Characterization of *Puccinia polysora* Epidemics in Pennsylvania and Maryland

R. N. Raid, S. P. Pennypacker, and R. E. Stevenson

Former graduate research assistant, associate professor, and research assistant, Department of Plant Pathology, The Pennsylvania State University, University Park 16802. Present address of the first author: Everglades Research and Education Center, University of Florida, Belle Glade 33430.

A portion of the first author's Ph.D. dissertation submitted to The Pennsylvania State University.

Contribution No. 1607, Department of Plant Pathology, the Pennsylvania Agricultural Experiment Station. Authorized for publication as Journal Series Paper No. 7553.

This research was supported in part by the U.S. Department of Agriculture under agreement 58-32U4-3-517.

We wish to thank Dr. J. S. Melching USDA, Plant Disease Research Laboratory, Frederick, MD, for technical assistance and support.

Accepted for publication 28 October 1987.

ABSTRACT

Raid, R. N., Pennypacker, S. P., and Stevenson, R. E. 1988. Characterization of *Puccinia polysora* epidemics in Pennsylvania and Maryland. Phytopathology 78:579-585.

Southern rust of corn epidemics, caused by *Puccinia polysora*, were initiated in Maryland and Pennsylvania by establishing a line source of inoculum across the width of 0.8-ha (64 × 128 m) field plots. The rust was most severe on lower leaves, and the severity generally decreased with successive leaf positions. Average apparent infection rates ranged from 0.110 to 0.268 and were greater ($p = 0.05$) at the Maryland study site than in Pennsylvania during both the 1983 and 1984 growing seasons. The velocity of spread ranged from 1.6 m/day in Pennsylvania to 9.1 m/day in Maryland. Grain yields were reduced 17.7 and 39.1%, respectively, at the Pennsylvania and Maryland field sites. These maximum reductions occurred during 1984 at a 3-m distance from the inoculum source. The yield reductions were significantly related to the area under the disease progress curves as calculated from time t_0 to 35 days after anthesis. Environmental data suggest temperature may be the factor most limiting to development of southern rust of corn in northern areas of the United States.

Of the three rusts that occur on corn (*Zea mays* L.) worldwide, southern rust of corn, caused by *Puccinia polysora* Underw., has been reported as the most destructive (15). *P. polysora* differs from the common rust pathogen, *P. sorghi* Schw., in that the resulting disease becomes progressively more severe as the plant develops, resulting in premature desiccation of plant tissues (27). Yield losses of 45–50% have been described (15,22), with destruction being greatest in late-season corn plantings (26).

Although southern rust was not identified on corn in the Western Hemisphere until 1941, its earlier presence has been ascertained by examining herbarium specimens dating back to 1879 (5). In 1949, an outbreak occurred in western Africa, rapidly spread across the continent in subsequent years, and caused significant yield losses (20). Breeding programs were rapidly initiated to incorporate resistance from Mexican and Central American lines (29). However, Vanderplank (33) attributed the rapid abatement of southern rust epidemics among open-pollinated African lines to selection pressure within corn populations with a broad genetic base.

In the United States, southern corn rust generally has been regarded as a minor pathogen of corn; however, epidemics were reported in 1972, 1973, 1974, and 1979 (6,13,25,26). The disease has been reported as far north as Wisconsin (18) and northern Indiana (25) and as far west as Kansas (28). However, its occurrence in northern areas was usually late in the season and therefore was of little consequence. The sudden appearance of these epidemics coincided with the increase in double cropping of corn in the lower Mississippi River Valley and on the coastal plain of the southeastern United States (6,34). The first crop is normally planted in February and harvested in June, while the second crop is planted in late June and harvested in October. This cropping practice may provide conditions for development of large quantities of inoculum to infect corn in the northern corn belt at an early growth stage.

Nine physiologic races of *P. polysora* have been described (21,24,30). Although resistance has been reported (7,27), the majority of commercial hybrids currently grown in the United States are rated as susceptible (23). *P. polysora* is more restrictive in the temperature and moisture requirements necessary for infection than is *P. sorghi* (15). However, favorable temperature and moisture conditions do exist throughout most of the U.S. corn production area (10).

This study was undertaken to evaluate the potential for disease development of southern corn rust in northern areas and to determine its effect on yield.

MATERIALS AND METHODS

Planting. Field studies were conducted at two locations during the 1983 and 1984 growing seasons to monitor crop growth and development of southern corn rust epidemics. Susceptible full-season corn hybrids, Pioneer 3369A and Pioneer 3358, were planted during 1983 and 1984, respectively. The 0.81-ha field plots (128 × 64 m) were located at the Plant Disease Research Laboratory at Frederick, MD (39° 26′42″N, 77° 25′32″W) and The Pennsylvania State University's Agricultural Research Center at Rock Springs Centre County, PA (40° 42′53″N, 77° 56′25″W). Plots were oriented with the long dimension parallel to the direction of the prevailing wind. Rows were spaced at 0.76 m and oriented perpendicular to the prevailing wind. Fields were overplanted and thinned to approximately 61,750 plants per hectare. During 1983 and 1984, sowing dates were 3 and 14 May at Frederick and 18 and 17 May at Rock Springs, respectively. Soil was classified as silty loam at both field sites. Lime and fertilizer were applied as recommended by soil analyses performed at The Pennsylvania State University's Merkle Laboratory. Starter fertilizer (10-30-10 NPK) was applied at a rate of 224 kg/ha at time of planting, except at Frederick in 1983, when 448 kg/ha was applied. Insects were controlled with timely applications of carbaryl (1.12 kg a.i./ha) at both sites. In addition, carbofuran (1.12 kg a.i./ha) was broadcast into the whorl for control of second-generation corn borer at Rock Springs during 1984.

