

Disease Progress of *Sclerotinia* Wilt of Sunflower at Varying Plant Populations, Inoculum Densities, and Environments

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ABSTRACT

Nelson, B. D., Hertsgaard, D. M., and Holley, R. C. 1989. Disease progress of *Sclerotinia* wilt of sunflower at varying plant populations, inoculum densities, and environments. *Phytopathology* 79:1358-1363.

Disease progress of *Sclerotinia* wilt of sunflower, caused by *Sclerotinia sclerotiorum*, was studied in varying plant populations over eight sites with different inoculum densities. Two prominent characteristics of disease progress were the absence of disease symptoms during the first 40–60 days after planting and the wilting of most plants after anthesis (~75 days after planting). Analysis of disease progress with the Weibull model showed that plant population affected the rate of disease progress, but

no consistent relationship between these factors was found. Lower plant populations, for example, did not consistently have lower or higher rates of disease progress. The differences in rates of disease progress between plant populations had no apparent effect on seed yield. Rates of disease progress were positively correlated with inoculum density, but not with precipitation and temperature (as growing degree days).

Additional keywords: *Sclerotinia sclerotiorum*, epidemiology, soilborne diseases.

Sclerotinia wilt caused by *Sclerotinia sclerotiorum* (Lib.) de Bary is a widespread, destructive disease of sunflower in North Dakota. Crop rotation to nonsusceptible hosts is, at present, the principal means of control because no resistant hybrids or economical chemical controls are known (21,30). *Sclerotinia* wilt of sunflower is the only crop disease caused by *S. sclerotiorum* in which root infection consistently occurs (1,9); all other diseases caused by this pathogen are primarily initiated on aboveground plant parts by ascospores or mycelium from sclerotia near the soil surface (1).

The quantitative epidemiology of *Sclerotinia* wilt has received limited study. Hoes and Huang (7,10) reported the effect of between-row and within-row spacing on development of disease, and Holley and Nelson (8) studied the effect of plant population and inoculum density on disease incidence. The progress of *Sclerotinia* wilt over time has not been intensively studied or analyzed with epidemiological models. Such studies could further our understanding of the dynamics of this disease, may provide information useful in control, and would add to the general knowledge about the epidemiology of diseases caused by soilborne pathogens.

The objective of this research was to study disease progress of *Sclerotinia* wilt in commercially used plant populations on

different sites with varying inoculum densities and environments. A preliminary report of this study was published (20).

MATERIALS AND METHODS

Description of field experiments. Eight field experiments were established at five locations, four in eastern North Dakota and one in western Minnesota, over 2 yr, from 1981 to 1982. These locations were Carrington, Galesburg, Northwood, and Wyndmere in North Dakota and Fargo-Moorhead in Minnesota. The sites chosen were fields naturally infested with *S. sclerotiorum* that had been planted to susceptible crops within the previous 3 yr. The site designations (and year) and respective soil types were as follows: Carrington irrigated (1982) and Carrington dryland (1982), Heimdahl loam; Galesburg east (1981) and Galesburg west (1981), Swenoda sandy loam; Northwood south (1981) and Northwood north (1982), Gardena silt loam; Wyndmere (1981), Overly silty clay loam; Fargo-Moorhead (1982), Fargo silty clay. The two experiments conducted at each Carrington, Galesburg, and Northwood location were located in different areas of the same field. All sites were dryland except Carrington irrigated, which was furrow-irrigated with a total of 23 cm of water during July and August.

