Epiphytology of Puccinia striiformis at Five Selected Locations in Oregon During 1968 and 1969

Robert G. Emge and Robert D. Shrum

Research Plant Pathologist, Agricultural Research Service, U.S. Department of Agriculture, Northeastern Region, Plant Disease Research Laboratory, Frederick, Maryland 21701; and Assistant Professor, Department of Plant Pathology, University of Minnesota, St. Paul 55108.

Accepted for publication 15 April 1976.

ABSTRACT

EMGE, R. G., and R. D. SHRUM. 1976. Epiphytology of Puccinia striiformis at five selected locations in Oregon during 1968 and 1969. Phytopathology 66: 1406-1412.

Epiphytological studies were conducted on stripe rust of wheat in northeastern Oregon during 1968-69. Five plot locations provided varied climates. Plants in the three- to four-leaf stage at the center of each plot were inoculated with uredospores of *Puccinia striiformis* at a dosage rate of 25 g/hectare. The spread and build-up of the stripe rust pathogen was closely followed during the growing season. Temperature, relative humidity, and dew formation were monitored within the plant stand. The pathogen-spread patterns indicated that the inoculated centers served as the only or the predominant source of initial inoculum. The epiphytotics of 1969 progressed more rapidly than those of 1968 with disease increase rates varying from 0.28 to 0.04. Upwind and downwind infection gradients and rates of disease increase were calculated for all plots. Yields varied sharply within and among plots. The data apply to the development of a predictive model of a stripe rust epiphytotic.

Additional key words: climate, predictive model, epidemiology.

Stripe rust, which is caused by *Puccinia striiformis* West, destroys wheat (*Triticum aestivum* L.) in Eurasia, the higher elevations of Africa, the Andean highlands, and the Pacific intermountain areas of the USA. From 1892 to 1957, its occurrence in this country was sporadic. Some damage occurred in the 1920's, and then a period of quiescence followed until 1957, when stripe rust was reported as far east as central Kansas. Damaging outbreaks occurred again in Washington during the 1959-60 and 1960-61 crop seasons. These epiphytotics were favored by fall infection, mild winters which favored overwintering, and periods of weather that were warmer than normal in the spring, followed by weather cooler than normal in May and early June. No appreciable damage was done to the 1962 crop by stripe rust, presumably because the severe winter with little or no snow cover reduced the amount of overwintering inoculum.

MATERIALS AND METHODS

The build-up and spread of *P. striiformis* from infection foci were studied in five Oregon locations which differed from each other climatologically. Plots were located at Madras (Jefferson County) and Hermiston, Rew Station, Pendleton, and Weston (Umatilla County), Oregon. Land use was secured through a cooperative agreement between the Plant Disease Laboratory, Frederick, Maryland, and Oregon State University, Corvallis.

The Madras plot was in a high valley (elevation 700 m) on the eastern slope of the Cascade Mountain range, about 160 km south of the Columbia River. This region has a growing season of 101 days and an average annual precipitation of 32 cm. Irrigation is used extensively through the valley (flood irrigation for the test plot).

The Hermiston plot was at the Umatilla Experiment Station (192 m). The area has a growing season of 163 days, with an annual precipitation of 32 cm. Sprinkler irrigation is used extensively. We abandoned this location in 1969 owing to heavy infestation of powdery mildew.

The plot at Rew Station (375 m) was on the Rew Ranch about 16 km west of Pendleton. The growing season is about 155 days, with an annual precipitation of 32 cm. Irrigation is not used in this area.

The Pendleton plot was on the Pendleton Experiment Station (457 m), about 13 km east of the city of Pendleton. This area has a growing season of 155 days, with an annual precipitation of 55-63 cm and no irrigation.

The Weston plot was on the Wood's Ranch (625 m), about 37 km northeast of Pendleton. This area has a growing season of 158 days, with an annual precipitation of 95 cm and occasional irrigation.

Each plot was 0.2 hectare(ha), measuring 45.75×45.75 m. The plots were seeded in rows 25 cm apart and at the rate of 67 kg/ha to Omar wheat, a white club winter cultivar which is susceptible to stripe rust. Until the stripe rust epiphytotics of 1959-61, Omar comprised the bulk of the wheat acreage of the Pacific Northwest. A border of at least one drill width (3 m) of the wheat cultivar Moro (a white club winter cultivar similar in appearance and growth habit to Omar, but highly resistant to stripe rust) was planted around each plot to nullify edge effects and help confine *P. striiformis* to the plot area. Land

preparation, fertilization, seeding, and weed control were handled by the supervisor of the Pendleton Experiment Station so our test plots would be comparable to wheat fields in that particular area.

