There was only 6.4 cm of rain between 17 March and 1 May which is 12.7 cm below normal for that period. Wheat cultivars reached anthesis between 1-10 May, 1976, at Experiment and rain was frequent throughout May. Glume blotch reduced yield by 45% on the susceptible, late-maturing Holley L. However, the occurrence of dry weather during the jointing stage in 1976 did not favor inoculum buildup so that losses from glume blotch were lower than in 1975. The higher percentage of shriveled seed in 1976 was attributed largely to dry weather. Highest percentage of seed infection was 33% in 1976 compared to 59% in 1975 (Table 1). However, the overall incidence of infection in 1976 was only moderately lower than 1975.

Control with fungicides.—In 1975, the season with severe glume blotch, foliar application of fungicides did

not greatly reduce the incidence of seed infection in McNair 1813 (Table 3). In contrast, on Holley the incidence of seed infection was reduced by greater than 50% with the treatments which included mancozeb (Table 3). Although the reduction was statistically significant, seed infection still was relatively high, 17%. There also was no significant increase in yield or 1,000-kernel weight as a result of any fungicide treatment (B. M. Cunfer and L. R. Nelson, *unpublished*).

In 1976 captafol + mancozeb gave significant increases in yield, test weight, and 1,000-kernel weight for greater than 50% of the cultivar-treatment combinations (Cunfer and Nelson, *unpublished*). Percentage seed infection was less than 5% for all cultivars protected by fungicides, including Holley L, and 12-24.5% for control plots (Table 4).

DISCUSSION

Hewett (4) considered wheat seed infection by *S. nodorum* in England to be a significant source of inoculum. Although percentage infection varied widely among cultivars and years, the average infection among most samples did not exceed 5% (4). This compares with an average of 9.8% for all samples in the present study. Seedlings were counted as infected only when sporulating pycnidia of *S. nodorum* were found. Therefore these data should be considered as minimum seed infection percentages.

There were no differences in germination percentages of *S. nodorum* infection. Similarly, there were no differences in germination based on seed size. This confirms several reports (4, 5, 14) that *S. nodorum* has little effect on germination except at high infection levels.

Shriveled seed most likely came from tillers infected most severely by *S. nodorum*. Shriveled and slightly shriveled seed had the highest infection in both years. However, the high incidence of infection of plump, well-filled seeds in 1976 when glume blotch was less severe may mean that even relatively light *S. nodorum* infection on heads can lead to relatively high seed infection. Shriveled seed also may result from severe infection on the flag leaf or on adjacent florets. Severe flag leaf infection by *S. nodorum* at the time of heading contributes greatly to reduction of 1,000-kernel weight (2). When reduction in seed size is caused by flag leaf infection rather than head infection by *S. nodorum*, shriveled seed would not be infected.

Von Wechmar (14) found no correlation between glume discoloration and seed infection. For several years McNair 1813 and Holley consistently have been rated susceptible and Arthur and related cultivars have been rated tolerant (Nelson and Cunfer, *unpublished*). Although direct correlations cannot be made between discoloration of individual heads or the condition of individual seeds and seed infection, the rankings of cultivars for glume blotch susceptibility are reflected in seed infection data.

The high percentage of wheat seed infection by *S. nodorum* represents a substantial source of inoculum for early infection in the southeastern United States. Foliar-applied fungicides currently available that control glume blotch require several applications. Therefore, their use may not be economical in commercial fields but yield

TABLE 3. *Septoria nodorum* seed infection of wheat following foliar applications of fungicide at Experiment, Georgia in 1975

Cultivar	Treatment	Seed infection ^a (%)
McNair 1813	Control	44.5 A ^c
McNair 1813	Mancozeb (3) ^b	32 A
McNair 1813	Captafol (3)	38 A
McNair 1813	Benomyl + mancozeb (2)	34 A
Holley	Control	39 A
Holley	Mancozeb (3)	17 B
Holley	Captafol (3)	34 A
Holley	Benomyl + mancozeb (2)	17 B

^aPercentage based on 100 seeds assayed.