Each experiment had 12–24 plots, hereafter referred to as experimental units, of various plant populations. An experimental unit consisted of four to six rows, each 6 m long with row spacing of 76.2 cm (Carrington, Northwood, and Wyndmere sites) or 91.4 cm (Galesburg and Fargo-Moorhead sites). Plant populations of 37.0, 49.4, and 61.7×10^3 plants per hectare were replicated four to six times at each site. At the two Northwood sites an additional population of 74.1×10^3 plants per hectare was included. Sites were planted at the rate of 98.8×10^3 plants per hectare, and plant populations were established at the three- to four-leaf stage by carefully thinning to the appropriate plant spacing by using a graduated measuring stick. Poor stand development at Galesburg west prevented the establishment of 61.7×10^3 plants per hectare. No disease symptoms occurred before thinning. The experimental design was a randomized complete block in 1981 and a completely randomized design in 1982. Conventional cultural practices for sunflower production were followed in each experiment (16). Sunflower hybrid 894A (Sigco Sun Research, Breckenridge, MN) was planted at Wyndmere and both Carrington sites, and hybrid 894 (a USDA release) was planted at all other sites. These two hybrids are closely related and do not differ in susceptibility to Sclerotinia wilt (B.D. Nelson, unpublished). Disease incidence in each plant population was monitored at 1–2-wk intervals from plant emergence to maturity and recorded as the proportion of wilted plants. Sclerotinia wilt was recognized by the wilting of leaves and the presence of a basal stem canker (29). The date of anthesis (i.e., when 50% of the stand was in anthesis) was recorded for each experiment.

Determination of inoculum density. The inoculum density (ID) of *S. sclerotiorum* in the upper 16.5 cm of soil in each experimental unit was determined from four soil samples taken in a zigzag pattern with a soil bucket auger (Arts Machine Shop, American Falls, ID) (7.6 cm diameter \times 16.5 cm deep, volume = 800 cm³) within 3 wk after planting. Samples were stored at 5 C until processed. Sclerotia were extracted as described by Holley and Nelson (8). An ID for each site, expressed as the number of sclerotia recovered per 800 cm³ soil, was calculated based on the total number of sclerotia recovered from experimental units. Holley and Nelson (8) have demonstrated that, when studying the effect of ID on this disease, it is acceptable to express ID as sclerotia recovered. They examined the relationship between the incidence of Sclerotinia wilt and the ID of *S. sclerotiorum* using regression analysis and found that the results were the same when ID was expressed as sclerotia recovered or was adjusted for germinability of sclerotia.

Analysis of disease progress data. Disease progress curves from each plant population at each site were plotted arithmetically and subjectively compared to observe similarities or differences that might be apparent in nontransformed data. Data were then

analyzed with three mathematical models: the monomolecular, the logistic, and the Weibull (12,23,28). These models have been used extensively in studies on disease progress (4,15). Data from the replicates were averaged before the fitting of disease progress models. The scale (*b*) and shape (*c*) parameters of the Weibull model were estimated by using a maximum likelihood technique, after the value of the location parameter (*a*) was set at the date when the first disease assessment was made, less 1 day.

The monomolecular and logistic models were performed using the General Linear Model procedure (GLM) of the Statistical Analysis System (SAS) (27). Curve fitting with the Weibull model was done by using the least squares program for nonlinear models (NLIN) of SAS. All computing was performed at the North Dakota State University Computer Center.

Since we intended to statistically compare the rates of disease progress between plant populations at each site and determine whether a correlation could be found between rate of disease progress and inoculum density and environmental factors, it was necessary to choose one model for analysis and presentation of this data. Therefore, the results of curve fitting with the three models were compared using subjective evaluation of plots of residuals and model error mean squares. The Weibull was judged the model that best fit the data.

To compare the rates of disease progress between plant populations at each location, the 95% confidence intervals for the scale (*b*) parameters of the Weibull model were determined and examined for overlap (2). If the confidence intervals overlapped, then the rates of disease progress were not significantly different. The scale parameter (*b*) is inversely proportional to the rate of disease increase (23).

The Weibull model has been used to differentiate epidemiological types based on the variation in the shape parameter (*c*) from 1.0 for monocyclic diseases to 3.6 for polycyclic diseases (23). For this data, the statistical equality of the Weibull *c* values to 1.0 or 3.6 was examined by calculation of a 95% confidence interval about the *c* value (2) for each disease progress curve and by inspection to determine whether the value 1.0 or 3.6 was included in this confidence interval.

Correlation of inoculum density, precipitation, and temperature with disease progress. The relationship between disease progress and ID, precipitation (in millimeters) and temperature as growing degree days (GDD, in C) was examined with regression analysis. The inverses of the Weibull scale parameters for plant populations were regressed against each factor using a standard linear equation $y = \alpha + \beta x$, in which *y* = inverse of the rate parameter and *x* = ID, precipitation, or GDD.

