Quantifying the Effects of Wheat Residue on Severity of *Stagonospora nodorum* Blotch and Yield in Winter Wheat

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Accepted for publication 1 July 2015.

ABSTRACT

Mehra, L. K., Cowger, C., Weisz, R., and Ojiambo, P. S. 2015. Quantifying the effects of wheat residue on severity of *Stagonospora nodorum* blotch and yield in winter wheat. *Phytopathology* 105:1417-1426.

Stagonospora nodorum blotch (SNB), caused by the fungus *Parastagonospora nodorum*, is a major disease of wheat (*Triticum aestivum*). Residue from a previously infected wheat crop can be an important source of initial inoculum, but the effects of infected residue on disease severity and yield have not previously been quantified. Experiments were conducted in Raleigh and Salisbury, North Carolina, in 2012, 2013, and 2014 using the moderately susceptible winter wheat cultivar DG Shirley. In 2014, the highly susceptible cultivar DG 9012 was added to the experiment and the study was conducted at an additional site in Tyner, North Carolina. Four (2012) or six (2013 and 2014) wheat residue treatments were applied in the field in a randomized complete block design with five replicates. Treatments in 2012 were 0, 30, 60, and 90%

residue coverage of the soil surface, while 10 and 20% residue treatments were added in 2013 and 2014. Across site-years, disease severity ranged from 0 to 50% and increased nonlinearly ($P < 0.05$) as residue level increased, with a rapid rise to an upper limit and showing little change in severity above 20 to 30% soil surface coverage. Residue coverage had a significant ($P < 0.05$) effect on disease severity in all site-years. The effect of residue coverage on yield was only significant ($P < 0.05$) for DG Shirley at Raleigh and Salisbury in 2012 and for DG 9012 at Salisbury in 2014. Similarly, residue coverage significantly ($P < 0.05$) affected thousand-kernel weight only of DG 9012 in 2014 at Raleigh and Salisbury. Our results showed that when wheat residue was sparse, small additions to residue density produced greater increases in SNB than when residue was abundant. SNB only led to effects on yield and test weight in the most disease-conducive environments, suggesting that the economic threshold for the disease may be higher than previously assumed and warrants review.

Stagonospora nodorum blotch (SNB), caused by the ascomycete fungus *Parastagonospora nodorum* (Quaedvlieg et al. 2013), occurs in wheat-growing areas worldwide, but is more prevalent in areas with warm and moist conditions such as the southeastern United States, parts of Europe, southern Brazil, and Australia (Eyal et al. 1987; Murray and Brennan 2009). Temperatures of 20 to 25°C with high relative humidity (> 75%) and frequent rainfall favor the development of the disease (Verreet and Hoffmann 1990). The pathogen is capable of infecting wheat (*Triticum aestivum* L.) at both the seedling and adult stages. Typical disease symptoms include a necrotic leaf blotch as well as discoloration of the head, which is usually known as glume blotch. After about a week following infection and under high humidity, pycnidia begin to form in the necrotic lesions.

SNB affects both the quantity and quality of wheat grain (Eyal 1999). Reduction in yield can occur due to reduced photosynthetic area of the upper leaves, which leads to a decrease in the level of carbohydrates available for seed fill (Verreet and Hoffmann 1989), and hence, lower thousand-kernel weight (Gilbert and Tekauz 1993). Yield losses up to 50% due to SNB have been reported in Europe and the United States (Bhathal et al. 2003; Fried and Meister 1987; King et al. 1983; McKendry et al. 1995; Shipton et al. 1971), while in Australia, the disease has been reported to account for 12% of yield losses in wheat (Murray and Brennan 2009). SNB reduced test weight, which is a measure of grain quality, by about 6% in

untreated compared with fungicide treated plots in Arkansas, United States (Milus and Chalkley 1997). The milling quality of grain can also be reduced by infection of the developing seed, causing shriveling, discoloration, and reduced flour yield (Bhathal et al. 2003; Gilbert and Tekauz 1993).

The primary sources of inoculum for initial infection in the field can include infected seed harboring mycelia of the fungus, pycnidiospores or conidia (asexual spores) from infected wheat debris, or windborne sexual ascospores (Arseniuk et al. 1998; Eyal 1999; Shah et al. 1995). Pseudothecia, which are the sexual fruiting bodies of *P. nodorum*, contain asci and play a major role in the survival of the pathogen. A mark-release-recapture study in Switzerland showed that sexual recombination occurs in the field, and airborne immigrant ascospores can be responsible for primary infection (Sommerhalder et al. 2010). Several other studies have also indicated that ascospores are important as a source of primary inoculum (Bathgate and Loughman 2001; Bennett et al. 2007; Sommerhalder et al. 2006). In North Carolina, ascospores could play a role in initiating SNB epidemics (Cowger and Silva-Rojas 2006), while both ascospores and seedborne inoculum are involved in initial plant infections in New York (Shah and Bergstrom 2000; Shah et al. 1995, 2001).

SNB is managed through cultural practices, fungicide application, and planting resistant cultivars (Engle et al. 2006; Luke et al. 1983; Milus and Chalkley 1997). The primary cultural management practices include crop rotation and burying infected wheat residue by tillage to reduce the survival of the pathogen from one season to the next (Luke et al. 1983; Milus and Chalkley 1997). Wheat straw infected with *P. nodorum* can serve as a major source of primary inoculum (Holmes and Colhoun 1975), especially in the case of wheat planted no-till with wheat debris from the previous crop remaining on the soil surface.

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<http://dx.doi.org/10.1094/PHYTO-03-15-0080-R>

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The widespread adoption of conservation tillage to reduce soil erosion and conserve moisture (Shaver et al. 2013) has resulted in an increase of crop residue on the soil surface, hence availability of more primary inoculum, and has been linked to an increase in the severity of SNB in the eastern United States (Shaner and Buechley 1995). The effect of tillage on the epidemiology of a disease is complex, but the impact on disease intensity generally relates to the amount of residue remaining after planting (Boosalis et al. 1986). Although the amount of wheat debris on the ground has been shown to influence severity of SNB (Luke et al. 1983; Milus and Chalkley 1997), a quantitative relationship between the level of soil-surface residue and SNB severity is still lacking. These previous studies (Luke et al. 1983; Milus and Chalkley 1997) have also not related levels of residue to yield in wheat. For example, Luke et al. (1983) reported that residue from previous crops was an important inoculum source that influenced the level of SNB, but they neither specified the amount of residue in their plots nor quantified the relationship between wheat residue and severity of SNB. Similarly, Milus and Chalkley (1997) reported a higher incidence and severity of SNB in plots infested with wheat residue than in clean plots, but they did not specify the amount of residue in the infested plots nor relate residue levels to disease intensity.