Experimental units consisted of four- and three-row blocks for 1983 and 1984, respectively. The blocks were located 6, 26, 46, 87, and 106 m downwind from the inoculum source during 1983 and 3, 12, 36, and 72 m from the inoculum source during 1984. In addition, one block was located 12 m upwind of the inoculum source during 1984. Each three- and four-row block was divided into four subplots 9.7 m in length, and these observational subplots were 5 m apart within each experimental unit.

Sampling and growth measurements. Four plants from each subplot (one from each row) were selected for growth measurements and disease assessments throughout the 1983 growing season. In 1984, six plants (two from each row) were measured and assessed. One subplot at each distance was sprayed weekly with Dithane M-45 80WP (zinc ion manganese ethylene bisdithiocarbamate) (1.68 kg a.i./ha) and served as a rust-free control in 1983. Two subplots were sprayed at each distance during 1984. Fungicide applications were made with a backpack sprayer to optimize foliar coverage and minimize plant injury.

The length, width, and percentage of green area of each leaf were determined at weekly intervals for all sampled plants. A 25-leaf sample at each leaf position was also obtained from non-subplot areas to determine leaf length, width, and area as measured by a leaf area meter (Li-Cor, Inc., Lincoln, NE). Regression analysis was then used to develop equations for estimating leaf area from length and width dimensions (14,31).

Establishment of disease. Urediospores of a pathogenic isolate of *P. polysora* were provided by Dr. J. Stanley Melching of the USDA Plant Disease Research Laboratory at Frederick, MD. The isolate was obtained originally from rusted leaves in a commercial corn field near Frederick. Plants of the same hybrids used in the field were greenhouse grown in 11-cm pots and inoculated at the six- to seven-leaf stage. Seventy-five infected plants were transported to the field before pustule break and were transplanted at 85-cm intervals within a single row across the field width on 13 June 1983 and 23 June 1984 at Frederick, and on 2 July 1983 and 24 June 1984 at Rock Springs. The transplants were hand-watered for several days to limit transplant shock. The line source of inoculum was situated approximately 10 and 20 m from the western edge of the field plots during 1983 and 1984, respectively. Erumpent pustules were observed on inoculated source plants 2–4 days after transplanting in the field. The inoculum source plants were assessed for disease after pustule break to determine the amount of initial inoculum available for development of rust epidemics. This initial assessment, made at growth stage 2 to 3 (8), disclosed the source plants averaged 426 and 376 pustules per plant in 1983 and 514 and 499 in 1984 at Frederick and Rock Springs, respectively.

Disease measurement. Plants selected for nondestructive growth measurements were examined weekly for southern corn rust. Disease measurements began 9–12 days after exposure of field plots to inoculum and continued until harvest. Measurements were made proceeding from the least to most severely rusted areas, and care was taken to minimize spread through physical contact with pustules. All leaves of plants in treatment and sprayed subplots were examined in a similar manner each week to minimize effects caused by differences in handling (2). Direct pustule counts were made where densities did not exceed 500 pustules per leaf. Because pustules of *P. polysora* are found predominantly on the adaxial surface of the leaf blade (4), pustule counts were performed only on this surface. When densities exceeded approximately 500 pustules per leaf, a template marked with four 1-cm^2 transparent areas was placed at three leaf positions: the base, middle, and tip. Pustules within the four areas were counted to estimate the average pustule density. This average value and the estimated leaf area were used to calculate pustules per leaf.

Thirty-five leaves exhibiting 500 or more pustules were collected from non-subplot areas to confirm pustule density calculations. Direct counts of pustule populations and leaf areas as measured by a leaf area meter provided actual pustule densities. These actual densities were correlated with estimates produced by the method previously outlined.

Final yield. Grain was harvested after black layer formation. Ears were hand-harvested from 20 plants per subplot-row, dried, and shelled mechanically. Grain yield was calculated as metric tons of shelled corn per hectare at 15.5% moisture.

Environmental monitoring. Environmental sensors were located in close proximity to the fields. At Frederick, a Campbell CR21 micrologger (Campbell Scientific Inc., Logan, UT) was used for data collection, while at Rock Springs an existing environmental acquisition system was used (19). Meteorological measurements included: air temperature, relative humidity, wind speed, wind direction, radiation, dew period, and rainfall. Hygrothermographs served as partial backup units.

Data analysis. Linear regression analysis using actual number of pustules as the independent variable and estimated number of pustules as the dependent variable was performed with the line being forced through the origin (Fig. 1). A coefficient of determination of 0.98 for the 35 leaves sampled indicated a strong correlation between the two variables. The regression coefficient of 1.07 indicated that, on the average, estimated pustule counts underestimated actual pustule densities by 7%.

Results from these and other field experiments with southern corn rust on these particular hybrids (J. S. Melching, *personal communication*) indicate that pustule densities may reach a maximum of approximately 200,000 pustules/plant before total foliar desiccation and death of the plant. This value was used as a basis for converting pustule counts to disease severity expressed as a proportion. Because leaf area was maximized before significant disease development and leaf area index was not different between sites, leaf area was not a factor in determining disease severity. These calculated proportions (0–1.0) of disease were used to estimate apparent infection rates (32) and areas under disease progress curves (AUDPC). The areas with units of 'disease proportion-days' were estimated by integrating the disease progress curves from time t_0 to 35 days subsequent to anthesis. Apparent infection rates, *r*, were calculated for each distance at both locations and for both years. In some instances, disease was detected at the more distant locations 2 to 3 wk later than in blocks close to the inoculum source. To facilitate comparisons, apparent infection rates were calculated using linear regression analysis, over a time period that started when disease was in excess of 0.01% and ended when disease had reached a maximum, or plants were harvested for yield. Differences in infection rates at the various distances were tested for significance at the 0.05 level for each year and location using Student's *t*-test.

