Epidemiology of Leaf Blotch of Soft Red Winter Wheat Caused by *Septoria tritici* and *Stagonospora nodorum*

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ABSTRACT

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Septoria tritici and *Stagonospora nodorum* frequently cause leaf blotch of wheat in the soft red winter wheat region east of the Mississippi River. Since 1955, leaf blotch has been the most destructive disease of wheat in Indiana. *Septoria tritici* was initially the principal pathogen responsible for this disease. Since 1986, *S. nodorum* has also been a devastating pathogen, causing both leaf and glume blotch. In order to understand the dynamics of this disease, we have monitored disease development on wheat cultivar Monon in replicated wheat performance trials each year since 1973. Monon has served as a long-term check cultivar in these trials, and it is very susceptible to both *Septoria tritici* and *S. nodorum*. The percent leaf area showing symptoms of Septoria leaf blotch was used to quantify disease severity, using a scale that reflected the vertical gradient of symptoms. Plots were usually assessed weekly for disease from the time of flag leaf emergence until crop maturity. During the 19 years of disease observation reported here, we rarely saw symptoms of leaf blotch in the fall. Initial symptoms usually appeared early in the spring on leaves touching or near the soil surface, suggesting that primary infection takes place during autumn, before the onset of cold weather, and that *Septoria tritici* and *S. nodorum* overwinter as asymptomatic infections. In most years, disease incidence was 100% by the time of flag leaf emergence; there was considerable variation in severity, but symptoms usually progressed at least to the third leaf below the flag leaf. The time from spike emergence to harvest (average = 49 days) was the main period of disease increase on the upper four leaves of the culm. For many of the years, the increase in severity with time, as recorded on the assessment scale, was approximately linear. In every year but 1982, the model based on plotting severity scale values vs. days relative to spike emergence yielded a reasonably linear relationship between severity and time.

Septoria tritici Roberge in Desmaz. and *Stagonospora* (= *Septoria*) *nodorum* (Berk.) Castellani & E.G. Germano are pathogens of wheat (*Triticum aestivum* L). Both cause a leaf blotch of wheat; *S. nodorum* also infects spikes of wheat, causing glume blotch. In this paper, we refer to the foliar disease caused by either or both of these pathogens as leaf blotch. Leaf blotch has long been a problem in Indiana and other states of the humid Corn Belt, and since 1955 it has been the most destructive disease of wheat in Indiana (21,25). *Septoria tritici* was historically the principal pathogen responsible for this disease. Until recently, glume blotch was rarely a problem, usually occurring only on wheat that had lodged severely. Since 1986, *S. nodorum* has also been a devastating pathogen, causing both leaf and glume blotch. In order to understand the dynamics of these diseases, we began to monitor their annual development in 1973 and have continued this every year since.

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The wheat produced in Indiana is exclusively soft red winter wheat. It is sown in the fall, overwinters in a vegetative state, and resumes growth in the spring. During the period 1973 to 1991, the area of wheat harvested in Indiana ranged from 243,000 to 607,000 ha. State average yields ranged from 2,353 to 3,966 kg/ha (e.g., 11). Most wheat is grown in rotation with soybeans or maize.

Wheat sowing in Indiana takes place from early September through November, but most of the crop is sown during October (11). Growers are advised to plant after the Hessian fly–free date, which ranges from 22 September in northern Indiana to 8 October in southern Indiana. Although this guideline was designed to allow the wheat crop to escape fall infestation by the Hessian fly (*Mayetiola destructor* (Say)), it also serves to reduce the risk of fall infection by several fungi and viruses (21).

Normally, weather is sufficiently warm after planting to allow wheat to tiller well before the onset of winter dormancy. Wheat resumes growth in March, the exact time depending on temperature. Spikes emerge during May, the time depending on latitude and temperature. Wheat is normally ready for harvest from mid-June through mid-July. Maturity is earliest in southern Indiana.

Wheat performance trials have been conducted for many years at several locations in Indiana by Purdue University staff (e.g., 6). Until 1991, the wheat cultivar Monon, released in 1960 (20), served in these trials as a long-term standard against which the performance of newer cultivars was measured. Monon is very susceptible to both *Septoria tritici* and *S. nodorum*. In this paper, we report the characteristics of leaf blotch development from 1973 to 1991 in the wheat performance trial conducted at the Purdue Agronomy Farm near West Lafayette, Indiana. This includes an analysis of disease progress and relation of disease to host developmental stage. We investigated the utility of critical point models, rate of disease progress, and area under the disease progress curve for comparing epidemics. Such comparisons are of value for evaluating the effect of weather on disease and for comparing cultivar resistance, fungicide treatments, and cultural control measures.

MATERIALS AND METHODS

Plant culture. Wheat was sown in early October in most years, but occasionally in late September (Table 1). Planting and harvest methods and cultural practices were representative of commercial wheat production in Indiana and are described each year in a bulletin that presents the results of the performance trials (6). Wheat entries, usually somewhat more than 40 cultivars and experimental lines, were sown in a randomized complete block design with four replicates. Individual plots were 10 m long and 1.9 m wide. In recent years, all of the fertilizer was applied in the fall before planting the wheat. Nitrogen was applied as anhydrous ammonia with nitropyrin, for a total nitrogen application rate of about 130 kg/ha. In earlier years, nitrogen application was split between a fall preplant application of about 22 kg/ha and a spring topdressing of about 90 kg/ha (5). The crop preceding the wheat trial each year was soybeans.

Plant growth stages were assessed according to a decimal scale (31). Throughout this paper, reference will be made to specific leaf positions on the wheat culm. These leaves are numbered relative to the uppermost leaf, the flag leaf. Thus, the leaf immediately below the flag leaf (F) is designated F-1, the second leaf below the flag leaf is F-2, and so on. A key phe-

nological stage for comparing relative maturity of wheat cultivars is the date of spike emergence, defined as the day on which spikes have just emerged from the leaf sheath (boot) on at least half of the culms. The date of spike emergence, recorded each year for Monon and all other cultivars in the test, serves as a phenological reference point for comparing development of leaf blotch among years. Day 0 is the day of spike emergence for Monon each year.

Disease assessment. The percent leaf area showing symptoms of leaf blotch was used to quantify disease severity. From the inception of this project it was evident that disease symptoms progressed up the plant with time. Thus, at any given time until the entire leaf area showed symptoms, there was a gradient of decreasing severity from lower to upper leaves on the stem. A leaf disease severity scale that would reflect this gradient of symptoms seemed appropriate for rating plants. Such a scale was published by Saari and Prescott (22) as a general scale for evaluating foliar diseases of wheat. We found that this scale did not work well for leaf rust or powdery mildew in Indiana, but it did work well for leaf blotch. Based upon detailed observations of percent severity on each leaf layer in the canopy, we refined and quantified the Saari and Prescott scale (24). With experience over the years, we found that this modified scale served well to describe severity on the upper four leaves of the plant and was sufficiently quick and accurate to permit assessment of hundreds of plots in a day. We made some changes to the original scale (24)—slight changes in severity intervals on leaf 4 and the addition of a scale value of 9.5 to accommodate severe symptoms on the flag leaf (Table 2). This assessment scale worked well to quantify the disease. Scale values indicate not only the total severity of disease, but also the vertical progression of symptoms. In this paper, severity on the 0 to 9.5 scale is denoted by S. Proportion severity is denoted by Y. The quadratic formula in the footnote of Table 2 provides a means to convert scale value of severity to percent disease (100Y). The mean percent severity in Table 2 is the average severity per leaf. It is not quite the same as percent severity on the total leaf area represented by leaves F through F-3 because the leaf blade at each of these positions on the stem has a different area. The relative blade areas of leaves F, F-1, F-2, and F-3 are not constant among cultivars, however, so it is not feasible to use relative leaf areas for converting scale values to percent symptomatic area of the upper four leaves collectively.