Precipitation and temperature data were collected for the months June through September from five climatological data gathering sites of the National Oceanic and Atmospheric Administration (NOAA) (17,18) closest to the five experiment locations. Growing degree days were calculated by averaging the daily minimum and maximum temperatures and subtracting 7.2 C (the base temperature), then summing the daily GDDs for the period. Regression analyses were performed with data compiled from June through September (the growing season) and July through August (when most Sclerotinia wilt develops).

Seed yields of plant populations. Each experimental unit was harvested and the seed yield determined. Analysis of variance procedures were used to determine whether significant differences in seed yields existed among the plant populations. Fisher's protected least significant difference (27) was used as a *posteriori* multiple comparison test. Yields were expressed as kilogram of seed per hectare at 10% seed moisture.

RESULTS

Disease progress curves. The arithmetic plots of the disease progress curves (Fig. 1) indicated two prominent characteristics of the progress of Sclerotinia wilt of sunflower. One was the absence of disease symptoms during the first 40–60 days after planting, and the other was the wilting of most plants after anthesis. With the exception of the Fargo-Moorhead site, the

average proportion of plants wilting after anthesis for all plant populations at the seven sites was 0.78 (range = 0.63–0.92) (Table 1). At the Fargo-Moorhead site, 0.40–0.47 of the plants in the various plant populations wilted after anthesis.

Evidence was found at six sites that plant population affected the rate of disease progress (Table 2). No consistent relationship was found, however, between plant population and rate of disease progress. Lower plant populations, for example, did not consistently have lower *b* values. At four sites, there was at least one population with a *b* value significantly different from the *b* values of the other plant populations: 37.0×10^3 plants per hectare at Carrington Dryland, 61.7×10^3 plants per hectare at Fargo-Moorhead, 37.0×10^3 plants per hectare at Carrington Irrigated, and 74.1×10^3 plants per hectare at Northwood North. At two other sites one population had a *b* value significantly different from the values for one or two of the other plant populations: the *b* value for the 61.7×10^3 plants per hectare at the Northwood south site was significantly different from two (49.4 and 74.1×10^3 plants per hectare) out of the other three plant populations, and the *b* value for the plant population of 49.4×10^3 plants per hectare at Galesburg east was significantly different from the *b* value for plant populations of 37.0×10^3 plants per hectare but not that for 61.7×10^3 plants per hectare.

Differentiation of epidemiological types. Of the 25 disease progress curves, five had no inflection point (from Galesburg east and west sites) ($c = 1$), 17 had an inflection point but were asymmetrical (maximum rate at $y \leq 0.5$) ($1 < c < 3.6$), and three curves (all from the Wyndmere site) had *c* values that overlapped or nearly overlapped both 1 and 3.6 (Table 2). The wide confidence interval for *c* was due to the low final level of disease.

Correlation of inoculum density, precipitation, and temperature with rate of disease progress. Data from three populations, 37.0 , 49.4 , and 61.7×10^3 plants per hectare, were used to examine the relationship between disease progress and ID, precipitation, and temperature (Table 1). For the three plant populations, a significant linear correlation was found between the rate of disease progress (the inverse of the scale *b* parameter of the Weibull model) and the inoculum density of sites ($r = 0.84$, 0.87 , and 0.88 [$P > 0.05$] for 37.0 , 49.4 , and 61.7×10^3 plants per hectare, respectively). No significant linear correlations were found, however, between the rate of disease progress and precipitation or GDD for the periods June through September or July through August.

Seed yields. ANOVA indicated no significant effect of plant population on seed yield in seven of the eight sites (Table 1).