Preplanting management factors that influence the risk of SNB include crop residue, crop rotation, disease history, cultivar selection, seed treatment, and location of field. Quantifying the effects of residue on the severity of SNB and yield is fundamental in developing models that can be used to make SNB management decisions prior to planting of a wheat crop. Therefore, the specific objectives for this study were to quantify the effect of amount of wheat residue on severity of SNB, and determine the impact of wheat residue on yield and yield components in soft red winter wheat. This work was part of a larger study designed to determine how preplanting disease management factors influence the risk of SNB on winter wheat during the growing season.

MATERIALS AND METHODS

Field sites and wheat cultivars. Field experiments were conducted during the 2011–2012, 2012–2013, and 2013–2014 growing seasons (hereafter referred to as 2012, 2013, and 2014, respectively) at the Lake Wheeler Road Field Laboratory in Raleigh and at the Piedmont Research Station in Salisbury, North Carolina. In 2014, experiments were also conducted at a grower's field in Tyner, North Carolina. All field plots were prepared using conventional tillage and within each location, experiments were conducted in different field plots every year. The previous crops in the field before conducting the experiments were as follows: fallow (no crop) at Raleigh, corn at Salisbury, and soybean at Tyner. The cultivar Dyna-Gro Shirley (hereafter referred to as DG Shirley), which is moderately susceptible to *P. nodorum*, was used at all sites due to its resistance to other wheat diseases such as powdery mildew and leaf rust that are prevalent in the region. In addition, a highly susceptible cultivar DG 9012 was used in the 2014 experiments to determine whether results obtained on DG Shirley (in 2012 and 2013) were broadly similar to those of a more susceptible cultivar. DG 9012 is moderately resistant to leaf rust but moderately susceptible to powdery mildew.

Certified seed was used in this study and seed for the two cultivars was purchased from commercial seed suppliers in eastern North Carolina. All production practices, such as seeding rate, seed treatment, and planting date, were performed according to the Small Grain Production Guide for North Carolina (Weisz 2013). Prior to planting, seed was treated with Vitavax (Chemtura, Australia), a product that is moderately effective in controlling *P. nodorum* in seed. Additionally, three replicate samples of 20 kernels were arbitrarily selected and plated on SNAW (a selective medium) as described below were found to be free of *P. nodorum*. Seed was planted using a research plot planter with 0.20-m row spacing.

Experimental plots were 1.0 m wide and ranged from 5.5 to 6.4 m in length, except in Salisbury in 2012, where the plots were 7.3 m long. To minimize interplot interference, each plot was surrounded by buffer plots of the same size as treatment plots. In 2012, buffer plots were planted with wheat cultivar NC-Neuse, which is moderately resistant to *P. nodorum*, while barley, which is a nonhost of *P. nodorum*, was planted in buffer plots in 2013 and 2014.

Weather data in Raleigh and Salisbury were acquired using standard automated weather stations located less than 1.5 km away from the field plots. Temperature and relative humidity at Tyner were recorded using portable data loggers (WatchDog, Spectrum Technologies, Plainfield, IL), while rainfall was monitored from the nearest automated national weather station. Typically, weather data were captured from late February until the last disease assessment date. When necessary, irrigation was conducted at the Raleigh and Salisbury sites during dry periods to promote disease development, and the amount of irrigation water was recorded.

Wheat residue treatments and experimental design. In 2012, four wheat residue treatments were tested, namely 0, 30, 60, and 90% of plot area covered with residue. The 0% treatment corresponded to conventional tillage representing complete burial of wheat residue; 30 and 60% residue treatments simulated gradations of minimum tillage; and the 90% residue treatment simulated no-tillage (Stubbs et al. 2004). In 2013 and 2014, residue levels of 10 and 20% soil surface coverage were added to the experiment to better investigate the low end of the range, for a total of six treatments. Wheat residue used in each year was obtained from a single source, with a different commercial wheat grower providing the residue used in each year. The residue was obtained from Rowan (2012), Forsyth (2013), and Stokes (2014) counties in the Piedmont region of North Carolina, where SNB epidemics occur frequently, and in each case the residue was baled immediately after harvest of the crop. The baled residue was stored under cover and then transported to the experimental sites for application to plots in midwinter. In all cases, SNB was present in the previous crop and the presence of *P. nodorum* pycnidia was confirmed on the straw by microscopic examination prior to application of treatments.

The percentage of residue cover in a plot for each treatment was estimated as follows. First, dry weights of residue needed to provide 95 to 100% soil surface coverage were established, using the line transect method (Wollenhaupt and Pingry 1991). From this relationship, the equivalent dry weight of residue to be applied for each treatment was then determined. To avoid smothering of seedlings, residue was applied to plots between mid-January and mid-February when the wheat was at Zadoks growth stage (GS) 21 to 23 (main stem and one to three tillers, Zadoks et al. 1974). Residue application in *Septoria* screening nurseries has shown that the application of residue at planting or before GS 21 results in patchy stands (C. Cowger, *personal observation*). At each site, the experiment was laid out in a randomized complete block design, with five replicates of each residue treatment.

Disease assessment. To quantify disease severity, the percentage of leaf area showing symptoms of SNB was estimated on a whole-plot basis. Briefly, disease severity in each plot was assessed in five distinct sections ($\sim 0.1 \text{ m}^2$) spread across the entire plot. The percent leaf area with SNB symptoms in each section was estimated visually and the mean leaf area across all the five sections was the overall disease severity for the plot. The lowest leaves that senesced due to maturity or abiotic factors were not included in the visual assessment. Typically, the first disease assessment was recorded when SNB lesions at least 1 cm in length were visible on lower leaves. This first assessment was between Zadoks GS 37 (flag leaf visible) and GS 45 (swollen boot) growth stages. Disease was assessed every 2 weeks for a total of three to four assessment records depending on the intensity and progression of the disease. Final disease severity was recorded around Zadoks GS 85 (soft dough) when the flag leaf and the F-1 leaf started to senesce. The same evaluator conducted all assessments of disease severity at all locations.

Harvest and grain assay. In each year, grain from all plots was harvested at plant maturity using a small-plot research combine. Grain moisture and test weight were then determined using a grain analysis computer (GAC 2100, Dickey-John Corp., Auburn, IL). Subsequently, grain weight from each treatment plot was used to determine the corresponding yield after adjusting the grain weight to moisture content of 13.5% (Cowger et al. 2010). In the 2013 and 2014 trials, 1,000 kernels were also randomly selected from each treatment plot and weighed to determine the thousand-kernel weight. Thousand-kernel weight is a measure of seed size, and is thus related to both yield and test weight.