In the earlier years of this study, when we evaluated only a few cultivars in the trial, we assessed severity on 20 arbitrarily chosen culms per plot. In later years, when we began assessing all cultivars in the trial, time did not permit examination of individual culms. Also, by that time we felt confident enough about the uniformity of severity within a plot to use whole-plot assessments. To do this, two or three experienced observers, stationed at different places around the plot, would each examine an area of several square meters and call out the rating to the person recording the data. If observers differed in their ratings, more areas of the plot were examined to determine if one observer had happened to view an area of unusually mild or severe disease, or if there was indeed a gradient of disease in the plot. Based on this inspection, an average assessment was recorded for the plot. It was rare for observers to differ by more than one scale value when viewing a plot.

In 1990, assessment of individual culms was compared to whole-plot assessments. Ten arbitrary culms per plot of four cultivars were rated individually for leaf blotch before all plots in the cultivar trial were assessed by the whole-plot method. We used these data (four cultivars and four assessment times) to compare whole-plot estimates with mean plot values calculated from subsamples.

Although growth stage was recorded when disease severity was assessed, detailed records of the time of emergence of leaves from successive nodes of the culm were not routinely kept. However, such observations were made on Monon in 1990 and on cultivar Clark in a foliar fungicide trial in 1991. These data were used for a more detailed analysis of progression of symptoms up the culm than was possible from the data collected during most years.

Plots were usually assessed weekly for disease from flag leaf emergence until crop maturity. In some years, assessments began earlier. In years without much disease development, Monon and a few other susceptible cultivars would be checked regularly, but quantitative assessments would not begin until sometime after emergence

Table 1. Cardinal phenological dates and time periods for Monon wheat used for a leaf blotch epidemiology study, Purdue Agronomy Farm, West Lafayette, Indiana, 1973–1991

Crop year	Date			Planting to heading (days)	Spike emergence to harvest (days)
	Planting[z]	Spike emergence	Harvest		
1973	5 Oct	24 May	6 Jul	231	43
1974	28 Sep	21 May	8 Jul	235	48
1975	2 Oct	23 May	7 Jul	233	45
1976	3 Oct	14 May	8 Jul	224	55
1977	4 Oct	13 May	6 Jul	221	54
1978	13 Oct	4 Jun	17 Jul	234	43
1979	2 Oct	26 May	17 Jul	236	52
1980	9 Oct	25 May	11 Jul	229	47
1981	30 Sep	22 May	9 Jul	234	48
1983	13 Oct	24 May	8 Jul	223	45
1984	30 Sep	1 Jun	10 Jul	245	39
1985	6 Oct	7 May	29 Jun	213	53
1986	7 Oct	13 May	16 Jul	218	64
1987	11 Oct	14 May	24 Jul	215	71
1988	7 Oct	16 May	27 Jun	222	42
1989	7 Oct	20 May	6 Jul	225	47
1990	2 Oct	15 May	3 Jul	225	49
1991	1 Oct	10 May	21 Jun	221	42
Average				227	49

[z] Planting was during the autumn preceding the crop year.

Table 2. Leaf blotch severity scale for wheat

Scale value	Range in percent severity on indicated leaf[y]				Mean severity[z]
	Flag	Flag-1	Flag-2	Flag-3	
1				0-5	0.1
2				5-20	2.9
3				20-40	8.1
4			1-10	40-70	15.6
5		0-1	10-25	70-90	25.5
6		1-10	25-75	90-100	37.8
7		10-50	75-100	100	52.3
8	1-20	50-90	100	100	69.3
9	20-90	90-100	100	100	88.5
9.5	90-100	100	100	100	99.1

[y] The flag leaf is the leaf that subtends the spike. Flag-1 is the leaf immediately below the flag leaf, Flag-2 is the second leaf below the flag leaf, etc.

[z] Mean severity is the average for the four leaves, based on midpoint values for each range. Mean severity (P) can be calculated from the scale value (S) according to: $P = -0.38253 - 0.69435\ S + 1.17499\ S^2$ ($R^2 = 0.999$).

of the flag leaf. In some years, disease ceased to develop before the end of the season owing to dry weather, and assessments were terminated before the crop was mature. In most years, all four replicate plots were assessed for disease, but in two years only two replicates were assessed, and in 1986 only three replicates were assessed because winter killing had largely eliminated the fourth block. Original data for 1973 and 1980 were lost, but the means for each assessment date were available. Therefore, 1973 and 1980 are included in some analyses but not in others.

RESULTS

Descriptive epidemiology. During the 19 years of disease observation reported here, we rarely saw symptoms of leaf blotch in the autumn. We would occasionally find a lesion of *Septoria tritici* on a lower leaf. In the spring, symptoms appeared first on the lowest leaves, those that had emerged in the autumn. Initial symptoms usually appeared on leaves that were near or touching the soil surface. A tan lesion bearing pycnidia and with a yellow margin were typical symptoms and signs of infection. Because primary lesions formed so early in the spring and only on leaves that had emerged in the autumn, we infer that these lesions were from infections that took place during autumn, before the onset of cold weather, and that *Septoria tritici* and *S. nodorum* overwintered as asymptomatic infections. Incidence of primary infection was often high, on the order of 20% or greater.

Rainfall and high humidity from the moist soil surface in the early spring assured that leaf blotch developed to some extent on lower leaves every year. By the time the flag leaf had emerged, lower leaves were senesced and withered, although pycnidia could still be seen on them. In most years, disease incidence on lower leaves was 100% by the time of flag leaf emergence. On average, there were 49 days between spike emergence and harvest. In years when leaf blotch became severe, this was the period of greatest increase in the amount of disease on the main leaves (F-3 through F) of the culm. If rainfall and temperature were conducive (4,25), conidia from infections on lower leaves were carried by splashing raindrops to upper leaves, where they infected and produced secondary lesions.

The time of symptom appearance in the spring varied among years. In 1981, 50% incidence of symptoms was noted on 25 March. In 1983, 100% incidence was noted on 21 April. In contrast, in 1985 no symptoms were evident on 13 May, but by 28 May symptoms were evident on F-1 and F-2 as well as on lower leaves. In 1989, no symptoms were detectable on 26 April, when wheat was at the 3-node stage; but by 4 May, when flag leaves were starting to emerge, incidence was 55% and the average scale value was 0.55. By 11 February 1990, there was some yellowing on leaves, but no pycnidia. Two months later (14 April), when the first aboveground node had formed, severity was 5 to 15% on leaves F-4 and F-5.

From 1973 through 1985, the leaf blotch that we monitored in plots at the Purdue Agronomy Farm, and that we observed generally in Indiana, was caused by *Septoria tritici*. We occasionally found pycnidia of *S. nodorum* on leaves late in the season, and we found glume blotch on lodged wheat or on certain semidwarf breeding lines; but this species seemed never to be more than an incidental pathogen. The winter of 1985–86 was severe, and winter killing of wheat was extensive (5). Rainfall was frequent in the spring, especially about the time of spike emergence and flowering, and leaf blotch developed rapidly. The principal pathogen was *S. nodorum*. Since 1986, *S. nodorum* has been the predominant leaf blotch pathogen on wheat in Indiana. It caused major epidemics in 1990 and 1991, and was a significant pathogen in 1989. *Septoria tritici* was detected frequently in the early spring from 1986 to 1991 and often predominated on lower leaves. But as the season progressed, *S. nodorum* gained ascendancy and was responsible for the blotching of upper leaves and spikes.

