Ecology and Epidemiology

The Influence of Tillage Systems on Incidence
and Spatial Pattern of Tan Spot of Wheat

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ABSTRACT


The severity and spatial pattern of *Pyrenophora tritici-repentis*, the causal agent of tan spot, was assessed at four locations (two conventional and two conservation tillage) two times during 1987 and three times during 1988. Tan spot was detected earlier in 1988 and had a higher final disease severity in 1987 under conservation tillage. Spatial pattern analysis, using Morisita’s index of dispersion, revealed a shift from clumped to random distribution over time, indicating the importance of residue-borne inoculum under conservation tillage systems. Random spatial patterns under conventional tillage indicated airborne inoculum as the source of infection. Results from this experiment suggest that straw residue on the field surface will lead to increased levels of tan spot.

Tan spot of wheat is caused by *Pyrenophora tritici-repentis* (Died.) Drechs. The fungus grows saprophytically on host debris. Ascospores, conidia, and hyphal fragments all serve as primary inoculum.

Historically, in wheat (*Triticum aestivum* L.) culture, plant residue remaining after harvest was incorporated into the soil by conventional tillage practices, e.g., with the use of a moldboard plow. This facilitated colonization and degradation of the straw by saprophytic organisms, and planting of the new seedbed was not hampered by great amounts of residue on the soil surface. Use of conservation tillage was first emphasized shortly after the dust bowl years of the 1930s as a method to minimize soil erosion due to wind and rain. Renewed interest in conservation tillage systems arose in the 1970s. The motivation was primarily economical, e.g., to substantially reduce purchased production inputs, as well as water and soil conservation issues. Since then, crop acreage in the United States under conservation tillage has steadily increased. Predictions by Crosson (1) estimate that 50–60% of the cropland will be managed by some sort of conservation tillage by the year 2010.

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The objective of this study was to determine the potential for increased till density resulting from a low tillage management technique. An increase in disease levels could negate gains realized through conservation tillage systems. An additional objective of this study was to test the suitability of spatial pattern analysis for this type of experiment.

**MATERIALS AND METHODS**

**Locations.** Four commercial fields (two conservation [L1, L2] and two conventional [S1, S2] tillage) were selected for this study. The fields were located in north central Oklahoma in close proximity of each other (approximately six-km radius) to minimize differences due to climate factors. The soil in the two conservation tillage fields consisted of 27% sand, 50% silt, and 24% clay with a pH of 4.1 for L1; and 57% sand, 20% silt, and 24% clay with a pH of 4.3 for L2. The respective values for the locations under conventional tillage were 37% sand, 44% silt, and 20% clay with a pH of 5.9 for S1; and 17% sand, 60% silt, and 24% clay with a pH of 5.8 for S2. Soil testing described the levels of nitrogen, phosphorus, and potassium adequate for a yield goal of 40 bushels.

Planting dates in 1987 and 1988 were during the second and third weeks of October for all locations. The fields were planted with the winter wheat cultivar TAM W-101.

The conventional tillage fields were prepared by a deep plowing in the fall with a moldboard plow, followed by secondary seedbed preparation. Conservation tillage fields were prepared by disking and chisel plowing. Seed was planted without any additional seedbed preparation. Seeding rate was approximately 45 kg/ha.

**Disease assessment.** Disease severity was assessed two times during 1987. The first assessment date was from 8-11 April and the second from 11-14 May (one day per location). The growth stages of the wheat were 6 and 10.5 on the Feekes scale (5), respectively. In 1988, the first assessment was from 10-13 April, the second from 30 April-3 May, and the third from 12-15 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. A binary series of quadrat sizes (1, 4, 8, 16, 32, 64, and 128 cm) 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 square meters, 10 tillers were randomly selected from each replicate, and 20 tillers were chosen for the remaining quadrat sizes. The disease severity values were then averaged from the 10/20 tillers for each quadrat size/replicate on a leaf basis. For the statistical analysis, the averaged severity values were sine transformed because of unstable variances.

