Effect of Nitrogen Fertilizers on Severity of Tan Spot of Winter Wheat

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ABSTRACT

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Field experiments were conducted over a 3-yr period in a split-plot design with the winter wheat (*Triticum aestivum*) cultivar TAM 105. Main-plot treatments were no fertilizer, NH_4SO_4, and $CaNO_3$ (each at 112 kg/ha total N); and subplots were presence or absence of tan spot. Fertilizers were applied in the fall (one-third of total N applied), late March (one-third), and late April (one-third). Disease evaluation was at the soft dough stage. Application of N fertilizers significantly increased tissue N levels; however, no consistent differences were observed in either disease severity or yield loss from tan spot. In addition, three experiments in the greenhouse were conducted with the same six treatments. Fertilizer application in the greenhouse significantly reduced disease severity in the inoculated treatments. However, there was severe physiologic chlorosis and necrosis from N deficiency in the nonfertilized, noninoculated check which was indistinguishable from the disease. These symptoms were alleviated with both N forms in noninoculated treatments. Thus, the apparent reduction of disease from N fertilizers observed in the inoculated treatments was due to alleviating physiologic chlorosis and necrosis. These data suggest that NH_4SO_4 and $CaNO_3$ fertilizers appear to reduce disease by delaying natural leaf senescence but do not have a direct effect on tan spot.

Tan spot is a foliar disease of winter wheat (*Triticum aestivum* L.) which is especially severe under reduced tillage (1,10–12,16,17). It is caused by the fungus *Pyrenophora tritici-repentis* (Died.) Drechs. (anamorph = *Drechslera tritici-repentis* (Died.) Shoemaker), which survives between crops as mycelia or pseudothecia in or on infested wheat residue. Because of the adoption of farming practices that reduce soil erosion, tan spot has increased in importance in Kansas and has caused an estimated average annual loss of $17 million during the past 10 yr (14).

Symptoms consist of necrotic lesions often surrounded by yellow halos (7). The fungus produces a cultivar-specific toxin (6,18) which is responsible for the necrotic symptom (7). This toxin, and/or another as yet uncharacterized one, is probably also responsible for symptoms observed on host cells beyond the hyphae within an infected leaf (8).

Older leaves are more susceptible to *P. tritici-repentis* than are younger ones in field and greenhouse environments (2,9,19). Because of these leaf-age effects, the symptomatology described above, and the involvement of at least one toxin, treatments that alter the natural senescence of leaves would be expected to affect disease severity. Nitrogen fertilizers, which tend to delay senescence, would be expected to reduce tan spot.

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The rate, form, timing, and efficiency of cultivar use of N can profoundly affect many plant diseases (4). For example, take-all root rot of cereals is affected by the amount of N; therefore, fertilizers can be used for effective control of this disease in certain circumstances (3). However, only the ammoniacal form reduces take-all, and nitrate sources may increase the disease (15). Similarly, application of NH_3 has been reported to dramatically reduce tan spot in Indiana (5). Leaf necrosis on one cultivar was reduced from 75 to 3% with nitrogen fertilizer stabilized by nitrapyrin, although nonstabilized NH_3 still decreased necrosis from 75 to 20% (5). Such levels of control would make this a highly effective management practice for the Great Plains, where tan spot is so important.

The experiments reported here were conducted to augment the finding in Indiana and to quantify the effect of nitrogen fertilizers on tan spot in Kansas. Both the ammoniacal and nitrate forms of N were included to determine the effect of the source of N on disease. Additionally, disease-free checks were included to differentiate between indirect and direct effects of nitrogen on tan spot.

MATERIALS AND METHODS

Field experiments were conducted for 3 yr (1987–88, 1988–89, and 1990–91) on Chase silty clay loam (pH 6.5, 2.3% organic matter) which had been cropped to winter wheat the previous season. During the summer prior to planting, the sites were moldboard plowed and disked as necessary to control weeds. The experimental design was a split plot with six treatments, subplots 1.22 × 7.62 m, and five replications. Main plots were not fertilized, or fertilized with NH_4SO_4 (21% N) or $CaNO_3$ (15.5% N) (each at 112 kg/ha N); and subplots were either without or with foliar fungicide. For the latter treatment, mancozeb (Dithane M-45) at 2.24 kg/ha in 187 L H_20/ha was applied weekly from mid to late March until maturity.

Seeding (67 kg/ha) was the first week in October with winter wheat cultivar TAM 105. This cultivar has been a popular commercial one in Kansas and is highly susceptible to *P. tritici-repentis*. Fertilizers were applied by broadcasting in the fall (one-third of total), late March (one-third), and late April (one-third). Oat kernels colonized by *P. tritici-repentis* were used as inoculum and were applied in early November at the rate of 225 g per plot (9). The fungus on the kernels produces pseudothecia, which releases ascospores to initiate disease. Afterwards, the conidial stage produced on infected leaves causes the epidemic (13). Disease evaluation was at the soft dough stage; the top three leaves of 25 randomly selected tillers per plot were rated for percentage of leaf area affected by necrosis and chlorosis. Immediately after disease evaluation, 30–40 randomly selected flag leaves were sampled from each plot and oven dried (60 C) for analysis of tissue N. Tissue analyses were performed by the Soil Testing Laboratory, Department of Agronomy, Kansas State University.

