Tan Spot Effects on Yield and Yield Components Relative to Growth Stage in Winter Wheat

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ABSTRACT

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Losses in yield and yield components caused by tan spot were measured at different growth stages of wheat in the field and greenhouse. In greenhouse experiments, plants were kept uninoculated or were exposed to a single inoculation period of *Pyrenophora tritici-repentis* at one of five different growth stages. Yield, 100-kernel wt, grain number per head, and number of heads per plant were determined. The highest yield losses were recorded for inoculations at the boot and flowering stages, indicating that plants were most susceptible physiologically to losses at those stages. Losses were a result of a significant reduction in kernel wt and number of grains per head and not of reduced number of heads per plant. In the field, tan spot epidemics were terminated at each of five growth stages by starting a fungicide spray program (4–7 day interval) that continued until crop maturity. About 17% of the total yield loss from tan spot occurred from early season infections by ascospores. Furthermore, about half of the total yield loss had occurred by the boot stage. Thus, tan spot activity before boot is important, because multiple infection periods at that time cause significant loss even though the plants may not be as physiologically prone to loss as at later growth stages. Fungicide spray programs to control tan spot in winter wheat should begin sooner to prevent early season disease.

Tan spot, a foliar disease of wheat caused by *Pyrenophora tritici-repentis* (Died.) Drechs. (anamorph *Drechslera tritici-repentis* (Died.) Shoem.) is worldwide in distribution (2,3,12). In Kansas, the first symptoms of the disease usually appear on lower leaves in early April as small, yellow-brown spots that develop into tan to brown blotches surrounded by yellow borders. The primary inoculum of the fungus consists of ascospores ejected from pseudothecia that mature on wheat stubble on or above the soil surface during the fall and winter. Conidia formed on the previous year's wheat residue or on killed leaves function as secondary inoculum. The disease spreads from the lower to the upper leaves under moist weather conditions and causes necrotic spots and ultimately death of infected leaves. Thus, severity of tan spot tends to be higher with reduced tillage cropping and is favored by prolonged periods of rain or dew (2).

Tan spot can cause substantial losses to the wheat crop under severe epidemic conditions. Sharp et al (11) and Rees et al (9) reported about 20 and 50% losses in grain yield from this disease, respectively. Severe tan spot was observed to cause 9–20% reduction in 1,000-kernel wt in seven of 30 cultivars of spring wheat (11). In Kansas, where tan spot was a minor problem 20 years ago, it has become the fourth most important wheat disease during the past 10 years; only soilborne mosaic, speckled leaf blotch, and leaf rust are more important. It is assumed that the increasing acceptance of practices that retain wheat residue on the soil surface has resulted in this increase in tan spot severity.

Four factors are primary determinants for losses in wheat caused by tan spot. The first is the inoculum level of the fungus. Rees et al (9) observed a logarithmic relationship between the loss in wheat grain yield and the amount of infested stubble on the soil surface. Second is the effect of a postinoculation wet period on susceptibility of wheat cultivars and their losses. Hosford and Busch (4) evaluated the cultivars Wells, Chris, and Waldron for tan spot reaction and yield response under field conditions and found that Wells had less tan spot and related yield loss than Chris, and Chris had less than Waldron. By correlating the performance of these genotypes in the field with their postinoculation wet period requirements in the greenhouse, it was observed that Wells required an 18–24 hr wet period, Chris 12–18 hr, and Waldron 6–12 hr. This indicated that susceptible cultivars require shorter durations of wet periods to suffer yield losses from tan spot. The third major determinant of loss is the host genotype. In the field, Raymond et al (7) observed that the cultivar Red Chief (resistant) sustained only 7.2% loss compared with TAM-105 (susceptible), which had 27.7% loss under moderately severe epidemic conditions. In another study (4), tan spot reduced the yield of Hercules, Wells, Chris, and Waldron by 8, 12, 23, and 28%, respectively, when these genotypes were grown in plots with infested straw. The fourth factor is the growth stage at which wheat is infected by tan spot. Two different growth stages were used by Rees and Platz (8), who estimated 13 and 35% loss in yield of cultivar Banks (susceptible) from severe attack of tan spot at the seedling and jointing stages, respectively. Cox and Hosford (1) reported that, for determining resistance response, reactions on flag leaves of adult plants in the greenhouse were the most accurate indication of reaction in the field, although reactions on seedlings were also useful in this regard.

