Triazole Seed Treatments Suppress Spore Production by *Puccinia recondita*, *Septoria tritici*, and *Stagonospora nodorum* from Wheat Leaves

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ABSTRACT

Sundin, D. R., Bockus, W. W., and Eversmeyer, M. G. 1999. Triazole seed treatments suppress spore production by *Puccinia recondita*, *Septoria tritici*, and *Stagonospora nodorum* from wheat leaves. Plant Dis. 83:328-332.

Treatments of winter wheat seed with the systemic triazole fungicides triadimenol (31 g a.i./100 kg = Baytan 30F at 1.5 fl oz/cwt) and difenoconazole (24 g a.i./100 kg = Dividend 3FS at 1.0 fl. oz/cwt) were tested for effect on asexual sporulation by *Puccinia recondita*, *Septoria tritici*, and *Stagonospora nodorum*. Spore production was measured on seedlings grown in a growth chamber (24°C day/15°C night, 12-h photoperiod) and inoculated with the pathogens 3, 5, or 7 weeks after sowing. Spore production was converted to a percentage of the non-treated control and regressed against weeks after planting when plants were inoculated. Linear models fit data for both fungicides against all three pathogens. According to the models, difenoconazole suppressed sporulation levels of *P. recondita* and *Septoria tritici* to 10% of the levels on plants from non-treated seed for about 3 weeks after sowing. Spore production for all three fungi was suppressed to 25% of the non-treated level for at least 4.2 weeks and to 50% for at least 6.5 weeks. Similarly, triadimenol suppressed all three pathogens to 50% of the non-treated level for at least 3.2 weeks. The two fungicides showed similar effects against *S. tritici*; however, difenoconazole showed significantly greater suppression of sporulation by *P. recondita* and *Stagonospora nodorum* compared with triadimenol. Responses occurred even though large concentrations of spores were used to inoculate plants and environmental conditions were optimized for spore production. Reduced sporulation should help protect fall-planted wheat seedlings and may significantly delay epidemics in the following spring.

Based upon crop survey data, annual loss estimates due to various wheat diseases are made in Kansas (24). From 1976 through 1997, the average yield loss due to leaf rust (caused by *Puccinia recondita* Roberge ex Desmaz.) and the "Septoria complex" (caused by *Septoria tritici* Roberge in Desmaz. and *Stagonospora nodorum* (Berk.) Castellani & E. G. Germano) has been 5.3%; however, yearly losses ranged from a low of 1% to a high of 14% (2). During 1992 and 1993, for example, leaf rust alone caused 11% yield loss. Similarly, the Septoria complex produced a 7.4% loss in 1995. These losses have occurred in spite of all the current disease control measures practiced by producers. Clearly, additional controls are needed to help manage these foliar wheat pathogens.

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Fungicide seed treatments are used extensively on almost all corn, sorghum, peanut, rice, and cotton seed planted in the United States. In addition, more than half of all small grain and vegetable seed are treated (20). Fungicide seed treatments have been used to suppress seed rot, prevent damping off, and inactivate seed-borne pathogens. On wheat (*Triticum aestivum* L.), fungicide seed treatment has traditionally been used to control the seed-borne pathogens causing common bunt (caused by *Tilletia caries* (DC.) Tul. & C. Tul.) and loose smut (caused by *Ustilago tritici* (Pers.) Rostk.; 11,13), with systemic fungicides being necessary to control loose smut. For many years, carboxin (trade name Vitavax) has been the standard systemic seed-treatment fungicide for wheat (21,25). More recently, two systemic triazole fungicides, difenoconazole (trade name Dividend) and triadimenol (trade name Baytan), have been registered in the United States for use on wheat seed. The systemic triazoles inhibit the biosynthesis of ergosterol and have a broad spectrum of activity, which includes activity on the bunt and loose smut pathogens (6).

One of the advantages of the sterol inhibitor fungicides is that they have the ability to control root diseases that previously were not controlled by seed treatments. For example, triadimenol-treated

wheat seed resulted in plants with less infection by *Rhizoctonia solani* Kühn (17). Similarly, difenoconazole and triadimenol reduced take-all (caused by *Gaeumannomyces graminis* (Sacc.) Arx & D. Olivier var. *tritici* J. Walker) and common root rots (caused by *Bipolaris sorokiniana* (Sacc.) Shoemaker) of wheat (4,5,12,18). Some disease control was associated with protection of young roots from infection by these fungi.

