Influence of Soil Infestation, Seed Infection, and Seed Treatment on Septoria Nodorum Blotch of Wheat

H. H. LUKE, Research Plant Pathologist, USDA, ARS, University of Florida, Plant Pathology Department, Gainesville 32611, R. D. BARNETT, Professor of Agronomy, IFAS, Agricultural Research and Education Center, Rt. 3, Box 638, Quincy, FL 32351, and P. L. PFAHLER, Professor, IFAS, Agronomy Department, University of Florida, Gainesville 32611

ABSTRACT

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 Disease 69:74-76.

Field experiments were conducted to determine the effects of soil infestation, seed infection, and seed treatment on the development of Septoria nodorum blotch of wheat. In 1981, disease development in plants grown from uninfected seed in infested soil and those grown from infected seed in uninfested soil was similar, but in 1982, disease severity was higher in infested soil plots. Disease in the control plots (uninfested soil/uninfected seed) and seed-treatment plots (uninfested soil/infected seed treated with fungicide) was significantly less than in other treatments. Seed treatment did not reduce the amount of disease on the leaves in infested soil plots. Grain harvested from seed-treatment plots in uninfested soil had lower percentages of seed infection than those harvested from infested soil plots. In 1982, the control and seed-treatment plots had higher grain yields in uninfested soil than the other treatments. In 1983, there were no yield differences among treatments at either location; however, seed treatment increased kernel weight at one location.

According to Shipton et al (15), Townsend was the first to report yield loss attributable to *Leptosphaeria nodorum* Müller, the causal agent of Septoria nodorum blotch of wheat, in 1898 in Maryland. Townsend indicated that Septoria nodorum blotch caused a 43–50% yield loss. In the early part of this century, yield loss estimates ranged from 10% in Maryland to 5% in North Carolina (15). More recently, yield losses of 31–50% (2,6) have been reported. Although yield losses are negligible during years of low rainfall, Septoria nodorum blotch has become a serious disease and has been reported to be the major factor that limits the profitable production of wheat in the southeastern United States (12).

Attempts to control Septoria nodorum blotch with resistant cultivars have not been successful because of a wide range of virulence in the pathogen and the lack of adequate resistance in the host (1,9,13,14). The failure to develop resistant cultivars led to attempts to control the disease with fungicides. Seed treatment with mercurial fungicides effectively controlled Septoria nodorum blotch (8,15). Mercurial fungicides can no longer be used in the United States; therefore, other fungicides have been tested. Kucharek (10) demonstrated that two or three foliar sprays of mancozeb resulted in a significant yield increase. Whitney (17) also reported that mancozeb increased yield. Foliar fungicides increase yield but application costs are high; therefore, their use is profitable only when wheat prices are high and disease is severe. Because of the high cost of foliar spray, a more efficient method of disease control is needed.

L. nodorum is both seedborne and soilborne. Seed is an important source of inoculum (2,3,12), but soilborne inoculum may also be a major source of infection (5,8,16). The effects of different inoculum sources must be determined in order to develop a more efficient control system. Therefore, in this paper, we report on the influence of soil infestation, seed infection, and seed treatment on disease development, seed quality, and yield loss.

Florida Agricultural Experiment Station Journal Series Paper 5557.

Accepted for publication 28 June 1984.

MATERIALS AND METHODS

Experiments were conducted at two locations (Gainesville and Quincy, FL) for 3 yr (1981–1983). Tests were arranged in a randomized block design and consisted of two soil treatments (infested and uninfested) and three seed treatments (uninfected seed, infected seed, and infected seed treated with 5 g of benomyl [Benlate 50WP] per kilogram of seed). In the 1983 test, 0.9 g of triadimenol (Baytan 30F) per kilogram of seed was also used as a seed treatment. All experiments were conducted in areas where wheat had not been grown (uninfested soil plots). Infested soil plots were established by placing infested wheat seed (0.5 g of seed per 0.3 m of row) in specified plots 1 or 2 days before planting. Seed used to infest the soil was sterilized in large flasks and infested with the pathogen about 2 mo before planting. Seed infection was determined to be 35–40% with a method reported by Cunfer (4). Seed free of *L. nodorum* was obtained by a method described in another report (12).