A measure of the disease gradient, *g*, defined as the slope of the logit of disease severity regressed on distance (16), was calculated at various times for each location and year. All *g* values are based on data obtained downwind from the original inoculum source. As suggested by Minogue and Fry (17), data from the subplots closest to the inoculum source were not used in calculating *g*, except early in the epidemic. A single value of *g* was obtained for each location

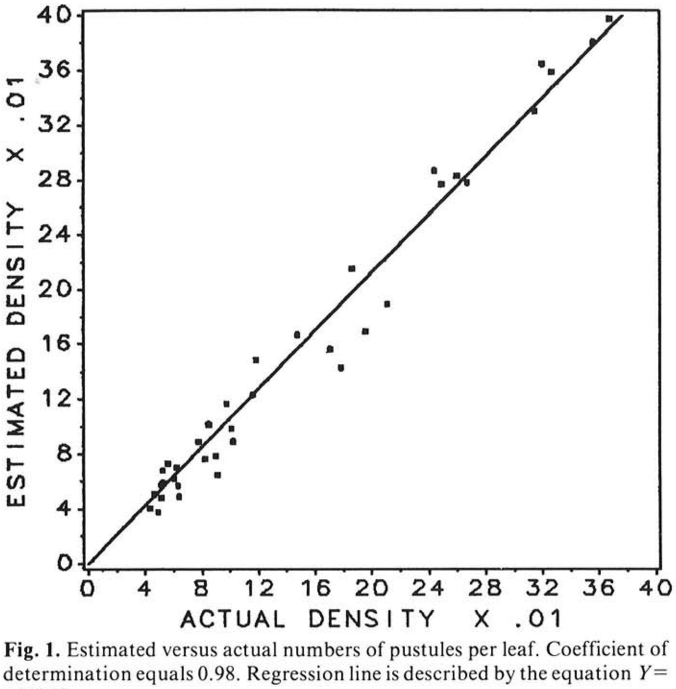

Fig. 1. Estimated versus actual numbers of pustules per leaf. Coefficient of determination equals 0.98. Regression line is described by the equation $Y = 1.07(X)$.

and year by pooling data from various times after gradients had stabilized; i.e., slopes were not significantly different. In addition, *r* values were calculated for the corresponding time period and for the range of distances used in calculating the pooled disease gradient. These data were likewise pooled for calculations of common *r* values for each location and year. A parameter representing velocity of spread, *v*, was calculated using the gradient and rate parameters (16).

RESULTS

Establishment of disease. Secondary infections in experimental blocks were observed a minimum of 14 days after pustule break on the source plants. As expected, secondary infections were most numerous on plants close to the line source of inoculum. Disease progress curves with disease severity represented as number of pustules per plant are illustrated in Figure 2A and B for Frederick and Fig. 2C and D for Rock Springs during 1983 and 1984, respectively. Decreases in numbers of pustules late in the season are due to foliar desiccation. Southern corn rust was more severe at Frederick than at Rock Springs, and greater pustule densities developed at both locations during 1984 than in 1983. Three meters from the inoculum source there were 75,000 pustules/plant at Rock Springs (Fig. 2D), in contrast to 186,000 pustules/plant at Frederick during 1984 (Fig. 2B). Differences in disease severity between locations were more pronounced in 1983, with maximum severities of 199,000 and 25,000 pustules/plant at the 6-m distance in Frederick and Rock Springs, respectively. Although rust pustules were observed in all nonsprayed subplots, severities did not exceed 25,000 pustules/plant in subplots at distances greater than 12 m from the inoculum source at Rock Springs. However, at Frederick severities exceeded 25,000 pustules/plant in all subplots at distances of 72 m or less.

Apparent infection rates characterizing epidemics at various distances from the original inoculum source are shown in Table 1. In general, *r* values were higher at Frederick than at Rock Springs, with a maximum value of 0.274 units/day being recorded at Frederick during 1984. This compares to a maximum of 0.195 at