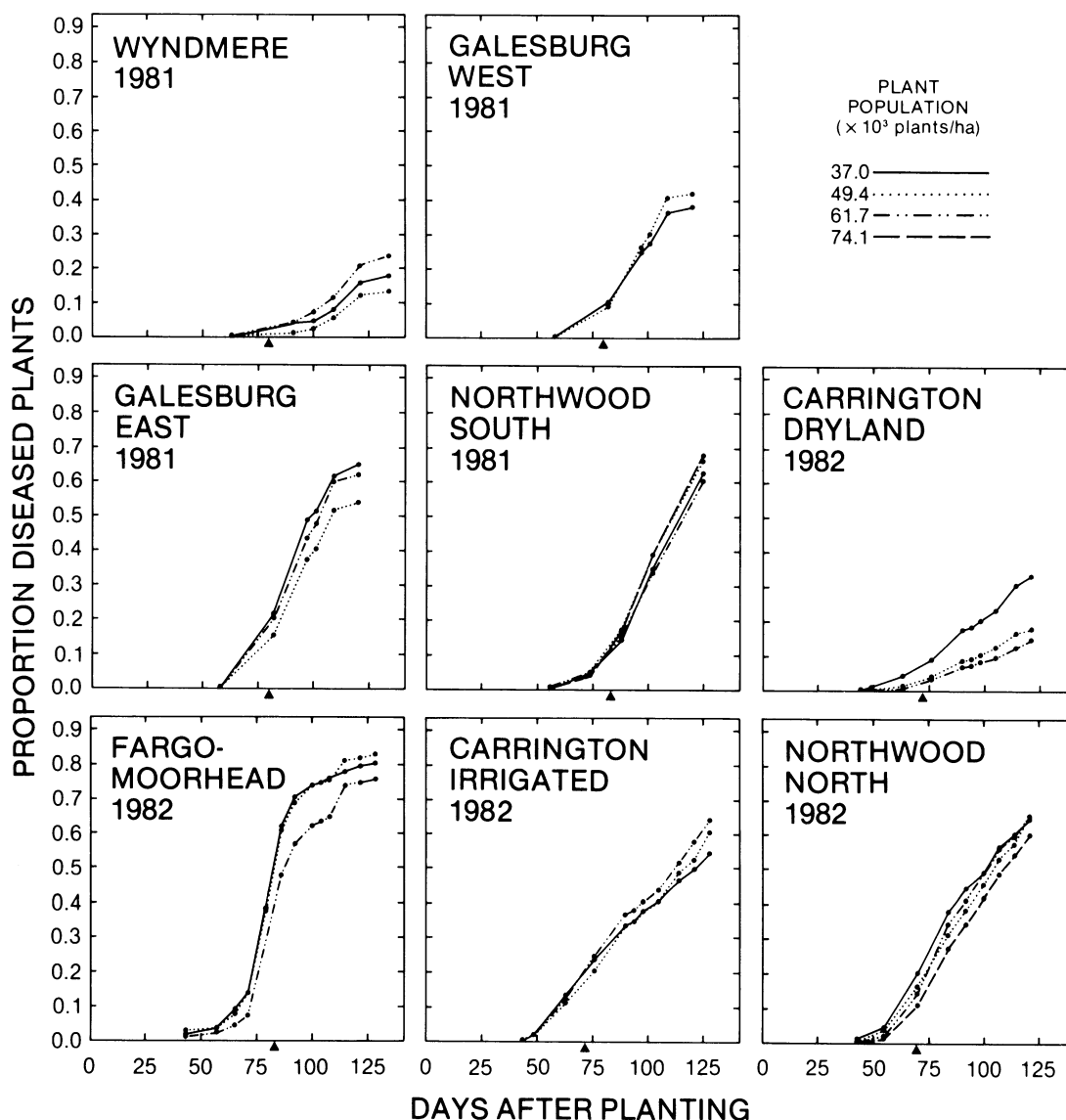


Fig. 1. Disease progress (proportion of wilted plants) of *Sclerotinia* wilt of sunflower at different plant populations at eight sites with different inoculum densities. Site inoculum densities as sclerotia per 800 cm^3 of soil were as follows: Wyndmere, 0.13; Galesburg west, 0.25; Galesburg east, 0.52; Northwood south, 1.67; Carrington dryland, 0.11; Fargo-Moorhead, 0.78; Carrington irrigated, 1.19; and Northwood north, 1.46. The arrow indicates the time of anthesis.

Only at Northwood south were differences in yield detected, but this site had substantial lodging of seed-bearing plants due to a September windstorm. The lodging was greatest (35% lodged plants) at the highest plant population, which had the lowest yield, and least (14.8%) in the lowest plant population, which had the greatest yield.