The four-row test plots (1.3×4 m) were separated on all sides by 9.25-m buffer zones to reduce interplot infection. Buffer zones were planted with oats (a nonhost) about 2 wk earlier than the test plots. Tests were established after a summer crop of corn or soybeans that was plowed under in September or early October. Cultural and fertilizer practices recommended for wheat culture in Florida were used. Coker 68-19 (CI 15229) wheat, a moderately susceptible cultivar, was seeded at the rate of 1 g/0.3 m of row. Tests (3 yr at two locations) were planted between 12 and 23 November.

The assessment key developed by James (7) was used to estimate the severity of disease on the glumes, flag leaf (terminal leaf), and second leaf. Disease severity was estimated on 20 plants per plot at plant growth stages 10.5–11.2 (11). Three meters of the two center rows from individual plots were hand-harvested in mid-May. Kernel weights were determined by weighing 500 or 1,000 seeds obtained from an electronic seed counter.

RESULTS

1981. Disease severity was very low because of inadequate rainfall in March and April 1981. The low percentage of disease that occurred did not affect grain yield. Nevertheless, there were significant differences among treatments in the severity of disease on the uppermost leaves and the head (Table 1). Plants originating from uninfected seed and benomyl treatment of infected seed had significantly reduced disease in uninfested soil. There were no significant differences among treatments for disease severity in infected seed plots (uninfested soil/infected seed) or infested soil plots (infested soil/uninfected seed).

1982. Weather conditions were conducive for development of Septoria nodorum blotch, and significant differences in disease, yield, and seed infection were observed (Table 2). At both locations, disease was less in control plots (uninfested soil/uninfected seed) and seed-treatment plots (uninfested soil/infected seed treated with benomyl) than in other treatments. Disease severity was higher in infested soil plots than in plots originating from infected seed. Seed infection was lower in control and seed-treatment plots than in other treatments. Erratic plant growth in some plots at Gainesville precluded yield measurements, but yields were determined in the Quincy test. The control and seed-treatment plots had higher yields than the other treatments. Grain yields and kernel weights from the infested soil (infested soil/uninfected seed) and infected seed (infected seed/uninfested soil) plots were similar.

1983. Adequate rainfall occurred, but below-normal temperatures delayed the onset of the epidemic; therefore, a low percentage of disease occurred at the end of the growing season. Although maximum disease severities were low, significant differences among treatments occurred at both locations (Table 3). The control and the seed-treatment plots had less disease on the leaves and the heads than the other treatments. A similar trend was observed for percent seed infection. At Quincy, there were no statistical differences among treatments for yield and kernel weight. The kernel weights of seed from the control plots and both seed-treatment plots at Gainesville were significantly higher than in the other treatments.

DISCUSSION

Although there is some disagreement about the importance of different sources

Table 1. Effects of infested soil, infected seed, and seed treatment on Septoria nodorum blotch development at Gainesville and Quincy, FL, during the spring of 1981

Treatments		Gainesville		Quincy	
Soil	Seed[x]	Second leaf	Terminal leaf	Second leaf	Glumes
Infested	NI	8.9[y] a[z]	4.2 a	10.1 a	5.4 a
Infested	I	10.2 a	4.9 a	9.8 a	4.1 b
Infested	I-T	8.0 a	4.7 a	10.6 a	3.8 b
Uninfested	NI	0.0 b	0.0 b	0.0 b	0.0 c
Uninfested	I	8.8 a	5.2 a	9.2 a	4.2 ab
Uninfested	I-T	0.0 b	0.0 b	0.0 b	0.0 c

[x] NI = not infected, I = 40% infected, I-T = infected and treated with 5 g of benomyl 50WP per kilogram of seed.
[y] Data are expressed as percent disease, which was estimated using an assessment method (7) that designated the maximum amount of disease as 50%. Each value was derived from 120 assessments (20 assessments per replicate, six replicates).
[z] Within columns, means with the same letter are not significantly different ($P = 0.05$) according to the new Duncan's multiple range test. Yield data were not presented because there were no statistical differences among treatments.