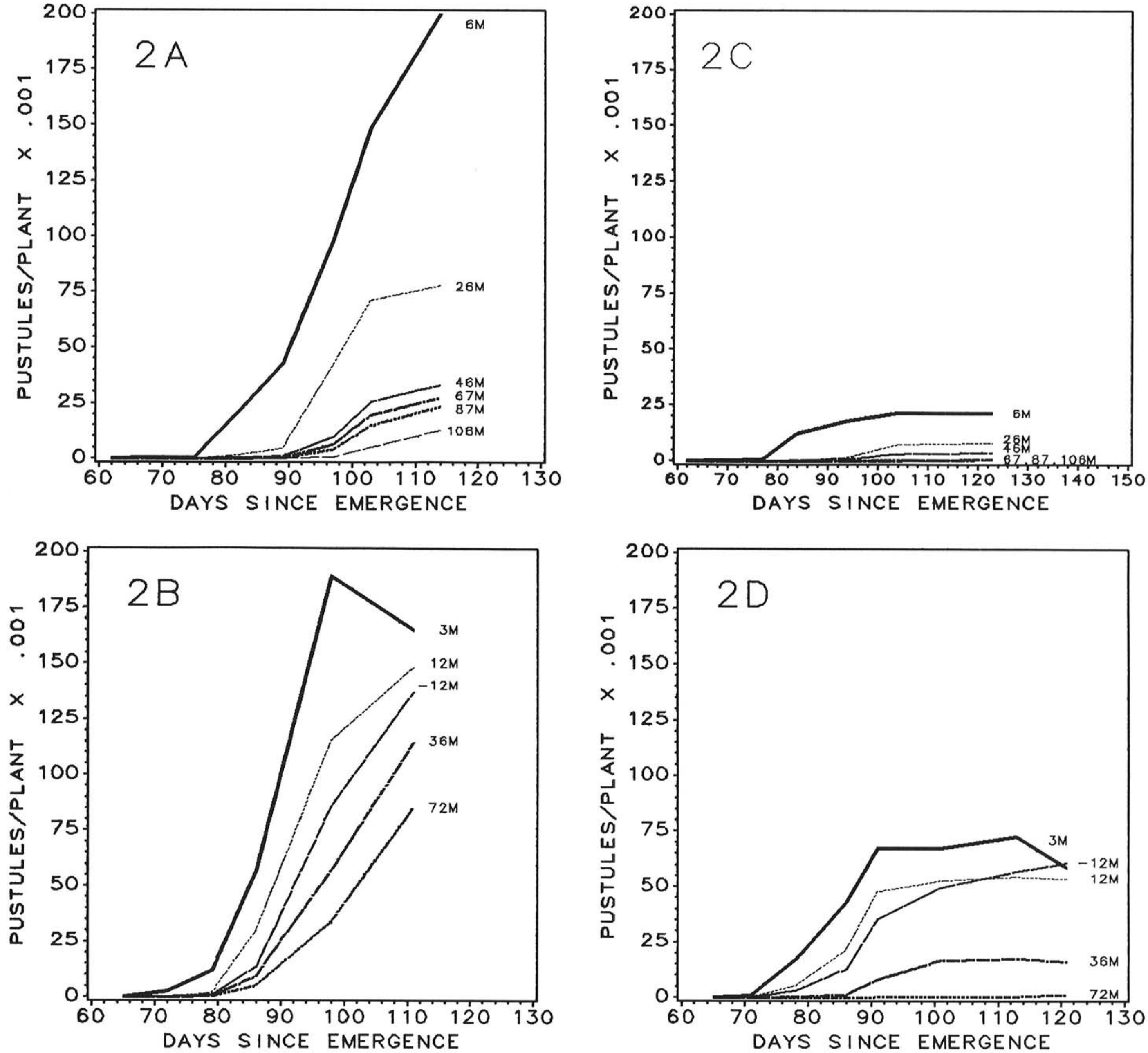

Fig. 2. Progression of southern corn rust at various distances from a line source of inoculum. Curve numbers represent distances (m) measured perpendicularly from the inoculum source. **A,** Frederick, MD, in 1983. **B,** Frederick, MD, in 1984. **C,** Rock Springs, PA, in 1983. **D,** Rock Springs, PA, in 1984.

Rock Springs. Although r values tended to be highest in blocks closest to the inoculum source, differences between infection rates with respect to distance were significant only at Rock Springs during 1984 at the 3- and 72-m distances. All other differences among infection rates with respect to distance were insignificant. Data were then pooled from all distances for each year and location, with the exception of 1984 data from the 72-m blocks at Rock Springs for the calculation of common r values. Values of 0.205 and 0.221 units/day for Frederick during 1983 and 1984, respectively, are not significantly different. At Rock Springs, the rates of 0.096 and 0.153 for 1983 and 1984, respectively, are different ($p = 0.05$). Differences in r occurred between locations during both 1983 and 1984.

The fungicide used to control southern rust was very effective. Even at the 3-m distance at Frederick, where inoculum pressure was extremely high (186,000 pustules/plant in nonsprayed subplots) disease severity of sprayed plants never exceeded 1,000 pustules/plant. Because of the general lack of disease progress, r values were not calculated for sprayed controls.

Estimated values of g, r, and v are presented in Table 2 for each location and year. The time and distance over which r and g were calculated are also listed. Although sprayed subplots were located throughout the field plot, the total area sprayed was relatively small, representing 1.2 and 1.5% of the total area in 1983 and 1984, respectively. It is unlikely that these small sprayed areas exerted a significant influence on disease spread. Disease gradients were steeper at Rock Springs than at Frederick during both 1983 and 1984. In general, gradients were initially steep and flattened over time. The rate of slope leveling was initially rapid, slowed with spread of the pathogen to all distances, and eventually reached a steady state, as predicted by Minogue and Fry (17). Velocity of spread ranged from 9.1 m/day at Frederick to 1.6 m/day at Rock Springs during 1984.

Disease development on individual plants. Disease development on individual plants was examined by comparing pustule densities (pustules per square centimeter) at various leaf positions on the plant (Fig. 3A and B). Because of foliar desiccation, maximum

TABLE 1. Apparent infection rates (r) for disease progress at various distances from an inoculum source at Frederick, MD, and Rock Springs, PA, during 1983 and 1984

Location (year)	Distance (m)	Time[a] (days)	r[b] units/day)	R^2
Frederick	6	68–103	0.232	0.94
(1983)	26	75–114	0.204	0.85
	46	75–114	0.197	0.91
	67	75–114	0.210	0.93
	87	75–114	0.200	0.95
	106	82–114	0.183	0.97
Frederick	3	59–98	0.274	0.99
(1984)	12	72–111	0.210	0.90
	−12	72–111	0.233	0.92
	36	72–111	0.200	0.89
	72	79–111	0.189	0.90
Rock Springs	6	68–103	0.146	0.81
(1983)	26	76–122	0.117	0.81
	46	76–122	0.089	0.68
	67	83–122	0.083	0.98
	87	83–122	0.082	0.91
	106	83–122	0.067	0.95
Rock Springs	3	65–101	0.195	0.84
(1984)	12	71–113	0.163	0.62
	−12	71–113	0.152	0.74
	36	78–113	0.125	0.79
	72	78–113	0.077	0.90

[a] Days since emergence during which the apparent infection rate was calculated.