To determine the incidence of kernels colonized by *P. nodorum* in grain, a sample of 20 arbitrarily selected kernels from each replicate was plated on SNAW (Manandhar and Cunfer 1991). Two Petri dishes (100 mm × 15 mm), each containing 10 kernels, were used per replicate for each treatment, and there were five replicates per treatment. Kernels colonized by *P. nodorum* were assessed 7 days after plating under a 15-watt near-ultraviolet light (model TL-D BLB, Philips, Somerset, NJ). *P. nodorum* produces metabolites in oxgall (an ingredient of SNAW medium) that fluoresce under near-ultraviolet light (Manandhar and Cunfer 1991). The percentage of infected kernels was calculated as the number of colonized kernels divided by the total number of plated kernels and multiplied by 100.

Data analysis. Disease severity over time was used to calculate the area under the disease progress curve (AUDPC) as described by Campbell and Madden (1990). To allow for direct comparison of AUDPC values obtained from different epidemic durations at different site and years, relative AUDPC (RAUDPC) was calculated as $RAUDPC = AUDPC/\Delta t$, where Δt = time between the first and last disease assessment.

The effects of residue level on maximum disease severity, RAUDPC, yield, test weight, thousand-kernel weight, and incidence of colonized kernels were determined by analysis of variance using the GLIMMIX procedure in SAS 9.3 (SAS Institute, Cary, NC). Due to differences in the number of treatments and cultivars used in different years, the analysis was conducted separately for each site and year. Replicates were treated as blocks and specified as a random effect, while treatments were specified as a fixed effect in the model. Further, to account for possible extraneous variation due to trends in soil moisture or fertility that could be associated with rows and columns in the plots, a row error model (Kehel et al. 2010) was implemented within the GLIMMIX procedure to separate independent random effects associated with rows of the trial and the independent plot error. The row error model was used to increase accuracy and precision in the estimates of the effect of residue treatment on yield. The random row error model and the base model were compared using the Akaike information criterion (Kehel et al. 2010). Treatment means were separated using Fisher's least significant difference (LSD) test for variables where the treatment effect was significant in the analysis of variance.

Orthogonal polynomials were also calculated where residue levels significantly affected disease severity or yield to determine linear, quadratic and cubic changes in these responses as residue level increased. Given that residue levels in the 2013 and 2014 trials were not equally spaced, the ORPOL function in PROC IML was used to obtain the appropriate coefficients for the CONTRAST statement in the GLIMMIX procedure. Following preliminary analyses to determine the relationship between RAUDPC and residue level, data were fitted to a two-parameter logarithmic model of the form, $y = a + b \cdot \ln(x + 1)$, where y = RAUDPC and x = residue level. In this model, the parameter a is the intercept and b is the rate parameter. The model was fitted to the disease data by linear regression using PROC REG in SAS. Goodness-of-fit of the model was assessed based on correlation of observed with predicted y values and statistical significance of the overall model.

Weather variables. In this section, the conduciveness of temperature, relative humidity, and rainfall (Bergstrom 2010; Eyal et al. 1987) is compared among years for the epidemic period, defined here as the interval from 2 weeks prior to disease onset until the final disease assessment. Temperature, relative humidity and rainfall were more favorable for disease development in 2012 than in 2013 or 2014. During the epidemic at Raleigh, mean daily temperatures ranged from 4 to 26°C, 5 to 24°C, and 1 to 26°C, in 2012, 2013, and 2014, respectively (data not shown). The number of days with average daily temperature between 20 and 25°C, the optimum range for disease development, was also higher in 2012 than in 2013 at Raleigh. A similar trend in days with conducive temperatures was also observed at Salisbury, where the number of days with optimum temperature for disease development was higher in 2012 than in 2013. In 2014, the number of days with optimum temperature was higher at Raleigh compared with Salisbury and Tyner (Table 1).

The trend in relative humidity was similar to that for temperature between sites and epidemic years. Relative humidity ranged from 37 to 98% during the epidemic in 2012 at Raleigh, while the corresponding range in 2013 and 2014 was 30 to 95% and 37 to 90%, respectively, at the same site (data not shown). Generally, relative humidity increased during the growing season across all sites and epidemic years. However, the number of days with favorable relative humidity varied across sites and epidemic years. For example, there were more days with $RH \geq 75\%$ in 2012 than in 2013 and 2014. The number of days with precipitation events and the cumulative precipitation were higher in 2012 than in 2013 or 2014 (Table 1). For example, there were more days with measurable precipitation in 2012 than in 2013 or 2014. The cumulative precipitation at Raleigh was 292, 135, and 372 mm in 2012, 2013, and 2014, respectively, and the corresponding rainfall amounts at Salisbury were 338, 290, and 260 mm, respectively.

Disease development. Severity of SNB increased over the experimental period during the epidemics in 2012, 2013, and 2014 at all sites (Fig. 1). Disease onset occurred much earlier in 2012 than in either 2013 or 2014 across all sites. For example, disease onset on DG Shirley occurred 20 to 30 days earlier at Raleigh and Salisbury in 2012 than in 2013 (Fig. 1). In 2014, disease onset on DG 9012 occurred 15 days earlier at Raleigh and Tyner than at Salisbury. Mean maximum disease severity on DG Shirley was approximately 15 to 30% higher in 2012 than in 2013 or 2014 at both Raleigh and Salisbury (Fig. 1). Disease severity on DG 9012 was higher at Raleigh than at Salisbury or Tyner in 2014 (Fig. 1). As expected from individual disease severity values, RAUDPC values were also considerably higher in 2012 than in 2013 or 2014.

The effects of residue level on maximum disease severity and RAUDPC were highly significant ($P < 0.001$) across all sites and epidemic years for DG Shirley (Table 2). In 2012, disease severity

TABLE 1. Number of days of weather conducive to the development of *Stagonospora nodorum* blotch during the epidemic period^v in North Carolina

Site	Weather variable								
	Temperature ^w			Relative humidity ^x			Rainfall ^y		
	2012	2013	2014	2012	2013	2014	2012	2013	2014
Raleigh	16	14	21	25	19	10	26	17	19
Salisbury	14	10	15	21	16	8	27	23	14
Tyner ^z	—	—	10	—	—	18	—	—	15

^v The epidemic period was defined as the interval starting 2 weeks prior to disease onset until final disease assessment.