Quantitative epidemiology. *Whole-plot estimates of severity.* The analysis of variance of data from the experiment con-

Table 3. Growth and development of wheat in terms of standard growth stages and the sequence of leaf emergence, Purdue Agronomy Farm, West Lafayette, Indiana

Date	Growth stage[x]		Uppermost leaf[y]	Leaf blotch[z]
Cultivar Monon, 1990				
4 March	2	Tillering		
14 April	31,14	1 node, 4 leaves	F-2	
18 April	31,14	1 node, 4 leaves	F-2	3.6
24 April	32,15	2 nodes, 5 leaves	F-1	4.2
1 May	39,16	F blade expanded	F	4.4
7 May	45	Mid-boot	F	2.4
15 May	52	Spikes just emerging	F	4.8
21 May	68	Late anthesis	F	6.0
29 May	71	Watery ripe	F	6.6
6 June	77	Late milk	F	7.6
13 June	79	Late milk	F	8.2
19 June	85	Soft dough	F	8.9
Cultivar Clark, 1991				
21 March	2	Tillering		
2 April	30,16	Pseudostem erection, 6 leaves on main culm	F-3	
9 April	31	1 node	F-2 (1/3)	
15 April	31	1 node	F-1 (1/3)	
22 April	32	2 nodes	F-1 (2/3)	
1 May	33,38	3 nodes	F (1/2)	
10 May	51	Spikes emerging	F	2.0
11 May	54	Spikes 1/2 emerged	F	
13 May	58	Spikes completely emerged	F	
15 May	67	Anthesis 3/4 complete	F	3.4
21 May	71	Watery ripe	F	4.3

[x] See Tottman, 1987.

[y] Number in parentheses is the proportional extension from the whorl of the indicated leaf blade. F = flag leaf, F-1 = 1st leaf below the flag leaf, etc.

[z] Severity on the upper four leaves, on the 0–9.5 scale (see Table 2). The 18 April 1990 assessment refers to leaves F-2 through F-5; the 24 April and 1 May 1990 assessments refer to leaves F-1 through F-4; the remaining assessments during 1990 refer to leaves F through F-3. In 1991, quantitative assessments did not commence until 10 May. Prior to that time, symptoms were confined to leaves below F-3.

Table 4. Vertical progression of Septoria leaf blotch on wheat cultivar Monon during 1990 at the Purdue Agronomy Farm, West Lafayette, Indiana

	Days before or after spike emergence[z]								
Leaf	–27	–21	–14	–8	0	6	14	22	29
F	0	0	0	0	0	1	4	24	37
F-1	0	0	0	0	5	30	33	16	3
F-2	0	0	3	5	34	8	3	0	0
F-3	0	3	36	35	1	1	0	0	0
F-4	22	23	1	0	0	0	0	0	0
F-5	18	14	0	0	0	0	0	0	0

[z] Day 0 is the day of spike emergence, May 15. Forty culms were examined on each date. Each number is the frequency of culms on which symptoms had progressed as high as the indicated leaf. For example, on day 22 symptoms had progressed to F-1 on 16 culms and to F on 24 culms. Leaf F is the flag leaf, leaf F-1 is the leaf immediately below the flag leaf, etc.

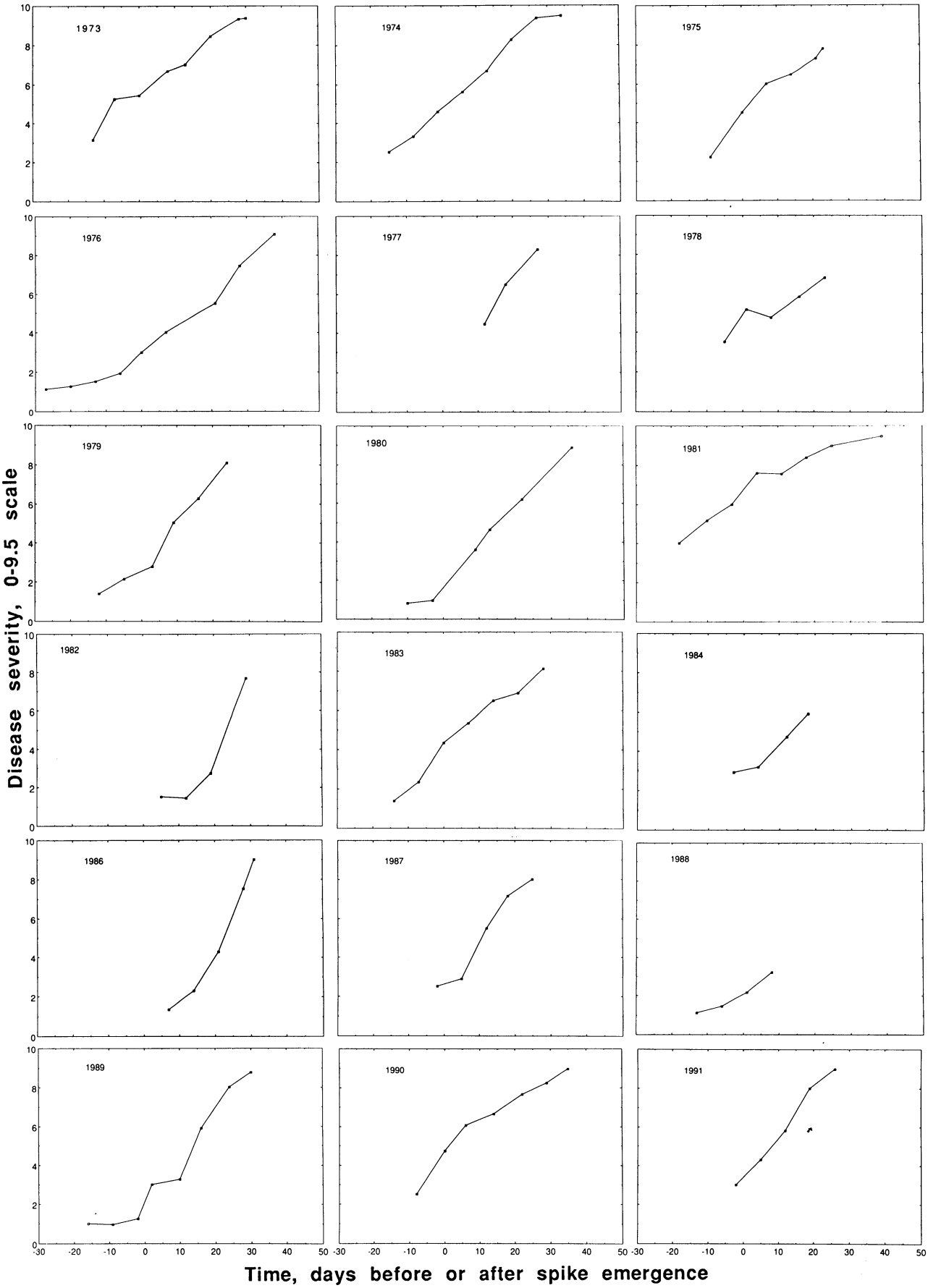

Fig. 1. Disease progress curves for leaf blotch on the upper four leaves of wheat cultivar Monon at the Purdue Agronomy Farm, West Lafayette, Indiana, 1973 to 1991. Severity is expressed on a scale from 0 to 9.5 (see Table 2). Time is expressed as days before or after spike emergence to facilitate comparisons among years. The curve for 1985 is omitted because there were only two dates of observation that year, and final severity reached only 4.

ducted in 1990 to compare whole-plot estimates of severity with means based on 10 plants per plot showed that cultivar, time of assessment, and their interaction were all highly significant, but assessment methods and interaction terms involving assessment method were not significant. The 64 standard deviations for plot means based on the 10 subsamples ranged from 0 to 1.08, with a mean value of 0.58. Eighty percent of the standard deviations were less than 0.7; 97% were less than 1.0. Among the 16 pairs of treatment means across cultivar and assessment time, five pairs of means differed by more than the range of their standard errors. Individual *t* tests on these data sets indicated significant differences for only two pairs: Monon at assessment time 2 had a value of S = 7.6 for the subsample method (Y = 0.62) and S = 7.2 for the whole-plot method (Y = 0.56), and Monon at assessment time 4 had a value of S = 8.9 for the subsample method (Y = 0.86) and a value of S = 9.0 for the whole-plot method (Y = 0.88). Although these differences are significant, they are small, especially the difference for time 4. This analysis leads us to conclude that whole-plot estimates, made as described in Materials and Methods, are as accurate as plot mean estimates based on subsamples.