In addition to control of root diseases, fungicide seed treatments have the potential to affect foliar diseases. *Stagonospora nodorum* is a seed-borne and residue-borne pathogen of wheat that causes Stagonospora blotch. Seed treatments with difenoconazole and triadimenol have resulted in less disease on flag leaves and heads (19). In those experiments, triazole seed treatments also reduced disease even when wheat was planted into infested residue. Although the relative importance of residue- and seed-borne inocula was not determined in that experiment, the results imply that the fungicides were translocated from the seed into the leaves to protect foliage, thus delaying the epidemic. Similarly, triadimenol can suppress the foliar diseases powdery mildew (caused by *Blumeria graminis* (DC.) Golovin ex Speer) and leaf rust on wheat (9,15). Therefore, even for foliar pathogens that have rapid repeating cycles, seed treatment can reduce epidemic development on adult wheat plants.

Although seed treatments with difenoconazole and triadimenol appear to reduce some foliar wheat diseases in the field, their effect upon pathogen spore production has not been determined. The objective of this research was to quantify the duration of control that seed treatment with these two triazoles could provide. The measured parameter was spore production from leaves in a controlled environment over time by three important wheat pathogens, *P. recondita*, *Septoria tritici*, and *Stagonospora nodorum*.

MATERIALS AND METHODS

Plant culture. All experiments were conducted in a growth chamber with 12 h of light at 24°C followed by 12 h of dark at 15°C. The hard red winter wheat cultivar used in this study (Newton) is susceptible to leaf rust, Septoria tritici blotch (caused by *Septoria tritici*), and Stagonospora

blotch. Each experimental unit consisted of six seeds planted in a 13-cm-diameter pot containing 50% soil by volume (Kennebeck silty clay loam) and 50% vermiculite. Plants were thinned later to three per pot for uniformity. Experiments were a 3-by-3 factorial with five replications (pots). Treatments included a non-treated control, triadimenol (31 g a.i./100 kg = Baytan 30F at 1.5 fl oz/cwt), and difenoconazole (24 g a.i./100 kg = Dividend 3FS at 1.0 fl oz/cwt), each with three inoculation times (3, 5, and 7 weeks after sowing). To apply fungicides, a lot of seed first was treated in a glass canning jar, discarded, and the "conditioned" jar used to treat seed for experiments. A total of 45 pots were planted for each run of an experiment for a single pathogen, 15 pots of each seed treatment. At each inoculation time, five pots of each seed treatment were inoculated with *P. recondita*, *Septoria tritici*, or *Stagonosporum nodorum*.

Fungal culture and quantification. For experiments involving *P. recondita*, an isolate virulent to cv. Newton that had been collected from the field was obtained from the United States Department of Agriculture-Agriculture Research Service in Manhattan, Kansas. An ampoule of urediniospores was removed from liquid nitrogen and heat shocked by immersion in a 40°C water bath for 5 min. Spores then were suspended in a light mineral oil (Soltrol) at a concentration of 9×10^6 per ml, and 5.0 ml of the suspension was applied with an atomizer to 15 pots. After inoculation, plants were placed into a mist chamber at 25°C for 16 h to maintain continual leaf wetness and then returned to the growth chamber for 7 days. At that time, many pustules had erupted through the epidermis but significant loss of spores from pustules due to wind currents in the growth chamber had not occurred. The lower three leaves of each plant were then removed, cut into 7-cm pieces, and placed in 500-ml Erlenmeyer flasks containing 50 ml of distilled water with one drop of Tween 20. Flasks were shaken manually for 20 s, an aliquot of the rinsate was removed, and urediniospores were counted using a hemacytometer. For each plant, the numbers of spores in four 0.0001-ml subsamples of the rinsate were determined and averaged.

For experiments with *Septoria tritici*, a single-conidial isolate was obtained from infected wheat in a field in Harvey County, Kansas and stored on one-quarter-strength potato dextrose agar at 4°C. For inoculations, the isolate was grown in one-half-strength potato dextrose broth for 6 days at 24°C. Cultures were filtered through cheesecloth and adjusted with distilled water to a concentration of 2.0×10^6 spores/ml. The spore suspension (30 ml) was applied to 15 pots using an atomizer (DeVilbiss Co., Somerset, PA) operated at 69 kPa. Inoculated plants were placed in a mist chamber at 25°C for 96 h to maintain

continual leaf wetness and then returned to the growth chamber. After 3 weeks, the bottom three leaves of each plant were removed and processed as described below.