Three additional experiments were conducted in the greenhouse from 1988 through 1991. The six treatments described above were used with four or five replications arranged in a randomized complete-block design. A soil mix consisting of one part Kennebec silt loam, one part sand, and one part vermiculite by volume was used; and nitrogen was applied at the rate of 224 kg/ha. Additionally, noninoculated controls were used instead of the foliar-fungicide treatment to produce disease-free checks. A replication consisted of a 15-cm pot with 10 seedlings.

After 5–7 wk growth at 13–26 C (three to four fully expanded leaves), each inoculated pot was atomized with 3.0 ml of a conidial suspension (20,000 spores per milliliter). Conidia were produced on V8 agar as described previously (13). After inoculation, leaves were allowed to dry to encourage spores to stick. All pots were then placed in a mist chamber in the greenhouse without supplemental light for 48 hr, with misting for 1.5 min every 10 min. After the mist treatment,

pots were moved to benches in the greenhouse (13–26 C, without supplemental light) and rated 4–5 days later. There is no spore movement in the greenhouse or mist chamber, and noninoculated plants remained free of disease. The top three fully expanded leaves of all tillers in a pot were evaluated for percentage of leaf area affected. Immediately after disease evaluation, above-ground plant parts were harvested, oven dried (60 C), and analyzed for percentage of N in the tissue.

Individual leaf-disease ratings were averaged to obtain a mean score for each replication. Disease-severity means were arcsine square root transformed for analysis, and back transformed for presentation. Statistical analysis involved either the GLM (general linear models) or ANOVA (analysis of variance) procedures of SAS (SAS Institute, Cary, NC) followed by LSD ($P = 0.05$). Within an environment (field or greenhouse) where there were no significant year-by-treatment interactions, the years were combined for analysis.

RESULTS AND DISCUSSION

Moderate to severe tan spot developed in the field during all 3 yr. Application of fertilizer significantly ($P = 0.05$) increased the tissue N (0.34–0.62%) of flag leaves at the soft dough stage. This was a 23.1–42.0% increase over levels for nonfertilized plants. No significant differences occurred among the four treatments receiving fertilizer (*data not shown*).

The disease-severity data showed significant year-by-treatment interactions. This was because severity for the nonfertilized treatment with disease was significantly higher than that for NH_4SO_4 with disease in 1988, but significantly lower in 1991 (Table 1). Besides these inconsistencies, nitrogen fertilizer had no effect on tan spot severity in the field.

Grain yields each year were higher than those normally obtained (2,354 kg/ha) in the Great Plains (Table 2). There were no significant year-by-treatment interactions for yields. Nitrogen was not a yield-limiting factor for the field experiments, despite the fact that soil tests prior to planting recommended applying 22–45 kg/ha additional N (20). Although fertilization significantly increased tissue N, it did not affect yields; nonsprayed plots had similar yields regardless of the nitrogen treatment (Table 2). Additionally, damage (yield loss) from tan spot was severe in inoculated plots regardless of whether they were treated with additional N. Therefore, as with disease severity, nitrogen fertilization above a sufficiency level did not affect yield loss from tan spot in the field.

In the greenhouse, nitrogen fertilizers significantly increased tissue N levels by 1.21–1.52%. This was a 63.7–80.0% increase over levels for nonfertilized treatments, but there was no significant difference among the four N treatments (*data not shown*). There were significant year-by-treatment interactions for disease severity. This was because severity for the inoculated NH_4SO_4 treatment was significantly less than that for the noninoculated, nonfertilized treatment in 1988, but significantly greater in 1989 and 1991 (Table 3). This interaction did not influence the conclusions.

Unlike in the field experiments, in the greenhouse either form of N consistently reduced leaf-disease ratings for the inoculated treatments (Table 3). Reductions in severity scores ranged from 6.9 to 27.3%; however, the inclusion of noninoculated checks in these experiments allowed the differentiation of indirect and direct effects of N fertilizers on tan spot. In the noninoculated checks, reductions in severity scores from nitrogen application also occurred, ranging from 16.5 to 35.1% (Table 3). The growth medium used in the greenhouse was very low in N, which resulted in significant chlorosis and necrosis of leaves in nonfertilized treatments, simulating damage from tan spot. Therefore, the apparent benefit from nitrogen fertilization in the greenhouse was actually the result of its reduction of natural leaf senescence, not the result of a direct effect on tan spot.