Plants in the three- to four-leaf stage were inoculated within a 2.3 m^2 area at the center of each plot. In Umatilla County, this stage occurred in middle to late March; in Jefferson County, it was almost a full month later. The inoculum was an endemic strain of *P. striiformis* supplied by Oregon State University. At about 1700 hours, we inoculated plants within the center at the rate of 25 g of viable uredospores per hectare by introducing a spore-talc aerosol into a 2.3 m^3 plastic settling tent placed over them. The tent was left in position for at least 30 minutes after inoculation and then removed. To induce the formation of free moisture and infection, we placed a plastic sheet [0.102-mm (4-mil) thick] over the inoculated plants, fixed it to the ground at the periphery of the inoculated center, left it overnight and removed it by 0800 hours the next morning. Even though the plastic restricts heat loss, moisture retention under the plastic almost always resulted in wetting of the plant surfaces under these conditions, particularly in areas where there was wide diurnal temperature fluctuation and possibility of air movement during the night. Temperature, relative humidity, and "dew" formation were monitored under the plastic during the incubation phase and within the plant canopy during the rest of the experiment. The record of environmental conditions under the plastic was of value in measuring the success of the inoculation. Epidermal strips also were inspected (2) to further confirm the success of the inoculation.

Sampling points were established along lines radiating from the inoculated center. These lines were laid out in the eight compass directions, and sampling points along these lines were marked off at 0.8, 1.5, 5, 10, and 30 m from the edge of the inoculated center (Fig. 1, 2). To record the increase and spread of *P. striiformis,* we periodically counted lesions, estimated severity, or both, within the centers and at each of the 44 sampling points. Readings were made at least once a week from the time of initial sporulation within the center, until 100% severity was attained or until the plants matured. The actual sample consisted of all leaves on all the plants in a 15-cm section of row. To reduce variation from reading to reading, each sample was marked so the same plant areas were observed throughout the study.

It was necessary to be able to equate counts of early sparse infections with later estimates of dense infections, for a meaningful and continuous record of disease increase. With stem rust of wheat, this task is relatively simple, since pustules are discrete and fairly uniform in size. Thus, it is possible to equate numbers of infections directly to a severity reading. Kingsolver et al. (5) estimated that 10 pustules per culm were equivalent to 1% severity in stem rust. With stripe rust, however, sporulating areas continue to expand and often fuse with adjacent sporulating areas, so counts alone are incompatible with severity estimates. Anticipating this complexity, a detailed sampling procedure was devised so that information could be obtained to establish the proper relationship between early counts and severity estimates.

Lesion counts began at the first sign of sporulation. Each lesion was assigned a coded value for size and one for density of sporulation. After all lesions were coded for one leaf, the entire leaf was similarly coded. This coding was repeated until all lesions and every leaf in the 15-cm row sample were coded. From this coding, average values for the sample were computed. The following codes were used throughout the studies:

Size of sporulating area:

Code 1—less than one-fifth of the leaf surface covered.
Code 2—one-fifth to two-fifths of the leaf surface covered.
Code 3—two-fifths to three-fifths of the leaf surface covered.
Code 4—three-fifths to four-fifths of the leaf surface covered.
Code 5—more than four-fifths of the leaf surface covered.

Density of sporulation:

Code 1—epidermis erumpent, but not ruptured.
Code 2—epidermis ruptured and uredia circular.
Code 3—uredia becoming oblong, but not yet fused with adjoining uredia within the same lesion.
Code 4—uredia oblong and beginning to fuse lengthwise with other uredia within the same lesion.
Code 5—uredia within a given lesion appearing to fuse laterally due to abundant sporulation.

When the amount of disease at a sampling point had increased so that the lesion counts were unduly difficult or impracticable, a visual estimate of severity was assigned to the sample. When disease levels permitted, lesion counts and severity estimates were made on the same sample. These double readings were used to derive a mathematical function to relate the counts to severity estimates.