^w The number of days when average daily temperature ranged between 20 and 25°C.

^x The number of days with relative humidity $\geq 75\%$.

^y The number of days with measurable rainfall.

^z Experiments were conducted at Tyner only in 2014.

was significantly ($P < 0.05$) lower in plots with no residue than in plots with residue at both Raleigh and Salisbury (Table 3). In addition, disease severity in plots with 60 and 90% residue levels was significantly higher than that in plots with 30% residue (Table 3). The effects of residue level on disease severity were slightly less variable at Salisbury than Raleigh as evidenced by lower standard errors at the Salisbury site (Table 3). Significant ($P < 0.05$) differences in disease severity in plots with no residue and those with $>20\%$ residue were also observed in 2013. However,

disease severity in plots with 10 and 20% residue level was not significantly different from that in plots with no residue except at Salisbury where SNB in plots with 20% residue was significantly different from no residue (Table 3). Similar effects of residue levels on disease severity were also observed in 2014 but residue effects on SNB were less variable in 2014 than in 2012 (Table 3). The effects of residue level on disease severity for DG 9012 in 2014 (Table 4) were similar to those observed on DG Shirley except that mean disease severity was higher on DG 9012 (Table 5) than on DG Shirley.

Fig. 1. Effect of different levels of wheat residue on the soil surface on the development of *Stagonospora nodorum* blotch in 3 years at three experimental sites in North Carolina. The wheat cultivar DG Shirley was evaluated in all site-years (A to G), while the cultivar DG 9012 was evaluated only in 2014 at all sites (H to J). Data points represent means of disease severity (%) values from five replicates; the bars are standard errors of the mean.

Trend analysis using orthogonal polynomials showed that disease severity on DG Shirley increased linearly with residue level as indicated by a highly significant ($P < 0.005$) linear orthogonal polynomial (Table 6). In addition, the increase in disease severity with residue level also displayed a significant ($P < 0.05$) quadratic trend with the exception of Raleigh in 2013 (Table 6). Similar results were observed for DG 9012 in 2014 (data not shown). The quadratic contrast is a test of whether a quadratic term can be included given that a linear term is already in the model. It serves as a hierarchical test of a quadratic model (with both linear and quadratic terms) versus a linear model. Thus, the significant results of the general ANOVA hypothesis were primarily explained by both a linear and quadratic relationship between residue level and disease severity.

Relationship between disease intensity and residue level.

The effects of residue levels on disease intensity were well described ($P < 0.05$) by the two-parameter logarithmic model (Fig. 2). The parameters a and b were significantly ($P < 0.05$) different from zero for data collected in all sites and epidemic years (Table 7). The correlations between observed versus predicted disease intensity values were high and significant ($P < 0.05$) and ranged from $r = 0.76$ to 0.98 (Table 7). In addition, plots of residuals versus observed or predicted values did not reveal any specific pattern.

Yield, yield components, and infected kernels. Effects of residue levels on grain yield varied among experimental sites and seasons of this study (Tables 2 and 4). For example, the effects of residue level on grain yield were significant ($P < 0.05$) at Raleigh and Salisbury in 2012 on DG Shirley (Table 2) and at Salisbury in 2014 on DG 9012 (Table 4). Marginal effects of residue on yield were observed at Raleigh in 2013 ($P = 0.0782$) and at Tyner in 2014 ($P = 0.0857$), while no significant ($P > 0.4622$) effects were observed at the other site-years (Tables 2 and 4). Where the effects of residue level were significant, yield in plots with no residue was significantly higher than yield in plots that had residue. In the latter case, yield generally decreased with increasing residue level, although mean yields were not always significantly different (Tables 3 and 5). Residue level only had a significant effect on test weight at Raleigh in 2012 (Tables 2 and 4). Similarly, the effects of residue on thousand-kernel weight were not significant except at

Raleigh and Salisbury in 2014 on DG 9012 where thousand-kernel weight generally decreased with increasing residue level (Tables 4 and 5).

Residue treatments did not significantly ($P > 0.05$) affect incidence of kernel infection for DG Shirley (Table 2). However, significant ($P < 0.05$) differences in kernel infection were observed on DG 9012 in 2014 at Raleigh and Salisbury but not at Tyner (Table 4). At Raleigh, kernel infection for DG 9012 in 2014 ranged from 40 to 75%, and residue levels $\geq 30\%$ resulted in kernel infection levels that were significantly higher than those in residue-free control plots. At Salisbury, the 90% residue level resulted in DG 9012 kernel infection percentages significantly higher than in the untreated plots or lower residue levels. Percent infected kernels at Salisbury was significantly lower than that at Raleigh, ranging from 15 to 45% (Table 5).

DISCUSSION

To the best of our knowledge, this is the first study to quantify the effects of the amount of residue on soil surface on the severity of SNB and yield in winter wheat. Our results showed that disease severity was significantly affected by the amount of residue on the soil surface at all experimental sites and growing seasons. In addition, we observed a nonlinear relationship between the amount of residue on the soil surface and disease severity, where fairly small increases in residue density produced a rapid increase in disease severity. The amount of residue on the soil surface also affected grain yield, but the effects were only significant when disease onset occurred early and weather conditions during the season were conducive for rapid disease development. The latter has an important implication since in the Mid-Atlantic region of the United States, SNB is one of the foliar diseases most frequently targeted for fungicide applications to wheat.

Infected wheat residue on the soil surface harbors *P. nodorum* between crops and can serve as a source of primary inoculum for initiation of new disease epidemics at the start of the growing season (Duczek et al. 1999; Krupinsky et al. 2007). Several studies have linked infected residue on the soil surface to increased severity of SNB in the field (Holmes and Colhoun 1975; Luke et al. 1983; Milus and Chalkley 1997). However, in all these reports, the amount

TABLE 2. Effect of wheat residue levels on the intensity of *Stagonospora nodorum* blotch and yield characteristics for winter wheat cultivar DG Shirley^y

