Effect of Seeding Date of Winter Wheat on Incidence, Severity, and Yield Loss Caused by Cephalosporium Stripe in Kansas

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ABSTRACT

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A 2-yr field trial was established to study the effects of delayed planting of winter wheat on Cephalosporium stripe (Cs) incidence, severity, and yield loss. There was a significant reduction in Cs incidence with delayed planting in one of the years; however, in the other year, incidence, disease severity, and percentage of yield loss caused by Cs were not significantly affected. Furthermore, there was a 13.7% yield reduction for uninoculated plots with each week of delay beyond the optimum planting date. Thus, in some years under Kansas conditions, a reduction in Cs incidence can be expected with delayed seeding, but this benefit is negated by loss of crop yield potential associated with this practice. The influence of planting date on host resistance reaction to Cs, host ontogeny, and rate of systemic symptom expression was also determined.

Additional key words: *Cephalosporium gramineum*

Cephalosporium stripe (Cs) of winter wheat (*Triticum aestivum* L.) is a severe systemic disease caused by the soilborne fungus *Cephalosporium gramineum* Nisikado & Ikata (*Cg*) (=*Hymenula cerealis* Ell. & Ev.). Cs is widely distributed in the United States (8) and can be a yield-reducing factor in a continuous-cropping system (2) or under summer fallow conditions (4). First reported in Kansas in 1972 (18), Cs has increased in importance so that there was an estimated loss of 136 million kilograms each year from 1976 through 1981 (15). Incidences approaching 100% have been observed in some production fields.

The most effective controls for Cs are crop rotation (4,7) and proper management of residue (2). Although effective levels of resistance to Cs have been reported (10,12), there are currently no popular commercial cultivars in Kansas with high levels of resistance and there are no chemicals registered for Cs control.

Another reported control for Cs is delayed fall planting. Bruehl (4), working in Washington, stated that losses to Cs were more severe in early-seeding-date trials as a result of increases in percentage of *Cg* infection. He observed more severe stunting and more complete systemic invasion of the host plant in infected early-seeded plants. Likewise, Pool and Sharp (14), demonstrated that early fall planting in Montana allowed for development of a more extensive root system, which resulted in higher Cs incidences than late plantings. The higher incidence of Cs was due to higher average soil temperatures 14 days after seeding, which they hypothesized provided more potential infection sites as a result of root injury caused during soil heaving (1,4) and root freezing (1). In a 4-yr study conducted in Michigan, Wiese and Ravenscroft (17) also reported reduced Cs incidence in late-planted winter wheat.

This study was undertaken to determine the effects of delayed seeding of winter wheat on 1) incidence of Cs under Kansas conditions, 2) yield loss to Cs, 3) crop yield potential in the absence of Cs, 4) resistance reaction of cultivars to Cs, 5) the effect of Cs on host ontogeny, and 6) the rate and degree of systemic spread of *Cg* in infected tillers.

MATERIALS AND METHODS

A 2-yr field experiment was conducted at Rocky Ford Experiment Field near Manhattan, KS, in a Chase silty clay loam soil (pH 6.2) that had not been cultivated to wheat for 2 yr and was not infested with *Cg*. Four planting dates were staggered 2 wk apart in both years, starting at the end of September and ending during the first week of November. The recommended optimum planting date for the Manhattan area is 7 October.

For the 1980–1981 season, three hard red winter wheat cultivars were selected on the basis of their agronomic characteristics and host reaction to Cs: Sturdy (CI13684), a semidwarf, early-maturing cultivar highly susceptible to Cs; Newton (CI17715), a medium-maturing semidwarf grown extensively in Kansas for its soilborne wheat mosaic virus resistance, moderately susceptible to Cs; and CC18-4, a tall, late-maturing breeding line with relatively low susceptibility to Cs.