Various transformations of the data were tested for the best combination of factors that would accurately describe severity estimates. The relationship yielding one of the best least-squares fits also was intuitively appealing:

$$\% \text{ severity} = 100 \times \left(X \times \frac{Y}{5} \times \frac{Z}{5}\right)$$

Where: X = percentage of leaves infected;
Y = coded value for average size of infected area per leaf; and
Z = coded value for average density of sporulation per leaf.

This transformation allows conversion of the coded values into a percentage of leaf surface covered by sporulating lesions. Thus, the early, low-level disease values could be converted directly into a percentage severity so that disease increase could be plotted with time as a continuous curve from the first to the last reading.

Yields for each of the five study plots were measured within the inoculated center, at each of the 44 sampling points, and within control plots. The control plots were sprayed weekly with maneb [manganous ethylenebis (dithiocarbamate)] at the rate of 370 g/ha to prevent the

development of rust. Yield of the control was the average computed from samples taken in the middle and at each end of the control plot. Among the yield data collected were values for kg/ha, total straw weight, plant height, number of heads, total grain weight, test weight, and 1,000-grain weight.

RESULTS AND CONCLUSIONS

Acceptable levels of infection were obtained in all inoculated centers. Actual values for the initial level of infection in these centers are recorded as the first point in the respective progress curves (Fig. 3, 4). These varied from a low of 0.18% at Hermiston to a high of 3% at Rew Station in 1968.

After inoculation, but before the appearance of infections, we carefully examined the plot area to determine if natural inoculum had been present. Immediately after the infections appeared within the centers, we searched again to determine if any effective spores had escaped during the inoculation procedure. No infection was found outside the inoculated centers at any location until at least 13 days (the minimum latent period) after the initial sporulation in the centers, except in the 1969 Weston plot. While examining the field and surrounding areas after the inoculation, we found a field of heavily infected volunteer wheat a few hundred meters upwind from the Weston plot. On April 30, 14 days after lesion break in the center, several infections were found at distances and in a direction that indicated their source was not the center. The later spread pattern in this field (Fig. 2) was not typical of the point-source patterns found in other plots (Fig. 1). In most plots, the effect of Oregon's prevailing westerly winds was obvious because spread of inoculum was most rapid eastwards and southwards. Also apparent from the patterns of spread was that the inoculated centers served as the only or predominant source of initial inoculum.

To quantitatively describe the epiphytotic dynamics depicted in Fig. 1 and 2, we measured the area within each isopleth of infection as the epiphytotics progressed through the season (Table 1). Isopleth areas of test plots not graphically presented in Fig. 1 and 2 are recorded in Table 1.

At all test plot locations, the epiphytotics of 1969 progressed more rapidly than did those of 1968. The slower rate of increase of the 1968 epiphytotic is the result of near-drought conditions. There was less year-to-year difference in the rate of increase of the epiphytotic at the Madras plot than at the Umatilla County plots (Table 1). The wide diurnal temperature fluctuations, adequate soil moisture, and still air at night resulted in regular occurrences of conditions conducive to infection (at least 5 hours dew at 15.6 C or below). Results of work at this laboratory and others (1, 6, 8) have indicated that the germination of *P. striiformis* uredospores varied with the strain (race or biotype) and they germinate over a temperature range of −4 to 20 C. Conditions conducive for infection were determined for the particular strain at each location. In the Umatilla area, because the nightly temperature reductions were not as great, the soil moisture levels were lower, and the night air movements were greater, the dew formation was less consistent and fewer infection-favoring periods occurred there than at Madras. These conditions considered favorable for infection are incorporated in Fig. 5. The differences in yield between Rew and Weston, 1969 (Table 2) can be attributed to the frequency of the dew periods. The dew

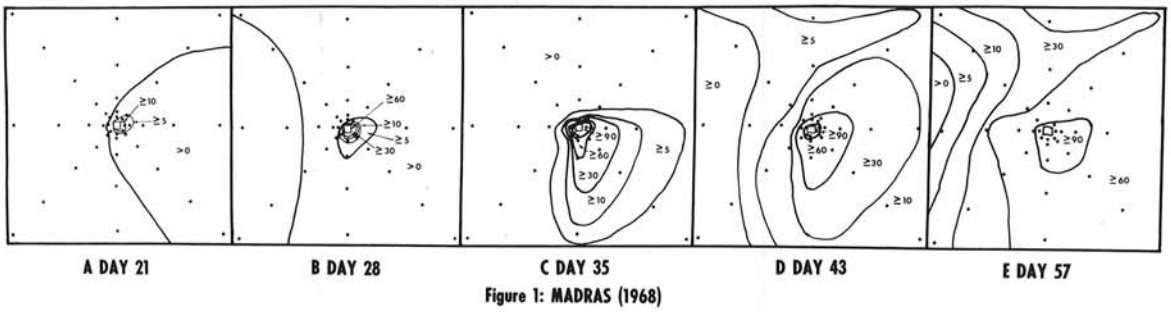

Figure 1: MADRAS (1968)