Site/variable ^z	2012				2013				2014			
	ndf	ddf	<i>F</i>	<i>P</i> > <i>F</i>	ndf	ddf	<i>F</i>	<i>P</i> > <i>F</i>	ndf	ddf	<i>F</i>	<i>P</i> > <i>F</i>
Raleigh												
MaxDS	3	12	80.55	0.0001	5	20	8.09	0.0003	5	20	31.37	0.0001
RAUDPC	3	12	181.07	0.0001	5	20	18.62	0.0001	5	20	108.77	0.0001
PKC	3	16	0.43	0.7358	5	20	1.53	0.2259	5	20	1.84	0.1507
Yield	3	16	6.81	0.0036	5	20	2.35	0.0782	5	20	0.83	0.5454
TW	3	12	9.50	0.0017	5	20	1.01	0.4374	5	20	0.54	0.7420
TKW	—	—	—	—	5	24	1.01	0.4337	5	24	1.23	0.3268
Salisbury												
MaxDS	3	12	245.51	0.0001	5	20	8.82	0.0002	5	20	25.26	0.0001
RAUDPC	3	12	392.15	0.0001	5	24	16.82	0.0001	5	20	55.77	0.0001
PKC	3	12	0.30	0.8229	5	24	1.00	0.4406	5	24	0.71	0.6225
Yield	3	9	9.17	0.0041	5	19	0.97	0.4622	5	24	0.77	0.5828
TW	3	12	2.53	0.1065	5	19	0.64	0.6748	5	24	0.56	0.7323
TKW	—	—	—	—	5	20	0.80	0.5623	5	20	1.26	0.3177
Tyner												
MaxDS	—	—	—	—	—	—	—	—	5	20	6.54	0.0009
RAUDPC	—	—	—	—	—	—	—	—	5	20	21.22	0.0001
PKC	—	—	—	—	—	—	—	—	5	20	2.62	0.0557
Yield	—	—	—	—	—	—	—	—	5	20	2.28	0.0857
TW	—	—	—	—	—	—	—	—	5	20	0.99	0.4488
TKW	—	—	—	—	—	—	—	—	5	24	1.03	0.4224

^y Four residue levels were evaluated in 2012, while six residue levels were evaluated in the 2013 and 2014 growing seasons. Thousand-kernel weight was not recorded in the 2012 experiments and the experiments were conducted at Tyner only in 2014.

^z MaxDS is maximum disease severity (%), RAUDPC is relative area under the disease progress curve, and PKC is percent kernels colonized by *Parastagonospora nodorum*. Yield is yield (t ha⁻¹), TW is test weight (kg m⁻³), while TKW is thousand-kernel weight (g).

of residue on the soil in field experiments was not specified. In the present study, disease severity increased with increasing amounts of residue on the soil surface and then leveled off above 30% residue. An increase of residue level up to 30% resulted in greater increase in disease severity than when residue coverage levels were increased to 60 or 90%. It is possible that the amount of inoculum on the surface that is available for splash-dispersal increases as residue levels increase until 30% residue coverage. However, at higher residue levels, a higher proportion of total inoculum is trapped in the lower layers of residue and therefore unavailable for dispersal to

cause infection. In some cases, a residue level of 10% resulted in disease severity that was not different from that in plots with 30% residue level. In other words, the 10% residue level provided adequate inoculum for an epidemic equivalent to that initiated from 30% residue. This suggests that relatively small amounts of residue on the soil surface can provide sufficient inoculum for the development of SNB epidemics. Similar observations have also been reported for gray leaf spot in corn (de Nazareno et al. 1993). The 10% residue level on the soil surface is equivalent to conditions of conventional tillage with incomplete burial of residue (Stubbs

TABLE 3. Impact of levels of wheat residue on the development and severity of *Stagonospora nodorum* blotch and yield characteristics for winter wheat cultivar DG Shirley^x

Variable ^y	Residue level (%) ^z	2012		2013		2014		
		Raleigh	Salisbury	Raleigh	Salisbury	Raleigh	Salisbury	Tyner
MaxDS	0	18.8 c	20.4 c	25.4 d	19.2 e	19.8 d	8.0 d	19.4 d
	10	—	—	26.4 d	20.6 de	27.0 c	20.8 c	20.4 cd
	20	—	—	29.4 cd	24.2 cd	29.2 bc	24.2 bc	24.4 ab
	30	36.6 b	29.8 b	36.8 ab	25.2 bc	30.0 b	23.4 bc	22.6 bc
	60	44.2 a	34.8 a	33.6 bc	28.6 ab	33.4 a	26.4 ab	24.4 ab
	90	47.2 a	36.0 a	40.0 a	29.6 a	34.2 a	28.8 a	26.4 a
	Mean	36.7	30.3	31.9	24.6	28.9	21.9	22.9
	SE	2.71	1.43	1.31	0.90	0.96	1.37	0.69
RAUDPC	0	8.8 c	9.4 d	8.0 d	7.8 d	4.8 d	3.5 d	6.7 d
	10	—	—	8.6 cd	9.7 c	9.5 c	9.3 c	9.6 c
	20	—	—	10.2 c	10.9 c	11.7 b	12.8 b	10.9 bc
	30	23.6 b	17.2 c	13.8 ab	11.3 bc	12.3 b	12.2 b	10.2 bc
	60	25.4 b	21.1 b	13.0 b	13.1 b	14.3 a	14.9 a	11.4 b
	90	27.6 a	23.3 a	15.9 a	15.3 a	14.8 a	16.1 a	13.2 a
	Mean	21.3	17.7	11.6	11.4	11.2	11.5	10.3
	SE	1.73	1.22	0.60	0.50	0.65	0.81	0.41
PKC	0	25.0	17.0	16.0 b	7.00	43.0 b	29.0	32.0 b
	10	—	—	17.0 b	5.00	45.0 b	35.0	24.0 b
	20	—	—	22.0 ab	7.00	55.0 ab	34.0	35.0 ab
	30	31.0	20.0	27.0 a	10.0	53.0 ab	26.0	30.0 b
	60	28.0	17.0	21.0 ab	11.0	53.0 ab	40.0	32.0 b
	90	24.0	21.0	23.0 ab	14.0	63.0 a	32.0	44.0 a
	Mean	27.0	18.8	21.0	9.00	52.0	32.7	32.8
	SE	2.31	1.81	1.50	1.34	2.39	2.31	1.88
Yield	0	8.7 a	8.0 a	11.1 a	9.4	8.6	8.5	6.9 a
	10	—	—	10.4 ab	9.7	8.6	8.1	6.5 ab
	20	—	—	10.1 b	9.5	8.1	8.9	6.1 b
	30	8.1 ab	7.6 b	10.4 ab	9.9	8.0	8.7	6.8 a
	60	7.3 c	7.4 b	9.8 b	9.7	8.0	8.6	6.6 ab
	90	7.8 bc	7.4 b	10.4 ab	9.4	8.4	8.3	6.2 b
	Mean	7.9	7.6	10.3	9.6	8.3	8.5	6.5
	SE	0.15	0.12	0.13	0.11	0.19	0.13	0.11
TW (×10)	0	70.2 a	73.1 a	69.7	68.4	74.9	72.0	72.9
	10	—	—	68.5	69.0	75.2	72.2	73.8
	20	—	—	70.0	68.6	75.2	72.0	73.7
	30	68.5 b	72.9 ab	68.8	69.2	75.4	72.3	74.2
	60	68.0 b	72.7 ab	69.2	68.3	75.2	72.6	74.0
	90	68.2 b	72.6 b	69.9	68.5	75.1	72.3	72.9
	Mean	68.7	72.8	69.4	68.7	75.2	72.2	73.6
	SE	0.26	0.08	0.30	0.17	0.10	0.12	0.25
TKW	0	—	—	37.0	40.9	38.0 ab	38.0 a	37.7
	10	—	—	35.2	42.1	39.4 a	37.9 ab	36.7
	20	—	—	35.8	39.9	38.5 ab	37.7 ab	37.1
	30	—	—	34.2	41.7	37.8 b	37.9 ab	37.5
	60	—	—	35.8	41.8	38.5 ab	38.0 ab	36.9
	90	—	—	36.5	41.2	37.9 ab	37.1 b	36.1
	Mean	—	—	35.8	41.3	38.3	37.7	37.0
	SE	—	—	0.41	0.37	0.23	0.14	0.23