For experiments with *Stagonosporum nodorum*, a single-conidial isolate was obtained from infected wheat collected from a field in Harvey County. For storage purposes, spore suspensions of the isolate were kept in 15% glycerol at -20°C. For inoculations, spores of the isolate were streaked onto plates of V-8 agar and grown at 25°C, 30 cm below fluorescent lights (constant light). After 7 days of growth, plates were flooded with 5 ml of distilled water to suspend pycnidiospores and the suspension was passed through cheesecloth. Spore suspensions were adjusted to 4.5×10^6 spores/ml and 30 ml were applied to plants in 15 pots as described above. Inoculated plants were placed in a mist chamber for 48 h and then returned to the growth chamber for 2 weeks. At sampling time, the bottom three leaves of each plant were harvested and processed.

To obtain spore counts for *Septoria tritici* and *Stagonosporum nodorum*, leaves were cut into 7-cm pieces, which were placed into glass petri dishes with moistened filter paper and held in an incubator under 12 h of light at 16°C followed by 12 h of dark at 12°C. Preliminary experiments were conducted to determine the optimum time to incubate leaves. As a result of those experiments, leaf pieces from each plate were blended in a tissue homogenizer with 25 ml of distilled water after 7 (*Septoria tritici*) or 4 days (*Stagonosporum nodorum*) of incubation. An aliquot was removed from the homogenate and spores were counted in a hemacytometer under 100× magnification. For each petri dish, the numbers of spores in four 0.0001-ml sub-samples were determined and averaged.

Statistical analyses. Sporulation data for the two fungicide treatments were converted to a percentage of the non-treated control and analyzed using SAS (SAS Institute, Cary, NC). For each of the three pathogens, percentage values of all individual replicates were regressed against the number of weeks after planting when leaves were inoculated with spores of the pathogens. Linear regression models were fit to the data and two linear models were produced for each pathogen, one for each fungicide. To determine the relative efficacy of the fungicides against a pathogen, the slopes of the models for triadimenol and difenoconazole were compared. When slopes were not significantly different, the estimates of the *Y* intercepts were compared. All experiments were conducted at least twice with similar results and were combined.

RESULTS

Average spore concentrations in leaf rinsates from non-treated plants were $0.18 \times$

10^6 spores/ml for *P. recondita*, 1.63×10^6 spores/ml for *Septoria tritici*, and 0.22×10^6 spores/ml for *Stagonosporum nodorum*. After treatment of seeds with triadimenol or difenoconazole, suppression of sporulation by all three pathogens from leaves was significant. Spore production relative to the non-treated control was reported as the time after planting that leaves were challenged with the pathogens. To allow time for growth and reproduction of the fungi, a 1.1-, 3.6-, or 2.3-week lag period was allowed between inoculation and measurement of sporulation for *P. recondita*, *Septoria tritici*, and *Stagonosporum nodorum*, respectively. Therefore, a conservative estimate of the duration of suppression is presented.

With one exception, linear models significantly ($P < 0.05$) fit the responses observed for all three pathogens to the two fungicides (Figs. 1–3). For the effect of triadimenol on sporulation by *P. recondita*, the linear model was not significant at $P < 0.05$ but was significant at $P = 0.0674$ (Fig. 1). A repeat experiment also showed similar *P* values (data not shown); nevertheless, the response of *P. recondita* to triadimenol was considered to follow a linear relationship ($P < 0.10$).

For *P. recondita* and *S. nodorum*, the slopes of the response to the two fungicides were not significantly different; however, estimates of the *Y* intercepts were significantly ($P < 0.05$) less for the difenoconazole response curves than for the triadimenol response curves (Figs. 1 and 3). For *Septoria tritici*, no significant differences occurred between fungicides in the slopes or estimates of the *Y* intercepts for the linear models (Fig. 2).

The models generated from data (Figs. 1–3) were used to estimate the time (in weeks) that each seed-treatment fungicide would suppress sporulation of each of the three pathogens to certain selected levels. The levels chosen were 10, 25, 50, and 75% of the non-treated control level, representing 90, 75, 50, and 25% suppression of sporulation, respectively (Table 1).