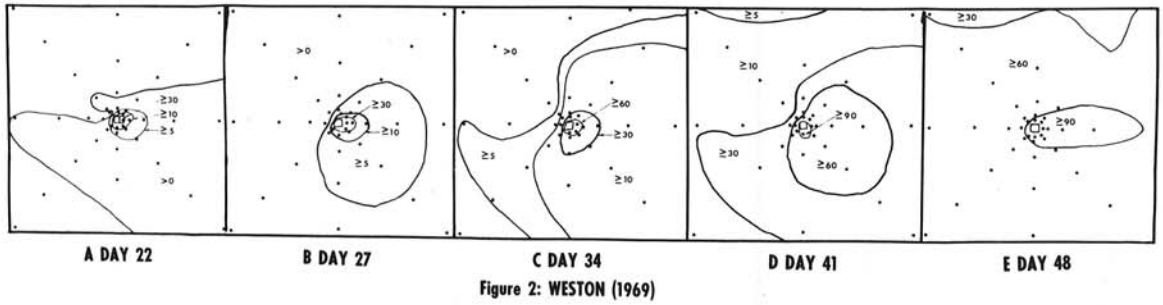

Figure 2: WESTON (1969)

Fig. 1-2. Spread and increase of stripe rust of wheat at Madras, Oregon, in **1)** 1968 and Weston, Oregon, in **2)** 1969 at indicated time intervals after initial sporulation within the inoculated center. Infection isopleths were drawn for the 0, 5, 10, 30, 60, and 90% severity levels.

periods at Rew occurred early in the season with none occurring after the joint stage, whereas those at Weston were well distributed throughout the growing season.

Curves of disease increase with time are plotted in several ways to illustrate the different features of these Oregon epiphytotics. Figures 3 and 4 show disease increase in inoculated centers at all test plot locations for 1968 and 1969, respectively. Data such as these could be used for comparison of overall favorability of environmental conditions from year to year and from place to place. In 1968, 100% disease severity was reached in both the Madras and Hermiston plots in about 50 days. The other plots rusted more slowly and reached plant maturity before 100% severity was reached. In 1969, 100% severity occurred in 55 days at the Madras plot but the other plots did not reach this level. Rates (apparent infection rate, r) (9) varied from 0.04 at Rew (1969) to 0.28 at Madras (1968). All rates were calculated by analyzing the regression of $\log x/(1-x)$ vs. time (9).

Figure 5 shows the pattern of disease increase at

TABLE 1. Epidemiology of stripe rust of wheat in centrally inoculated field plots (45.75 m × 45.75 m) in Oregon. Areas (m^2) within isopleths of given stripe rust severity levels relative to the number of days after initial sporulation within the inoculated center