The effect of growth stage on yield loss from tan spot could be a major factor in understanding and controlling the disease in Kansas. In the past, most control strategies for foliar diseases of wheat have been modeled after the rust diseases. Much of the damage done by rusts occurs after flag leaf emergence and when the temperature has increased past some critical value (12). Consequently, controls are aimed at reducing losses during the last few weeks of crop growth, e.g., foliar fungicide applications for rust control are applied after flag leaf emergence. Also, early maturing cultivars have been one of the most effective means of reducing rust losses in the Great Plains. However, because of differences in epidemiology, these practices may not be as effective for controlling tan spot.

The objectives of this study were to determine the growth stages and time of the year at which most losses from tan spot occur. This study compared five different growth stages in the greenhouse to determine which stage was most prone physiologically to tan spot losses, and five different times of terminating the epidemic in the field to determine important times of the year for losses. A preliminary report has been published (10).

MATERIALS AND METHODS

Preparation of inoculum and method

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of inoculation in the greenhouse. The five isolates of *P. tritici-repentis* selected for this study (HV-2, MC-1, PTF, TS-1, and TS-6) are highly virulent isolates, each originating from a single conidium obtained from infected wheat leaves collected across Kansas. Spore suspensions of each isolate were prepared by the method of Raymond et al (7). Briefly, the method consists of transferring a mycelial plug of the fungus to a medium consisting of overlapping agar wedges of one-quarter strength potato-dextrose agar and V-8 agar. The inoculated plates were incubated at 21 ± 2 C for 7 days, 10 cm under continuous fluorescent light (six 20-watt bulbs). Then, the advancing aerial mycelium of the fungus was knocked down with a sterile, bent glass rod and the petri plates were kept under a 24-hr light/12-hr dark regime at 15 ± 2 C. The light period stimulated conidiophore formation, while the dark period caused production of conidia on the conidiophores (5). When the conidia were formed, 5 ml of sterile distilled water was added to each petri dish, and the colony surface was rubbed gently with a glass rod. Spore suspensions of all five isolates were collected into a container, mixed, and the concentration was adjusted to 3×10^3 spores/ml.

Before inoculating the plants, a few drops of Triton B-1956 were added to the spore suspension to aid in adhering and distributing the spores on the leaf surface. Plants were inoculated with an atomizer operated at 0.7 kg/cm^2 pressure (10 psi), and leaves were sprayed to the drip stage. After inoculation, the plants were allowed to dry for 1 hr and then moved to a mist chamber for 48 hr. Two centrifugal humidifiers in the mist chamber were controlled by a timer and operated for 1.5 min during every 10 min to provide continual leaf wetness. After the 48-hr mist period, the plants were placed on greenhouse benches, watered, and fertilized as needed until maturity. In this experiment, the uninoculated plants were sprayed with distilled water to which Triton B-1956 was added. These plants were then dried, misted, and treated the same as the inoculated plants of the same treatment.