^x Residue cover levels of 10 and 20% were not tested in 2012; thousand-kernel weight was not recorded in 2012.

^y MaxDS is maximum disease severity (%), RAUDPC is relative area under the disease progress curve, and PKC is percent kernels colonized by *Parastagonospora nodorum*. Yield is yield (t ha⁻¹), TW is test weight (kg m⁻³), while TKW is thousand-kernel weight (g). Values presented are means and for each variable within a site and year, values followed by the same letter are not significantly different based on Fisher's least significant difference at $\alpha = 0.05$. Mean separation is shown only for variables with significant treatment effects in analysis of variance.

^z Percentage of residue cover on the soil surface and SE = standard error of the mean.

et al. 2004). We predict that SNB will progress at a fast rate and reach a high maximum severity in such a field and thus, fields would need to be plowed in a manner that ensures complete burial of infected residue if conventional tillage is expected to result in a decrease in SNB inoculum.

The disease intensity (RAUDPC) at all experimental sites was higher in 2012 than in 2013 or 2014 for DG Shirley, irrespective of residue treatment. Similarly, disease onset was earlier in 2012 than in 2013 and 2014 growing seasons, regardless of the residue treatments. Given that SNB is a polycyclic disease, earlier onset was likely responsible for the higher disease intensities in 2012. Weather factors probably account for differences in timing of disease onset and severities of SNB epidemics observed in this study. More days with optimal temperatures, relative humidity, and rain were observed in 2012 than in 2013 or 2014. Although grain yield in plots with no residue was generally higher than yield in plots with residue, significant effects of residue level on yield were only observed during the 2012 season. The levels of disease severity in 2013 and 2014, which were limited by less favorable weather, were not high enough to significantly reduce grain yield especially for DG Shirley. These findings are similar to those of Milus and Chalkley (1997) who also observed significant differences in yield between plots with residue and plots with no residue only during severe epidemics of SNB. Similarly, Eyal (1981) reported significant reductions in grain yield only during severe epidemics of SNB in Israel.

In our study, residue-associated impacts on yield occurred in environments where disease levels in residue-treated plots were elevated early in the epidemic (Fig. 1A, B, and I). Since residue was applied in late January or early February, conducive weather in February was likely an important determinant of whether yield would be affected by SNB. It is reasonable that if disease severity is elevated early in the growing season, the risk of disease-related yield impacts may be greater than if the same disease level is not reached until later in the season, due to polycyclic disease development. Infected residue used in the present study was stored under shelter prior to field application and thus did not undergo the weathering to which residue left in the field is exposed after harvest. Thus, the residue used in this study may have had a higher inoculum efficiency than residue left in the field. However, the increase in SNB with residue application is qualitatively similar when either

artificially inoculated residue is introduced in the field or naturally weathered residue left in the field is evaluated as an inoculum source (Holmes and Colhoun 1975; Milus and Chalkley 1997).

Our results support the conclusion that yield and test weight, the variables of interest to wheat growers, are only negatively impacted by the highest levels of disease intensity, despite the disease-elevating effects of unincorporated wheat residue. Although further work on the economic threshold for SNB in the Mid-Atlantic states is needed, our results suggest that the threshold may be higher than is often presumed. In this study, we selected cultivars with moderate or high susceptibility in order to maximize the chances of detecting

TABLE 5. Impact of amounts of wheat residue on the development and severity of *Stagonospora nodorum* blotch and yield characteristics for winter wheat cultivar DG 9012 in 2014

Variable ^y	Residue level (%) ^z	Site		
		Raleigh	Salisbury	Tyner
MaxDS	0	29.6 d	15.0 d	23.4 c
	10	34.0 cd	27.2 c	24.0 bc
	20	39.0 bc	28.0 bc	26.0 abc
	30	42.4 b	31.0 b	26.8 ab
	60	49.2 a	34.8 a	26.8 ab
	90	49.8 a	36.8 a	27 a
	Mean (SE)	40.7 (1.61)	28.8 (1.38)	25.7 (0.45)
RAUDPC	0	7.5 f	6.4 d	11.1 d
	10	13.3 e	14.3 c	13.2 c
	20	16.6 d	16.1 c	15.3 b
	30	18.5 c	18.3 b	15.4 b
	60	20.4 b	21.8 a	16.5 ab
	90	22.4 a	23.1 a	16.9 a
	Mean (SE)	16.5 (0.85)	16.7 (1.07)	14.7 (0.43)
PKC	0	41.0 b	16.0 b	32.0
	10	62.0 ab	28.0 b	47.0
	20	59.0 ab	26.0 b	45.0
	30	75.0 a	24.0 b	41.0
	60	74.0 a	28.8 ab	35.0
	90	75.0 a	45.0 a	38.0
	Mean (SE)	64.3 (3.56)	27.9 (2.58)	39.7 (2.46)
Yield	0	8.1	8.5 ab	5.6
	10	8.1	8.0 bc	5.6
	20	7.9	7.9 c	5.8
	30	8.2	8.6 a	5.6
	60	7.8	7.9 c	5.8
	90	8.0	8.0 bc	5.6
	Mean (SE)	8.0 (0.11)	8.2 (0.14)	5.7 (0.12)
TW (×10)	0	77.2	74.3	76.2
	10	77.2	74.1	75.9
	20	77.2	74.0	76.2
	30	77.0	74.2	74.3
	60	76.9	73.9	76.2
	90	77.1	74.5	75.2
	Mean (SE)	77.1 (0.10)	74.2 (0.17)	75.6 (0.34)
TKW	0	38.0 a	38.2 a	35.7
	10	37.0 ab	38.2 a	34.7
	20	36.7 abc	38.1 ab	35.6
	30	35.9 bc	37.5 abc	34.9
	60	35.4 c	37.0 bc	34.9
	90	36.5 abc	36.8 c	35.4
	Mean (SE)	36.6 (0.27)	37.6 (0.23)	35.2 (0.16)