Table 2. Effects of infested soil, infected seed, and seed treatment on Septoria nodorum blotch development, grain yield, and seed infection at Quincy and Gainesville, FL, during the spring of 1982

Treatments		Yield	Kernel weight	Disease (%)[x]		
Soil	Seed[y]	(kg/ha)	(mg)	Terminal leaf	Glumes	Seed
			Quincy			
Infested	NI	3,694 c[z]	26 c	24 a	26 a	45 a
Infested	I	3,934 b	26 c	22 a	26 a	44 a
Infested	I-T	3,760 c	26 c	23 a	27 a	48 a
Uninfested	NI	4,944 a	30 a	6 c	15 c	10 b
Uninfested	I	3,978 bc	27 bc	18 b	20 b	46 a
Uninfested	I-T	4,529 a	28 ab	10 c	21 b	12 b
			Gainesville			
Infested	NI	...	...	34 a	28 a	28 a
Infested	I	...	...	34 a	28 a	26 a
Infested	I-T	...	...	36 a	30 a	28 a
Uninfested	NI	...	...	18 b	15 c	13 b
Uninfested	I	...	...	29 a	23 b	24 a
Uninfested	I-T	...	...	16 b	11 c	10 b

[x] Data are expressed as percent disease (disease severity) and percent seed infected. Percent disease was estimated using an assessment method (7) that designated 50% as the maximum amount of disease.
[y] NI = not infected, I = 40% infected, and I-T = infected and treated with 5 g of benomyl 50WP per kilogram of seed.
[z] Within columns and each location, means with the same letter are not significantly different ($P = 0.05$) according to the new Duncan's multiple range test.

Table 3. Effects of infested land, infected seed, and seed treatment on Septoria nodorum blotch development, kernel weight, and seed infection at Quincy and Gainesville, FL, during the spring of 1983

Treatments		Kernel weight (mg)	Disease (%)[x]			
Soil	Seed[y]		Second leaf	Terminal leaf	Glumes	Seed
			Quincy			
Infested	I	28 a[z]	25 a	12 a	15 a	44 a
Uninfested	I	28 a	17 a	11 a	13 a	34 a
Uninfested	NI	28 a	5 b	4 b	9 b	16 c
Uninfested	I-Tr	29 a	5 b	2 b	7 b	11 c
Uninfested	I-Be	29 a	6 b	4 b	9 b	12 c
			Gainesville			
Infested	I	25 a	...	13 a	15 a	11 a
Uninfested	I	25 a	...	9 a	15 a	9 a
Uninfested	NI	26 b	...	2 b	5 b	2 b
Uninfested	I-Tr	26 b	...	1 b	6 b	2 b
Uninfested	I-Be	27 b	...	0 b	5 b	1 b

[x] Data are expressed as percent disease (disease severity) and percent seed infected. Percent disease was estimated using an assessment method (7) that designated 50% as the maximum amount of disease.

[y] NI = not infected, I = 40% infected, I-Be = infected and treated with benomyl 50WP (5 g/kg of seed), and I-Tr = infected and treated with triadimenol 30F (0.9 g/kg of seed).

[z] Within columns and each location, means with the same letter are not significantly different ($P = 0.05$) according to the new Duncan's multiple range test. Yield data were not presented because there were no statistical differences among treatments.

of inoculum of *L. nodorum* on subsequent disease development, it is generally accepted that this fungus is both seedborne and soilborne (15). A 31% yield reduction occurred in North Carolina when plants were grown from infected seed (2). Other workers reported that seed infection was not related to disease development (5,18). In our tests, both soil infestation and seed infection resulted in about the same amount of disease when disease was light to moderate. Infested soil induced more disease than seed infection when disease was severe. It is very difficult to assess the effectiveness of soil infestation on subsequent disease because of the different methods used to infest soil and differences in environmental conditions during the development of an epidemic. Inoculum placed in the soil at planting (mid-November) caused severe disease and significantly reduced grain yield (Table 2). In North Carolina, infested straw placed in the plots at planting (October–November) had no more disease than the uninoculated control (13). Soilborne inoculum seems to influence disease severity in Florida, but this inoculum source may be of little consequence in North Carolina.

