

## Quantitative Relationships Between Climatic Variables and Stripe Rust Epidemics on Winter Wheat

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## ABSTRACT

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Climatic variation at Pullman, WA, since 1958 has contributed to an increase in the frequency of epidemics and severity of stripe rust (caused by *Puccinia striiformis*) on winter wheat. For 1963-1979, rust intensities on cultivar Omar (CI 13072) (very susceptible) and on cultivar Gaines (CI 13448) (susceptible in the seedling stage at all temperatures, but resistant at later growth stages at high temperatures) were positively correlated with January temperatures and negatively correlated with April and June temperatures. Spring temperatures were more highly correlated with disease development in Gaines than in Omar. Frequency of precipitation in June was correlated with stripe rust intensity in both Gaines and Omar. To mathematically relate stripe rust intensity to cumulative temperatures, we calculated negative degree days (NDD) and positive degree days (PDD) by

using a 7°C base. Disease intensity was negatively correlated with NDD and PDD; the best correlations were with NDD that accumulated between 1 December and 31 January and PDD that accumulated between 1 April and 30 June. Slopes of linear regression equations for disease intensity on Gaines vs NDD in December and January and vs PDD in April through June differed significantly from zero at  $P < 0.001$ . Regression equations for disease intensity on Omar as a function of NDD or PDD had significantly different Y-intercepts but slopes that were similar to the comparable equations for Gaines. These relationships help explain why stripe rust was not severe between 1941 and 1958, and may be useful for predicting stripe rust intensity in the Pacific Northwest.

*Additional key words:* model for disease prediction, quantitative epidemiology, statistical model for stripe rust prediction.

In the USA, stripe rust of wheat (*Triticum aestivum* L. em Thell) by *Puccinia striiformis* West., is most important in the Pacific Northwest (PNW). Coakley (1) reviewed the history of stripe rust occurrence and its relationship to the climatic factors that limit the development of epidemics to that region and concluded that climatic variation, determined from long-term averages, had resulted in increased frequency and severity of stripe rust epidemics since 1960 at Pendleton, OR. Epidemics developed during years when two or more winter months had higher than normal temperatures and when April temperatures were below normal. The climatic trends at Pendleton since 1960 were toward higher winter temperatures and lower spring temperatures. Those trends were generally true for the entire PNW (2). The earlier work related meteorological data to qualitative and quantitative disease data for Oregon and Washington but did not quantify the climatic limitations for development of stripe rust epidemics.

Line et al (4) showed that most of the cultivars presently grown in the PNW have a durable type of resistance that has not been vulnerable to new races of the fungus but is affected by temperature. The cultivars are highly susceptible in the seedling stage at a wide range of temperatures, but become increasingly more resistant at later stages of growth at high temperatures. The resistance has been designated as adult-plant, temperature-sensitive resistance; climatic variation may have a greater effect on the increase of stripe rust in these cultivars than in other cultivars.

The objectives of this research were: to quantify the climatic conditions that influence the development of stripe rust on winter wheat at Pullman, WA; to determine how climate affects disease development in locally grown cultivars with different types of resistance to stripe rust; and to develop a model by which meteorological data can be used to predict the intensity of stripe rust on wheat cultivars grown in the PNW.

## MATERIALS AND METHODS

Stripe rust development and stages of plant growth were recorded for several hundred cultivars and breeding lines of wheat planted in single rows 1.5-3.0 m long at numerous sites in the PNW. Disease intensity (percent of the total leaf and glume surface covered by rust) and infection type were recorded periodically at various stages of growth. The time and frequency of observations varied from year to year and from site to site, but usually were made between stage 3 (jointing) and stage 8 (dough) identified according to the scale described by Zadoks and Konzak (10). Disease intensities were usually low at stage 3. Rust intensity beyond stage 8 was seldom recorded because disease development beyond that stage had little additional effect on wheat yields. The date at which plants had developed to stage 8 and the rate of rust development varied from year to year.

Four cultivars grown commercially in the PNW were selected for the research reported here. They are Elgin (CI 11755) and Omar (CI 13072), widely grown in the 1950s and 1960s and susceptible to *P. striiformis* at all growth stages, and Gaines (CI 13448) and Nugaines (CI 13969), major cultivars widely grown since 1962 that are susceptible in the seedling stage, but have adult-plant, temperature-sensitive resistance.