^bNumber in parentheses represents number of fungicide applications.

^cNumbers followed by the same letter do not differ significantly ($P = 0.05$) using Duncan's multiple range test.

TABLE 4. *Septoria nodorum* seed infection of wheat following three foliar fungicide applications with captafol + mancozeb at Experiment, Georgia in 1976

Cultivar	Percentage seed infection per treatment	
	Sprayed (%)	Control (%)
Arthur 71	1.3* ^b	13.5
Holley	2.7*	13.5
Holley L	4.5*	37.0
Oasis	0 *	12.0
McNair 1813	4.0*	24.5

^aPercentage based on 200 seeds assayed.

^bAsterisks (*) indicate that the figure is significantly different from the corresponding control ($P = 0.05$) using Duncan's multiple range test.

increases combined with lowered seed infection may prove valuable in certified seed fields. Fungicides applied as seed treatments offer a more practical method. Several compounds have been tested but more precise data on their value are needed. Further work is needed to determine the benefits of using combinations of seed production site and seed and foliar-applied fungicides to reduce seedborne *Septoria nodorum* and increase yields of wheat grown for certified seed.

LITERATURE CITED

1. BRÖNNIMANN, A. 1968. On *Septoria nodorum* Berk., the pathogen causing leaf blotch and glume blotch of wheat. *Phytopathol. Z.* 61:101-146.
2. BRÖNNIMANN, A. 1969. Causes of different degrees of tolerance to attack by *Septoria nodorum* Berk. in wheat. *Phytopathol. Z.* 66:353-364.
3. CUNFER, B. M. 1976. Incidence of *Septoria nodorum* in wheat seed. Pages 41-42 in B. M. Cunfer and L. R. Nelson, eds. Proceedings of the Septoria diseases of wheat workshop. Ga. Agric. Exp. Stn. Spec. Publ. No. 4. 69 p.
4. HEWETT, P. D. 1965. A survey of seed-borne fungi of wheat. I. The incidence of *Leptosphaeria nodorum* and *Griphosphaeria nivalis*. *Trans. Br. Mycol. Soc.* 48:59-72.
5. KIETREIBER, M. 1961. The diagnosis of *Septoria* disease of wheat grains by seed testing. *Pflanzenschutz-Berichte* 26:129-157.
6. KIETREIBER, M. 1966. Atypical symptoms of *Septoria nodorum* on wheat seedlings. *Proc. Int. Seed Test. Assoc.* 31:179-186.
7. KIETREIBER, M. 1971. Gesundheitszustand der Saatgutproben. *Landw. Forschung* 22:53-65.
8. KIETREIBER, M. 1975. Seed health situation. Pages 62-69 in Yearbook, Fed. Inst. Agric. and Seed Test., Vienna. 212 p.
9. LIMONARD, T. 1968. Ecological aspects of seed health testing. *Proc. Int. Seed Test. Assoc.* 33:343-513.
10. MACHACEK, J. E. 1945. The prevalence of *Septoria* on cereal seed in Canada. *Phytopathology* 35:51-53.
11. NELSON, L. R., D. D. MOREY, and A. R. BROWN. 1974. Wheat cultivar responses to severe glume blotch in Georgia. *Plant Dis. Rep.* 58:21-23.
12. SCHAREN, A. L. 1964. Environmental influences on development of glume blotch in wheat. *Phytopathology* 54:300-303.
13. SCHAREN, A. L. 1966. Cyclic production of pycnidia and spores in dead wheat tissues by *Septoria nodorum*. *Phytopathology* 56:580-581.
14. VON WECHMAR, M. B. 1965. Seed transmission of *Septoria nodorum* Berk. in the Western Cape Province. *S. Afr. J. Agric. Sci.* 8:737-744.
15. WEBER, G. F. 1922. *Septoria* diseases of cereals. *Phytopathology* 12:449-470.