^y MaxDS is maximum disease severity (%), RAUDPC is relative area under the disease progress curve, and PKC is percent kernels colonized by *Parastagonospora nodorum*. Yield is yield (t ha⁻¹), TW is test weight (kg m⁻³), while TKW is thousand-kernel weight (g). Values presented are means and for each variable within a site and year, values followed by the same letter are not significantly different based on Fisher's least significant difference at $\alpha = 0.05$. Mean separation is shown only for variables with significant treatment effects in the analysis of variance.

^z Percentage of residue cover on the soil surface and SE = standard error of the mean.

TABLE 4. Effect of wheat residue levels on intensity of *Stagonospora nodorum* blotch and yield characteristics for winter wheat cultivar DG 9012 in 2014

Site	Variable ^z	ndf	ddf	F	P > F
Raleigh	MaxDS	5	20	16.60	0.0001
	RAUDPC	5	20	115.26	0.0001
	PKC	5	20	3.52	0.0190
	Yield	5	20	0.94	0.4751
	TW	5	20	0.42	0.8291
	TKW	5	20	2.86	0.0418
Salisbury	MaxDS	5	20	52.86	0.0001
	RAUDPC	5	20	82.54	0.0001
	PKC	5	23	3.30	0.0216
	Yield	5	20	2.93	0.0382
	TW	5	20	0.24	0.9378
	TKW	5	20	2.88	0.0407
Tyner	MaxDS	5	24	2.54	0.0555
	RAUDPC	5	20	24.74	0.0001
	PKC	5	20	0.99	0.4511
	Yield	5	20	0.31	0.9020
	TW	5	24	0.83	0.5422
	TKW	5	20	1.39	0.2694

^z MaxDS is maximum disease severity (%), RAUDPC is relative area under the disease progress curve, and PKC is percent kernels colonized by *Parastagonospora nodorum*. Yield is yield (t ha⁻¹), TW is test weight (kg m⁻³), while TKW is thousand-kernel weight (g).

TABLE 6. Orthogonal polynomial contrasts to test the hypothesis of linear, quadratic and cubic response in *Stagonospora nodorum* blotch severity and yield with increasing levels of wheat residue cover in plots of cultivar DG Shirley^y

Variable ^z	Site	Test	2012			2013			2014		
			ndf,ddf	F	P > F	ndf,ddf	F	P > F	ndf,ddf	F	P > F
Disease severity	Raleigh	General	3,12	80.55	0.0001	5,20	8.09	0.0003	5,20	31.37	0.0001
		Linear	1,12	213.69	0.0001	1,20	30.80	0.0001	1,20	115.82	0.0001
		Quadratic	1,12	27.18	0.0002	1,20	1.40	0.2501	1,20	29.67	0.0001
		Cubic	1,12	0.78	0.3950	1,20	2.18	0.1558	1,20	6.59	0.0184
	Salisbury	General	3,12	245.51	0.0001	5,20	8.82	0.0002	5,20	25.26	0.0001
		Linear	1,12	654.45	0.0001	1,20	39.38	0.0001	1,20	75.66	0.0001
		Quadratic	1,12	82.00	0.0001	1,20	4.11	0.0561	1,20	25.65	0.0001
		Cubic	1,12	0.09	0.7721	1,20	0.01	0.9131	1,20	16.92	0.0005
	RAUDPC	General	3,12	181.07	0.0001	5,20	18.62	0.0001	5,20	108.77	0.0001
		Linear	1,12	422.86	0.0001	1,20	76.38	0.0001	1,20	389.58	0.0001
		Quadratic	1,12	97.97	0.0001	1,20	3.85	0.0639	1,20	119.35	0.0001
		Cubic	1,12	22.39	0.0005	1,20	3.07	0.0953	1,20	26.62	0.0001
Yield	Raleigh	General	3,16	6.81	0.0036	—	—	—	—	—	—
		Linear	1,16	12.63	0.0026	—	—	—	—	—	—
		Quadratic	1,16	5.98	0.0264	—	—	—	—	—	—
		Cubic	1,16	1.81	0.1973	—	—	—	—	—	—
	Salisbury	General	3,9	3.30	0.0041	—	—	—	—	—	—
		Linear	1,9	5.95	0.0007	—	—	—	—	—	—
		Quadratic	1,9	3.21	0.0651	—	—	—	—	—	—
		Cubic	1,9	0.75	0.9963	—	—	—	—	—	—

^y Residue levels significantly affected yield in 2012 but not in 2013 and 2014; thus, orthogonal contrasts were conducted only for yield data in the 2012 growing season.
^z Disease severity refers to maximum disease severity (%) recorded in the respective treatment plots; RAUDPC is relative area under the disease progress curve; yield is measured as t ha⁻¹.

Fig. 2. Relationship between *Stagonospora nodorum* blotch intensity (relative area under the disease progress curve) and levels of wheat residue on soil surface in three growing seasons at three experimental sites in North Carolina. The wheat cultivar DG Shirley was evaluated in all years (A to C), while the cultivar DG 9012 was evaluated only in 2014 at all sites (D). Symbols (circles and triangles) represent the observed disease data, while the curves represent the predicted disease intensity based on a two-parameter logarithmic model. In each year, the coefficient of correlation (r) between observed and predicted values is a range depicting the goodness-of-fit of the model to the data across experimental sites.

significant effects of residue level on disease symptoms, yield, and test weight. However, many high-yielding wheat cultivars are moderately resistant to *P. nodorum*, and use of these cultivars is more likely to prevent yield and test weight reductions attributable to SNB.