Disease data for these cultivars were collected at four sites near Pullman from 1968-1979. Most data were from sites 1 and 2 because plots at those sites existed for more years. The source of inoculum was from naturally occurring stripe rust. Site 1 also received some inoculum each year from adjacent inoculated plots. The additional inoculum, however, influenced the results only in 1979 when little or no naturally occurring inoculum survived the winter. In years when water for plant growth was limiting, plants at site 2 were irrigated once or twice in June to supplement natural rainfall. Disease data from the sites were averaged for each year (Fig. 1). Disease data for 1963-1967 consisted of quantitative and qualitative data obtained from annual reports of the "Results from cooperative wheat varietal experiments in the Western Region"

and unpublished records at Washington State University and were based on the observations of several plant breeders and plant pathologists. No reliable data were obtained for Gaines, Nugaines, or Omar in 1966 or for Omar in 1968; therefore, none are presented. To simplify comparison of disease intensity (severity) with climatic changes and to minimize errors in correctly ranking the years, we grouped average percent intensities and converted them to a 0–9 disease index (Fig. 2).

Daily meteorological data for December 1940 to August 1979 for Pullman, WA (latitude 46°46' N, longitude 117°12' W, elevation 775 m) were obtained from the National Climatic Center at Asheville, NC. The data consisted of minimum and maximum temperatures, total precipitation, snowfall, and depth of snow on the ground. April 1950 to September 1951 and July 1974 were omitted from calculations of monthly and seasonal averages because no data were available. To calculate moving-average lines to show long-term trends, monthly averages for all years were inserted for the missing values. Temperature data were missing for 8 days in December 1956 to January 1957, and in December 1957. To calculate the monthly average temperature, we averaged the data for the last day preceding and the first day succeeding the missing data and used that value to replace the missing data. This method was a better estimate of the monthly average temperature than the average of available data.

Degree days were used to determine the cumulative effect of temperature on disease development. Sharp (7) reported that the optimum temperature for both *in vivo* germination of urediospores and infection of the wheat plant was 7 C. Therefore, 7 C was selected as the base for calculating negative and positive degree days. Daily average temperature was determined by adding the day's minimum and maximum temperatures and dividing by two; degree days were then calculated as follows:

$$\text{Daily average temperature} - 7 \text{ C} = \text{degree days}$$

When the average temperature was below 7 C, values were less than 0 and the absolute values were recorded as negative degree days (NDD); values greater than zero were recorded as positive degree days (PDD). The number of PDD and NDD were calculated for each month, starting in September, and for each successive month in combination with all succeeding months in the growing season.

## RESULTS

**Cultivar response to stripe rust.** The mean rust intensities on cultivars Nugaines, Gaines, Omar, and Elgin at Pullman were recorded at anthesis (stage 6) in 1977, at the milk stage (stage 7) in 1972, 1974, and 1978, and at the dough stage (stage 8) in 1968–1971, 1973, 1975, 1976, and 1979 (Fig. 1). The drought in 1977 so severely affected plant growth and disease development that no later measurements were made. From 1968–1978, the mean disease intensity at stages 7 or 8 was 69% on Omar, 68% on Elgin, 37% on Gaines, and 27% on Nugaines. In 1975 and 1976, rust intensities on Gaines and Nugaines were greater than one standard deviation above the mean. In 1969 and 1977, Nugaines and Gaines had disease intensities more than one standard deviation below the mean. For any given year, average disease intensities on Omar and Elgin were essentially equal and were usually considerably higher than disease intensities on Gaines and Nugaines.

The average ratio of disease intensity of Gaines to Omar was 0.50 with a range of 0.10 to 0.96; the mean ratio of disease on Nugaines to Gaines was 0.55 with a range from 0.01 to 1.06. The ratio of disease intensity on Nugaines to Gaines was 1.0 in 1976 and 1.06 in 1975, years when rust was especially severe on both cultivars. Thus, the overall rust intensity on Omar was two times greater than on Gaines, and rust intensity on Gaines was 1.82 times greater than on Nugaines.

**Relation of disease intensity to climatic parameters.** The analyses of the relationship between meteorological and disease intensity

TABLE 1. Correlation between climatic parameters analyzed by various methods and stripe rust intensity index on cultivars Gaines and Omar winter wheat at dough stage (stage 8) and statistical significance (*P*) of the correlation coefficient. Based on data collected at Pullman, WA, 1963–1979

Parameter	Basis of analysis	Time period	Gaines		Omar	
			Correlation coefficient	<i>P</i>	Correlation coefficient	<i>P</i>
Minimum temperature	Season	Winter	0.71	0.01	0.68	0.01
Average temperature	Season	Spring	-0.61	0.01	-0.50	0.05
Minimum temperature	Month	April	-0.52	0.05	-0.43	> 0.05
Average temperature	Month	January	0.81	0.001	0.80	0.001
Average temperature	Month	June	-0.60	0.05	-0.61	0.05
Negative degree days	Cumulative	1 Dec to 31 Jan	-0.85	0.001	-0.76	0.001
Positive degree days	Cumulative	1 Apr to 30 Jun	-0.79	0.001	-0.75	0.001
Precipitation frequency	Month	June	0.59	0.05	0.53	0.05