Sequence of leaf development and vertical progression of disease. Although growth stage was recorded when disease severity was assessed, detailed records of the time of emergence of leaves from successive nodes of the culm were not routinely kept. However, such observations were made on Monon in 1990 and on cultivar Clark in a foliar fungicide trial in 1991; and these illustrate the timing of leaf emergence relative to standard growth stages (Table 3). The progress of symptom appearance up the plant canopy in 1990 is shown in Table 4.

In 1990, the first aboveground node was detectable and there were four fully expanded leaves per stem on cultivar Monon on 18 April, i.e., day –27. These leaves were F-2 through F-5. Of 40 individual culms examined, 22 had a severity of 4 or 5, meaning that symptoms were apparent on leaf 3 (F-4). The other culms had a

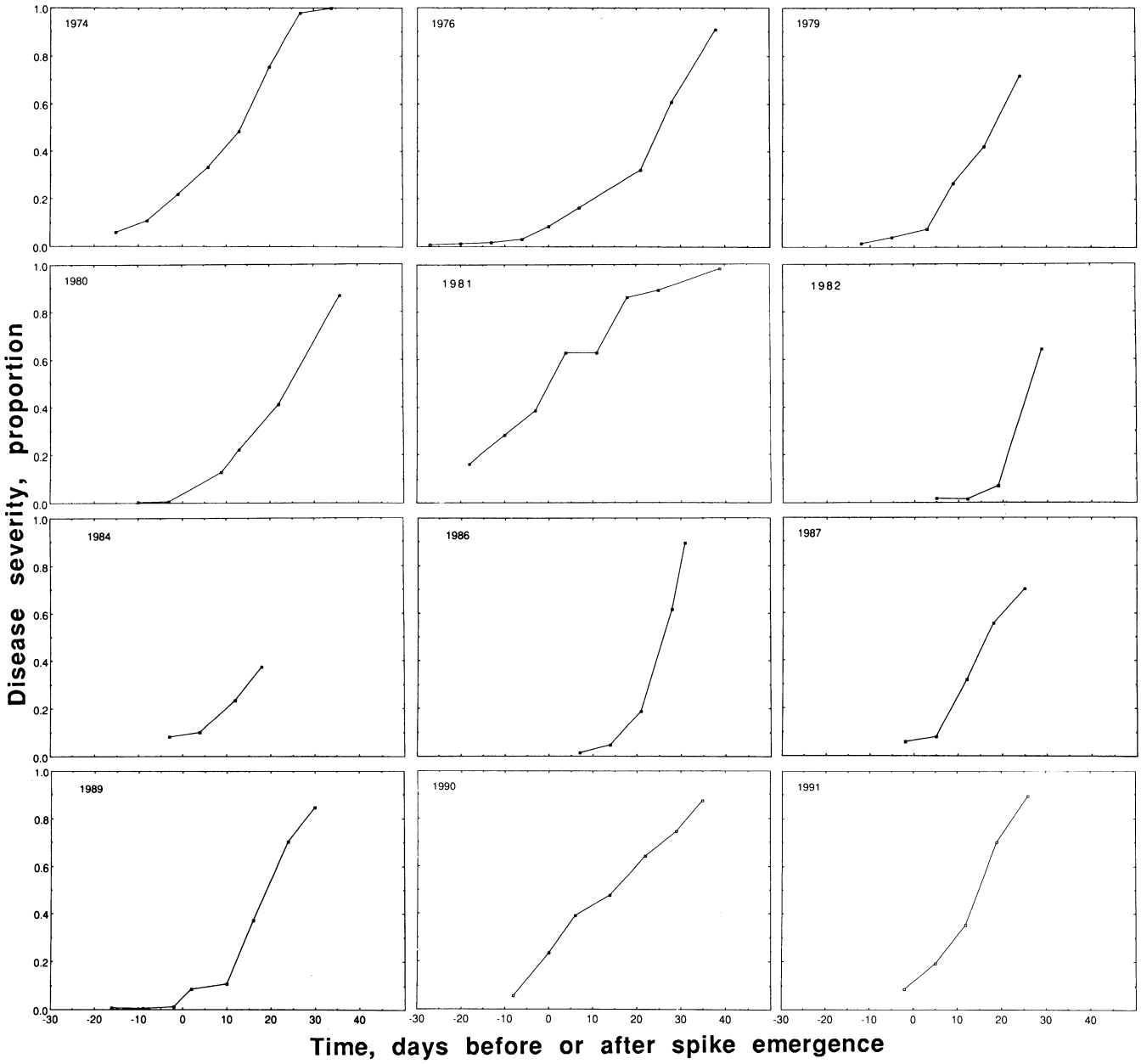

Fig. 2. Disease progress curves for leaf blotch on wheat cultivar Monon at the Purdue Agronomy Farm, West Lafayette, Indiana, for selected years from 1974 to 1991. Severity is expressed as the average proportion of leaf area showing symptoms for the upper four leaves (flag leaf and the three leaves below). Disease proportion was calculated from scale severity values according to the equation given in the footnote of Table 2. Time is expressed as days before or after the day of spike emergence, to facilitate comparisons among years.

severity of 3 or 2, meaning that symptoms were confined to F-5 (Table 4). By 24 April, day –21, the second node was clearly evident and the leaf blade of F-1 was fully expanded. Most plants still had symptoms no higher than F-4; a few plants had symptoms on F-3. On 1 May, day –14, the flag leaf blade was fully expanded, but symptoms had reached F-2 on only three of 40 culms. Symptoms were evident only up to F-3 on most of the remaining culms. On 7 May, day –8, when plants were in midboot, symptoms had reached F-2 on only five culms. Spikes were emerging on 15 May, day 0. Most culms had symptoms on F-2, but only five had symptoms on F-1. Anthesis was complete by 21 May, day 6, and symptoms had progressed to F-1 on 30 culms, but only one culm showed symptoms on F. Over the next three observation times, days 14, 22, and 29, the frequency of culms with symptoms on F increased greatly.

An examination of weather data in 1990, along with the timing of emergence of each leaf, allows us to infer the time of infection and the length of the incubation period. F-2 was fully expanded on 18 April (day –27), and symptoms first appeared on a few of these leaves on 1 May (day –14). There were 7 days with rain from 14 to 21 April, so we may assume that infections occurred on this leaf as it emerged from the whorl. If we consider that the apical portion of the leaf was exposed by 16 April, the mean temperature for the incubation period (16 April to 1 May) was 14.7°C. Leaf F-1 was fully expanded on 24 April (day –21), but symptoms were not seen on it until 15 May (day 0). However, rain was not sufficient for infection after this leaf emerged until the period 4 to 6 May. For the 12-day period 4 to 15 May, the mean temperature was 13.8°C. The flag leaf blade was fully emerged on 1 May (day –14), and we first saw symptoms on it on 21 May (day 6). If the rain that fell 4 to 6 May permitted infection of this leaf, this implies an incubation period of about 18 days. On 4 May, symptoms were still mainly at the level of F-3 or below. Although F was exposed to wetness and high humidity from 4 to 6 May, inoculum from the lower canopy might not have been capable of reaching F directly. Inoculum can be transported a considerable vertical distance by a sufficiently hard rain (26), but it is not possible to determine from standard meteorological records how hard the rains of early May were. Twenty-four hour precipitation totals for 4, 5, and 6 May were 15, 7, and 2 mm, respectively. The next period of rain was 10 to 17 May. Lesions were abundant on F-2 and beginning to appear on F-1 by then. This is more likely the period when F was initially infected by inoculum from midcanopy and suggests an incubation period of 12 days (10 to 21 May). The mean temperature during this period was 14.9°C. The three incubation periods under discussion had similar mean temperatures and were of similar duration (16, 12, and 12 days for F-2, F-1, and F, respectively). The estimate of 16 days for F-2 may be slightly long, because disease observations were only made every 7 days. Symptoms were not seen on F-2 on 24 April and were seen on 1 May, so they actually appeared sometime between these days, which would include a 12-day incubation period, as seen on F-1 and F.

