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

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ABSTRACT

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 susceptibility to Cs. There was a significant reduction in virus resistance, moderately susceptible, to Cs. Incidences approaching 100% injury caused during soil heaving (1,4) (MT 7579). Sturdy is susceptible, whereas Arkan and Crest LRC 40 (12) are moderately susceptible 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-m² areas. An equivalent amount of uninocfied 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...
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 that date (Table 3). 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).

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 planting past the optimum planting date (7 October). The yields of a reduced compared with the yields of planted wheat compared with early-season. Yield loss during 1980-1981 was November planting date was severely considerable lag in development of late-

![Fig. 1. Influence of planting date and Cephalosporium gramineum inoculation on yield of three winter wheat cultivars by C. gramineum in 1980-1981](image)

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

<table>
<thead>
<tr>
<th>Planting date</th>
<th>Sturdy</th>
<th>Newton</th>
<th>CC1078-4</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>23 September</td>
<td>69.2 a</td>
<td>56.0 ab</td>
<td>29.2 c</td>
<td>51.5 d</td>
</tr>
<tr>
<td>7 October</td>
<td>60.6 ab</td>
<td>45.2 ab</td>
<td>42.4 abc</td>
<td>50.4 d</td>
</tr>
<tr>
<td>21 October</td>
<td>69.2 a</td>
<td>36.4 bc</td>
<td>31.2 c</td>
<td>45.7 d</td>
</tr>
<tr>
<td>3 November</td>
<td>79.8 a</td>
<td>45.6 ab</td>
<td>33.6 bc</td>
<td>50.0 d</td>
</tr>
<tr>
<td>Mean</td>
<td>68.3 c</td>
<td>45.8 f</td>
<td>34.1 g</td>
<td></td>
</tr>
</tbody>
</table>

1Percentage of infection determined between heading and flowering.
2Values followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.
3Row 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

<table>
<thead>
<tr>
<th>Planting date</th>
<th>Sturdy</th>
<th>Arkan</th>
<th>CLR 40</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>27 September</td>
<td>99.6 a</td>
<td>96.4 ab</td>
<td>85.2 cd</td>
<td>93.7 a</td>
</tr>
<tr>
<td>12 October</td>
<td>98.0 a</td>
<td>89.2 bcd</td>
<td>82.4 d</td>
<td>89.9 a</td>
</tr>
<tr>
<td>26 October</td>
<td>91.6 a</td>
<td>70.8 e</td>
<td>69.2 c</td>
<td>77.2 b</td>
</tr>
<tr>
<td>4 November</td>
<td>69.2 e</td>
<td>62.4 ef</td>
<td>58.8 f</td>
<td>63.5 c</td>
</tr>
<tr>
<td>Mean</td>
<td>89.6 g</td>
<td>79.7 h</td>
<td>73.9 i</td>
<td></td>
</tr>
</tbody>
</table>

1Percentage of infection determined between heading and flowering.
2Values followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.
3Row 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

<table>
<thead>
<tr>
<th>Planting date</th>
<th>Treatment</th>
<th>Yield (g/plot)</th>
<th>Yield reduction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sturdy</td>
<td>Newton</td>
</tr>
<tr>
<td>23 September</td>
<td>Control</td>
<td>242 b</td>
<td>486 b</td>
</tr>
<tr>
<td>7 October</td>
<td>Inoculated</td>
<td>109 cd</td>
<td>264 cd</td>
</tr>
<tr>
<td>21 October</td>
<td>Control</td>
<td>375 a</td>
<td>506 a</td>
</tr>
<tr>
<td></td>
<td>Inoculated</td>
<td>63 d</td>
<td>222 cd</td>
</tr>
<tr>
<td>3 November</td>
<td>Control</td>
<td>472 a</td>
<td>307 bc</td>
</tr>
<tr>
<td></td>
<td>Inoculated</td>
<td>42 d</td>
<td>153 de</td>
</tr>
<tr>
<td></td>
<td>Inoculated</td>
<td>184 bc</td>
<td>138 e</td>
</tr>
</tbody>
</table>

1Average yield for five replicates in grams per plot.
2Values within a column followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

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 Cs 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 Matthe (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.

LITERATURE CITED