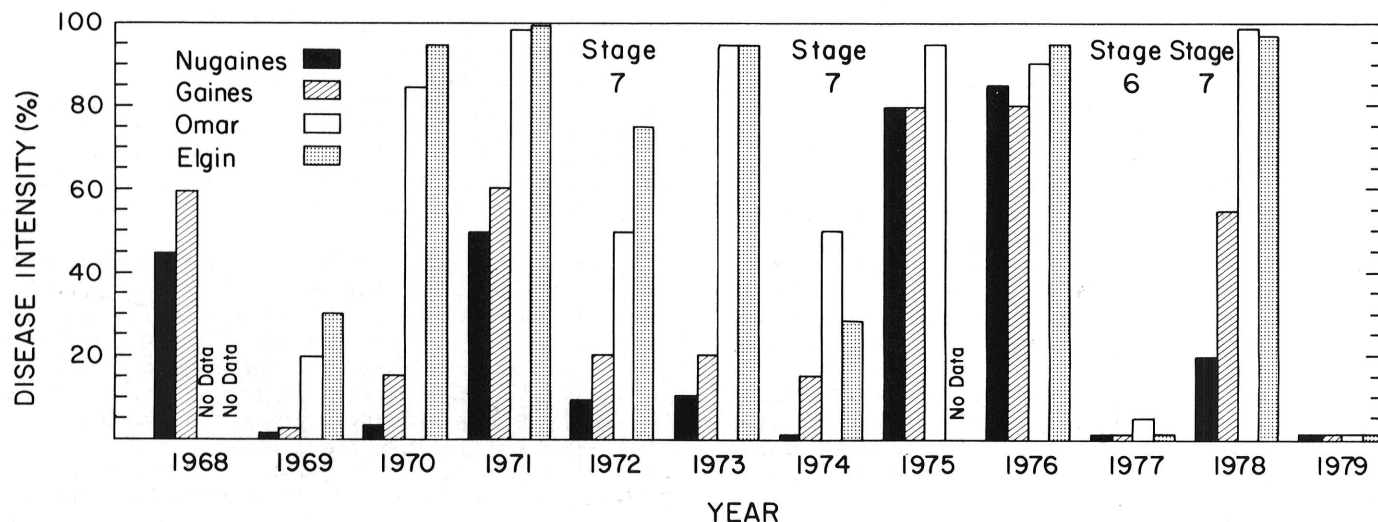


Fig. 1. Stripe rust intensity on winter wheat cultivars Nugaines, Gaines, Omar, and Elgin at Pullman, WA, 1968–1979. Data were recorded at the dough stage of growth (stage 8) for all years except 1972, 1974, and 1978, which were in the milk stage (stage 7) and 1977, which was in the anthesis stage (stage 6).

data that resulted in quantitative methods for identifying the influencing meteorological conditions are reported here. Of the cultivars with temperature-sensitive resistance, Gaines has been grown for the longest period of time (since 1961); therefore, subsequent results will emphasize that cultivar unless otherwise indicated.

The correlations between the disease index (Fig. 2) and the monthly and seasonal mean maximum, minimum, and average temperatures, total precipitation, frequency of precipitation, PDD and NDD, and cumulative PDD and NDD were calculated. The more significant correlation coefficients ( $r$ ) are presented in Table 1. Disease intensities in Gaines and Omar were favored to the same extent by high January and low June temperatures. Minimum temperatures during the winter season were positively correlated with subsequent disease development in Gaines and Omar. Average spring temperatures were more negatively correlated with disease development in Gaines than in Omar. During the spring, April minimum temperatures were significantly inversely correlated with disease development in Gaines but not in Omar.

The only precipitation parameter that was significantly correlated with the disease index was the frequency of precipitation during June, which was positively correlated with disease intensity in Gaines and Omar (Table 1). The correlation between precipitation frequency and disease index is not independent of the inverse correlation ( $r = -.59$ ,  $P < 0.01$ ) between June precipitation frequency and June average temperature.

January, April, and June temperatures had the highest correlation with disease intensity (Table 1). The average temperatures for those months in years with severe, moderate, and light stripe rust on Gaines (see Fig. 2) are shown in Fig. 3. The average January temperature in years with severe rust was 6.69 C above the average temperature in years with light rust. In years with severe disease, the average temperatures in April and June were, respectively, 2.34 C and 2.33 C lower than in years with light disease.