DISCUSSION

The two prominent characteristics of the progress of Sclerotinia wilt of sunflower, the absence of disease symptoms during the first 40–60 days after planting and the wilting of most plants after anthesis, have also been noted by other researchers in North America (6,7,29) and are consistent with our observations of this disease in growers' fields since 1980 (21). Although the reasons for these characteristics are not clear, there are three plausible explanations. One is that the susceptibility of sunflower tissues to *S. sclerotiorum* increases after flowering. Noyes and Hancock (22) have reported that whole leaf cells of four sunflower genotypes became more susceptible to damage from oxalic acid after flowering began. Oxalic acid is a toxin produced by *S. sclerotiorum* in sunflower and other crops (14,22). A second explanation, proposed by Nelson (19), and supported by measurements of root growth, is that a greater root volume at, and decreased root activity after, flowering favors infection (through increased root contact with sclerotia) and dispersal of the pathogen between plants, which results in most disease occurring after flowering. Such an explanation would agree with Huisman's (11) concepts regarding the interrelation of root growth dynamics to the epidemiology of root-invading fungi. The third explanation is that the expression of disease symptoms is environmentally con-

ditioned and the critical factors (or accumulation of them) only occur in July through September. There is no evidence yet to support this last explanation.

Plant population within the commercial range used in North Dakota does not appear to be an important factor affecting disease progress of Sclerotinia wilt. Although some differences were found in the rates of disease progress between plant populations as indicated by the Weibull scale parameters, those differences had no apparent effect on seed yields. The seed yield differences at the Northwood south site were directly due to the severe lodging and not to differences in disease progress. Also, lower rates of disease progress were not associated with lower plant populations, as might be expected. Burdon and Chilvers (3), in their review article on host density as a factor in plant disease ecology, state that "raising host density will increase infection rates and lowering host density will reduce infection rates." However, relatively few studies have been made that have examined the effect of plant population on the progress of soilborne diseases (3). Koch et al (13) studied the effects of plant density on the progress of Phymatotrichum root rot in cotton and found no consistent relationships between rate of progress and overall plant density within plots or rows. They evaluated an even wider range of plant densities than we used in this study. As pointed out by Koch et al (13), each unique combination of initial and maximum disease incidence results in a unique rate of disease progress, thus confounding trends in the rate with respect to plant density. Further studies may reveal that the above statement by Burdon and Chilvers is not universally true for soilborne diseases.

Hoes and Huang (7), in Manitoba, reported that the use of plant spacing of 36 cm or greater and plant populations of 26–49 × 10³ plants per hectare would minimize yield loss due to

TABLE 1. Inoculum density, incidence of Sclerotinia wilt, seed yield, precipitation, and growing degree days at eight different sites planted to various populations of sunflowers

Year/ site	Sclerotia per 800 cm ³ of soil	Plant population (× 10 ³ plants/ha) ^a	Disease incidence in proportion		Percent of disease occurring after anthesis	Seed yield (kg/ha) ^c	Precipitation (mm) ^d		Degree growing days (C)	
			At anthesis ^b	At maturity			June- Sept.	July- Aug.	June- Sept.	July- Aug.
1981										
Wyndmere	0.13	37.0	0.03	0.18	83	977.5	290.8	161.8	1,461	888
		49.4	0.01	0.13	92	879.0				
		61.7	0.03	0.24	87	847.0				
Galesburg west	0.25	37.0	0.09	0.38	76	1,619.5	319.6	147.1	1,378	867
		49.4	0.09	0.42	79	1,605.8				
		61.7	0.19	0.65	71	852.5				
Galesburg east	0.52	37.0	0.13	0.54	76	984.4	319.6	147.1	1,378	867
		49.4	0.19	0.62	69	920.3				
		61.7	0.12	0.63	81	1,029.5				
Northwood south	1.67	37.0	0.12	0.66	82	802.4	362.4	154.9	1,242	787
		49.4	0.12	0.60	80	769.0				
		61.7	0.12	0.68	82	551.7				
		74.1								
						LSD = 145.2				
1982										
Carrington dryland	0.11	37.0	0.08	0.33	76	2,041.0	236.0	130.8	1,234	801
		49.4	0.03	0.18	83	2,247.9				
		61.7	0.03	0.15	80	2,111.8				
Fargo-Moorhead	0.78	37.0	0.49	0.81	40	359.6	165.0	95.6	1,289	847
		49.4	0.49	0.83	41	353.6				
		61.7	0.40	0.76	47	427.5				
Carrington irrigated	1.19	37.0	0.20	0.54	63	1,793.8	466.0	360.8	1,234	801
		49.4	0.17	0.60	72	1,734.3				
		61.7	0.20	0.64	69	1,776.1				
Northwood north	1.46	37.0	0.19	0.65	71	1,232.1	234.6	129.2	1,083	679
		49.4	0.16	0.65	75	1,341.1				
		61.7	0.14	0.66	79	1,315.2				
		74.1	0.11	0.60	82	1,257.6				

