Influence of Tillage Systems on Disease Intensity and Spatial Pattern of Septoria Leaf Blotch

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ABSTRACT


The disease severity and spatial pattern of leaves with Septoria leaf blotch (Septoria tritici) symptoms was assessed two times in 1987 and three times in 1988 under conservation and conventional tillage (two locations each). Disease severity was higher under conventional tillage in 1987 (first assessment 86.2 and 39.8% vs. 22.2 and 7.5% [leaf 4], 24.3 and 11.5% vs. 4.3 and 3.6% [leaf 3]; second assessment [1987] 19.6 and 29.4% vs. 3.5 and 0.0% [leaf 3], 2.1 and 4.6% vs. 0.4 and 0.0% [leaf 3]) and at the first assessment in 1988. It was concluded that the amount of straw residue remaining in the field following different tillage practices did not have a strong effect on disease level. Spatial pattern analysis using Morisita’s index of dispersion revealed predominantly random pattern over all tested quadrat sizes, at all dates. Results implicate airborne propagules, consisting of ascospores of Mycosphaerella graminicola, as the source of primary inoculum.

Disease assessment. Disease severity was assessed during 1987 from 8 to 11 April and from 11 to 14 May (one day per location). The growth stages of the wheat at assessment were 6 and 10.5 on the Feekes scale (8), respectively. In 1988, the first assessment was from 10 to 13 April, the second from 30 April to 3 May, and the third from 12 to 15 May. The growth stages of the wheat were 5, 10, and 10.5, respectively.

In each field an area of approximately 0.75 ha was demarcated in each year. Fields had been under wheat monoculture. A binary series of quadrat sizes (1, 2, 4, 8, 16, 32, 64, and 128 m) was used to assess the spatial pattern. Ten replicates of each quadrat size were randomly placed in the survey area. For quadrat sizes up to 32 sqm, 10 tillers were randomly selected from each replicate; 20 tillers were chosen for the remaining quadrat sizes. The 10 or 20 severity values were then averaged for each quadrat size or replicate on a leaf basis. For the statistical analysis, the averaged disease severity values were arcsine transformed because of heterogeneous variances.

A total of 1,000 tillers were assessed per location per assessment date. Tillers were removed from the field, bagged, stored on ice, and transported to the lab. The top four leaves of each tiller were then rated for percent leaf area diseased in the laboratory using a standard area diagram (6). At each assessment, the youngest, fully developed leaf was designated as leaf 1, the next was leaf 2, etc. Periodically, isolations were made from diseased leaves to confirm the presence of S. tritici.

Statistical analysis. Disease severity among the locations was compared for each assessment date and leaf position. To minimize the influence of quadrat size on the disease assessment, statistical analysis was restricted to results obtained from the 1- and 2-m quadrat size. This difference in assessment area was assumed to be negligible. Therefore, the data were combined—that is, 20 data points per leaf per location per time were used. Analysis of the data as a nested design (location nested in tillage) would give 2 df for the appropriate error term to test the influence of tillage systems. Therefore, the influence of tillage systems on the disease severity was compared using linear orthogonal contrasts of locations in the form La1 + La2 (conventional tillage) = SF1 + SF2 (conventional tillage) for each leaf and assessment time separately.

The index of dispersion (10,11), I, was computed according to the formula

\[ I_3 = \frac{n \sum (x)^2 - \Sigma x}{(\Sigma x)^2 - \Sigma x^2} \]

in which x is the disease incidence (number of infected leaves)
for each replicate at a given quadrat size and n is the number of sampling units, in this case 10. The index was computed for all quadrat sizes in the binary series. If the index could not be computed for all quadrat sizes at a location and date, the analysis was dropped. For SF2 (leaf 4 and 3) and SF1 (leaf 3) at the second assessment in 1988, for SF2 (leaf 1) and SF2 (leaf 2 and 1) at the third assessment in 1988, and for LA1 (leaf 3 and 2) at the second assessment in 1987, disease was absent or at very low incidences. The data sets contained 0 (no infected leaves) and 1 (infected leaf). In this case, Morrisita's equation is undefined because the numerator becomes 0. Values of I correspond to three different interpretations of a spatial distribution: $I_0 = 1$ indicates a uniform, $I_0 \geq 1$ a random, and $I_0 > 1$ a clumped pattern. Formulas are available to test the statistical significance of departure from a random expectation. No appropriate statistical methods are available to test whether index values are different from each other.