By early April of 1991, cultivar Clark wheat had reached the pseudostem erection stage of growth, and there were six fully expanded leaves on the main shoot (Table 3). The uppermost fully expanded leaf was F-3, which is the leaf destined to be leaf 4 on the leaf blotch assessment scale. Over the next 33 days, the remaining leaves emerged at about 12-day intervals. Spike emergence took place over a 4-day period. The four aboveground nodes on these Clark plants are typical of what we see on Monon and other soft red winter wheat cultivars adapted to Indiana. These nodes, from the base of the culm and moving apically, subtend leaves F-3, F-2, F-1, and F. The node subtending F-3 may be near the soil line or a few centimeters above it. By the time F has fully emerged, F-5 and lower leaves are typically withered, and F-4 withers soon after.

Disease progress. Our objective in assessing disease throughout the spring was to learn more about how disease severity increases with time. Septoria leaf blotch incidence was consistently high by the time flag leaves emerged. Thus, disease progress during flowering and grain filling was mainly a matter of increase in severity by vertical spread of infections.

Plotting disease progress curves with scale value severities (S) depicts the epidemic in the original units of measurement and allows one to infer the vertical distribution of disease as well as total severity on the upper four leaves. A severity value of 4 indicates that symptoms have reached leaf F-2; a severity of at least 6 indicates that symptoms are on leaf F-1; a severity of at least 8 indicates that symptoms are on F (Table 2). For many of the years, the increase in S with time was approximately linear (Fig. 1).

Because the day of spike emergence is a cardinal phenological event, familiar to most farmers, we consider it useful to examine severity of leaf blotch at that stage of growth. In some years, the day of disease assessment did not happen to fall on the day of spike emergence, so a scale value was interpolated from readings on either side of this day. In 3 years when there was little disease development, assessments did not commence until after spike emergence, and in these cases severity for the day of spike emergence was estimated by extrapolation of the equation relating scale value to time. In all 3 years, the extrapolated value of S_0 was 0. There was considerable variation in severity at the time of spike emergence (day 0 on the time scale). Symptoms had reached F-2 by this time in 1973, 1974, 1975, 1978, 1981, 1983, and 1990 (i.e., average severity was at least 4; see Fig. 1). Symptoms were confined to F-3 or below at this time in the other 12 years. The greatest amount of disease at spike emergence was in 1981, when symptoms had already progressed to leaf F-1.

A typical method of graphing disease progress is to plot disease proportion against time (3). For each field plot and observation date, we converted the scale value, S, to disease percent, P (see footnote of Table 2). At low values of S (<1.5),

Table 5. Area under the disease progress curve ($AUDPC^{x,y}$) for leaf blotch epidemics on wheat cultivar Monon, Purdue Agronomy Farm, West Lafayette, Indiana, 1974–1991

Year	Replicates[z]	$AUDPC_Y$	Year	Replicates[z]	$AUDPC_S$
1985	4	0.65 a	1985	4	21.0 a
1988	4	0.81 a	1988	4	35.4 a
1984	4	3.41 ab	1982	2	68.7 ab
1982	2	3.48 a-c	1984	4	81.9 b
1986	3	4.81 a-d	1986	3	93.4 bc
1976	4	5.27 b-d	1977	4	97.4 bc
1977	4	6.98 cd	1975	4	106.6 b-d
1975	4	7.29 cd	1987	4	131.2 c-e
1978	4	7.87 d	1976	4	136.1 c-e
1987	4	8.10 de	1978	4	143.8 d-f
1989	4	11.13 ef	1989	4	175.1 e-g
1991	4	12.36 f	1991	4	183.0 fg
1983	4	12.56 f	1983	4	207.7 g
1990	4	20.22 g	1990	4	274.8 h
1974	4	22.56 g	1974	4	299.9 h
1981	4	29.54 h	1981	4	360.5 i
LSD 0.05		3.18			45.6

[x] $AUDPC_Y$ is calculated from disease proportion (Y) plotted against time; $AUDPC_S$ is calculated from scale value (S) plotted against time.

[y] Within a column, means followed by a letter in common are not significantly different at $P = 0.05$ according to Fisher's protected LSD.

[z] The number of replicate plots analyzed.

the conversion formula gives lower values of P than those given in Table 2. The few values in the data set that were less than 1.5 were transformed to $P = 0.6\%$. Percent severity was converted to a proportion, and these values were plotted against the same time scale used for plotting S vs. time. Thus, the y-axis is the average proportion of the area of the upper four leaves (F through F-3) visibly diseased. Disease progress varied as depicted for selected years (Fig. 2). Few of the disease progress curves were sigmoidal, although curves for 1974, 1987, and 1989 have a roughly sigmoid shape. Several others (1976, 1979, 1980, 1982, 1984, 1986) showed an exponential increase early in the epidemic but did not flatten out before the wheat had ripened (Fig. 2). Most curves were essentially linear once Y = 0.1.

Area under the disease progress curve was calculated for each epidemic, using the formula: $\text{AUDPC} = \Sigma[(Y_i + Y_{i+1})/2][t_{i+1} - t_i]$, in which t is time in days relative to date of spike emergence and Y is disease proportion. The total time period and number of observations (n) varied among years (see Fig. 1). AUDPC was calculated for both disease severity scale values (AUDPC_S) and proportion of disease (AUDPC_Y). AUDPC_S values ranged from 21 to 360; AUDPC_Y values ranged from 0.30 to 29.5 (Table 5).

AUDPC_Y and AUDPC_S were calculated for each replicate each year and subjected to analysis of variance. Differences among years were highly significant (Table 5). The most severe epidemic occurred in 1981. Disease appeared especially early that year and increased steadily in intensity (Figs. 1 and 2). By the day of spike emergence, the scale value was 6.7 (=48.6%). The least disease developed in 1985. Symptoms first appeared 3 weeks after spike emergence and remained confined to lower leaves in the canopy. There were some slight changes in ranking of years according to whether AUDPC_Y or AUDPC_S was analyzed, and some slight changes in significant groupings according to Fisher's protected LSD.

To further compare leaf blotch epidemics among years, various models of disease progress were compared. Disease proportions (Y_i) were subjected to log, logistic, Gompertz, and monomolecular transformations (3). Mean values for each observation time were calculated and regressed on time and expressed as days relative to day of spike emergence. Also, means of the original scale values (S_i) and disease proportions (Y_i) were regressed on time. The purpose of regression with S_i, Y_i, or a transformation of Y_i (all designated generically as Y_*) was to see if any of these models resulted in a linear relation between Y_* and t, by which epidemics could be characterized with rate and position parameters. Even though many of the disease progress curves in which proportion of disease was plotted against time were not curvilinear, we thought it worthwhile to examine the effects of these various transformations because they are so commonly used for disease progress analysis.