On the whole, 1,000 tillers were assessed per location per assessment date. Tillers were removed from the field, stored on ice, and transported to the lab. The top four leaves of each tiller were then visually rated for percent leaf area diseased in the laboratory using a standard area diagram (4). The uppermost, fully developed leaf was designated as leaf 1, the next leaf 2, etc., progressing down the tiller. Periodically, isolations were made from diseased leaves to confirm the presence of *Pyrenophora tritici-repentis*.

Disease severity was compared among the locations for each assessment date and leaf separately. To avoid problems associated with disease assessments based on widely different quadrat sizes, only severity values of the 1- and 2-m quadrat size were used. Since the difference in quadrat size (1 m vs. 2 m) was assumed to be negligible in regard to the disease assessment, data were combined, i.e., 20 data points per the time per location were used per data set. Analysis of the data as a nested design (location nested in tillage) would have left 2 df for the appropriate error term to test the influence of tillage. Therefore, data were analyzed based on a model using disease severity as the dependent and location as the independent variable with $n = 80 (4 \times 20)$ for each assessment time. The disease severities between tillage systems were then compared with linear orthogonal contrasts of the form \( (L1 + L2) [\text{conservation tillage}] - (S1 + S2) [\text{clean tillage}] \) for each leaf and assessment time separately.

**Morisita's index of dispersion.** Morisita's index of dispersion \( I_p \) was computed according to the formula

$$I_p = \frac{(\Sigma(x^2) - \Sigma x)}{\Sigma(x^2) - \Sigma x} * n$$

in which \( x \) is the number of tillers showing disease symptoms for each replicate at a given quadrat size and \( n \) is the number of sampling units, in this case, 10. The index was computed separately for all quadrat sizes. If the index could not be computed for all quadrat sizes at a location and date, the analysis was not performed. Especially during the second assessment of 1988, disease incidence was low. Several data sets contained only 0 and 1. In this case, the above equation is undefined because the numerator becomes 0. Values of \( I_p \) correspond to three different interpretations of a distribution: \( I_p < 1 \) indicates a uniform, \( I_p = 1 \) a random, and \( I_p > 1 \) a clumped pattern. Indices were tested for the statistical significance of departure from random expectations (6,7). No appropriate statistical methods are available to test if index values are significantly different from each other.

**RESULTS**

**Disease severity.** No disease was detected at any location during the first assessment date in 1987. At the second assessment, disease severity was significantly higher in the conservation tillage plots on leaf 3 (Table 1) and leaf 2, using linear orthogonal contrasts

<table>
<thead>
<tr>
<th>Assessment time</th>
<th>GS*</th>
<th>Location</th>
<th>Leaf</th>
<th>Diseased (%)</th>
<th>Leaf</th>
<th>Diseased (%)</th>
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<td>10.5</td>
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<td>26.7</td>
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<td>3</td>
<td>9.2</td>
<td>2</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S1</td>
<td>3</td>
<td>5.8</td>
<td>2</td>
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<tr>
<td></td>
<td></td>
<td>S2</td>
<td>3</td>
<td>1.9</td>
<td>2</td>
<td>1.5</td>
</tr>
<tr>
<td>10-13 April 88</td>
<td>5</td>
<td>L1</td>
<td>4</td>
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<td>3</td>
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</tr>
<tr>
<td></td>
<td></td>
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<td>4</td>
<td>5.1</td>
<td>3</td>
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<tr>
<td>30 April-3 May 88</td>
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<tr>
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<tr>
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</tr>
</tbody>
</table>

*Growth stage according to Feekes scale.

*Percent leaf area diseased.

*L1, L2 = Conservation tillage; S1, S2 = conventional tillage.
for the difference of \((L_1 + L_2) - (S_1 + S_2)\). When the disease severity was compared across locations, \(L_1\) had the highest disease severity on leaf 3 with 26.7\% and leaf 2 with 10.7\% (Table 1). On leaf 2 and leaf 3, both reduced tillage locations had significantly higher disease severity than conventional contrasts. No disease was observed on leaf 1 (flag leaf). Leaf 4 was already senescent and therefore omitted.