Inoculum consisted of autoclaved oat kernels infested with six isolates of *Cg* (9). Twenty grams of inoculum was mixed with 7.8 g of wheat seed at planting and introduced in the furrow. An equal amount of autoclaved oats not colonized by *Cg* was used in control plots.

The experimental design was a randomized split plot with five replicates. Each cultivar was hand-planted on a given seeding date in single 3.85-m rows, with the inoculated treatment paired with the control. Those treatments were separated from each other by single rows of uninoculated wheat, and all rows were spaced 0.3 m apart. The cultivar/planting date made up the main plot while the treatment comprised the subplot.

During the 1981–1982 season, the three cultivars used were Sturdy, Arkan, and Crest Line Row Component (LRC) 40 (MT 7579). Sturdy is susceptible, whereas Arkan and Crest LRC 40 (12) are moderately susceptible, to Cs.

The experimental design was a randomized split plot with five replicates; the main plot was the treatment (inoculated/control) and the subplot was the planting date and cultivar. Each cultivar was planted in a single row 3.85 m long and separated by single border rows spaced 0.3 m apart. The inoculum consisted of 5 kg of infested oats tilled to a 10-cm depth in each of five 10-m^2 areas. An equivalent amount of uninfested autoclaved oats was tilled into the soil in five separate areas to serve as controls.

Infection percentages were estimated in late spring, from heading through flowering (growth stages [gs] 13–18) (16), by counting the number of symptomatic tillers in a population of 50 randomly chosen tillers in each plot.

Observations of disease severity were made every 3–4 days on each plot, starting at the end of jointing (gs 9) and continuing until harvest. Twenty-five randomly chosen tillers showing Cs symptoms were tagged at gs 9 (16) and scored periodically for disease severity based on the degree of systemic symptom

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expression caused by the pathogen (3). Because the system of measuring disease severity used here uses only infected tillers, the severity score has been termed systemic spread index (SSI).

The effect of planting date and Cs infection on host ontogeny was assessed by estimating the gs (16) of each plot every 3–4 days. All plots were hand-harvested when mature and yields adjusted to 12% moisture. Heavy rain and hail shortly before harvest prevented yield determinations for the 1981–1982 season. Yield loss during 1980–1981 was calculated by subtracting the yield of a given cultivar/planting date from the appropriate control yield and represented as a percentage.

RESULTS

During the 1980–1981 season, infection percentages did not differ among planting dates (Table 1). During the 1981–1982 season, however, a significant decrease in percentage of infection was observed with delayed fall planting (Table 2). Control plots had less than 5% incidence.

The presence of *Cg* significantly reduced yields relative to uninoculated treatments; however, the percentage of yield reduction for each planting was not significantly different (Table 3).

During the 1980–1981 season, the yield of the uninoculated plots for the 3 November planting date was severely reduced compared with the yields of earlier planting dates (Table 3). For the three cultivars combined, an average 13.7% yield reduction resulted for every week of delay in planting past the optimum planting date (7 October). The highest yields for the inoculated plots came from the earliest planting date (23 September), with an average of 11.1% reduction in yield for each week of delay in planting past that date (Table 3).

Table 1. Influence of planting date on percentage of infection of three winter wheat cultivars by *Cephalosporium gramineum* in 1980–1981

	Percentage of infection[x]			
Planting date	**Sturdy**	**Newton**	**CC1078-4**	**Mean**
23 September	69.2 a[y]	56.0 abc	29.2 c	51.5 d[z]
7 October	63.6 ab	45.2 ab	42.4 abc	50.4 d
21 October	69.6 a	36.4 bc	31.2 c	45.7 d
3 November	79.8 a	45.6 ab	33.6 bc	50.0 d
Mean	68.3 e[z]	45.8 f	34.1 g	

[x] Percentage of infection determined between heading and flowering.

