Development of Septoria Nodorum Blotch on Wheat from Infected and Treated Seed

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ABSTRACT

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Effects of seed infection on development of Septoria nodorum blotch on the upper leaves and heads of wheat was assessed at two locations over 3 yr. As the incidence of seed infection at planting increased from 1 to 40%, the intensity of subsequent disease increased but the relationship of seed infection to disease was nonlinear. About 10% seed infection supplied sufficient inoculum to cause a severe epidemic. In 1983, disease severity on the head was similar at two widely separated locations but percentages of seed infection at harvest were greatly different. Thus, percent seed infection at harvest seems to be influenced by environmental conditions in the heading phase of crop development. Seed treatments (benomyl or triadimenol) resulted in reduced disease severity on the upper leaves and heads at both locations in all 3 yr.

Increased wheat production in the southeastern United States has favored serious disease (Septoria nodorum blotch) caused by *Leptosphaeria nodorum* E. Müller (*Septoria nodorum* Berk.). Losses attributed to this pathogen have been reported to range from 31 (1) to 53% (6). Septoria nodorum blotch is one of the factors that limits the profitable production of wheat in the southeastern United States (10). Environmental conditions ideal for disease development, lack of resistant cultivars, and chronic seed infection are the major factors that contribute to severe disease.

Seed infected by *L. nodorum* was considered to be the primary source of inoculum (1,2,10). In the southeastern United States, seed infection was chronic and varied from 40 to >50% (3,10). Although mercurial fungicides effectively controlled Septoria nodorum blotch (8,13), these compounds cannot be used in the United States. We therefore designed experiments to determine the effects of percent seed infection and seed treatment on disease severity.

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MATERIALS AND METHODS

Experiments were conducted at two locations (Gainesville and Quincy, FL) for 3 yr (1982–1984). Tests were arranged in a randomized block design and consisted of five levels of seed infection and two seed treatments. Either five or six replicates were used in these experiments. Different percentages of seed infection were obtained by mixing infected with noninfected seed. Levels of seed infection were 0, 1, 10, 20, and 30%. Seed free of *L. nodorum* was obtained with a method described in another report (10). Benomyl (Benlate 50WP, 5 g/kg of seed) was used to treat the seed in 1982, and triadimenol (Baytan 30F, 0.9 g/kg of seed) was used in 1983 and 1984. Before treatment, infection of seed was determined to be about 30% with a method reported by Cunfer (3).

Each four-row test plot (1.3×4 m) was separated on all sides by a 9.25-m buffer zone to reduce the spread of the pathogen between plots. Oats (*Avena sativa*), a nonhost species, were planted in the buffer zones about 2 wk before the wheat.