The temperature parameters (Table 1) that correlated best with disease intensity in Gaines and Omar were the total negative degree days (NDD) from 1 December to 31 January ( $r = -.85$  for Gaines and  $-.76$  for Omar) and the total positive degree days (PDD) from 1 April to 30 June ( $r = -.79$  for Gaines and  $-.75$  for Omar). The regression equations for the relationship between disease index on Gaines and Omar and degree days are shown in Fig. 4. Disease intensity decreases as NDD increases (which corresponds to lower winter temperatures) (Fig. 4A and C), and as PDD increases (Fig. 4B and D). The slopes of the regression lines for Gaines (Fig. 4A and B) and for Omar (Fig. 4C and D) are significantly different from zero, at  $P < 0.001$  and  $P < 0.005$ , respectively, according to Student's  $t$ -test. The slopes for Gaines are not significantly different from the comparable slopes for Omar, but the Y-intercepts for these cultivars differ significantly (for NDD,  $P < 0.001$  and for PDD,  $P < 0.01$ ).

The ranges of NDD or PDD that were associated with the development of severe, moderate, or light disease intensity (see Fig. 2) were determined from the data for 1963–1979 (Fig. 4). The range of NDD or PDD values which allow the development of a specific disease intensity are different for Gaines and Omar (Table 2).

**Long-term climatic trends.** An intensive search of the research files on stripe rust at Washington State, Plant Disease Reporter Supplements (1919–1959) that dealt with disease surveys or crop losses, and the 1951–1979 annual "Reports from cooperative wheat varietal experiments in the Western Region" (compiled by the USDA-ARS in cooperation with state agricultural experiment stations) indicated that stripe rust was not an important disease in the USA between the mid-1930s and 1957. It was present and described on various grasses, but was reported to be widespread on wheat only in 1942. Stripe rust is an easily identified disease, especially when it develops to epidemic levels. Research on stripe rust was widely reported through the late 1930s (1). From that period to the late 1950s there were no significant reports or research in the USA or Canada. Therefore, we are confident that the general absence of reports reflects the low level of disease during that period. Beginning in 1958, the above sources indicate an

increase in the occurrence and severity of stripe rust in Washington. Because the climatic record for Pullman begins in 1941, it is appropriate to compare the long-term climatic means for 1941–1957 (when stripe rust was not considered important) with those for 1958–1978 (when stripe rust was important) to determine how climatic variation may have affected stripe rust.

Daily precipitation and temperature data for December 1940 to November 1978 were summarized on a monthly and seasonal basis. The average temperature (Fig. 5A–C) and total precipitation for each season were calculated, and the seasonal means for December 1940 to November 1957 were compared with those for December 1957 to November 1978.

The mean winter temperature (December, January, and February) was  $-1.39$  C for 1941–1957 and  $-0.10$  C for 1958–1978 (Fig. 5A). Thirteen of 16 winters from 1941 to 1957 were below the mean temperature for 1958–1978. From 1958 to 1979 only 4 of 22 winter mean temperatures were below the mean temperature for 1941–1957. Thus, winter temperatures were highest during the period when rust was frequently severe.

In contrast, the mean spring temperature (March, April, May) was higher ( $7.55$  C) for 1941–1957 and lower ( $7.21$  C) for 1958–1978 (Fig. 5B). The decrease in the upper limit that actual spring temperatures reached was of greater magnitude than the

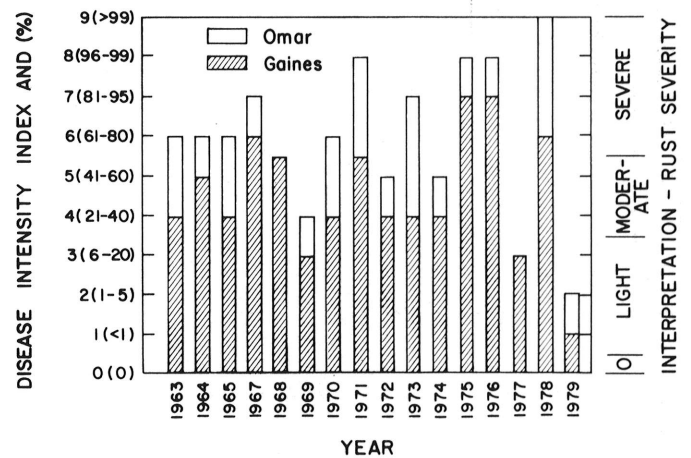


Fig. 2. Stripe rust at the dough stage of growth on winter wheat cultivars Gaines and Omar at Pullman, WA, 1963–1979. No data were available for either cultivar in 1966, and for Omar in 1968. In 1977, intensity was the same for Gaines and Omar. The disease intensity index (0–9) represents a range in intensity from 0 to 100% infection.