DISCUSSION

Data presented in the current study show substantial reductions in the sporulation of three important foliar wheat pathogens following seed treatment with triazole fungicides. Difenoconazole suppressed sporulation levels of *P. recondita* and *S. tritici* to 10% of the levels observed on plants from non-treated seed for at least 3.1 weeks after sowing. Similarly, this fungicide suppressed all three pathogens to 25% of the non-treated levels for at least 4.2 weeks, and to 50% for at least 6.5 weeks. Triadimenol suppressed all three pathogens to 50% of the non-treated level for at least 3.2 weeks and 75% for at least 6.6 weeks. These time periods are equivalent to a significant portion of the fall growth period for winter wheat in Kansas. Although one

Fig. 1. Effect of treatment of wheat seed with triadimenol or difenoconazole on urediniospore production by *Puccinia recondita* when inoculated 3, 5, or 7 weeks after planting, expressed as a percentage of the non-treated control. Linear regression models are $Y = 7.51X + 25.7$ ($r = 0.3315$, $P = 0.0674$) for triadimenol and $Y = 11.8X - 27.0$ ($r = 0.6144$, $P = 0.0004$) for difenoconazole. The slopes were not significantly different ($P = 0.3837$) but the estimates of the intercepts were significantly different ($P = 0.0003$).

Fig. 2. Effect of treatment of wheat seed with triadimenol or difenoconazole on spore production by *Septoria tritici* when inoculated 3, 5, or 7 weeks after planting, expressed as a percentage of the non-treated control. Linear regression models are $Y = 13.8X - 31.8$ ($r = 0.4997$, $P = 0.0032$) for triadimenol and $Y = 9.36X - 24.1$ ($r = 0.5836$, $P = 0.0007$) for difenoconazole. The slopes were not significantly different ($P = 0.3863$) and the estimates of the intercepts were not significantly different ($P = 0.1337$).

report (16) indicates that triadimenol does not affect *Stagonosporum nodorum*, our results corroborate the findings of Milus and Chalkley (19) that showed suppression of this fungus by triadimenol. The data presented here apparently are the first to quantify the effects of systemic seed-treatment fungicides on sporulation by the pathogens, as opposed to effects on disease severity.

The suppression estimates reported here are likely to be conservative, because our experiments were conducted in a controlled environment that would be expected to enhance the loss of fungicidal activity compared with a field environment (14,26). For example, the temperature regime used (24°C day and 15°C night) simulated temperatures usually encountered during early seedling emergence in the field; however, after emergence, temperatures in the field would rapidly decline as winter approached. Lower temperatures would help maintain fungicidal activity by slowing plant growth that dilutes the chemical and would slow chemical degradation by various processes (14,26). Similarly, we used concentrated spore suspensions to inoculate plants and optimum environmental conditions for infection and sporulation. Plants in the field usually are not exposed to such high inoculum pressure in the fall.

Suppression of sporulation by seed treatment with both fungicides followed linear progressions (Figs. 1–3). Pathogen sporulation on plants from treated seed relative to that on plants from non-treated seed increased over time, presumably because of inactivation of the fungicides and dilution as the plant absorbed and translocated the chemicals to new growth. For all three pathogens, the slopes of the response lines were similar for difenoconazole and triadimenol, indicating that both fungicides lost their suppressive activity at similar rates. Also, the estimates of the Y intercepts for *Septoria tritici* were similar for both fungicides, indicating no difference between them in suppressive activity against that pathogen. However, for *P. recondita* and *Stagonosporum nodorum*, the estimates of the Y intercepts were significantly different, indicating that difenoconazole provided significantly greater suppression than triadimenol. For example, difenoconazole reduced sporulation by *P. recondita* to about 10% of the non-treated level 3 weeks after planting, whereas triadimenol reduced it only to about 50%.

The reason for greater suppression of two of the three pathogens by difenoconazole is not known. Only one rate of each chemical was used (the highest labeled rate), so different results might occur at different rates. Furthermore, the patterns of translocation of the fungicides in the plant might differ. Quantitative measurements of the compounds in the leaves would have to be done to determine if different micro-

molar amounts of the fungicides are transported to the leaves. Additionally, the specific activities of the two chemicals against the pathogens might differ; however, 29% more triadimenol than difenoconazole was applied (31 and 24 g a.i./100 kg, respectively) and yet triadimenol provided lower suppression. Ultimately, the differences seen in inhibition of sporulation may be at the biochemical level, where difenoconazole might be more efficient at inhibiting demethylation of the sterol precursors in *P. recondita* and *S. nodorum* (14).