Greenhouse yield parameter experiments. Three seeds of TAM 105 (CI 17826) were planted in each of 40 7.5-cm-diameter plastic pots containing steam-sterilized soil. The soil consisted of Chase silty clay loam, sand, and sphagnum moss in a 2:1:1 ratio by volume, respectively. Plants were vernalized at 5 C for 6 wk at the seedling stage, then transplanted to 15-cm plastic pots, with a single plant per pot. Temperatures during the experiments ranged from 18 to 27 C with plants receiving natural light and water and fertilized as needed. Two identical experiments consisting of inoculation at five different growth stages were conducted: 1) tillering (Feekes growth stage 3 [6]), 2) elongation (stage 7), 3) boot (stage 10), 4) flowering (stage 10.5), and 5) milk (stage 11.1). Each treatment consisted of 20 plants; half the plants were inoculated and half were kept uninoculated. Disease severity was rated 7–10 days after inoculation using the following scale: 0 = no symptoms, 1 = flecks or minute lesions, 2 = lesions with distinct yellow halos covering less than 10% of the leaf area, 3 = lesions with distinct yellow halos covering between 10 and 50% of the leaf area, 4 = numerous coalescing lesions with more than 50% of the leaf area affected, and 5 = dead leaf. A score was given to each plant by averaging the readings for the top four leaves. Yield per plant (g), 100-kernel wt (g), number of kernels per head, and number of heads per plant were recorded at the time of harvest. Percentage loss of each parameter and *t* tests ($P = 0.05$) between inoculated and uninoculated plants within a treatment were calculated.

Experiments in the field. Experiments were conducted in each of 2 years (1985–1986 and 1986–1987) on Chase silty clay loam soil (pH = 6.2) in a Latin square design at the Rocky Ford Research Farm near Manhattan, KS. There were five treatments and five replications, using 1.2 × 7.5 m plots. Treatments involved different times of terminating the tan spot epidemic by starting a 4- to 7-day interval fungicide spray program that continued until crop maturity. Manzate 200 applied at 2.46 kg/cm^2 (35 psi) through hollow-cone nozzles at the rate of 2.3 kg in 187 L water per ha was used for each fungicide application date. Growth stages and times of epidemic termination were: 1) beginning of pseudostem erection (Feekes growth stage 4 [6], about 10 March, healthy check); 2) pseudostem fully erect (stage 5, about 10 April); 3) boot (stage 10, about 5 May); 4) milk (stage 11.1, about 20 May); and 5) no fungicide application. The plots were seeded in the last week of September with TAM 105 wheat and infested at the 4-leaf stage (1 November) with oat kernels colonized by *P. tritici-repentis*. During the 1985–1986 season, disease severity was determined five times from 13 April to 30 May. The top four leaves of 20 randomly selected plants per plot were rated with the scale used for the greenhouse experiments.

Inoculum for the field consisted of five isolates (HV-2, MC-1, PTF, TS-1, and TS-6) of *P. tritici-repentis* obtained from infected wheat collected across Kansas and was prepared as follows (7): 150 g of oat kernels and 130 ml of distilled water were placed into 1-L canning jars and held overnight. The jars were then capped with a perforated lid plugged with cotton and autoclaved for 90 min. When the jars were cool, 2–3 mycelial plugs were taken from the culture of a single isolate and introduced into each jar under aseptic conditions. The jars were kept at room temperature for a 3-wk period and shaken every 2–3 days to facilitate uniform colonization of the fungus on the oat kernels. The oat kernels were then spread out and air-dried. Equal volumes of inoculum of each of the five isolates were mixed and applied to the plots at the rate of 230 g per plot.

Yield loss determination. During the first year of the study, 25 heads were collected from each plot, and number of

Table 1. Disease severity, yield, and yield components of winter wheat plants inoculated with *Pyrenophora tritici-repentis* at different growth stages in the greenhouse

Growth stage at inoculation[w]	Disease severity[x]		Yield (% loss[y])		100-kernel wt (% loss)		Grains/head (% loss)		Heads/plant (% loss)	
	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2	Test 1	Test 2
Tillering	2.6*[z]	2.9*	−13	10*	0	1	−4	−5	−12	12
Elongation	2.8*	2.7*	12	3	10	−3	7	2	−9	5
Boot	3.4*	3.1*	33*	13*	25*	8*	26*	9*	−1	−5
Flowering	3.9*	3.7*	29*	17*	25*	11*	1	10*	−2	−5
Milk	4.6*	4.2*	6	11*	11	1	−6	−1	12	9

[w] Vernalized, potted plants inoculated at indicated growth stage with a spore suspension (3×10^3/ml) and placed in a mist chamber for 48 hr. Uninoculated checks sprayed with water and placed in mist chamber.