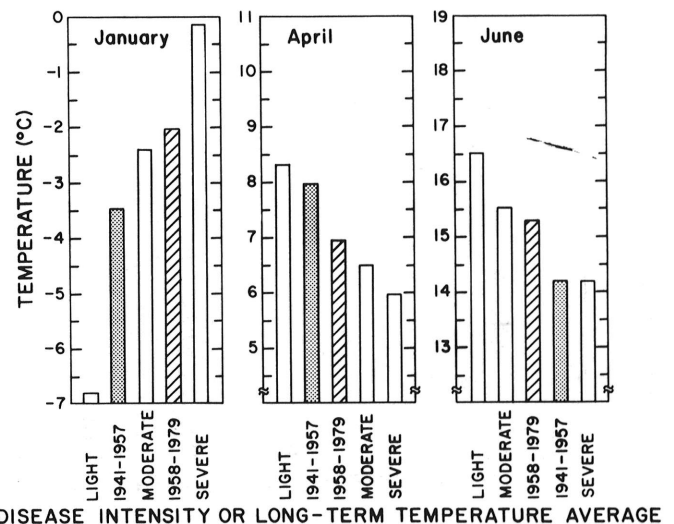


Fig. 3. Average temperatures for January, April, and June at Pullman, WA, for years during 1963–1979 when stripe rust was light, moderate, or severe, respectively, on Gaines winter wheat and for 1941–1957 and 1958–1979.

decrease in mean temperature. Between 1941 and 1957, the upper limit was 9.43 C and six of 15 springs had average temperatures  $\geq 8.21$  C. Since 1961, the highest spring temperature was 8.21 C. This decrease in variability of spring temperatures was even more evident at Walla Walla, WA, which has data available since the year 1900 (Fig. 5D).

The mean summer temperature (June, July, August) increased since 1958 (Fig. 5C). Fourteen of 15 summer temperatures between 1941 and 1957 were below the 1958–1978 mean. Since 1958, 18 of 22 summers had temperatures above the 1941–1957 mean.

Although average fall temperatures (September, October, November) decreased from a mean of 9.02 C during the 1941–1957

TABLE 2. Ranges of cumulative negative degree days (between 1 December and 31 January) and positive degree days (between 1 April and 30 June) associated with the development of different stripe rust intensities

Disease intensity	Disease index	Negative degree days <sup>a</sup>		Positive degree days <sup>a</sup>	
		Gaines	Omar	Gaines	Omar
Light	<3.5	>570	>560	>460	>460
Moderate	>3.5–<5.4	>470–<630	>500–<710	>360–<470	>420–<560
Severe	>5.5	<500	<630	<440	<470

<sup>a</sup>Calculated as the absolute value of daily average temperature – 7 C.