Location and year	Days	Severity class (%)[a]					
		T[b]	5	10	30	60	90
1968							
Madras	21	756.1	12.1	4.2	2.3[c]	0	0
	28	1,646.1	39.1	15.8	7.4	3.7	0
	35	2,093.0[d]	544.5	249.2	108.8	25.1	8.4
	43	2,093.0	1,731.7	1,264.8	636.6	113.5	9.8
	51	2,093.0	2,154.8	2,003.2	1,713.1	1,238.8	91.1
Pendleton	13	8.4	2.3	0	0	0	0
	31	20.5	2.3	2.3	0	0	0
	42	474.3	9.8	4.2	2.3	2.3	0
	51	1,928.8	91.1	14.0	7.4	2.3	2.3
Weston	26	12.1	2.3	2.3	0	0	0
	35	33.5	2.3	2.3	2.3	0	0
	44	894.7	6.0	2.3	2.3	2.3	0
	63	1,254.6	839.8	468.7	222.3	45.6	2.3
Hermiston[e]	25	103.2	2.3	2.3	2.3	0	0
	31	436.2	10.2	5.6	2.3	2.3	0
	39	901.2	61.8	13.0	4.7	2.3	2.3
	49	2,093.0	1,076.0	173.9	64.2	12.1	2.3
	65	2,093.0	2,093.0	2,093.0	2,093.0	1,925.1	1,424.8
1969							
Madras	22	1,002.5	34.4	8.8	3.7	0	0
	27	2,093.0	409.7	35.3	10.2	2.3	0
	34	2,093.0	1,483.4	1,010.9	49.3	9.3	0
	41	2,093.0	2,093.0	2,120.4	1,229.5	329.2	7.4
	48	2,093.0	2,093.0	2,093.0	2,093.0	2,033.9	142.8
Pendleton	27	1,925.1	325.5	213.0	2.3	0	0
	34	1,873.0	641.7	311.6	68.8	2.3	0
	40	2,093.0	2,093.0	2,019.0	943.0	74.4	0
	52	2,093.0	2,093.0	2,093.0	2,093.0	1,802.3	0
	62	2,093.0	2,093.0	2,093.0	2,093.0	2,093.0	0
Weston	22	714.2	2.3	2.3	0	0	0
	30	2,093.0	484.5	155.3	2.3	0	0
	41	2,093.0	1,939.1	796.1	2.3	0	0
	56	2,093.0	2,093.0	1,292.7	5.1	0	0
	70	2,093.0	2,093.0	2,093.0	1,988.3	884.4	0
Rew[e]	30	949.5	2.3	2.3	0	0	0
	37	1,383.8	7.4	5.6	0	0	0
	48	2,108.3	1,430.3	571.0	3.7	0	0
	57	2,093.0	1,881.4	1,005.3	2.3	0	0
	63	2,093.0	2,093.0	318.1	2.3	0	0

[a]Includes that area equal to or greater than the severity class.
[b]Trace, includes infection levels less than 5% severity.
[c]Area of the inoculated center.
[d]Area of the test plot.
[e]Data from the Rew 1968 and Hermiston 1969 plots were not included (see text).

Madras (1969) as distances from the inoculated center were increased. Each point on the curves represents the average severity reading of the eight points at a given distance from the center. At Madras in both 1968 and 1969, the rate of disease increase was rapid, and the disease progress curves at the various distances were well separated from each other. Although the disease increase rates were not significantly different with increasing distance from the center, the occurrence of infections were progressively delayed. This delay can be attributed to the dilution of inoculum levels with distance and the problems associated with the detection of extremely low levels of infection. Rates of infection at Pendleton and Weston were lower than those at Madras. This at least partially is related to the differing frequency and intensity of dew at the various locations. In those fields where conditions favorable for infection were infrequent, the rate of increase was low, but not as low as would be expected. This is due to the expansion of the sporulating zone of *P. striiformis* which results in an apparent increase of disease in the absence of conditions conducive to infection (3).

Figure 6 presents disease increase curves for the upwind and downwind halves of the same plot. Each point on an upwind curve represents the average severity reading of 22 sampling points located from the southwest sector clockwise through the north sector. Each point on the downwind curve is the average of the 22 points in the other half of the test plot. These data show that the effect of going from the downwind to the upwind side of the source is similar to the effect of increasing the distance from the source. The rate of disease increase is essentially unaltered, but the level of initial infection is lower and delayed in the upwind direction. As before, the differences are more distinct in those plots having the higher rates of disease increase.

We do not believe that the sampling technique had appreciable confounding effect on the disease spread and development data. We entered the field from the side having the least amount of infection and progressed toward the center inspecting sampling points in order of expected increasing rust incidence. Exit was made by a fixed path in an east southeast direction. Periodically we sampled portions of the plot away from the established sampling points, but did not detect any significant differences in rust incidence. Figures 1 and 2 are typical of the spread patterns developed in all plots for both years. These figures show no effect of the observer on rust spread. Personnel moving within plots dislodge and transport spores. Such movements of spores in these studies seem to be insignificant compared to natural means of spore transport.