At the first assessment date in 1988, no disease was observed in the fields under conventional tillage. At \(L_2\), leaf 4 had an average disease severity of 5.1\%, significantly higher \((P \leq 0.05)\) than \(L_1\) with 1.0\%. A similar situation was observed on leaf 3, where \(L_2\) had an average severity of 4.2\%, significantly higher \((P \leq 0.05)\) than \(L_1\).

At the second assessment date in 1988, disease severity was significantly higher \((P \leq 0.01)\) under conventional tillage on leaf 3 and leaf 4, using orthogonal contrasts as previously described (Table I). \(L_2\) had the highest severity with a 4.0\% for leaf 4 and 3.0\% for leaf 3 (Table I). At all locations except \(L_2\), a higher disease severity was observed for leaf 3 as compared to leaf 4. No disease was observed on leaf 1 and leaf 2.

At the third assessment date in 1988, the difference in disease severity, analyzed as previously described, was nonsignificant \((P \geq 0.05)\) when comparing locations in tillage systems, i.e., \(L_1 + L_2\) versus \(S_1 + S_2\). Leaves 3 and 4 were already senescent and therefore omitted from the analysis.

**SPATIAL PATTERN**

In 1987, the spatial pattern was analyzed for the second assessment date. Index values at all locations and leaves ranged from 0.9 to 1.1, indicating a random pattern of the disease in the field. The only exception was observed at \(L_2\) on leaf 2 (index = 1.5).

In 1988, on the first assessment date, indices of both conventional tillage fields indicated clumped disease pattern. At \(L_1\), index values denoting clumping were observed at the 2-, 4-, 8- and 16-m quadrat sizes on leaf 4 (Fig. 1). The shape of the curve is typical for a population arising from point sources. The point sources, or small clumps, were surrounded by areas with lower levels of disease. The further the distance from the point source, the lower the disease incidence. Thus, small clumps have large index values and large clumps small ones; this happens because large quadrats average areas with high and low disease incidence. On leaf 3, index values indicating a clumped pattern were detected at the 1-, 2-, 4-, 8- and 16-m size (Fig. 1). The shape of the curve is indicative of a population arising from larger clumps (6,7).

At \(L_2\), a clumped pattern was found at the 4-, 8-, and 16-m size on leaf 4 and leaf 3 (Fig. 2). The shape of the curve, when compared to the curve shapes developed by Morisita (6,7), indicate a population consisting of larger clumps.

At the second assessment in 1988, the index could not be computed at \(S_1\) on leaf 4 and the 1- and 16-m size on leaf 3, and at \(S_2\) for leaf 4 and leaf 3. The quadrat sizes on leaf 3 (\(S_1\)), for which the index is computable, indicated a random disease pattern. At \(L_1\), clumping was detected on leaf 4 at the 1- and 2-m size (index values of 1.9 and 5.0, respectively), indicating again a pattern arising from point sources. On leaf 3, all index values except the 8-m size (index 1.5) indicated a random pattern. At \(L_2\), clumping was detected at the 64-m size on leaf 4. All other index values, on both leaf 4 and leaf 3, indicated a random pattern.

At the third assessment in 1988, random disease patterns were observed at all locations, leaves, and quadrat sizes.

**DISCUSSION**

Disease severity generally was higher under conventional tillage systems as compared to conventional tillage at all assessment dates, except the third assessment in 1988. The absence of the disease at the first assessment in 1987 was in all probability due to the unfavorable weather that prevailed during April (primarily cold temperatures). Hosford et al (2) found the optimal temperature for infection and symptom expression to be in the range of 20–30°C, with the optimum for symptom expression being around 30°C. These data apply to conidial infection. Data for infection by ascospores, the most likely primary inoculum at this time, have not been established. The differences in severity levels declined from the first to the third assessment. This decrease is due to the growing importance of airborne conidia, which, due to their mobility, minimize the effect of inoculum produced within a field. The increasing role of airborne conidia over time could be further exemplified by the fact that at the second assessment date in 1988, the younger leaves had higher disease severities than the older ones, even though alternative explanations such as differential susceptibility of the tissue cannot be excluded.