**RESULTS**

**Disease assessment.** At the first date in 1987, disease was assessed on leaves 4 and 3. On both leaves, the disease severity was significantly higher under the conventional tillage systems using linear orthogonal contrasts ($P = .01$). SF2 was the location with the highest disease severity. The difference in mean disease severities (Table 1) were large. This strengthens the results obtained through the ANOVA, which is based on a small number of fields.

At the second assessment in 1987, leaves 3 and 2 (flagleaf = 1) were used in the statistical analysis. Again, when analyzed using contrasts comparing SF1 + SF2 vs. LA1 + LA2, the location under conventional tillage had a higher disease severity ($P \leq .01$) for both leaves. SF1 was the location with the highest disease severity on both leaves assessed. The difference in severities between the tillage systems was large for leaf 3 and declined strongly for leaf 2 (Table 1).

At the first disease assessment in 1988, disease severity was assessed on leaves 4 and 3. On leaf 4, the difference in disease severity was nonsignificant when comparing tillage systems, although actual disease severities were higher for the conventionally tilled fields. On leaf 3, there was a higher disease severity ($P \leq .01$) on the wheat plants in conventionally tilled fields. The differences in actual severities were rather small (Table 1).

Disease severity was based on leaves 4 and 3 at the second assessment date in 1988. On leaf 4, the influence of tillage systems was statistically significant ($P \leq .01$), with conservation tillage fields exhibiting higher disease levels. The situation is less clear compared to the previous assessments. Locations for each tillage system had high (La1 and SF1) and low (La2 and LF2) mean disease values (Table 1). On leaf 3, disease severity was low (2.0%) and differences were small.

At the third assessment in 1988, disease severity was assessed on the flagleaf (leaf 1) and the flagleaf-1 (leaf 2). Conservation tillage had a significantly higher disease severity on leaf 2. Only traces of the pathogen were detected under conventional tillage on leaf 2 and for all locations on leaf 1.

**Spatial pattern.** The spatial pattern analysis at the first assessment in 1987 indicated a random pattern of S. tritici at all locations and quadrat sizes on leaves 3 and 4. Morrisita's index ranged from 0.8 to 1.2.

At the second assessment in 1987, random spatial patterns were observed for all quadrat sizes for SF1 (leaves 2 and 3) and SF2 (leaves 2 and 3). At LA2, all quadrat sizes, with the exception of the 2-m size (index $= 1.8$), indicated random patterns on leaf 3. The spatial pattern on leaf 2 was not analyzed further because of noncomputable values for quadrat sizes of 8, 32, and 128 m.

At the first assessment in 1988, index values for SF1 and La2 indicated random pattern for all quadrat sizes on both leaves. The situation at SF1 was very similar; the only exception was SF2 at leaf 4-m size on leaf 4. At LA1, all index values showed randomness on leaf 4. On leaf 3, clumping was detected on leaf 3 at quadrat sizes of 1, 2, 4, and 128 m, typical for a population arising from point sources. The maximum index value was 2.7 at the quadrat size of 2 sqm (Fig. 1).

At the second assessment in 1988, index values could not be computed for SF1 on leaf 3 and SF2 on leaves 3 and 4. At SF1, all index values for leaf 4 indicated a random pattern. At LA and LA2, all quadrat sizes were random for leaf 4. For leaf 3 at LA, a pattern describing a population arising from point sources was observed. These point sources, or small clumps, are surrounded by areas with lower levels of disease. The further the distance to the point source, the lower the disease incidence.

**TABLE 1.** Disease severity (percent leaf area diseased) of wheat leaves (cv. Tam W-101) caused by Septoria tritici under two tillage systems