Upwind and downwind infection gradients (4) were calculated for all test plots (Table 2). Although steeper gradients were expected for the upwind direction, our data do not support this expectation. Upwind and downwind gradients were not significantly different. Wind data indicate that the prevailing westerlies of

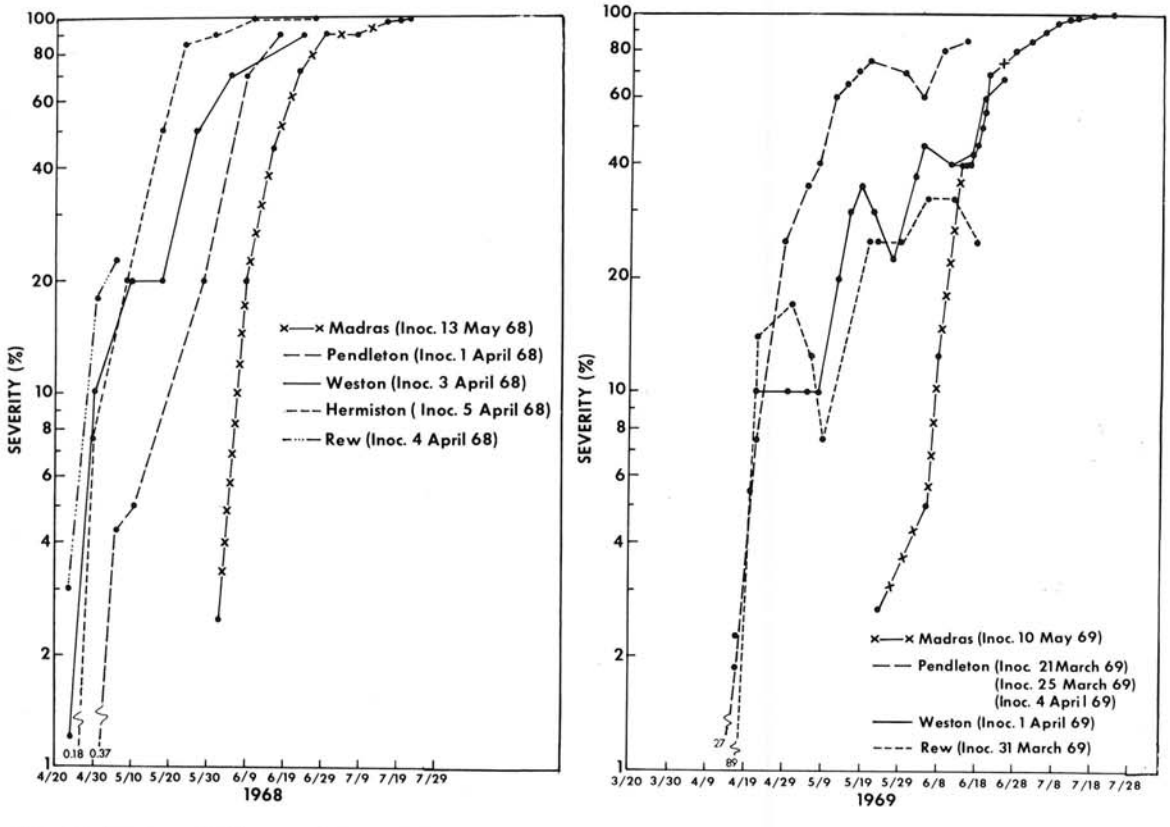

Fig. 3-4. Increase of stripe rust of wheat within the inoculated centers of the Oregon epiphytology plots for **3)** 1968 and **4)** 1969.