Rates of disease increase for S plotted against time (r_S) ranged from 0.105 to 0.301 per day (Table 6). Rate parameters for Y plotted against time (r_Y) ranged from 0.006 to 0.033 per day. These rates of disease progress were each compared to various cardinal severities (at the day of spike emergence, 26 days after spike emergence, and at the final observation time) and to AUDPC (see Table 6 for values of these parameters). r_S was correlated only to S_0 and AUDPC_S; in both cases the

Table 6. Epidemic parameters for Septoria leaf blotch development on wheat cultivar Monon in trials at the Purdue Agronomy Farm conducted from 1973 through 1991

	Severity at time[w]							AUDPC[y]		Rate parameter[z]	
Year	S_0	S_{26}	S_f	Y_0	Y_{26}	Y_f	T_8[x]	S	Y[x]	r_S	r_Y
1973	5.4	9.1	9.4	0.31	0.92	0.98	18	290	2.59	0.137	0.020
1974	4.7	9.2	9.5	0.22	0.87	0.99	22	300	22.56	0.161	0.021
1975	4.5	8.4	9.1	0.20	0.80	0.88	22	213	14.58	0.180	0.022
1976	3.0	6.9	7.8	0.09	0.51	0.59	35	170	7.65	0.124	0.010
1977	0.0*	8.1	8.3	0.00*	0.69	0.70	26	97	6.98	0.237	0.033
1978	4.3	7.0*	6.8	0.24	0.49*	0.48	36	144	7.87	0.105	0.012
1979	2.5	8.2*	8.1	0.04	0.79*	0.72	25	143	8.19	0.212	0.020
1980	1.6	7.0	8.9	0.02	0.52	0.87	33	204	13.29	0.185	0.019
1981	6.7	9.0	9.4	0.49	0.90	0.99	14	360	29.54	0.112	0.017
1982	0.0*	6.2	7.7	0.00*	0.44	0.62	34	69	3.48	0.275	0.025
1983	4.4	7.8	8.1	0.18	0.65	0.71	26	208	12.56	0.161	0.017
1984	3.0	6.9*	5.9	0.07	0.44*	0.36	34	82	3.41	0.152	0.014
1985	0.0	3.4	4.0	0.00	0.11	0.17	41	21	0.65	0.301	0.019
1986	0.0*	6.6	9.0	0.00*	0.53	0.80	31	93	4.81	0.272	0.027
1987	2.6	8.5*	8.0	0.04	0.69*	0.67	24	131	8.10	0.246	0.026
1988	2.1	4.8*	3.2	0.06	0.20*	0.09	60	34	0.81	0.114	0.006
1989	2.1	8.3	8.8	0.04	0.74	0.84	28	175	11.13	0.200	0.019
1990	4.7	8.0	9.0	0.24	0.69	0.87	26	275	20.21	0.139	0.018
1991	2.9	9.0	9.0	0.11	0.88	0.88	21	102	6.10	0.202	0.026
Unpaired *t* tests to compare years 1973–1985, when *Septoria tritici* predominated, with years 1986–1991, when *Stagonospora nodorum* predominated:											
Means											
1973–1985	3.2	7.5	7.9	0.14	0.62	0.70	28.2	178	11.80	0.180	0.019
1986–1991	2.7	7.5	7.8	0.08	0.62	0.69	31.7	135	8.53	0.196	0.020
Difference	0.5	0.0	0.1	0.06	0.00	0.01	–3.5	43	3.27	–0.015	–0.001
Probability	0.61	0.95	0.92	0.36	1.0	0.98	0.50	0.37	0.42	0.62	0.72

[w] S refers to severity on the 0–9.5 scale (see Table 2). Y refers to severity as proportion of diseased tissue. Subscripts 0, 26, and f refer to severities on day 0 (the day of spike emergence), 26 days after spike emergence, and the final date of observation, respectively. If the disease observation day did not happen to correspond to the day of spike emergence or to day 26, severity on these days was obtained by interpolating from values on either side of them. When no observation occurred before day 0 or after day 26, severity for these days was estimated by extrapolation with the regression equation relating severity to time. Values estimated by extrapolation are marked with an asterisk.

[x] T_8 is the time, in days after spike emergence, when symptoms appeared on the flag leaf, i.e., when the scale value severity reached 8. T_8 was calculated from the regression model that related S to time.

[y] AUDPC is the area under the disease progress curve. Subscript S refers to the AUDPC for severity expressed in scale values (S); subscript Y refers to the AUDPC for severity expressed as proportion of disease (Y). Both versions of AUDPC were calculated for the actual interval of time over which disease severity was recorded.

[z] Rate parameters for two models of disease progress: r_S is the rate parameter for the regression of scale value on time; r_Y is the rate parameter for the regression of per unit severity (Y) on time.

correlation was negative. Although the correlation between r_S and AUDPC was significant, r_S would not be a reliable predictor of $AUDPC_S$ (Fig. 3). A considerable range of r_S values was associated with values of $AUDPC_S$ of 150 or less. There was no correlation between r_Y and $AUDPC_Y$. r_Y was only correlated with Y_f, and this correlation was positive.

The negative correlations between r_S and S_0 or $AUDPC_S$, and the positive correlation between S_0 and $AUDPC_S$, suggest that severe epidemics have a slower rate of increase but begin at an earlier stage of wheat growth and have a longer period of development.

S_0 was correlated to S_{26} but not to S_f, whereas Y_0 was correlated to both Y_{26} and Y_f (Tables 7 and 8). The *R*-values for $Y_0 \cdot Y_f$ and $S_0 \cdot S_f$ were similar, although the latter just failed to be significant. S_{26} was correlated to S_f, as was Y_{26} to Y_f. This is expected because day 26 was usually near the final day of observation (See Figs. 1 and 2). $AUDPC_S$ was correlated to S_0, S_{26}, and S_f. The highest correlation was between $AUDPC_S$ and S_0 (Table 7 and Fig. 4). A similar pattern was seen for $AUDPC_Y$ and Y_0, Y_{26}, and Y_f (Table 8, Fig. 5). This suggests that severity at spike emergence would be an indicator of overall epidemic development as expressed by AUDPC.

It was stated earlier that since 1986, *S. nodorum* has replaced *Septoria tritici* as the predominant leaf blotch pathogen in Indiana. To see if epidemics caused by these two pathogens differed in any parameter of disease progress, we compared the years 1973 to 1985 with the years 1986 to 1991. On the basis of unpaired *t* tests, mean values for none of the parameters for the two groups of years differed (Table 6).

Rate parameters were calculated for several models, as described above. Appropriateness of a model can be judged by the magnitude of R^2, residual mean square, and pattern of residuals plotted against fitted values. The various models for each year's data were examined by these criteria. In every year but 1982, the model based on plotting S values vs. days relative to spike emergence yielded an acceptable linear relationship between severity and time. In some years, other models were also acceptable, but none was as generally appropriate as the scale value model. None of the models fit the 1982 data well. In 2 years, 1976 and 1989, the model was improved by excluding data early in the season when there was little change in severity. In 1976, this entailed dropping the readings for 17 and 24 April (days –27 and –20). These observations were among the earliest, relative to the day of spike emergence, of any year. For 1989, the fit of the models was improved by dropping the observation made 2 May (day –16). There was no change in severity from this time to 1 week later.

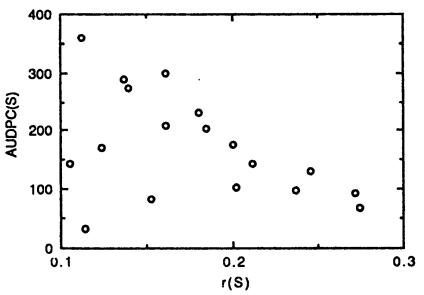

Fig. 3. The relation between area under the disease progress curve ($AUDPC_S$) and the rate parameter for scale value of leaf blotch (r_S) on Monon wheat at the Purdue Agronomy Farm, West Lafayette, Indiana, for the years 1973 to 1991. $R = 0.542$.

Because of the general acceptability of the model involving scale values, unpaired *t* tests were performed for every combination of 2 years to determine the significance of differences between r_S values (29, p. 258). A simple mean separation method of indicating differences could not be used because the variable number of data points that went into calculation of r_S each year affected the threshold value of *t* for each test of significance. Instead, results are presented in an array table (Table 9). Among the 153 combinations of years, there were 55 significant differences among r_S values.