[b] Apparent infection rate calculated using the logistic model of Vanderplank (32).

TABLE 2. Estimated values of the gradient parameter (g), apparent infection rate (r), and velocity of spread (v) for Frederick, MD, and Rock Springs, PA, during 1983 and 1984

Location (year)	g (units/m)	Distance[a] (m)	r (units/day)	Time[b] (days)	v (m/day)
Frederick (1983)	0.040	26–106	0.237	82–103	5.918
Frederick (1984)	0.029	12–72	0.268	79–98	9.119
Rock Springs (1983)	0.044	26–106	0.110	83–103	2.520
Rock Springs (1984)	0.087	12–72	0.139	78–101	1.604

[a] Distance from the original inoculum source over which the gradient parameter (g) was calculated.

[b] Days since emergence over which the rate parameter (r) was calculated.

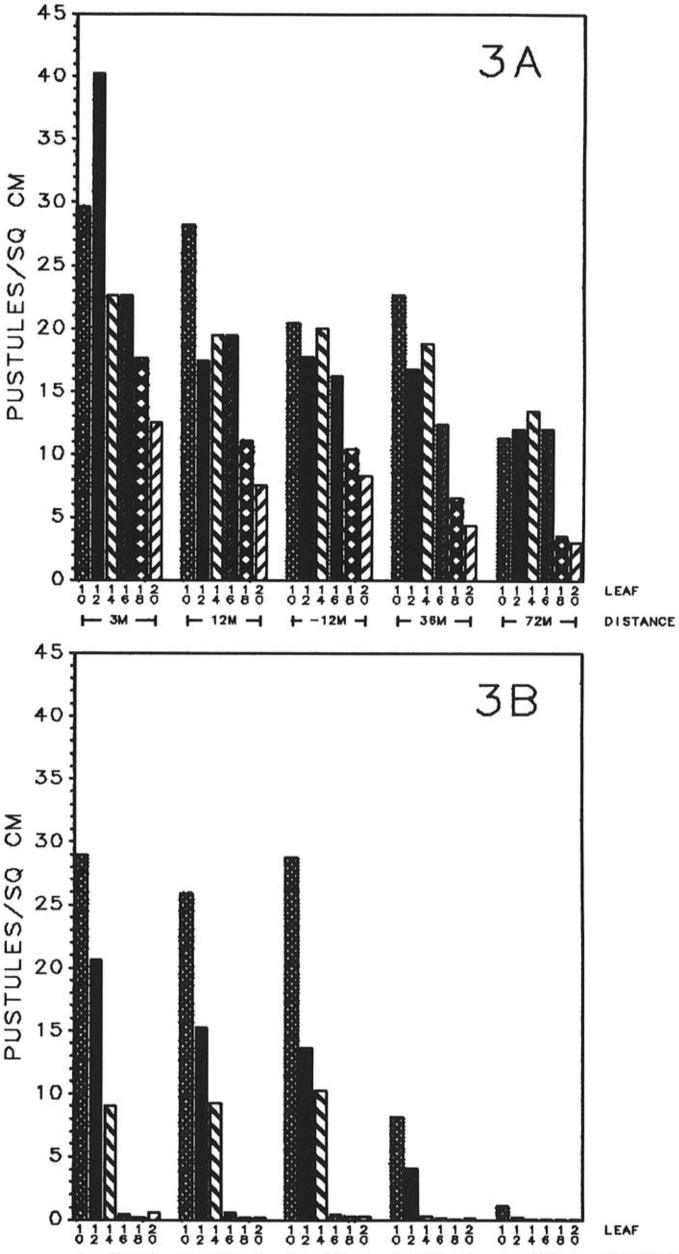

Fig. 3. Maximum pustule density of southern corn rust versus leaf position at five distances from a line source of inoculum. **A,** Frederick, MD, 1984. **B,** Rock Springs, PA, 1984. Leaf numbers represent position on plant with respect to order of development. Leaf position 12 was the ear leaf. Each bar represents the mean of 12 leaves.

densities did not occur on all leaves on the same date. Photosynthetic contributions from leaves above the ear leaf have been demonstrated to be most influential with respect to ear biomass (1). The ear and the flag leaf were identified as leaf 12 and leaf 20, respectively.

Southern rust was most severe on the lower leaves, with severity generally decreasing with successive leaf positions (Fig. 3A and B). A similar relationship between leaf position and disease severity was reported by King and Scott (12). A maximum mean pustule density of 40 pustules/cm^2 was recorded on the ear leaf at the 3-m distance at Frederick; however, observations indicate that localized densities of 50–60 pustules/cm^2 are possible with southern rust.

In contrast to the stepwise depression of pustule densities with leaf positions subsequent to the ear leaf at Frederick (Fig. 3A), pustule densities at Rock Springs exhibit a sharp decrease for positions above the 14th leaf (Fig. 3B). The lack of disease development in the upper leaf canopy at Rock Springs is also reflected in the flattening of disease progress curves (Fig. 2C and D).