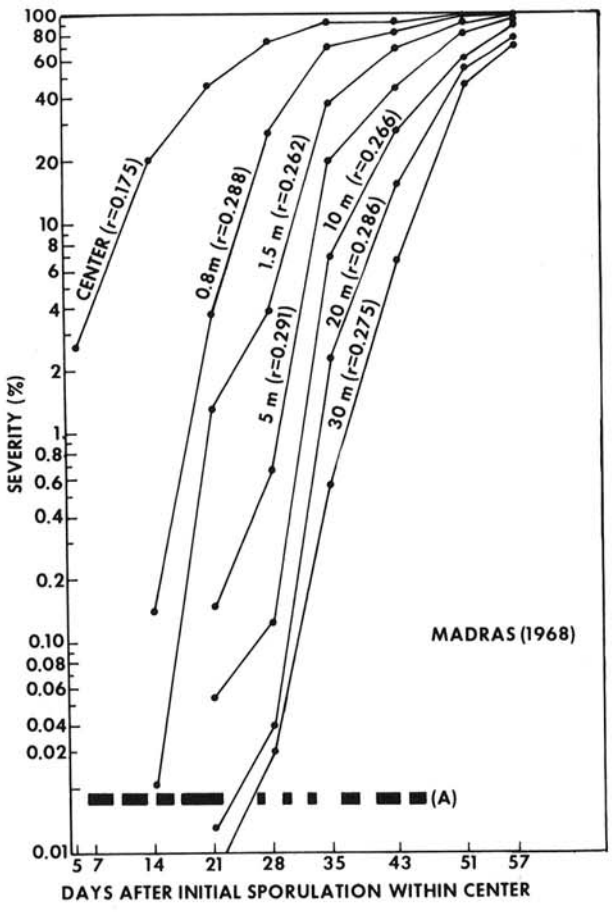

Fig. 5. Epidemiology of stripe rust of wheat in centrally inoculated Oregon epiphytology plots (45.75 m × 45.75 m). Rate (r) of disease development with increasing distance from the inoculated center as related to (A) number and frequency of dew periods of at least 5 hours at 15.6 C or below.

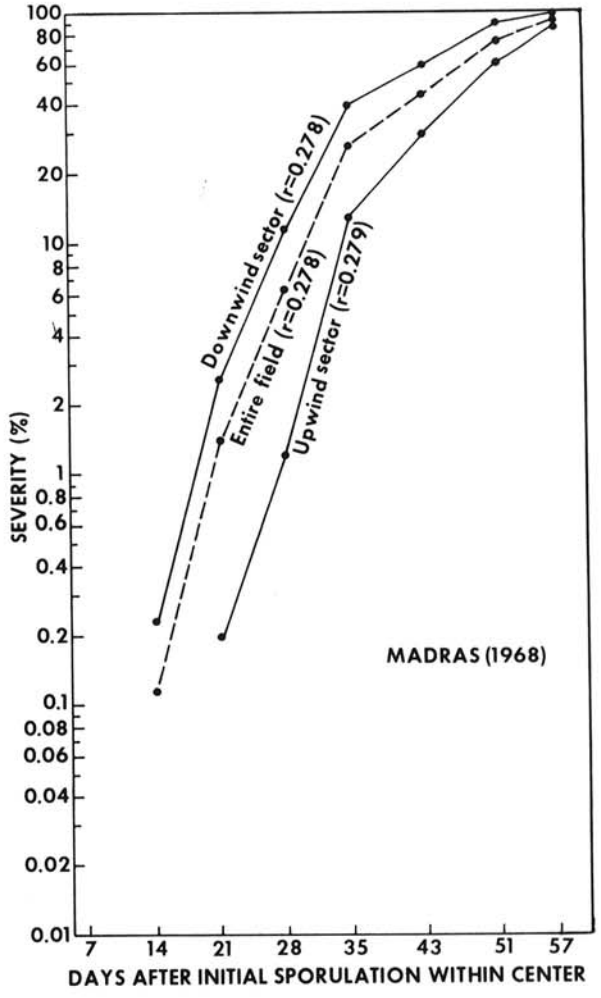

Fig. 6. Epidemiology of stripe rust of wheat in centrally inoculated Oregon epiphytology plots (45.75 m × 45.75 m). Rate (r) of disease development as influenced by prevailing winds.

TABLE 2. Epidemiology of stripe rust of wheat in Oregon. Data summary for centrally inoculated epiphytology plots (45.75 m × 45.75 m)

Location	Year	Days favorable for infection (%)	Slope of disease increase curves		Infection gradient average[a]		Yield loss (%)	
			Center	Field average	Upwind	Downwind	Center	Field average
Madras	1968	46	0.175	0.278	−1.488	−1.540	59	23
	1969	78	0.170	0.263	−0.773	−0.999	78	51
Hermiston[b]	1968	28	0.203	0.235	−2.220	−1.622	32	22
Rew[c]	1969	36	0.042	0.103	−0.496	−0.598	12	−11
Pendleton	1968	33	0.139	0.174	−2.062	−1.927	13	0
	1969	38	0.097	0.164	−0.254	−0.186	40	6
Weston	1968	24	0.098	0.194	−0.815	−1.389	−1	−11
	1969	37	0.052	0.096	−0.440	−0.328	25	14

[a]Weighted average of infection gradients along four sectors in each direction, calculated for each sampling date.
[b]Heavy powdery mildew infestations in 1969 forced elimination of the Hermiston site as a test plot.
[c]There was essentially no increase or spread of rust after a successful initial inoculation at Rew in 1968.