<table>
<thead>
<tr>
<th>Assessment time</th>
<th>GS</th>
<th>Location</th>
<th>Leaf</th>
<th>Dis</th>
<th>SEM</th>
<th>Leaf</th>
<th>Dis</th>
<th>SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>8–11 April 87</td>
<td>6.0</td>
<td>La1</td>
<td>4</td>
<td>8.0</td>
<td>1.0</td>
<td>3</td>
<td>3.7</td>
<td>0.3</td>
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<tr>
<td></td>
<td></td>
<td>La2</td>
<td>4</td>
<td>24.1</td>
<td>4.6</td>
<td>3</td>
<td>5.0</td>
<td>1.1</td>
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<tr>
<td></td>
<td></td>
<td>SF1</td>
<td>4</td>
<td>41.1</td>
<td>4.2</td>
<td>3</td>
<td>14.7</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SF2</td>
<td>4</td>
<td>81.4</td>
<td>4.0</td>
<td>3</td>
<td>25.0</td>
<td>2.2</td>
</tr>
<tr>
<td>11–14 May 87</td>
<td>10.5</td>
<td>La1</td>
<td>3</td>
<td>0.0</td>
<td>0.0</td>
<td>2</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>La2</td>
<td>3</td>
<td>4.9</td>
<td>1.0</td>
<td>2</td>
<td>0.8</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SF1</td>
<td>3</td>
<td>29.8</td>
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<td>2</td>
<td>6.1</td>
<td>1.3</td>
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<td></td>
<td></td>
<td>SF2</td>
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<td>20.4</td>
<td>2.3</td>
<td>2</td>
<td>2.3</td>
<td>0.3</td>
</tr>
<tr>
<td>10–13 April 88</td>
<td>5.0</td>
<td>La1</td>
<td>4</td>
<td>25.9</td>
<td>4.3</td>
<td>3</td>
<td>2.2</td>
<td>0.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>La2</td>
<td>4</td>
<td>32.2</td>
<td>4.7</td>
<td>3</td>
<td>1.4</td>
<td>0.4</td>
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<td></td>
<td></td>
<td>SF1</td>
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<td></td>
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<td>1.7</td>
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<tr>
<td>30 April–3 May 88</td>
<td>10.0</td>
<td>La1</td>
<td>4</td>
<td>40.3</td>
<td>3.2</td>
<td>3</td>
<td>1.0</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>La2</td>
<td>4</td>
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<td>1.6</td>
<td>3</td>
<td>2.0</td>
<td>0.3</td>
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<td></td>
<td></td>
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<td></td>
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<td>3</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>12–15 May 88</td>
<td>10.5</td>
<td>La1</td>
<td>2</td>
<td>9.7</td>
<td>2.1</td>
<td>1</td>
<td>2.7</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>La2</td>
<td>2</td>
<td>2.8</td>
<td>0.5</td>
<td>1</td>
<td>0.3</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SF1</td>
<td>2</td>
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<td>0.05</td>
<td>1</td>
<td>0.01</td>
<td>0.01</td>
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<tr>
<td></td>
<td></td>
<td>SF2</td>
<td>2</td>
<td>0.6</td>
<td>0.1</td>
<td>1</td>
<td>0.05</td>
<td>0.04</td>
</tr>
</tbody>
</table>

$^a$Growth stage according to Feeke's scale.

$^b$Percent leaf area diseased.

$^c$Standard error of the mean.

$^d$La1, La2 = conservation tillage; SF1, SF2 = conventional tillage.
Therefore, small quadrats have large index values and large quadrats small ones; this is attributable to the fact that large quadrats average areas with high and low disease incidence (Fig. 2).

At the third assessment in 1988, the index could not be computed for SF1 (leaf 2 and 1) and SF2 (leaf 1). With the exception of LA2, leaf 1, and LA1 at the 1- and 8-sqm size (leaf 1), all quadrats indicated random pattern. At LA2, leaf 1, a curve typical for a point source population was again observed (Fig. 3).

**DISCUSSION**

The amount of initial inoculum under both tillage systems was not quantified. In the conventionally tilled locations, straw residue was buried using a moldboard plow. According to Hilu and Bever (4), pycnidia of *S. tritici* disappeared from infected leaf tissue buried 76 mm deep in soil for 1 mo. Similarly, Von Wechmar (21) found no viable pycnidia of *S. nodorum* in infected wheat straw buried 152 mm deep in soil for 1 mo and suggested decomposition of pycnidial contents by soil microorganisms. Additionally, the use of a packer for seedbed preparation would decrease the survival of pycnidia not buried. Sanderson, et al (14) found that crop residues and pycnidia that remain in direct contact with the soil surface were very vulnerable to decay, similar to incorporated residues. The conventionally tilled fields (SF1 and SF2) were, therefore, considered free of within-field primary inoculum.

The primary inoculum in the conservation fields was not assessed, because quantification of pycnidia on crop residue would have to include incubation of the pycnidia found and observations on the appearance of the conidia. This is especially important in regard to the findings of Sanderson, et al (14). This was impractical when considering the size of the fields.