[y] Values followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

[z] Row and column means followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

Table 2. Influence of planting date on percentage of infection of three winter wheat cultivars by *Cephalosporium gramineum* in 1981–1982

	Percentage of infection[x]			
Planting date	**Sturdy**	**Arkan**	**CLR 40**	**Mean**
27 September	99.6 a[y]	96.4 ab	85.2 cd	93.7 a[z]
12 October	98.0 a	89.2 bcd	82.4 d	89.9 a
26 October	91.6 a	70.8 e	69.2 e	77.2 b
4 November	69.2 e	62.4 ef	58.8 f	63.5 c
Mean	89.6 g[z]	79.7 h	73.9 i	

[x] Percentage of infection determined between heading and flowering.

[y] Values followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

[z] Row and column means followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

Table 3. Influence of planting date and *Cephalosporium gramineum* inoculation on yield of three winter wheat cultivars in 1980–1981

		Yield (g/plot)[y]				Yield reduction
Planting date	**Treatment**	**Sturdy**	**Newton**	**CC1078-4**	**Mean**	**(%)**
23 September	Control	242 b[z]	486 b	393 a	374 ab	
	Inoculated	109 cd	264 cd	203 bc	192 c	49 a
7 October	Control	375 a	506 a	428 a	436 a	
	Inoculated	63 d	222 cd	182 b	155 c	64 a
21 October	Control	472 a	307 bc	277 b	352 b	
	Inoculated	42 d	153 de	201 bc	132 cd	62 a
3 November	Control	184 bc	138 e	271 b	198 c	
	Inoculated	15 d	56 e	122 c	64 d	67 a

[y] Average yield for five replicates in grams per plot.

[z] Values within a column followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

In either year, delayed seeding did not significantly change cultivar reaction to Cs with respect to percentage of infection, and the ranking of cultivars remained the same (Tables 1 and 2).

Date of planting had a marked effect on growth and development of the host in all plots, although only data for the cultivar Sturdy collected during 1980–1981 are presented (Fig. 1). There was a considerable lag in development of late-planted wheat compared with early-planted wheat so that heading date (gs 15) was delayed an average of 3 days for every 2-wk delay in planting (Fig. 1). The inoculated plots showed a retardation of growth and development compared with the control plots of the same planting date from the vegetative stages to kernel development (gs 25). This was followed by accelerated maturation of the caryopsis in Cs-infected tillers (Fig. 1).

Combined data for all three cultivars during 1980–1981 show that planting date did not significantly affect systemic development of symptoms (SSI) (Fig. 2). The small scattering between planting dates at a particular growth stage supported a hypothesis that they represented one line with a common slope and y intercept. The alternative hypothesis was tested and rejected ($P = 0.05$). Although not presented in this paper, similar data were generated for the 1981–1982 season.

DISCUSSION

Delayed planting significantly reduced the percentage of infection in one of the years studied. The different time of application and placement of inoculum for the 2 yr may explain the different results, although additional research is necessary to determine these effects on Cs incidence. Reduced incidence with late

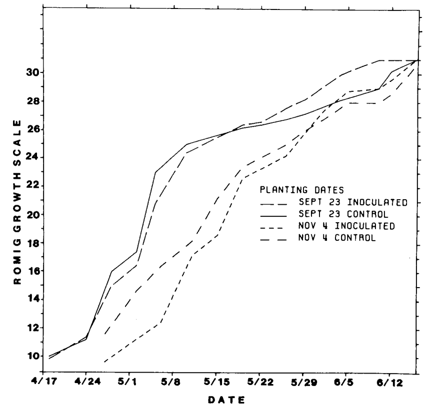

Fig. 1. Influence of planting date and *Cephalosporium gramineum* inoculation on development and maturation of the winter wheat cultivar Sturdy, where growth stage (gs) 15 = heading 95% complete, gs 26 = late milk, gs 29 = late dough, and gs 31 = harvest ripe. Each point is the mean of five replicates.