DISCUSSION

Primary inoculum does not appear to be a limiting factor for leaf blotch of wheat in Indiana. In the early spring, abundant infections can be seen in most fields examined. The source of this primary inoculum has not been unequivocally discovered and remains a point of conjecture. Indirect evidence suggests that ascospores of *Mycosphaerella graminicola* and *Phaeosphaeria nodorum* are the primary inocula. The high incidence of infection early in the season and the fact that most wheat is sown after soybeans or maize rather than wheat suggest that the primary inoculum is an airborne propagule that can be carried from field to field, at least tens of meters to a few kilometers. Because ascospores of *P. nodorum* and *M. graminicola* are airborne, but pycnidiospores are carried only short distances by wind (18), we surmise that ascospores are involved in primary disease establishment in Indiana, as they have been implicated in other areas of the world (18). Seedborne infection could also account for the high incidence of *S. nodorum* infection (18) but not the high incidence of *Septoria tritici* infection that we customarily see early in the spring. Although seed infection by *Septoria tritici* has been demonstrated (2), evidence is lacking that this is a natural means of pathogen carryover. Much of the wheat in Indiana is sown from purchased seed, and much of this is treated with a fungicide.

The time of primary infection is uncertain. There are about 3 months between harvest of a mature crop of wheat in the summer and emergence of the new crop in the autumn. If primary infection occurs shortly after the new crop emerges, then the inoculum must not only be capable of dispersal over some distance, but must also be produced or survive long after the previous season's wheat crop has been harvested. Pycnidiospores are readily discharged from pycnidia when wet, and with repeated wettings the contents of pycnidia are depleted (7,14). Faulkner and Colhoun (9) presented evidence that wheat residue in England could produce pycnidiospores of *S. nodorum* throughout the autumn, winter, and early spring, and that these spores could be caught 2 m above ground; so it is probable that for this pathogen,

Table 7. Correlation matrix for epidemic parameters[z] for leaf blotch development on wheat cultivar Monon in trials conducted from 1973 through 1991 at the Purdue Agronomy Farm, West Lafayette, Indiana

	S_0	S_{26}	S_f	$AUDPC_S$	r_S
S_0	1.0	0.706*	0.409	0.781*	–0.751*
S_{26}		1.0	0.808*	0.717*	–0.309
S_f			1.0	0.690*	–0.093
$AUDPC_S$				1.0	–0.542*

[z] Parameters are as defined in Table 6 and are based on assessment of disease on the 0–9.5 scale (see Table 2). Correlation coefficients marked by an asterisk are significant at a probability of ≤0.05.

Table 8. Correlation matrix for epidemic parameters[z] for leaf blotch development on wheat cultivar Monon in trials conducted from 1973 through 1991 at the Purdue Agronomy Farm, West Lafayette, Indiana

	Y_0	Y_{26}	Y_f	$AUDPC_Y$	r_Y
Y_0	1.0	0.550*	0.463*	0.840*	–0.262
Y_{26}		1.0	0.897*	0.732*	0.390
Y_f			1.0	0.768*	0.472*
$AUDPC_Y$				1.0	0.014

[z] Parameters are as defined in Table 6. Original data were recorded on the 0–9.5 scale (see Table 2) and converted to percent severity before analysis. Correlation coefficients marked by an asterisk are significant at a probability of ≤0.05.

conidia as well as ascospores function as primary inoculum. Evidence regarding the ability of *Septoria tritici* to produce pycnidiospores in host debris is conflicting (1). The summers in Indiana, from the time of wheat harvest in early July until the emergence of the new crop in October, are characterized by intermittent heavy rains. Normal precipitation for July, August, and September in west central Indiana, where our studies were conducted, is 277 mm, or 28% of the total annual precipitation. When we examine leaf residues at the end of the summer, we find mainly empty pycnidia. It seems unlikely that pycnidia would contain sufficient spores after three humid and often wet summer months to cause substantial numbers of primary infections on the new wheat crop in the autumn, especially considering that the new wheat crop is rarely planted into the residue that bears these pycnidia. Exposed pycnidiospores do not survive for many days unless relative humidity is very low (12). This also implicates airborne ascospores as the primary inoculum.

One reason for the greater frequency of severe leaf blotch in recent years may be the increasing adoption of reduced tillage as a soil conservation measure in Indiana and other states of the U.S. Corn Belt. Spores of *Septoria tritici* and *S. nodorum* in buried wheat residue lose viability rapidly (2,14). Tillage practices that leave wheat residue on top of the soil certainly increase the availability of primary inoculum. However, even in the early 1970s, when moldboard plowing was much more common, there were severe epidemics of Septoria tritici blotch, suggesting that plowing much of the cropland does not reduce primary inoculum to insignificant levels.

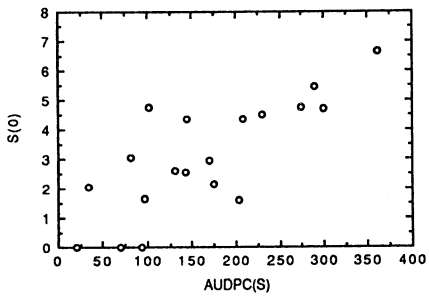

Fig. 4. The relation between scale value of leaf blotch on Monon wheat at the Purdue Agronomy Farm, West Lafayette, Indiana, on the day of spike emergence (S_0) and area under the disease progress curve ($AUDPC_S$) for the years 1973 to 1991. $R = 0.781$.

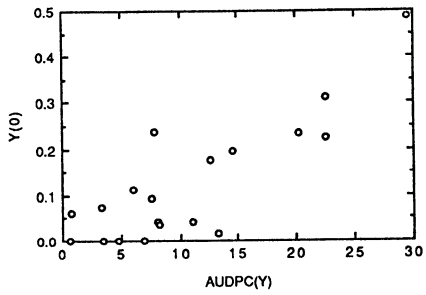

Fig. 5. The relation between severity of leaf blotch on Monon wheat at the Purdue Agronomy Farm, West Lafayette, Indiana, as average proportion of symptomatic tissue on the upper four leaves (Y_0) on the day of spike emergence and area under the disease progress curve ($AUDPC_Y$) for the years 1973 to 1991. $R = 0.840$.

Although in recent years we have found both *Septoria tritici* and *S. nodorum* infections on wheat early in the season, *S. nodorum* has predominated later in the season. This may be because of its greater tolerance of high temperature compared to *Septoria tritici*, and perhaps because of an increase in susceptibility of wheat to *S. nodorum* as it matures (32). Infections of *Septoria tritici* and *S. nodorum* often occur on the same plant and may even occur on the same leaf. Prior to 1986, *S. nodorum* was almost an incidental pathogen on wheat in Indiana. Occasionally we would see a flush of infection late in the season, but this apparently developed too late to cause any appreciable reduction in yield. Since 1986, *S. nodorum* has been the principal leaf blight pathogen on wheat in Indiana. There are three conditions that might explain this. From the late 1970s through the late 1980s, much of the wheat area in Indiana was planted to cultivars with resistance to *Septoria tritici*: Oasis, Sullivan, Caldwell, and Auburn. During this same period, farmers increasingly used reduced tillage. The combination of greater resistance to *Septoria tritici* and more wheat residue left on the soil surface after harvest may have favored *S. nodorum*. It is also possible that in recent years, the wet weather conducive to infection and spore dispersal has been in conjunction with warmer weather, which would favor *S. nodorum*. Wainshilbaum and Lipps (32) found that *S. nodorum* blotch developed well in the glasshouse at 19, 24, or 29°C, whereas *Septoria tritici* blotch developed poorly at 29°C. Hart et al. (15) reported that both fungi infected wheat in Michigan in 1982 and 1983. They speculated that *Septoria tritici* re-

Table 9. Significance at $P = 0.05$ of differences in r_S values among years for epidemics of Septoria leaf blotch on Monon wheat at the Purdue Agronomy Farm, West Lafayette, Indiana

		Year, r_S value, and number of points in the disease progress curve[z]											
		1976	1974	1983	1975	1980	1991	1979	1989	1977	1987	1986	1982
		0.159	0.161	0.161	0.180	0.181	0.202	0.212	0.227	0.237	0.246	0.272	0.275
Year	r_S	7	9	7	6	5	6	6	7	3	5	4	4
1988	0.096	*	*				*	*	*		*		
1978	0.105	*	*		*		*	*	*		*		
1981	0.112	*	*	*	*	*	*	*	*	*	*	*	*
1973	0.137				*		*	*	*	*	*	*	*
1990	0.139			*			*	*	*		*	*	
1984	0.152												
1976	0.159						*	*	*		*	*	
1974	0.161						*	*	*	*	*	*	*
1983	0.161								*		*		*
1975	0.180												
1980	0.181												
1991	0.202												
1979	0.212												
1989	0.227										*		
1977	0.237												
1987	0.246												
1986	0.272												
1982	0.275												

[z] A significant *t* test is indicated by an asterisk. Columns for 1988, 1978, 1981, 1973, 1990, and 1984 are omitted because there were no significant differences among any of the pairs of these years.

mained the dominant pathogen in 1983 because of a cool May. In 1982, *S. nodorum* gained ascendancy and was the principal cause of lesions on flag leaves.