[x] 0 = No symptoms, 1 = flecks or minute lesions without halos, 2 = lesions with distinct yellow halos covering no more than 10% of the leaf area, 3 = lesions with distinct halos covering between 10 and 50% of the leaf area, 4 = numerous coalescing lesions with more than 50% of the leaf area affected, and 5 = dead leaf. Values are the means of 10 plants with the top four leaves of each plant rated.

[y] Percent loss calculated by comparison of 10 replicate inoculated plants with 10 uninoculated plants.

[z] Asterisk indicates that the loss was significantly different ($P = 0.05$) from the uninoculated checks in a pairwise *t* test.

grains per head was recorded. Percentage of loss in number of grains from tan spot was calculated by comparison to the treatment protected by the fungicide throughout the season (treatment 1). For both years, the experiments were machine-harvested, and the actual plot yield was recorded. One-thousand kernel wt were determined from the means of five samples of 1,000 kernels per plot. The data on average number of grains per head, 1,000-kernel wt, and average grain yield were analyzed by analysis of variance. Duncan's multiple range test ($P = 0.05$) was used where the data showed significant differences between the treatments.

RESULTS

In both greenhouse experiments, no tan spot was observed on uninoculated plants, but significant disease developed on inoculated plants at all inoculation times (Table 1). Severities increased with increasing age of the plants at inoculation. Significant losses in yield, 100-kernel wt, and grains per head were associated with single tan spot inoculations at the boot and flowering growth stages (Table 1). In one experiment, significant yield reduction occurred at tillering and milk inoculation times, but with these two exceptions, no significant losses in yield or yield component parameters were measured after inoculation at the tillering, elongation, or milk growth stages. No significant reductions in the numbers of heads per plant were observed for any treatment. Very late tillers were occasionally produced by plants and were usually associated with the inoculation treatment. In one experiment, 24 out of 29 very late tillers were from inoculated treatments. These tillers usually produced no grain or very shriveled grain and were excluded from the results.

Significant losses in grain yield from tan spot occurred during each year of the field study (Table 2). When fungicide spraying began about 10 April, 1 mo after ascospores started to discharge but before secondary spread of the fungus was observed, there was 9.8 and 5.8% yield loss in years 1 and 2, respectively (Table 2). These values represented about 17% of the total loss. The treatment in which fungicide spraying began at the boot stage (about 5 May) sustained approximately half the total yield loss in both years. In year 2, there were no significant infection periods (rains) between the boot and watery ripe growth stages, so yields of these treatments were not significantly different. In year 1, a late stem rust epidemic occurred and caused an estimated 10% loss in yield, so that losses reported for treatments in which no fungicide was applied and in which fungicide application began at watery ripe are the result of rust and tan spot activity. In year 2, no disease activity, besides tan spot, was observed.

Thousand-kernel wt losses were not as high as those recorded for yield, but accounted for about 57% of the total yield loss (Table 3). Again, damage accounting for about one-half of the total loss in kernel wt had already occurred by the boot stage. Smaller losses were attributed to a decrease in the number of grains per head (Table 3). However, there was a significant loss between the treatment protected during the entire tan spot epidemic (10 March) and the unprotected treatment. The loss in grain number per head (11%) accounted for about 20% of the total yield loss for 1986 (57%). Most of the loss in grain number per head (69%) was due to damage occurring before the boot stage.