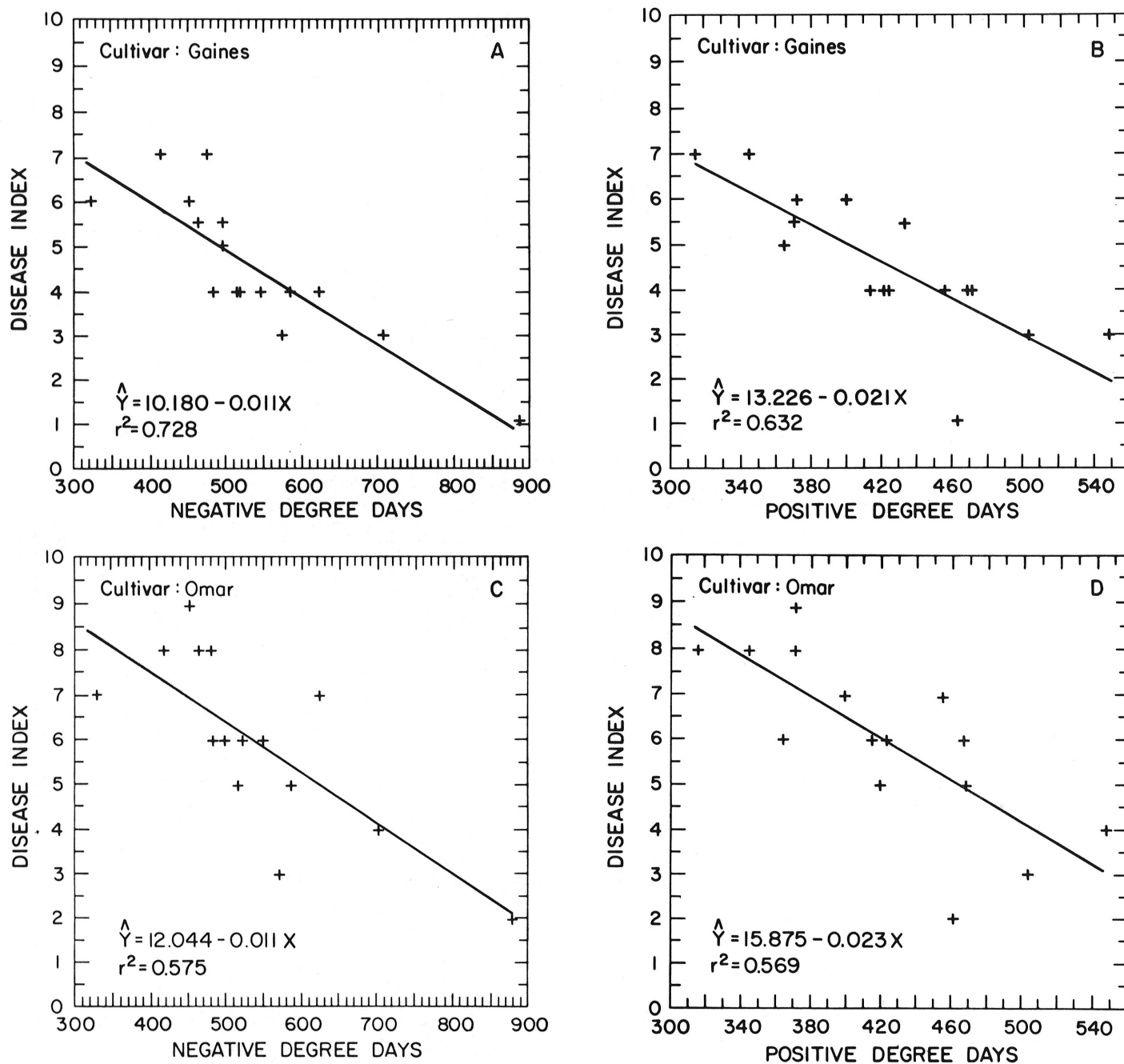


Fig. 4. Stripe rust index on winter wheat cultivars expressed as a function of negative degree days for A, Gaines and C, Omar and expressed as a function of positive degree days for B, Gaines and D, Omar. Disease and meteorological data were collected at Pullman, WA, for 16 yr between 1963 and 1979. Negative degree days were the total accumulated from 1 December to 31 January with a base of 7 C, and positive degree days were the total accumulated from 1 April to 30 June with a base of 7 C.  $\hat{Y}$  = predicted disease severity index, X = negative or positive degree days, and  $r^2$  = correlation coefficient squared.



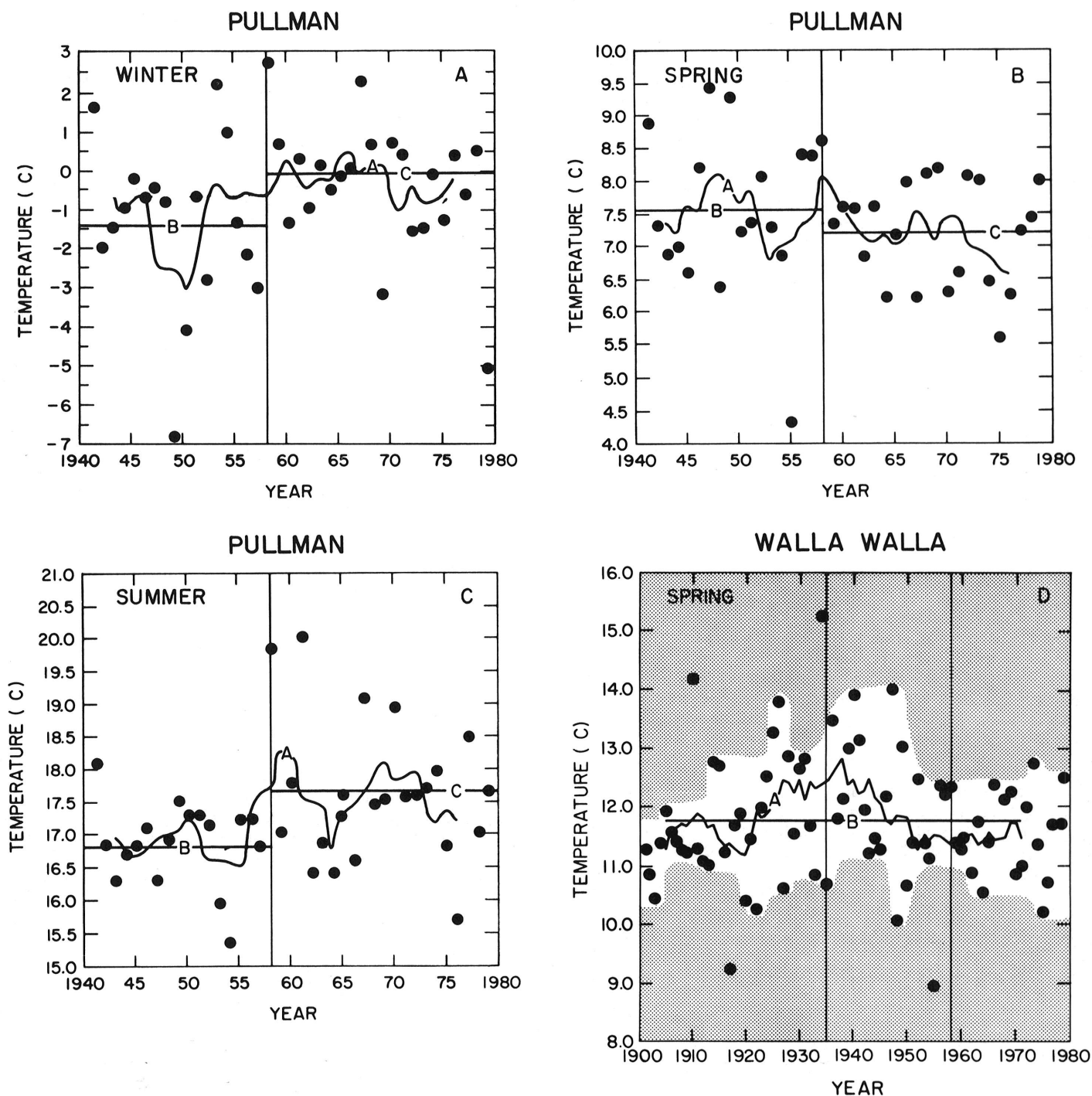
period to a mean of 8.82 C for 1958–1978, no marked temperature trends were evident.