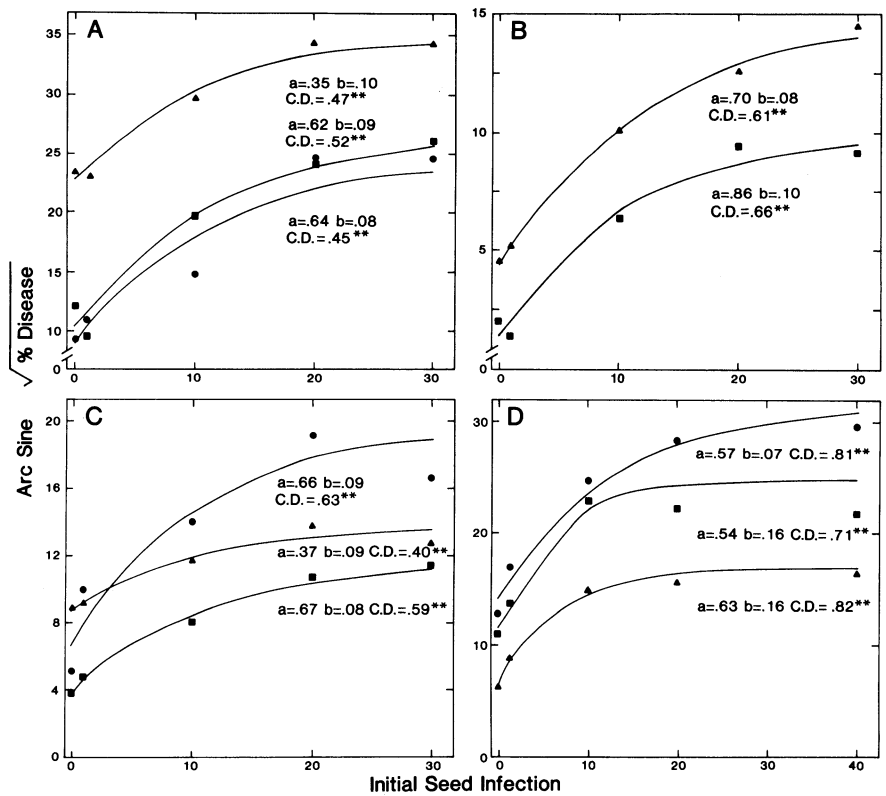

Fig. 1. Effects of percent seed infection at planting on the development of Septoria nodorum blotch on the upper leaves and heads of Coker 68-19 wheat at two locations in Florida. A = Gainesville, 1982; B = Gainesville, 1983; C = Quincy, 1983; D = Quincy, 1984. The model (12) used for data analysis had the following characteristics: $y = y_{max}\,[1 - a \exp(-bt)]$, where y = arc sine percent disease, y_{max} = arc sine maximum percent disease, a = position parameter for seed infection, b = rate parameter, and t = percent seed infection at planting. ▲ = % disease on head, ■ = % disease on flag leaf, and ● = % disease on second leaf.

Tests were established after the summer crop of either corn or soybeans was plowed under in September or early October. Cultural and fertilizer practices recommended for wheat culture in Florida were used. Wheat cultivar Coker 68-19 (CI 15229), a moderately susceptible cultivar, was seeded at the rate of 1 g/0.3 m of row. Tests were planted between 14 and 20 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 (9).

RESULTS AND DISCUSSION

When disease on the upper leaves and head was plotted versus percent seed infection at planting, the response was nonlinear, i.e., the disease intensity tended toward an asymptote at about 0.2 $< y_{max} <$ 0.35. Therefore, the data were fitted to a nonlinear model (von Bertanlanffy) of the form $y = y_{max}[1 - a \exp(-bt)]$, where y = arc sine percent disease, y_{max} = estimated maximum disease for the particular set of values set at about 105% of observed maximum disease, a = constant of integration, b = rate parameter, and t = percent seed infection at planting (12). This model provided an improved coefficient of determination and near-random distribution of residuals compared with a linear equation. The b parameter was useful because it gave a comparison of the relationship between seed infection at planting and disease development on the upper leaves and head throughout the epidemic. Because the b values (0.07–0.1) were similar, the relationship between percent seed infection at planting and disease development was fairly consistent from year to year and from location to location. The y_{max} parameter was useful because it gave a better fit of the data to the model. The use of y_{max} was justified because all curves were asymptotic at y values considerably <1.

When the maximum amount of disease on the head (Fig. 1) was compared with maximum amount of seed infection at harvest (Fig. 2), contrasting results were obtained. At Gainesville in 1982 and 1983, disease severity on the head was similar to the percentage of seed infection at harvest. At Quincy in 1983 and 1984, however, low percentages of disease on the head resulted in high percentages of seed infection at harvest. In 1983, disease severity was similar at both locations but the percentages of infected seed at Quincy were more than double that at Gainesville. Cunfer and Johnson (4) also observed wide variations in the amount of seed infection from different locations. The cause of wide variation in percent seed infection is unknown, but we suspect it is the differences in environmental conditions near the end of the growing season.

As the percentage of seed infection at planting increased, disease on the upper leaves and head also increased (Fig. 1). We could not, however, assess the effects of 1% seed infection because of contamination of the noninfected seed plots. Infection occurred in plots derived from noninfected seed even though distance between plots was about 10 m and a nonhost crop (oats) was planted in the interplot areas. We do not know the cause of interplot contamination. Distances >10 m and seed devoid of the