The disease severities and spatial patterns observed at the first assessment in 1987 and 1988 are not caused by the primary infection of the wheat. In all probability, several infection cycles had occurred. The number should be low (1-2 generations), however. According to Shaner (17,18), the optimal temperature for germination is 20-25 C, with a minimum of 2-3 C. Infection in the field is delayed if the temperature falls below 7 C during two consecutive nights. Low temperatures (4 C) affect spore germination, mycelial growth, and lesion and pycnidia development by lengthening the time required for each. Temperatures lower or equal to these reference points are common in Oklahoma from November to March, making a higher number of generations unlikely.

During 1987, which was characterized by a cool, wet spring, wheat in conventionally tilled fields had higher disease severities than the conservation tillage systems on all leaf levels assessed at both assessment times. The situation in 1988, when warm, dry weather predominated, was different, even though the general trend prevailed. Convention tillage locations had wheat with higher severities at the first assessment. At the second date, conservation-tilled wheat had the higher disease level, even though the ranking was mixed among the tillage systems. At the third assessment, the wheat in conservation fields had a higher disease severity, which, although statistically significant, was small when compared to the other dates. It is very unlikely that these differences in the later assessments in 1988 could have been caused by residue-borne inoculum. During this time of the year (May), secondary inoculum produced on the leaves is the most likely source (12,18). Differences in climatic conditions on or within a field level could account for the differences. Holmes and Colhoun (5) found, however, that when artificially infested straw residue was placed among seedlings, higher levels of disease severity were obtained as compared to control blocks. When examining straw residue left on the field surface for about 6 mo after harvest, the presence of *S. tritici* could not be confirmed. The role of field-produced straw thus remains doubtful. In general, disease severity during the season was not influenced by tillage systems, even though the conservation tillage fields had residue-borne, in-field inoculum. This inoculum did not cause higher disease levels, especially of the first assessments, which were relatively close to the primary infection in regard to the number of generations passed.

The spatial pattern of the disease at the first assessment of each growing season was random. This agrees with findings by
Shaw and Royle (19), who observed the disease to be uniformly distributed throughout the fields from the beginning of the growing season onwards. The fields were conventionally tilled. Their spatial determination, based on the spatial correlation function, found 15 negative regressions of the cross-product of deviations from the mean and eight positive ones. Overall, there was more correlation between disease amounts at close locations than at distant ones. Their data encompassed the period of mid-January to early July, and a distance scale extending to 50 m. They concluded that the initial inoculum must have been rather evenly distributed over wide areas. In this study the appearance of clumped quadrat sizes later in the growing season at some location and/or leaves could be attributed to the climatic conditions, which were unfavorable for disease development. Also, infiel variation of the microclimate, caused by low-lying areas and different plant densities, could be responsible for this appearance of clumping. The importance of leaf wetness for the disease epidemiology is well established (7). The statement is supported by the fact that the clumping occurred under both tillage systems and that, in general, high disease incidence on lower leaves was associated with high disease incidence on the upper leaves within the same field. Additionally, weather conditions in Oklahoma tend to be progressively less favorable for disease development as the season progresses in regard to temperature and leaf wetness, enhancing the importance of in-field variation in microclimate. As previously stated, tillage systems or the straw residue left in fields do not lead to different disease incidences at the beginning of or during the growing season. This opens the question of the origin of the disease in fields. In previous research on *Pyrenophora tritici-repentis* (Died.) Dresch. (15), residue-borne infection was associated with clumped disease patterns. With increasing importance of airborne conidia, the patterns changed to random over time. The random patterns observed in this study at the beginning of the growing season could, therefore, be interpreted as the result of airborne infection. This agrees with studies conducted by Shaw and Royle (20). They showed that the source of primary infection of winter wheat was evenly dispersed and airborne. Their conclusions implicated ascospores of *M. graminicola* as the most likely primary inoculum. Ascospore production on straw residue was observed by Scott, et al (16) in the United Kingdom. The sexual stage of the fungus has been observed very rarely in the United States, but results from this study would lead to similar conclusions. In 1988, frequent isolation of ascospores of *M. graminicola* from California wheat fields were reported (9). This should give new impetus to the reassessment of the sexual stage in the epidemiology of *S. tritici* disease in U.S. wheat production, especially in regard to disease forecasting.

**LITERATURE CITED**