January, February, April, and June temperatures differed more than 1 C between the two periods. Average temperatures in January, February, and June were, respectively, 1.81, 1.70, and 1.14 C higher for 1958–1978 than for 1941–1957. In contrast, the average April temperature for 1958–1978 was 1.03 C lower than that for 1941–1957.

The January, April, and June temperature averages for 1941–1957 and 1958–1979 were compared with the mean temperatures associated with severe, moderate, and light stripe rust intensity on Gaines for 1963 to 1979 (Fig. 3). For January and April, the 1941–1957 means were associated with a lower disease

intensity than were the comparable means for 1958–1979. The average January temperatures for 1941–1957 and 1958–1979 were, respectively, 3.34 C and 1.90 C below the mean temperature associated with severe disease. In April, the average temperature during 1958–1979 was 0.97 C higher than that associated with severe disease, but was 1.03 C lower than the average temperature for 1941–1957. The average June temperature for 1958–1979 was 1.09 C higher than the June average during 1941–1957. Average June temperatures for both time periods were between those temperatures associated with moderate and severe disease (Fig. 3).

Total precipitation for each season differed  $\leq 6.6$  mm between the two time periods. For winter, spring, summer, and fall, respectively, that was less than 3, 5, 10, and 5% of the total



**Fig. 5.** Seasonal temperatures at Pullman, WA, 1941–1979 for: **A**, winter (December, January, and February); **B**, spring (March, April, and May); and **C**, summer (June, July, and August); and at **D**, Walla Walla, WA, 1901–1975 for spring (March, April, and May). The 5-yr moving average at Pullman and the 9-yr moving average at Walla Walla (Line A) indicate temperature trends. Line B represents the average for 1941–1957 at Pullman and the average for 1901–1975 at Walla Walla. Line C represents the average for 1958–1978 at Pullman.

precipitation that occurred in those seasons during 1941 to 1957. Total precipitation for May and October 1958–1978 was 11.8 and 10.9 mm, respectively, lower than the average of 47.9 mm in May and 49.58 mm in October for 1941–1957.

To determine whether the frequency of years with degree days associated with severe, moderate, and light rust had changed since 1958, we calculated NDD and PDD values for 1941–1979. The percentage of years falling in each degree day range corresponding to each disease intensity (see Table 2) were calculated for 1941–1957 and 1958–1979 for Gaines and Omar (Fig. 6). Because the range boundaries overlap, some years were counted in two different degree day ranges and hence the total percent of years falling in the three disease categories were greater than 100% for each time period. For both Gaines and Omar, the percentage of years that could have a severe disease intensity increased from the 1941–1957 period to the 1958–1979 period. Using NDD ranges for estimating disease intensity on Gaines during 1941–1957, a maximum of 25% of the years could have had severe disease; in contrast, during 1958–1979, 55% of the years had a sufficiently low number of NDD to allow development of severe disease. The maximum number of years for light disease on Gaines decreased from 44% in 1941–1957 to 23% of the years in 1958–1979. Hence the 1941–1957 period was not favorable for frequent severe disease development on temperature-sensitive cultivars. Sixty-nine percent of the years between 1941 and 1957 and 91% of the years between 1958 and 1979 had NDD that would allow severe disease on Omar, and 60% in 1941–1957 and 82% in 1958–1979 had PDD corresponding to severe disease on Omar (Fig. 6).