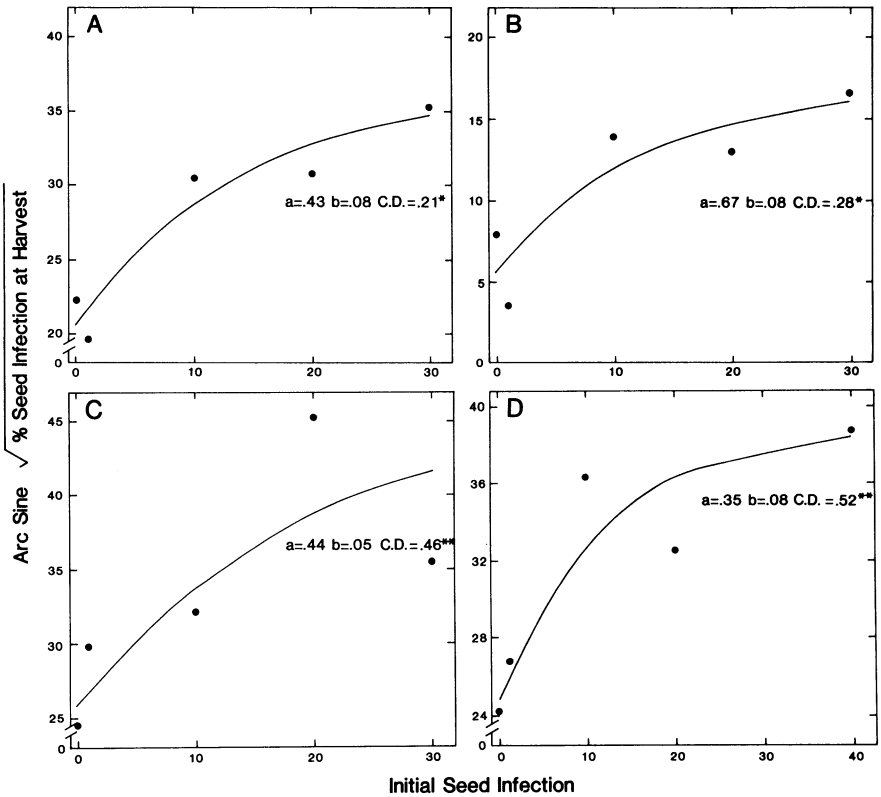

Fig. 2. Effects of percent seed infection by *Leptosphaeria nodorum* at planting on percent seed infection at harvest at two locations in Florida. A = Gainesville, 1982; B = Gainesville, 1983; C = Quincy, 1983; D = Quincy, 1984. The model (12) used for data analysis had the following characteristics: $y = y_{max}[1 - a \exp(-bt)]$, where y = arc sine percent seed infection at harvest, y_{max} = arc sine maximum percent seed infection at harvest, a = position parameter for seed infection, b = rate parameter, and t = percent seed infection at planting.

Table 1. Effects of seed treatment on the development of Septoria nodorum blotch on wheat at two locations in Florida

Location Plant part	Percent disease[a]		Significance (P)[c]
	Treated[b]	Untreated	
	1982		
Gainesville			
Head	23.0	34.4	<0.001
Flag leaf	15.1	26.1	0.020
Second leaf	10.0	24.8	0.001
	1983		
Gainesville			
Head	5.1	14.9	0.028
Flag leaf	0.4	9.3	0.009
Quincy			
Head	4.2	12.8	0.017
Flag leaf	4.1	11.5	0.001
Second leaf	5.6	16.8	<0.001
	1984		
Quincy			
Head	7.7	16.4	0.013
Flag leaf	12.9	21.7	0.017
Second leaf	17.4	29.9	0.019

[a] Data are expressed as arc sine of percent disease severity. Disease was estimated with an assessment method (7) that designated 50% as the maximum amount of disease.

[b] Benomyl 50WP (5 g/kg of seed) was used as a seed treatment in 1982 and triadimenol 30F (0.9 g/kg of seed) was used in 1983 and 1984. Treated and nontreated seed were 30% infected.

[c] Based on treated vs. untreated in paired comparisons.

pathogen are apparently needed for disease-free plots. Because *L. nodorum* occurs on native grasses (5,11) and may be dispersed by flying insects, disease-free plots could be difficult to obtain.

Seed treatment with benomyl and triadimenol effectively reduced Septoria nodorum blotch on the upper leaves and head at two locations over a 3-yr period (Table 1). These fungicides also significantly reduced the amount of disease even when the epidemic was severe. We therefore recommend seed treatment, because seed grown in the southeastern United States is heavily infected (3,4,10) and a low percentage (about 10%) of seed infection is sufficient to establish a severe epidemic (Fig. 1).

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