Commencement of a fungicide spray program on 10 March 1986, before the beginning of the tan spot epidemic (7), resulted in low disease severity ratings throughout most of the season (Table 4). By the 30 May rating date, natural senescence had begun on lower leaves and this influenced the ratings. On 13 April, when the first disease severity data were collected, the plots that had been protected with a fungicide from 10 March had significantly less disease than the other four treatments that were unprotected to this point (Table 4). At the 26 April rating time, the data were very similar to 13 April. The treatment where fungicide application commenced on 10 April did not yet show reduced disease severity. Furthermore, ratings tended to be lower at this time compared with the 13 April rating, probably due to the wheat "outgrowing" the disease because the ascospores are only ejected approximately 5 cm (B.L. Norman, *unpublished*). By the 2 May rating date the treatment where spraying began on 10 April was significantly lower than the treatment where spraying was to be started on 5 May. It had taken approximately 3 wk after initiating a spray program to notice a reduction in disease severity. Similarly, the treatment where application started on 5 May did not show reduced disease on 14 May but did have significantly reduced disease on 30

Table 2. Effect of time of epidemic termination on yield and yield loss from tan spot on winter wheat in the field

Time of epidemic termination[x]	Year 1 (1985–1986)			Year 2 (1986–1987)		
	Yield (kg/plot)	% loss	% of total loss	Yield (kg/plot)	% loss	% of total loss
10 March (healthy check)	3.72 a[y]	...	...	2.41 a[y]	...	...
10 April (pseudostem erect)	3.36 b	9.8	17.1	2.27 b	5.8	17.0
5 May (boot)	2.75 c	26.2	45.7	2.02 c	16.2	47.5
20 May (watery ripe)	2.13 d[z]	42.7	74.5	1.99 c	17.1	50.1
Epidemic not terminated (diseased check)	1.59 e[z]	57.3	100.0	1.59 d	34.1	100.0

[x] Fungicide spray program initiated at time indicated and continued at 4- to 7-day intervals until maturity.
[y] Averages of five replications and values followed by a common letter are not significantly different ($P = 0.05$), according to Duncan's multiple range test.
[z] There was a stem rust epidemic during this year that accounted for about 10% yield loss and affected the yields of these two treatments.

Table 3. Loss in 1,000-kernel weight and number of grains per head from tan spot of wheat in the field as influenced by time of epidemic termination

Time of epidemic termination[y]	1,000-kernel weight		No. of grains/head	
	Average	% loss	Average	% loss
10 March (healthy check)	31.4 a[z]	...	35.0 a	...
10 April (pseudostem erect)	30.7 a	2	34.0 ab	3
5 May (boot)	26.9 b	14	32.3 ab	8
20 May (watery ripe)	24.2 c	23	31.5 ab	10
Epidemic not terminated (diseased check)	21.2 d	33	31.1 b	11

[y] Fungicide spray program initiated at time indicated and continued at 4- to 7-day intervals until crop maturity.
[z] Means of five replications. Means within a column followed by different letters are significantly different, according to Duncan's multiple range test ($P = 0.05$).

May compared with the unprotected check.

DISCUSSION

The greenhouse experiments indicated that significant losses from tan spot can occur with just a single, 48-hr infection period. Losses in grain yield as high as 33% were measured in one treatment in one experiment. One very important determinant for significant loss is the growth stage at inoculation. Our results indicate that the wheat plant is more prone physiologically to injury from tan spot at the boot and flowering growth stages. Single inoculations at the tillering or elongation stages did not consistently reduce yield, probably because of the ability of the wheat plant to "outgrow" the infections and compensate for any earlier injury. Analogously, single inoculations at the milk stage, or later, probably occurred too late in the life of the plant to cause serious damage. Although high disease severities were obtained with inoculations at the milk stage (Table 1), by the time these tan spot infections became established the plant had already accumulated enough photosynthate to reach much of its yield potential. Furthermore, the amount of healthy leaf, sheath, and glume tissue that remained after late infections could supply some additional photosynthate to complete the grain filling process. Thus, single infection periods that occur in the field from shortly before boot until the milk stage would be expected to cause more loss than similar infection periods at other growth stages.