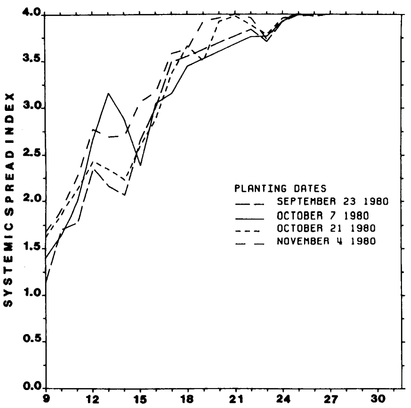

Fig. 2. Effect of planting date of winter wheat on systemic development of Cephalosporium stripe symptoms at various host growth stages (gs), where gs 15 = heading 95% complete, gs 26 = late milk, gs 29 = late dough, and gs 31 = harvest ripe. Systemic spread index determined by rating the top four leaves of infected tillers for symptoms, where 4 = flag leaves showing symptoms; 3 = flag leaf healthy, penultimate leaf showing symptoms; 2 = top two leaves healthy, third leaf showing symptoms; 1 = top three leaves healthy, fourth leaf showing symptoms; and 0 = top four leaves healthy. Each observation represents the mean rating from 375 infected tillers.

planting has been attributed to reduced root growth associated with cooler soil temperatures in late fall (4). Less growth in the fall could result in a reduction in potential infection sites caused by root breakage during winter or spring soil heaving (14) or root freezing (1).

The planting date of winter wheat greatly affects yield potential under Kansas conditions. Data collected during a 9-yr period at Manhattan, KS, showed an 11% reduction in yield for every week of delay past the optimum planting date (6). This is in agreement with work done in Canada, where planting date also has a large influence on the performance of winter cereals (5). Results from our study also showed a large (13.7%) loss in yield for every week of delay past the optimum planting date. Thus, the benefit of delayed fall planting of winter wheat, for whatever reason, should compensate for the loss in yield associated with this practice. Yield loss to Cs was not reduced with any of the planting dates for the 1980–1981 season (Table 3). Furthermore, in the presence or absence of severe Cs, the highest yield during that year was obtained with the earlier planting dates. Even though a reduction (30.2%) in the percentage of *Cg* infection was experienced in the 1981–1982 season, this reduction would not compensate for the loss in yield potential (54.8%) attributed to a 4-wk delay in planting past the optimum date. Thus, the decision to delay planting of winter wheat to partially control losses to Cs is not considered as effective in Kansas as it might be in other states (7,14,17).

Nevertheless, successful crop management must take into account other pressures that may limit crop productivity. Hessian fly, wheat streak mosaic virus, and take-all are examples of diseases where yield losses also are reduced by delayed seeding of winter wheat (16). In Kansas, these and other pests along with Cs in a producer's field may make delayed planting more economical than in fields infested with *Cg* alone.

Because the ranking of cultivar reaction of Cs with respect to percentage of infection and yield loss will not change with different planting dates, breeders and plant pathologists screening germ plasm for resistance to Cs will not need to consider this.

Results of this study indicate that for investigations involving different planting dates, Cs incidence, and/or severity should be conducted at the same growth stage and not the same calendar date. Late planting significantly delayed crop maturity, with an approximate 3-day delay in heading for each 2-wk delay in planting. Thus, because the extent of systemic spread has been closely linked with host ontogeny and xylem maturation (13), data for Cs incidence and severity will be biased toward later planting dates if collected on the same calendar date.

At all planting dates, *Cg* infection delayed host development until grain maturation, then rapid development occurred. This is in agreement with the premature ripening associated with Cs (16) and points to the "most severe effects of pathogenesis" expressed by Morton and Mathre (11) as reduced carbohydrate synthesis and disrupted transport of assimilates to the caryopsis during the reduced grain-filling period. In our study, planting date did not affect the rate of systemic symptom development when observed at particular growth stages, corroborating the correlation of host ontogeny with symptom expression (13). Thus, the benefits of delayed planting with regard to Cs are apparently due to reduced disease incidence and not reduced systemic spread of the pathogen once it has entered the host.

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