The amount of overwintering inoculum is a critical determinant of the amount of leaf rust in Kansas (8). The severity of the winter dictates the amount of pathogen survival. When harsh winters kill wheat

leaf tissue and, thus, the obligate parasite *P. recondita*, urediniospores must be blown into Kansas from wheat-growing areas in Oklahoma and Texas. This long-range introduction causes a sufficient delay in the epidemic to attenuate the impact of leaf rust in Kansas wheat crops (8). Therefore, in surviving leaf tissue, fungal mycelium from fall infections can sporulate early in the spring, enhancing leaf rust epidemics (8). Any management practice that would reduce infection of the wheat in the fall should help lower the amount of overwintering by *P. recondita* in Kansas. Based upon results presented here, seed treatments with triazole fungicides may help protect the wheat during the important fall growth period.

Both *Septoria tritici* and *Stagonosporum nodorum* survive the summer on infested host debris, and air-borne ascospores can serve as the primary inoculum (10,22,27). *S. nodorum* can be seed-borne and, in some locations, conidia of *S. nodorum* may serve as primary inoculum (19). Spores that function as primary inoculum for *Septoria tritici* are dispersed from autumn through winter (27), whereas those for *Stagonosporum nodorum* are dispersed mainly during August through October in Poland (3). If this pattern occurs in Kansas, primary inoculum is most abundant at the same time as the emergence of winter wheat seedlings. This inoculum produces mostly asymptomatic infections where the fungi overwinter. After the winter, asexual sporulation occurs from expanding lesions caused by primary inoculum, resulting in secondary spread (23). Several repeating cycles occur and produce leaf necrosis and yield losses. Therefore, as with *P. recondita*, protecting plants from infection by *Septoria tritici* and *Stagonosporum nodorum* during the fall could reduce the development of epidemics in the following spring. Both difenoconazole and triadimenol resulted in significant suppression of pathogen spore formation for several weeks on wheat seedlings. Such partial control should result in healthier plants entering the overwintering period.

Triadimenol has suppressed powdery mildew and leaf rust on wheat in the field (9,15). Therefore, seed treatment with triazoles apparently can affect foliar pathogens that have multiple repeating cycles. The fungicide is translocated from the seed into leaves, resulting in reduced disease. Measurement of carbon-14-labeled triadimenol showed that 7.7% of the compound applied to wheat seed was taken up by the leaves within 51 days, with the largest quantity in the first three leaves (14). Such translocation to the foliage early in the season apparently delays epidemic development.

Currently, only 22% of the wheat seed in Kansas is treated with a fungicide and over half of that is treated with carboxin (1). Our data indicate that more producers should investigate adopting triazole seed treatment as a production practice. Triazole fungicides provided significant suppression of sporulation by three of the four most important foliar pathogens in Kansas. However, our results were in a controlled environment, and additional experiments are necessary to substantiate this effect in producers' fields and quantify the potential economic return from this practice. Nevertheless, the models of sporulation suppression presented here should be useful in helping to predict and interpret responses to seed treatment observed in the field (7,9,15,19,25).

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Fig. 3. Effect of treatment of wheat seed with triadimenol or difenoconazole on spore production by *Stagonospora nodorum* when inoculated 3, 5, or 7 weeks after planting, expressed as a percentage of the non-treated control. Linear regression models are $Y = 8.02X + 19.2$ ($r = 0.4263$, $P = 0.0121$) for triadimenol and $Y = 8.83X - 12.5$ ($r = 0.4980$, $P = 0.0033$) for difenoconazole. The slopes were not significantly different ($P = 0.8494$) but the estimates of the intercepts were significantly different ($P = 0.0001$).

Table 1. Number of weeks after sowing that seed treatment with two triazole fungicides reduced spore production by three pathogens of winter wheat to 10, 25, 50, or 75% of the non-treated control

Pathogen (treatment)	Percentage of non-treated control			
	10	25	50	75
<i>Puccinia recondita</i>				
Difenoconazole	3.1 ^a	4.4	6.5	>7.0 ^b
Triadimenol	N/A ^c	N/A	3.2	6.6
<i>Septoria tritici</i>				
Difenoconazole	3.6	5.2	>7.0 ^b	>7.0 ^b
Triadimenol	3.0	4.1	5.9	>7.0 ^b
<i>Stagonospora nodorum</i>				
Difenoconazole	<3.0 ^b	4.2	>7.0 ^b	>7.0 ^b
Triadimenol	N/A	<3.0 ^b	3.8	7.0

^a Calculated from linear models.

^b Outside the observation range of experiments (3 to 7 weeks).

^c N/A = not applicable; fungicide cannot reduce sporulation to that level.

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