Environmental conditions. Average mean daily temperatures were 3.3 and 4.8 C degrees higher at Frederick than at Rock Springs during 1983 and 1984 (Fig. 4A and B). Average numbers of hours/day where relative humidity (RH) exceeded 90% were 7.5 and 11.0 in 1983 and 13.2 and 11.2 in 1984, at Frederick and Rock Springs, respectively. Relative humidity data indicate conditions

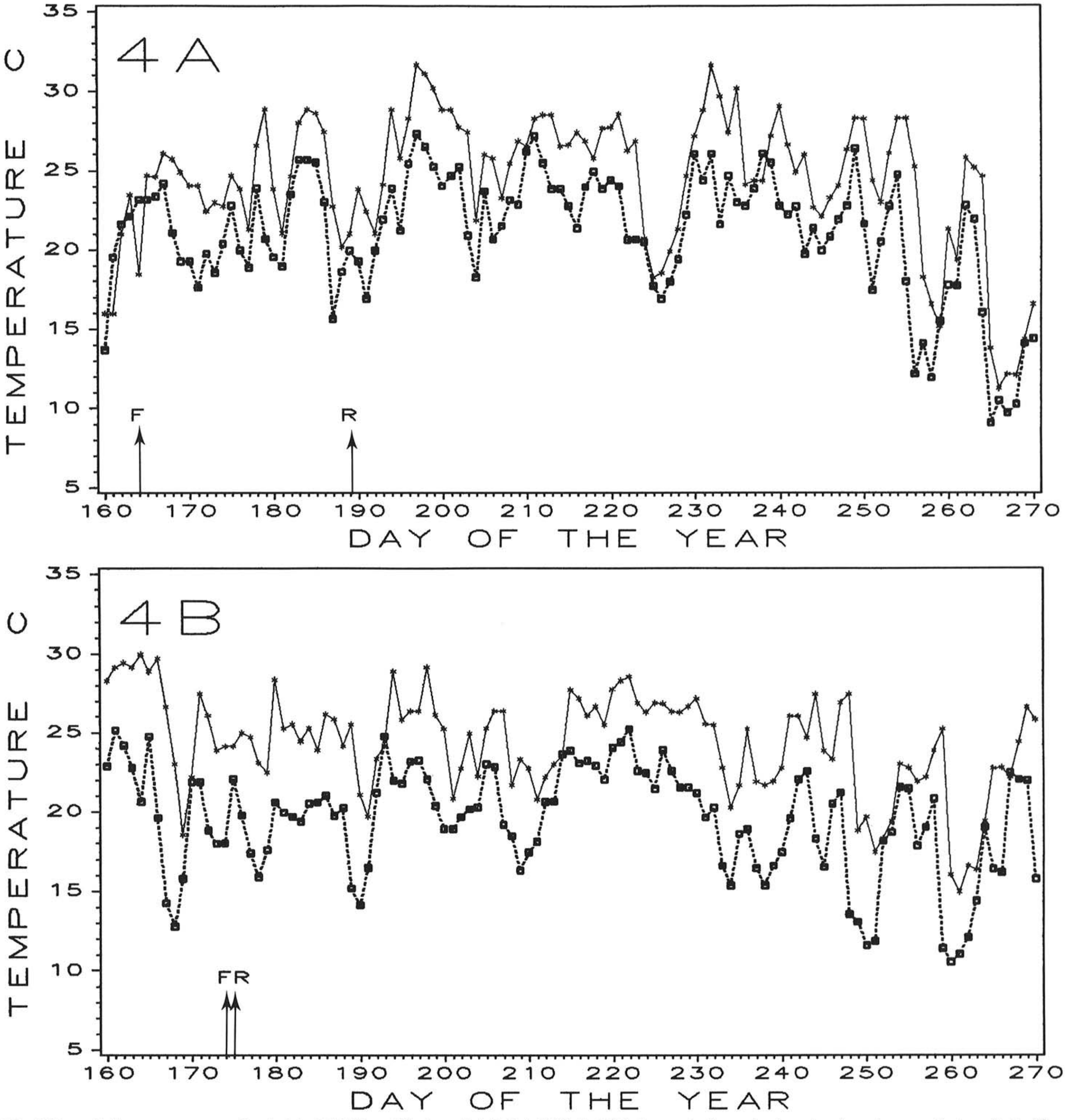

Fig. 4. Mean daily temperatures at Frederick, MD (*), and Springs, PA (□) **A,** 1983. **B,** 1984. Arrows indicate the day when inoculum was first available (F = Frederick, R = Rock Springs).

were more humid at Rock Springs than at Frederick during 1983 with the situation being reversed in 1984. Total rainfall and irrigation amounts were greater at Rock Springs during both years. Total amounts were 324 and 346 mm in 1983, and 341 and 482 mm in 1984, at Frederick and Rock Springs, respectively.

Grain yield. Grain yields in sprayed and nonsprayed subplots and percent yield reduction with respect to the sprayed controls at various distances from the inoculum source are presented in Tables 3 and 4 for 1983 and 1984, respectively. At Frederick, grain yields were reduced as much as 38.92% at the 6-m distance in 1983 and 39.11% at the 3-m distance in 1984. Maximum reductions of 13.18% at the 6-m distance in 1983 and 17.67% at the 3-m distance in 1984 were recorded at Rock Springs. The reductions in grain yield were related to the area under the disease progress curves (AUDPC), as calculated from time t_0 to 35 days subsequent to anthesis at both locations during both years (Table 5).