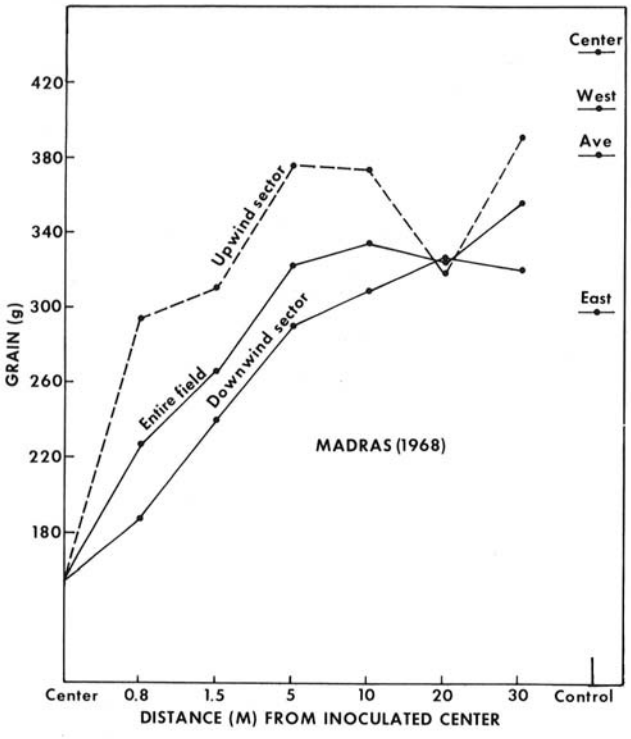

Fig. 7. The influence of distance and direction from the inoculated center of the plot on total grams of harvested grain.

northeastern Oregon occur about 60% of the time, accounting for the directional pattern of spread observed in these plots.

The effect of stripe rust on grain yield is shown in Table 2 and Fig. 7. Table 2 gives the yield for each plot and year as a percentage loss. The average of the three samples from the control area associated with a test plot was used as the potential yield of that test plot. At Weston (1968) and Rew (1969), total test plot yields were greater than yields of the controls. However, yield was always lower at the more heavily rusted centers than at other locations within the plot. All plots showed greater reductions in yield toward the more heavily rusted centers and downwind from the centers. At Madras, where the amount of rust was distinctly different from point to point within the test plot, yields varied greatly (Fig. 7). At Pendleton and Weston, where "rusting" was more uniform within the test plot, yields varied less with distance and/or direction from the center, but the same trends were evident.

Comprehensive disease severity and yield loss data are available for all plot locations. These data lend themselves to the verification of stripe rust disease prediction (7) and yield reduction models.

LITERATURE CITED

1. BURLEIGH, J. R. 1965. The winter biology of Puccinia striiformis West in the Pacific Northwest. Ph.D. Thesis. Washington State University, 102 p.
2. DELP, C. J. 1954. Effect of temperature and humidity on the grape powdery mildew fungus. Phytopathology 44:615-626.
3. EMGE, R. G., C. H. KINGSOLVER, and D. R. JOHNSON. 1975. Growth of sporulating zone of Puccinia striiformis and its relationship to stripe rust epiphytology. Phytopathology 65:679-681.
4. GREGORY, P. H. 1968. Interpreting plant disease dispersal gradients. Annu. Rev. Phytopathol. 6:189-212.
5. KINGSOLVER, C. H., C. G. SCHMITT, C. E. PEET, and K. R. BROMFIELD. 1959. Epidemiology of stem rust: II. Relation of quantity of inoculum and stage of growth of wheat and rye at infection to yield reduction by stem rust. Plant Dis. Rep. 43:855-862.
6. SHANER, G., and R. L. POWELSON. 1971. Epidemiology of stripe rust of wheat, 1961-1968. Oregon Agric. Exp. Stn. Tech. Bull. 117. 31 p.
7. SHRUM, R. D. 1975. Simulation of wheat stripe rust, Puccinia striiformis West., using EPIDEMIC a flexible plant disease simulator. The Pennsylvania State University, Agric. Exp. Stn. Prog. Rep. 347. 68 p.
8. TOLLENAAR, H., and B. R. HOUSTON. 1967. A study on the epidemiology of stripe rust, Puccinia striiformis West, in California. Can. J. Bot. 24:291-307.
9. VAN DER PLANK, J. E. 1963. Plant diseases: Epidemics and control. Academic Press, New York. 349 p.