It is noteworthy that at Raleigh and Salisbury where precipitation was higher than at Tyner in 2014, the susceptible cultivar DG 9012 had a higher incidence of infected grain associated with higher residue levels. While this was not accompanied by a negative test weight impact, it could lead to higher levels of disease in subsequent wheat crops via seed transmission if the harvested grain were saved for planting. The thousand-kernel weight of DG 9012 was negatively affected by residue level at those same two locations, indicating the impact of residue on both seed infection and seed size. This trend was, however, not observed for the moderately susceptible DG Shirley. In their work on the control of SNB using cultural practices, Luke et al. (1983) reported a significant negative correlation between SNB severity and thousand-kernel weight or seed infection when disease severity was low ($\leq 20\%$). In the present study, we observed no effects of SNB on thousand-kernel weight and seed infection under our epidemic conditions except in 2014 on the susceptible cultivar DG 9012, for which the mean disease severity was $>20\%$. Increased cultivar susceptibility to SNB was found to be positively correlated with increased seed infection by *P. nodorum* (Cunfer 1978). However, we are unable to directly compare our results with those of Luke et al. (1983) given that we assessed disease severity on a whole-canopy basis, while Luke et al. (1983) assessed disease severity only on glumes and flag, F-1 and F-2 leaves.

SNB developed in residue-free plots, although the resultant levels of disease severity were much lower than those in plots with residue. Development of SNB in clean plots with no wheat residue was also reported in a previous study (Milus and Chalkley 1997). Dispersal gradients of *P. nodorum* pycnidiospores from a source are usually shallow with a horizontal distance of up to 1 m (Brennan et al. 1985b) in the absence of free vegetation between plots (Holmes and Colhoun 1975), although the spores can also be dispersed to a horizontal distance of 10 m in splash microdroplets under windy conditions (Brennan et al. 1985a). The experimental plots in our study were surrounded with buffer plots and it is unlikely that disease in residue-free plots was due to pycnidiospores from adjacent plots. Given that pathogen-free seed was used for planting, it is possible that windborne ascospores played a role in initial infection in the residue-free plots. Ascocarps of *P. nodorum* have been reported in North Carolina albeit at low frequency (Cowger and Silva-Rojas 2006). Thus, both pycnidiospores and ascospores

may play a role in initiating SNB epidemics in North Carolina, although ascospores of *P. nodorum* have not been found in other parts of the eastern United States (Cunfer 1978; Shah et al. 1995). In other pathosystems, earlier onset of disease epidemics has been reported to occur in plots with residue than in residue-free plots (Payne et al. 1987), an observation that is attributed to the higher inoculum density in plots with residue. In the present study, however, timing of SNB epidemic onset was similar in both residue-free plots and plots with residue. This is probably because our experiments were conducted in residue-free sites and we introduced residue in plots only after plants were well-tillered to avoid smothering of plants. Our results are similar to those reported for gray leaf spot in corn, where disease epidemics started about the same time regardless of residue level in the experimental plots (de Nazareno et al. 1993). Given that residue remains relatively undisturbed in no-tillage or minimum-tillage fields, it is expected that onset of SNB epidemics will occur much earlier in no-tillage fields than observed in the present study.

Important conclusions that are relevant to the control and management of SNB can be drawn from the present study. First, the increase in disease severity with increasing residue level was nonlinear; residue levels of 10 to 30% produced a rapid increase in disease severity compared with high residue levels under favorable environmental conditions. This finding is relevant where residue is used as an input variable in assessing the likelihood and severity of SNB during the season. Residue levels and other cultural practices can be useful predictors in determining the preplanting risk for SNB that can be used to guide growers in making disease management decisions prior to planting (Paul and Munkvold 2004). Second, our results strengthen the previous recommendations to rotate wheat with a nonhost (Krupinsky et al. 2007; Luke et al. 1983; Milus and Chalkley 1997) to reduce the amount of initial inoculum in subsequent seasons when wheat is planted. The level of resistance to SNB will also influence the effects of residue on disease severity, with impacts being greater on more susceptible than moderately resistant cultivars. Where a susceptible cultivar is planted in the presence of infected residue, fungicides may be necessary to prevent negative impacts on yield when environmental conditions during the season are favorable for disease development. But the economic threshold for fungicides to manage SNB may be higher than previously believed, as yield and test weight losses appear to be associated with SNB only in more severe epidemics. The acreage under reduced tillage is increasing in wheat-growing areas of the United States (Horowitz et al. 2010), an outcome of which will be increased inoculum availability and the risk of more severe epidemics of SNB, particularly where wheat follows wheat. Thus, cultural practices aimed at reducing infected residue will remain important in the overall SNB management strategy.

ACKNOWLEDGMENTS

This study was supported by Hatch Funds from North Carolina Agricultural Experiment Station for Project Number NC02251, and by the USDA-ARS. We thank staff at Piedmont Research Station at Salisbury and Lake Wheeler Road Field Laboratory at Raleigh, North Carolina, for managing the research plots; and R. Parks, B. Van Staaldin, T. Cole, B. Clontz, I. Swaim, C. Glover, B. Stevens, J. Jeffreys, and J. Patton-Özkurt for their assistance in planting and harvesting.

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TABLE 7. Nonlinear regression analysis of the relationship between amounts of wheat residue cover on the ground and the development of *Stagonospora nodorum* blotch, using a two-parameter logarithmic model^x

Year	Location	Cultivar	Parameter (SE) ^y		Goodness-of-fit	
			<i>a</i>	<i>b</i>	<i>r</i> ²	<i>P</i> > <i>F</i>
2012	Raleigh	DG Shirley	8.83 (0.723)	4.15 (0.206)	0.978	0.0001
	Salisbury	DG Shirley	9.04 (0.615)	2.89 (0.176)	0.968	0.0001
2013	Raleigh	DG Shirley	6.75 (0.889)	1.66 (0.272)	0.756	0.0001
	Salisbury	DG Shirley	6.97 (0.653)	1.51 (0.200)	0.819	0.0001
2014	Raleigh	DG Shirley	4.57 (0.413)	2.29 (0.126)	0.960	0.0001
	Salisbury	DG Shirley	3.32 (0.635)	2.79 (0.194)	0.938	0.0001
	Tyner	DG Shirley	6.53 (0.455)	1.30 (0.139)	0.870	0.0001
2014	Raleigh	DG 9012	6.81 (0.675)	3.30 (0.207)	0.949	0.0001
	Salisbury	DG 9012	5.85 (0.770)	3.71 (0.236)	0.948	0.0001
	Tyner	DG 9012	10.8 (0.500)	1.33 (0.153)	0.855	0.0001

^x Two-parameter model fitted to the data were of the form: $y = a + b \cdot \ln(x + 1)$, where y = relative area under the disease progress curve (RAUDPC), x = amount of residue, a = intercept, and b = rate parameter.

^y Values in parentheses are standard errors (SE) of the parameter estimates.

^z Correlation coefficient between observed and predicted disease intensity (RAUDPC) values.

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