DISCUSSION

In general, disease progressed throughout the growing season at Frederick during both 1983 and 1984 (Fig. 2A and B), with progress being halted or slowed only by harvesting or depletion of susceptible green leaf tissue. At Rock Springs, however, disease levels reached a plateau before the end of the season (Fig. 2C and D) and were not severely limited by the amount of susceptible tissue remaining. Because only one rust isolate was used and identical hybrids were grown at both locations, environmental conditions appear to be the factor that limited disease development at Rock Springs.

TABLE 3. Grain yield (metric tons/ha) in sprayed and nonsprayed subplots at six distances from an inoculum source at Frederick, MD, and Rock Springs, PA, during 1983

		Grain yield[a]		
Location	Distance (m)	Sprayed (t/ha)	Nonsprayed (t/ha)	Reduction[b] (%)
Frederick	6	9.79	5.98	38.92
	26	9.66	7.85	18.74
	46	9.54	7.99	16.25
	67	8.81	7.51	14.75
	87	9.19	9.27	−0.87
	106	9.34	8.94	4.21
Rock Springs	6	8.04	6.98	13.18
	26	8.15	7.98	2.08
	46	8.07	8.21	−1.73
	67	6.61	6.87	−3.93
	87	7.45	7.48	−0.40
	106	7.58	7.69	−1.45

[a] Yields were hand-harvested, mechanically shelled, and adjusted to 15.5% moisture. Values do not reflect losses due to lodging.

[b] Percent reductions are with respect to the sprayed control at each distance.

TABLE 4. Grain yield (metric tons/ha) in sprayed and nonsprayed subplots at five distances from an inoculum source at Frederick, MD, and Rock Springs, PA, during 1984

		Grain yield[a]		
Location	Distance (m)	Sprayed (t/ha)	Nonsprayed (t/ha)	Reduction[b] (%)
Frederick	3	12.54	7.63	39.11
	12	13.20	9.55	27.68
	−12	12.89	10.21	20.78
	36	13.49	11.03	18.23
	72	13.34	11.68	12.48
Rock Springs	3	11.86	9.76	17.67
	12	11.45	10.46	8.65
	−12	10.98	10.70	2.50
	36	10.60	10.75	−1.41
	72	9.48	9.01	4.95

[a] Yields were hand-harvested, mechanically shelled, and adjusted to 15.5% moisture. Values do not reflect losses due to lodging.

[b] Percent reductions are with respect to the sprayed control at each distance.

Southern rust has been classified as a "warm weather disease," being favored by temperatures of 21–27 C (9,15). The large differences in apparent infection rates between locations are most likely caused by differences in temperature regimes rather than moisture conditions. Temperatures at Frederick were more conducive to epidemic progression than those at Rock Springs. With respect to moisture requirements, periods of leaf wetness were of sufficient length for infection and significant disease development, even though relatively dry conditions existed at Frederick during 1983, as suggested by the average number of hours (7.5) when relative humidity exceeded 90%. It is important to note that temperatures at Frederick are similar to those occurring throughout much of the Corn Belt. It is also significant that despite lower temperatures rust levels capable of causing significant yield reductions did develop at Rock Springs.

Calculated r values, particularly those for Frederick (Table 2), indicate that southern rust has a high r value in spite of its relatively long latent period (12–14 days) under field conditions (15). The ease with which its urediospores are wind disseminated enable *P. polysora* to have a higher velocity of spread than potato late blight, which has a higher r value (17) than southern rust. Leveling of disease gradients was similar to that reported by Cammac (3) for southern corn rust and Imhoff et al (11) for bean rust.

Infection rates calculated at the 6- and 3-m distances at Frederick during both years of this study are comparable to values calculated, by the authors, from data published by Melching (15) for disease progress in inoculated foci. The field research performed by Melching was also conducted at Frederick using the same rust isolate used in this study.

With respect to disease development on individual plants, the almost total lack of disease development in the upper leaf canopy at Rock Springs was in contrast to results at Frederick, where pustule densities decreased in a steplike manner as height within the canopy increased. This steplike progression could be due to leaf exposure to inoculum, with successively higher leaves being exposed for shorter periods of time. King and Scott (12) hypothesized that microenvironmental conditions such as duration of dew, light, leaf habit, and inoculum gradients could be contributing factors. They dismissed the possibility of increasing resistance with increasing plant maturity. Hollier and King (10) reported that exposures to low temperatures, particularly at high humidities, greatly reduced the infectivity of inoculum. It is possible that low night temperatures late in the growing season at Rock Springs reduced the viability of currently available inoculum and may have limited the organism's ability to produce more inoculum, thus limiting disease to the lower canopy and explaining the flattening of the disease progress curves (Fig. 2C and D).

The reduced yields encountered in this study demonstrate the capacity of *P. polysora* to cause significant reductions even as far north as central Pennsylvania. The magnitude of yield loss under natural conditions would be dependent on the amount of inoculum arriving from southern areas and the time of its arrival. It appears

TABLE 5. Regression models for the relationship between percent reduction in grain yield (Y) and area under the disease progress curve (X)[a]

Location (year)	Equation[b]	R^2	C.V.	P
Frederick (1983)	$Y = 2.437(X)$	0.892	46.62	0.0014
Frederick (1984)	$Y = 2.004(X)$	0.995	8.51	0.0001
Rock Springs (1983)	$Y = 3.174(X)$	0.850	188.76	0.0031
Rock Springs (1984)	$Y = 0.976(X)$	0.768	76.23	0.0220

[a] Coefficients of determination (R^2), coefficients of variance (C.V.), and significance levels (P) are also presented.