Results from the greenhouse experiments indicated that yield loss from tan spot was due primarily to a loss in kernel wt and number of grains per head. Rees and Platz (8) showed kernel wt to be more important than grains per head in the field and results of our field experiments also showed significant reductions in kernel wt and grains per head, with kernel wt being reduced more than grains per head. However, each parameter appeared to be equally important as a determinant of final yield loss in our greenhouse studies. On the other hand, the number of heads per plant was not significantly affected by inoculation with tan spot at any growth stage. We did observe a slightly increased tendency of inoculated plants to produce very late heads. In one experiment, 24 out of 29 very late heads observed were produced from inoculated plants, although this number is insignificant when compared with the 995 heads of normal maturity produced by the plants.

Field results substantiated other reports (7,9,11) that high yield losses (30–50%) can occur from moderate to severe tan spot epidemics. Significant losses (5–10%) were also measured for early season disease when only ascospores are active in Kansas (7). This was in spite of the fact that these infection periods were 2–3 mo before harvest. The wheat did not "outgrow" or compensate for the very early injury caused by ascospore infections. These results were not contrary to the greenhouse data in which consistent, significant losses were not obtained with inoculations at early growth stages. The greenhouse experiments involved a single infection period, whereas multiple infections occurred in the field. Presumably, these multiple infections produced enough cumulative injury to the crop that it did not fully recover by maturity, whereas a single infection period produced a small, nonsignificant loss in the greenhouse. Thus, control recommendations for tan spot should take into account that about 17% of the total yield loss occurs from ascospore infections very early in the season. As well as weather conditions, the actual amount of damage is probably related to the number of pseudothecia per unit area in the field, although our level of ascospore inoculum was typical of that encountered in growers' fields.

Previous reports by some workers (1,4,8) have shown that tan spot causes more severe losses to wheat after jointing, especially between the boot and dough stages. Our results also showed significant losses during this period. However, approximately half of the yield loss occurred from infections before the boot stage. These early season losses may have been even more important during the 1985–1986 season due to stem rust contributing to late-season losses. The importance of early season tan spot is significant with regard to timing of fungicide sprays. Foliar fungicide programs for leaf and stem rust control in Kansas are usually begun after boot or even after heading. The most control of tan spot one could expect from these programs would be about 50%, which is what is actually observed (Bockus, *unpublished*). Our data suggest, since tan spot has such a long period of activity in Kansas and about 3 weeks are required after initiating a fungicide spray program to notice a reduction in disease severity, that spray programs to control it should be altered by making applications earlier than those for the rusts.

Table 4. Effect of time of epidemic termination on tan spot disease severity on winter wheat in the field

Time of epidemic termination[x]	Disease severity[y]					
	13 April	26 April	2 May	14 May	30 May	Average
10 March (healthy check)	0.49 a[z]	0.58 a	0.75 a	1.98 a	3.29 a	1.42 a
10 April (pseudostem erect)	1.96 b	1.52 b	1.53 b	2.16 a	3.40 a	2.11 b
5 May (boot)	1.98 b	1.56 b	2.05 c	2.70 b	3.94 b	2.45 c
20 May (watery ripe)	1.85 b	1.49 b	1.78 bc	2.73 b	4.35 c	2.44 c
Epidemic not terminated (diseased check)	2.01 b	1.51 b	1.61 b	3.01 b	4.56 c	2.54 d

[x] Fungicide spray program initiated at time indicated and continued at 4- to 7-day intervals.
[y] 0 = No symptoms, 1 = flecks or minute lesions, 2 = lesions with distinct yellow halos covering less than 10% of the leaf area, 3 = lesions with distinct yellow halos covering between 10 and 50% of the leaf area, 4 = numerous coalescing lesions with more than 50% of the leaf area affected, and 5 = dead leaf.
[z] Averages of five replications with the top four leaves of 20 plants per replication rated. Values followed by a common letter are not significantly different ($P = 0.05$), according to Duncan's multiple range test.

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