^aRow spacing was 76.2 cm at all sites except the Galesburg east, Galesburg west, and Fargo-Moorhead sites, where it was 91.4 cm.

^bAnthesis was defined as 50% of the stand in anthesis.

^cAnalysis of variance indicated no significant effect of plant population ($P > 0.05$) on yields at all sites except Northwood south. Seed yield means for Northwood south were compared with Fisher's least significant difference (LSD) ($P = 0.05$).

^dData from the Carrington irrigated site includes 230 mm of water added in July and August by furrow irrigation.

Sclerotinia wilt because greater numbers of wilted, but still productive, plants (i.e., plants that produced seed) would be found. They ascribed this maintenance of some yield potential at lower populations to reduced competition with neighboring plants for nutrients and moisture. Our study included similar spacing and plant populations (35 cm spacing and 37×10^3 plants per hectare), but the results did not corroborate those of Hoes and Huang (7); lower plant populations did not reduce losses in seed yield. Plants in lower populations, however, do have more robust stems than those in higher populations and are less susceptible to lodging, even when decayed by *S. sclerotiorum* (B. D. Nelson, unpublished).

Disease progress curves for Sclerotinia wilt could not be categorized as either of the monomolecular or logistic type when analyzed by the Weibull model. The shape parameter values generally were closer to 1 than to 3.6, suggesting that disease progress was similar to the monomolecular type, i.e., maximum absolute rate of increase near the beginning of the epidemic (23). Sclerotinia wilt is, by Vanderplank's definition, a polycyclic disease (= compound interest disease [28]), because the pathogen spreads from plant to plant along the root system (10,29). This mode of spread is, however, a constraint on the rate of disease progress since the fungus spreads from an infected plant only to adjacent plants within the row and must traverse the root system of those adjacent plants before it can infect two more plants, and so on. The fungus does not spread between rows in the typical 76–91 cm row spacing used in commercial sunflower production in North Dakota (10,16). This two-dimensional spread is in contrast to foliar fungal pathogens, the spores of which can spread in multiple directions, resulting in disease progress curves characteristic of polycyclic diseases. Pfender and Hagedorn (25) have also pointed out the epidemiological importance of the constrained spread of *Aphanomyces* on pea roots. The mode of

spread of *S. sclerotiorum* on sunflower may explain why the disease progress curves generated by the Weibull function are intermediate between monocyclic and polycyclic. Our data support Pfender's argument (24) that the nature of a disease cycle should not be inferred from disease progress curves, but rather should be determined by direct investigation of the biology of the disease.

The positive correlation between inoculum density and rate of disease increase is in agreement with other similar studies on the epidemiology of soilborne diseases (5,25,26). Precipitation and temperature, however, were not correlated with disease progress, indicating that inoculum density is one of the most important factors affecting progress of Sclerotinia wilt. Environmental factors obviously affect disease progress, but how and to what magnitude are unknown. Our results concerning the effect of environment on disease progress should be considered preliminary since few site-years were studied. Further research is needed to understand the effects of temperature and precipitation on the epidemic.

The effect of Sclerotinia wilt on seed yield merits further mention. Few researchers have attempted to quantify the effect of this disease on seed yield (6,7); indeed, the relationship between seed yield and disease incidence was not previously investigated. Sclerotinia wilt generally reduces whole-plant yields by about 50% (29). In our study, the severity of this disease was clearly demonstrated in sites with high inoculum densities. For example, the seed yields at the Fargo-Moorhead and Carrington irrigated sites, when compared to yields of adjacent healthy sunflower plantings, were estimated to have been reduced 80 and 50%, respectively, by Sclerotinia wilt. Such losses emphasize the importance of avoiding sunflower production in fields with high densities of sclerotia of *S. sclerotiorum*.