[b] Areas under the disease progress curves (X) were calculated from time t_0 to 35 days subsequent to anthesis and have units of 'disease proportion-days.

that temperature is the environmental parameter most likely to limit disease development in areas similar to central Pennsylvania. Under normal circumstances, inoculum does not arrive in northern areas until late July or early August. It is unlikely that naturally occurring epidemics would reduce yields to the extent observed in this study. However, given the pathogen's ability to cause losses even though infections do not occur until after anthesis, it is possible that under ideal conditions *P. polysora* may cause significant yield reductions in northern areas of the United States.

LITERATURE CITED

1. Allison, J. C. S., and Watson, D. J. 1966. The production and distribution of dry matter in maize after flowering. Ann. Bot. 30:365-381.
2. Beardsell, M. F. 1977. Effects of routine handling on maize growth. Aust. J. Plant Physiol. 4:857-861.
3. Cammack, R. H. 1958. Factors affecting infection gradients from a point source of *Puccinia polysora* in a plot of *Zea mays*. Ann. Appl. Biol. 46:186-197.
4. Cammack, R. H. 1959. Studies on *Puccinia polysora* Underw. III. Description and life cycle of *P. polysora* in West Africa. Trans. Br. Mycol. Soc. 42:55-58.
5. Cummins, G. B. 1941. Identity and distribution of three rusts of corn. Phytopathology 31:856-857.
6. Futrell, M. C. 1975. *Puccinia polysora* epidemics on maize associated with cropping practice and genetic homogeneity. Phytopathology 65:1040-1042.
7. Futrell, M. C., Hooker, A. L., and Scott, G. E. 1975. Resistance in maize to corn rust, controlled by a single dominant gene. Crop Sci. 15:597-599.
8. Hanway, J. J. 1966. Growth stages of corn (*Zes mays* L.). Agron. J. 58:487-491.
9. Hollier, C. A., and King, S. B. 1985. Effect of dew period and temperature on infection of seedling maize plants by *Puccinia polysora*. Plant Dis. 69:219-220.
10. Hollier, C. A., and King, S. B. 1985. Effects of temperature and relative humidity on germinability and infectivity of *Puccinia polysora* urediospores. Plant Dis. 69:937-939.
11. Imhoff, M. W., Leonard, K. J., and Main, C. E. 1982. Analysis of disease progress curves, gradients, and incidence-severity relationships for field and phytotron bean rust epidemics. Phytopathology 72:72-80.
12. King, S. B., and Scott, G. E. 1982. Development of southern rust on maize at different stages of maturity. Plant Dis. 66:477-481.
13. Leonard, K. J. 1974. Foliar pathogens of corn in North Carolina. Plant Dis. Rep. 58:532-534.
14. McGee, W. E. 1964. A coefficient for computing leaf area in hybrid corn. Agron. J. 56:240-241.
15. Melching, J. S. 1975. Corn rusts: Types, races, and destructive potential. Pages 90-115 in: Proceedings of the 30th Annual Corn and Sorghum Research Conference.
16. Minogue, K. P., and Fry, W. E.. 1983. Models for the spread of disease: Model description. Phytopathology 73:1168-1173.
17. Minogue, K. P., and Fry, W. E.. 1983. Models for the spread of plant disease: Some experimental results. Phytopathology 73:1173-1176.
18. Pavgi, M. S., and Flangas, A. L. 1959. Occurrence of southern corn rust in Wisconsin. Plant Dis Rep. 43:1239-1240.
19. Pennypacker, S. P., and Stevenson, R. E. 1977. A data acquisition and process control system for agricultural investigations. (Abstr.) Proc. Am. Phytopathol. Soc. 4:195.
20. Rhind, D., Waterson, J. M., and Deighton, F. C. 1952. Occurrence of *Puccinia polysora* Underw. in West Africa. Nature (London) 169:631.
21. Robert, A. L. 1962. Host ranges and races of the corn rusts. Phytopathology 52:1010-1015.
22. Rodriguez-Ardon, R., Scott, G. E., and King, S. B. 1980. Maize yield losses caused by southern corn rust. Crop Sci. 20:812-814.
23. Ru-Hong, Z., King, S. B., and Scott, G. E. 1983. A study of slow rusting of southern rust of corn: Preliminary report. USDA Bull. 925.
24. Ryland, A. K., and Storey, H. H. 1955. Physiological races of *Puccinia polysora* Underw. Nature (London) 176:655-656.
25. Schall, R. A., McCain, J. W., and Hennen, J. F. 1983. Distribution of *Puccinia polysora* in Indiana and absence of a cool weather form as determined by comparison with *P. sorghi*. Plant Dis. 67:767-770.
26. Scott, G. E., and Futrell, M. C. 1976. Southern corn rust. Crops Soils Mag. April-May:16-18.
27. Scott, G. E., King, S. B., and Armour, J. W., Jr. 1984. Inheritance of resistance to southern corn rust in maize populations. Crop Sci. 24:265-267.
28. Sim IV, T. 1980. Southern rust of corn recognized in Kansas. Plant Dis. 64:500.
29. Stanton, W. R., and Cammack, R. H. 1953. Resistance to the maize rust, *Puccinia polysora* Underw. Nature (London) 17:505-506.
30. Ullstrup, A. J. 1965. Inheritance and linkage of a gene determining resistance in maize American race of *Puccinia polysora*. Phytopathology 55:425-430.
31. van Arkel, H. 1978. Leaf area determinations in sorghum and maize by the length-width method. Neth. J. Agric. Sci. 26:170-180.
32. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
33. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 207 pp.
34. Widstrom, N. W., and Young, J. R. 1980. Double cropping corn on the coastal plain of the southeastern United States. Agron. J. 72:302-305.