Under our conditions, leaf blotch, whether caused by *Septoria tritici* or *S. nodorum*, shows a clear vertical gradient as symptoms progress up the plant. This is because the disease becomes established in the crop at an early stage of growth and secondary infections on successively higher (and younger) leaves are the result of splash dispersal of pycnidiospores from leaves below (26). Our modified version of the Saari–Prescott scale proved to be effective and easy to use in assessing severity on the upper four leaves of the culm. The scale can be converted to percent severity values, but in its original form it denotes both severity (average percent area showing symptoms on the upper four leaves), and the leaf layer to which symptoms have progressed. The use of the scale in extension education programs and fungicide spray advisory schemes may be more effective than use of percent severity.

Detailed notes on the sequence of leaf emergence and appearance of symptoms on leaves in 1990 suggested that the incubation period was about 12 days when mean temperature was 14 to 15°C. These incubation periods for symptoms caused mainly by *S. nodorum* were somewhat longer than incubation periods reported for *Septoria tritici* by Hess and Shaner (16), but shorter than incubation periods for *Septoria tritici* reported from England (27,28).

Although in all but one year the severity scale value on the day of spike emergence was 5 or less, meaning that symptoms were confined to F-2 or below, symptoms reached the flag leaf 26 days later in 10 of the 19 years (Table 6). The time required for severity to reach a value of 8, i.e., for symptoms to appear on the flag leaf, averaged 29 days after spike emergence (standard error = 2.3 days).

It is not clear to what extent the increase in severity on a leaf is due to lesion growth rather than to new infections, once initial symptoms appear on it. In the glasshouse, we sometimes inoculate wheat leaves with *Septoria tritici* by pricking the leaf with a needle and rubbing a small droplet of spore suspension into the wound. Plants thus inoculated are not given a postinoculation moist period. The lesion that subsequently develops may extend 2 or 3 cm, suggesting that in nature a single lesion may extend considerably in length. When flag leaves of susceptible wheat cultivars were inoculated with 20-µl droplets of spore suspension of *S. nodorum*, lesions grew at a rate of 3.53 mm^2 day^{-1} and reached a final area of 36 mm^2 (33). Thus, we might conclude that a significant amount of increase in severity on a leaf, once it shows some degree of blotching, is the result of lesion expansion. However, if an extended period of moisture and high humidity occurs after an earlier infection event, additional new infections could occur and account for an increase in severity.

When disease severity on the 0 to 9.5 scale was plotted against time, disease progressed in a generally straight line (Fig. 1). In some years, there was a lag phase early in the epidemic, but in other years there was no lag phase over this same time relative to the day of spike emergence (e.g., compare 1973 through 1975 with 1982, 1987, and 1989). This suggests that the lag phase is not an inherent feature of disease progress on the upper four leaves but a consequence of weather conditions. There was generally no diminution in disease progress rate as severity approached the maximum each year.

Disease progress curves in which proportion of disease was plotted against time showed the same variation in existence or absence of a lag phase as curves of S vs. time (See Fig. 2). On either scale of severity, there were fluctuations in rate of disease progress during an epidemic that suggested variations in favorability of weather for infection or incubation.

Compared to some diverse epidemics used by Campbell and Madden (3) to illustrate disease progress curves (see their Fig. 8.1, p. 162), the epidemics of leaf blotch on wheat progressed rapidly, going from light infections on the fourth leaf to nearly complete blighting of foliage in 30 to 50 days. There have been few studies of leaf blotch progress in the field from natural infection, and among those that have been published, there is such diversity in presentation of data that comparisons are difficult. Eyal and Ziv (8) plotted cumulative percent severity of disease against time and obtained a straight line. Given the autocorrelation that would arise from using cumulative disease severity (19), a straight line is not unexpected. Forrer and Zadoks (10) observed a moderately exponential increase in average percent necrotic area of the upper four leaves with time in plots that were not inoculated. Of four models that we evaluated for their data—linear, exponential, logistic, and Gompertz—the exponential model gave the best fit to the data; and the rate parameter, r_E, was 0.06 per day, a value at the low end of the range we observed in our experiments. Thomas et al. (30) presented leaf blotch progress curves for several locations in England, but because they used a thermal time scale without providing the corresponding calendar times, their epidemics cannot be compared with others. From data Hampton and Close (13) presented on progress of Septoria tritici blotch on F and F-1 on wheat that was not treated with fungicide, we calculated an apparent infection rate, r_L, of 0.066 per day. The apparent infection rates on Monon at the Purdue Agronomy Farm in 1973 and 1974 were previously reported to be 0.118 and 0.116 per day, respectively (24). In an epidemic on cultivar Longbow in England, lesions appeared on the flag leaf about 20 days after spike emergence (28). This is similar to the time course we observed.

When we ranked years according to AUDPC, there was a fairly continuous gradation of leaf blotch intensity (Table 5). The mean separation tests showed overlapping ranges, except that years 1974, 1981, 1983, and 1990 stood out as having particularly severe epidemics. Epidemics in 1989 and 1991 could be described as moderately severe.

AUDPC was correlated to three cardinal severities (at spike emergence, 26 days later, and final time of observation) whether expressed as S or Y. Given the more or less linear progress of disease, this is expected. The rate parameter for the scale value disease progress curve was negatively correlated with S_0 and $AUDPC_S$. The higher the severity at the time of spike emergence, the slower the subsequent progress of disease as it approached its maximum. If the value of AUDPC is taken as an overall indicator of disease intensity, then disease progress rate (r^*) on the 0 to 9.5 scale is a weak negative predictor and on a disease proportion scale is no predictor at all. A more reliable indicator of AUDPC was severity at spike emergence.

The data in this report are all from one wheat cultivar, Monon, which is very susceptible to both *Septoria tritici* and *S. nodorum*. Our observations suggest that primary infection occurs in the autumn. Severity is generally low until after all of the leaves of the culm have expanded, even in years when a destructive epidemic develops. Once the flag leaf has emerged, disease progresses steadily to some maximum constrained by weather. Under favorable conditions, all of the foliage can be blotched within 25 to 35 days of spike emergence. This interval of time corresponds to the period of dry matter accumulation in wheat (17); and consequently, severe disease can greatly reduce yield and grain quality. In a fungicide trial with cultivar Clark, we measured a yield reduction of 38% between the most effective treatment and the unsprayed control (23). Until greater degrees of resistance to *Septoria tritici* and *S. nodorum* have been bred into soft red winter wheat cultivars adapted to Indiana and surrounding states, fungicides are needed to assure a good yield of high-quality grain in years that are favorable for leaf blotch. The patterns of disease development reported here may have value for determining the need for a fungicide. When used in conjunction with a weather-based disease forecast (4), it may be possible to advise growers, at about the time of spike emergence, whether a fungicide would be justified.

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