In recent reports from the southeastern United States, infected seed was a major source of inoculum (2), and benomyl seed treatment effectively reduced disease severity after 2 yr of rotation (12). In our tests, seed treatment of infected seed also significantly reduced the severity of disease on the leaves in uninfested soil plots. In 1982, benomyl seed treatment increased grain yield and kernel weight in uninfested soil plots but did not increase yield in infested soil. In 1983, seed treatment did not increase grain yield, but both benomyl and triadimenol seed treatments increased the kernel weight at one location. The increase in kernel weight occurred only when seed was planted in uninfested soil. This observation confirms reports that seed treatment was an effective measure against seed transmission of *L. nodorum* (8,16). It therefore appears that seed treatment effectively reduces the amount of disease when the major source of inoculum is from seed but may not be of much value when the soil is heavily infested.

A significant positive correlation between disease on the glumes and seed infection was obtained when disease severity was low (<20%) (4,12). At Gainesville (1983), the percentage of disease on the glumes (15%) and the percentage of seed infection (10%) was low, but a high percentage (44%) of seed infection was obtained at Quincy when disease severity on the glumes was low (15%). The wide differences in the amount of seed infection at the two locations (Table 3) was perhaps due to differences in the environment at the two locations toward the end of the growing season. This supposition is based on a report by Cunfer and Johnson (4), who stated that infections that occurred just before maturity did not cause symptoms on the glumes but resulted in seed infection that often exceeded 40%.

Seed treatment is effective when the primary source of inoculum is from seed but is ineffective when soil is heavily infested. We therefore recommend seed treatment in conjunction with 2 yr of crop rotation to control Septoria nodorum blotch of wheat. This practice should eliminate or reduce the number of fungicidal sprays required to control this disease.

LITERATURE CITED

1. Allingham, E. A., and Jackson, L. F. 1981. Variation in pathogenicity, virulence, and aggressiveness of *Septoria nodorum* in Florida. Phytopathology 71:1080-1085.
2. Babadoost, M., and Hebert, T. T. 1982. Seed-borne incidence of *Septoria nodorum* and the effect of environmental conditions on wheat seedling infections. (Abstr.) Phytopathology 72:975.
3. Baker, C. J. 1970. Influence of environmental factors on development of symptoms on wheat seedlings grown from seed infected with *Leptosphaeria nodorum*. Trans. Br. Mycol. Soc. 55:443-447.
4. Cunfer, B. M., and Johnson, J. W. 1981. Relationship of glume blotch symptoms on wheat heads to seed infection by *Septoria nodorum*. Trans. Br. Mycol. Soc. 76:205-211.
5. Doling, D. A. 1961. Glume blotch in wheat varieties, 1958. Plant Pathol. 10:24-27.
6. Eyal, Z. 1981. Integrated control of Septoria diseases of wheat. Plant Dis. 65:763-768.
7. James, W. C. 1971. A manual of assessment keys for plant disease. Can. Dep. Agric. Publ. 1458. 80 pp.
8. Jenkyn, J. F., and King, J. E. 1977. Observations on the origins of *Septoria nodorum* infections of winter wheat. Plant Pathol. 26:153-160.
9. Kleijer, G., Fossati, A., and Paccaud, F. X. 1980. Genetic analysis of tolerance to *Septoria nodorum* in wheat using chromosome substitution lines. Z. Pflanzenzuecht. 85:287-293.
10. Kucharek, T. A. 1977. Profitable control of foliar diseases of wheat by aerial application of zinc-maneb complex fungicides. Plant Dis. Rep. 61:71-75.
11. Large, E. C. 1954. Illustration of the Feekes scale growth stages in cereals. Plant Pathol. 3:128-129.
12. 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.
13. Rufty, R. C., Hebert, T. T., and Murphy, C. F. 1981. Variation in virulence in isolates of *Septoria nodorum*. Phytopathology 71:593-596.
14. Scharen, A. L., and Eyal, Z. 1983. Analysis of symptoms on spring and winter wheat cultivars inoculated with different isolates of *Septoria nodorum*. Phytopathology 73:143-147.
15. Shipton, W. A., Boyd, W. R. J., Rosielle, A. A., and Shearer, B. I. 1971. The common Septoria diseases of wheat. Bot. Rev. 37:231-262.
16. von Wechmar, B. 1965. Seed transmission of *Septoria nodorum* in the western cape province. S. Afr. Agric. Sci. 8:737-744.
17. Whitney, G. 1983. Cereal disease control research. Plant Dis. 67:587.