The importance of early infection in relation to yield loss was demonstrated by Shabeer and Bockus (10). They found that about 17% of the total yield loss from tan spot occurred from early season infection by ascospores. This corresponds to the first assessment date. Additionally, about half the total yield loss had occurred by the boot stage (second assessment, 1988). Rees and Platz (8) observed 13 and 35% yield loss from severe attacks of tan spot in the seedling and jointing stages, respectively. The earlier and higher infection levels under conventional tillage demonstrate the importance of straw residue. This corroborates findings by Rees et al (9), who determined a logarithmic relationship between the loss of wheat yield and the amount of infested stubble on the soil surface. No attempt was made in this study to quantify either amount of residue or degree of residue infestation. The higher disease levels and earlier date of appearance...
in fields under conservation tillage during developmental stages of the wheat plant, where significant yield loss from infection can occur, should be taken into consideration when tillage decisions are made.

The higher final disease severity in 1987 (across all locations) as compared to 1988, in spite of the shorter time period for epidemic development, is in all probability weather related. The summer of 1988 was unusually dry, and the pathogen requires extended periods of leaf moisture for infection (2).

The second objective of this study was to evaluate the importance of residue-borne inoculum using spatial pattern analysis. The hypothesis underlying the approach was that disease patterns arising from residue should be clumped, unless the area was saturated with infected straw, and should approach a random pattern with time, due to the influence of airborne conidia. Disease patterns originating from airborne inoculum only should be random, as exemplified by the conventional tillage locations. Trends and patterns of this type were observed in both years. In 1987, spatial patterns were assessed at the second date only due to the absence of disease. Patterns under both tillage systems were random. The most likely mechanism causing such patterns is infection through airborne conidia. This agrees with the fungal biology, i.e., only conidia are produced at this time of the year (3). The absence of tan spot in the conventionally tilled locations at the first assessment in 1988 points to residue-borne ascospores as the most likely propagule. The spatial patterns observed in the conservation tillage locations are typically for a population arising from point sources. These point sources consist of clumps of perithecia or straw residue in the fields. The shapes of the curves suggest larger clumps at La2. This can be explained through the higher disease severities observed at this location.

The spatial pattern at the second assessment described a random disease pattern at La2 on both leaf 4 and leaf 3. At Lai, the shape of the index curve suggests a point source population on leaf 4, changing to a random pattern on leaf 3. This could be interpreted as a change from ascospore to conidal infection. The higher disease levels could be the reason that this observation was not made at La2. The larger clumps detected at the first assessment and their expansion to sizes larger than the maximum quadrat size used in this experiment could account for this. The spatial pattern of SFI leaf 3 was random. The absence of disease at the first assessment in the conventionally tilled fields excludes ascospores as the causal agent for disease, thus pointing to airborne conidia. The spatial pattern observed and the leaf position (leaf 3) would confirm this conclusion. At the third assessment, random patterns were found at all locations and on all leaves. The low disease severities observed (Table 1) make saturation of the fields as the cause of the random patterns unlikely, again pointing to airborne conidia.

Residue-borne ascospores serve as the primary inoculum for tan spot of wheat, resulting in earlier disease onset and higher disease severities. Initial infection takes place during spring through ascospores as opposed to infection in the preceding fall through conidia. Conidia sustain the epidemic during the growing season as shown through the spatial analysis. Residue-borne ascospores generally caused a clumped disease pattern, typical for populations arising from point sources, whereas airborne conidial infection resulted in randomness. Spatial analysis therefore can be used to differentiate between spread mechanisms, i.e., residue-borne vs. airborne infection.

LITERATURE CITED