These results differed from an earlier

Table 1. Effects of nitrogen fertilizers on tan spot severity on winter wheat in the field over a 3-yr period

		Disease severity (%)[y]			
Nitrogen[w]	Disease[x]	1987–88	1988–89	1990–91	Avg.
None	−	6.4 c[z]	38.5 b	5.8 c	16.9
None	+	41.0 a	65.9 a	73.2 b	60.0
NH_4SO_4	−	7.6 c	38.8 b	4.3 c	16.9
NH_4SO_4	+	31.9 b	55.8 a	82.4 a	56.7
$CaNO_3$	−	8.6 c	29.6 b	3.0 c	13.7
$CaNO_3$	+	37.9 ab	58.9 a	80.8 ab	59.2

[w] Broadcast-applied at 112 kg N/ha (total N) in the fall (1/3), late March (1/3), and late April (1/3).
[x] Healthy plots were achieved by weekly applications of mancozeb (2.24 kg/ha).
[y] Necrosis and chlorosis of the top three leaves of 25 randomly selected tillers per plot.
[z] Means of five replications are not significantly different ($P = 0.05$) if followed by the same letter within a column. Year-by-treatment interactions were significant.

Table 2. Effects of nitrogen fertilizers and tan spot on grain yields of winter wheat in the field over a 3-yr period

		Grain yields (kg/ha)				
Nitrogen[x]	Disease[y]	1988	1989	1991	Avg.	Loss (%)
None	−	4,637	3,725	4,266	4,213 a[z]	...
None	+	3,592	3,268	3,444	3,435 b	18.5
NH_4SO_4	−	4,595	3,597	4,319	4,182 a	...
NH_4SO_4	+	4,128	3,226	3,215	3,500 b	16.2
$CaNO_3$	−	4,860	3,449	4,298	4,225 a	...
$CaNO_3$	+	3,597	3,062	3,093	3,238 b	23.4

[x] Broadcast-applied at 112 kg N/ha (total N) in the fall (1/3), late March (1/3), and late April (1/3).
[y] Healthy plots were achieved by weekly applications of mancozeb (2.24 kg/ha).
[z] There were no significant year-by-treatment interactions. Values within a year are the means of five replications. For the column averaged across years, values followed by a common letter are not significantly different ($P = 0.05$).

Table 3. Effects of nitrogen fertilizers on tan spot severity on winter wheat in the greenhouse over a 3-yr period

		Disease severity (%)[y]			
Nitrogen[w]	Inoculated[x]	1988	1989	1991	Avg.
None	−	40.6 b[z]	18.0 c	35.5 c	31.7
None	+	61.4 a	56.1 a	75.5 a	65.2
NH_4SO_4	−	5.9 d	1.5 d	1.3 d	2.5
NH_4SO_4	+	34.1 c	40.7 b	67.5 b	49.0
$CaNO_3$	−	5.5 d	1.0 d	1.3 d	2.8
$CaNO_3$	+	36.9 bc	36.6 b	68.6 b	49.0

[w] Applied at 224 kg N/ha.
[x] Sprayed with 3.0 ml of a suspension containing 20,000 spores per milliliter.
[y] Mean percent necrosis and chlorosis of the top three leaves of all tillers of 10 plants per replication with four or five replications.
[z] Means followed by the same letter within a column are not significantly different ($P = 0.05$). Year-by-treatment interactions were significant.

report (5) in which NH_3 fertilizer was shown to dramatically reduce tan spot. Several explanations for the discrepancy are possible, including the different rates and forms of N used (NH_4 and NO_3 vs. NH_3). Also, the inclusion of disease-free checks in our experiments enabled us to differentiate indirect from direct effects of N fertilization on disease-severity scores. Another possible explanation could be the soil type. In the Indiana study (5), the site chosen had a low level of residual or mineralizable N relative to the needs of the plant and a rapid rate of nitrification, whereas the soils used in these experiments provided enough N for optimum yield. Even though the earlier study had a nitrapyrin variable, this apparently could not explain the different results, because treatments without nitrapyrin also showed significant (although less) disease suppression (5). Another possible explanation is that the experiments used different wheat cultivars; however, we have conducted greenhouse experiments involving four additional cultivars with various levels of resistance to the necrosis and/or chlorosis symptoms (7) and obtained similar results to those presented (*unpublished*). Nevertheless, as was suggested in the earlier work (5), certain wheat genotypes may have a resistance mechanism that is sensitive to N fertility. This is a distinct possibility (3,4) and needs further evaluation.

The rates of nitrogen used in the experiments reported here are about the maximum a producer would consider applying in the Great Plains and were in excess of sufficiency for optimum yield. Similarly, splitting the rate into three applications would be the maximum. Because we could not demonstrate a reduction in tan spot from these management practices, we do not believe that excess ammoniacal or nitrate N would have a great effect on the disease in this area. However, we have shown that N fertilizers can produce an apparent benefit by reducing leaf senescence, although they do not directly reduce the disease. Tan spot is still capable of causing significant losses in the Great Plains, even when wheat is grown with adequate, or more than adequate, nitrogen fertilizer.

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