This information on the epidemiology of Sclerotinia wilt has

TABLE 2. Analysis of disease progress of Sclerotinia wilt of sunflower with the Weibull model

Year/site	Plant population ($\times 10^3$ plants/ha) ^d	Mean square error	R^2	Parameter estimates ^b					
				Scale			Shape		
				Lower b	Estimated b	Upper b	Lower c	Estimated c	Upper c
1981									
Wyndmere	37.0	0.000243	0.960	79.76	164.02	248.29	0.00	1.90	3.89
	49.4	0.000236	0.940	50.71	172.60	294.40	0.00	2.14	5.11
	61.7	0.000277	0.974	98.61	139.23	179.84	0.48	1.89	3.21
Galesburg west	37.0	0.000697	0.975	76.51	100.15	123.79	0.41	1.40	2.38
	49.4	0.001288	0.964	69.12	86.46	103.80	0.41	1.58	2.74
Galesburg east	37.0	0.001530	0.981	52.85	56.30	59.76	0.83	1.49	2.15
	49.4	0.001245	0.977	62.56	69.68*	76.79	0.72	1.50	2.28
	61.7	0.001214	0.983	55.93	59.72	63.51	0.87	1.50	2.14
Northwood south	37.0	0.000225	0.997	67.73	69.86	71.99	1.88	2.29	2.70
	49.4	0.000261	0.997	64.68	66.70	68.72	1.83	2.24	2.64
	61.7	0.000136	0.998	70.11	72.04*	73.98	1.86	2.18	2.51
	74.1	0.000326	0.997	63.41	65.46	67.50	1.88	2.34	2.80
1982									
Carrington dryland	37.0	0.000060	0.996	125.51	134.97*	144.43	1.37	1.61	1.86
	49.4	0.000019	0.996	171.77	197.67	223.58	1.36	1.69	2.02
	61.7	0.000015	0.995	186.83	229.27	271.70	1.28	1.69	2.10
Fargo-Moorhead	37.0	0.007507	0.934	50.85	53.81	56.76	1.30	1.91	2.51
	49.4	0.005947	0.950	51.07	53.60	56.13	1.45	2.01	2.58
	61.7	0.005013	0.950	60.34	63.33*	66.33	1.44	2.00	2.56
Carrington irrigated	37.0	0.000073	0.998	104.17	107.65*	111.14	1.02	1.12	1.22
	49.4	0.000184	0.996	90.80	93.81	96.83	1.25	1.41	1.57
	61.7	0.000226	0.996	84.79	87.44	90.09	1.20	1.36	1.52
Northwood north	37.0	0.000359	0.994	72.13	74.42	76.71	1.22	1.43	1.63
	49.4	0.000072	0.999	76.18	77.21	78.23	1.55	1.66	1.77
	61.7	0.000461	0.993	71.69	73.87	76.05	1.43	1.69	1.95
	74.1	0.000175	0.997	79.62	81.47*	83.32	1.61	1.81	2.00

^a Row spacing was 76.2 cm at all sites except Galesburg east, Galesburg west, and Fargo-Moorhead sites where it was 91.4 cm.

^b The data include the 95% confidence intervals for the scale (b) and shape (c) parameters. For plant populations at a specific site, if the confidence intervals of the scale parameters overlapped, then the rates of disease progress were considered not to be significantly different. Plant populations with b values significantly different from one or more of the other populations at a site are indicated by an asterisk. If a shape parameter value of 1.0 or 3.6 is included within the confidence intervals, the disease progression type would be considered monomolecular or logistic.

some practical applications of disease control. Monitoring of sunflower fields for disease, coupled with crop rotation to non-susceptible hosts is recommended to prevent buildup of sclerotia in soil (21). Since all infected plants can potentially contain sclerotia, which are returned to the soil during tillage, it is important to obtain a good estimate of disease incidence in the field before making decisions on rotation. The disease progress curves indicate that monitoring should be conducted as late in the season as possible to identify diseased plants. If monitoring is performed too early, for example, within several weeks after flowering, disease incidence would be substantially underestimated in most fields, even those with low inoculum densities where growers might plant sunflower.

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