## DISCUSSION

Severity of stripe rust on wheat in the PNW fluctuates from year to year, and since 1958, severe epidemics have been more frequent. The fluctuations and more frequent epidemics cannot be attributed only to changes in the cultivars or management methods. Several cultivars grown extensively in the period from 1941 to 1957, such as Elgin, Elmar, and Golden, are as susceptible as Omar, and since 1962, the more resistant cultivars with temperature-sensitive, adult-plant resistance have been predominantly grown in the region (4). Many cultivars grown prior to the 1950s also have various degrees of the high-temperature, adult-plant resistance (R. F. Line, unpublished).

We believe that the lower winter and higher spring temperatures in 1941–1957 reduced the frequency and severity of stripe rust epidemics and that a return to the 1941–1957 environment would reduce the severity of stripe rust in the future. Our hypothesis also helps to explain the infrequent occurrence of stripe rust in the central USA where winter temperatures are lower and the southeastern USA where spring temperatures are higher.

Since 1958, the trend has been toward higher winter temperatures. All years with severe stripe rust have had mean winter temperatures above the 1941–1957 mean. From 1963 to 1979 rust severity varied greatly, and that variation correlated directly with winter temperatures. Of the winter months, January temperature was the most highly correlated with disease index (Table 1). The high temperatures since 1958 would have favored survival of overwintering inoculum for most years and may even have allowed some fungal growth. The mycelium can survive in the host plant unless infected leaves of the host are killed by low temperatures (5,9). Snow cover may modify survival, but when temperatures are very low for several weeks, such as in 1969 and 1979, snow cover is not adequate for protection of wheat foliage and most of the inoculum is eliminated.

Low spring temperatures were more highly correlated with the disease index in Gaines than in Omar, and disease development in both Gaines and Omar had the greatest negative correlation with April temperatures. The low temperatures not only affect resistance of temperature-sensitive cultivars but also retard growth of the wheat plant, thus providing a longer period for the increase of rust intensity.

Years with severe stripe rust had summer temperatures lower than the 1958–1978 mean, but of the summer months, only June temperatures had a significant negative correlation with disease intensity. Because the response of Omar was similar to the response of Gaines, it seems probable that the activity of the fungus may be limited by the higher temperatures in June. Data (R. F. Line, unpublished) collected under controlled temperatures agree with this interpretation. In most years, winter wheat is in the dough stage of growth by mid-July and ripens in August. Rust develops more slowly after the dough stage; therefore, July and August temperatures had little effect on rust epidemics.

The fall climate does not appear to have a major effect on stripe rust epidemics. No fall climatic conditions were significantly correlated with subsequent disease development, and our observations since 1968 indicate that available inoculum during the fall has not limited the development of stripe rust epidemics in the PNW.

Tollenaar and Houston (8) stated that stripe rust epidemics in coastal valleys of California were closely related to winter rainfall. Frequent precipitation in the spring was reported to be important to the development of stripe rust epidemics in the PNW (3). However, our results indicate that except under drought conditions, no precipitation parameter was sufficiently correlated with disease intensity to be useful for predicting disease intensity. Precipitation frequency in June was significantly correlated with disease index (Table 1), but precipitation frequency was more highly correlated with mean temperatures in June than with disease index. It appears that differences in disease intensity from year to year may be directly related to temperature and only indirectly related to precipitation frequency.

The equations in Fig. 4 are proposed as tentative models for predicting rust intensity on cultivars similar to Gaines and Omar. The models may be more useful for predicting disease extremes than for predicting intensities in the middle ranges. Accuracy can be increased by using a combination of both NDD and PDD models. For example, in 1979, the greatest number of NDD (887) and the lowest disease index (1.0) since 1963 occurred; however, total PDD in the spring were 462 and the PDD regression equation predicted a final disease index of 3.52. This shows that in years with exceptionally low winter temperatures, that subsequent spring temperatures had less effect on disease intensity. When winter temperatures were not limiting, spring temperatures were more important. In 1975, ~480 NDD accumulated, which would result in a predicted index of ~4.9. However, the spring of 1975 had the fewest PDD (314) of any year and the disease index on Gaines was 7. This demonstrates that a long period of low temperatures in the spring can compensate for a moderate number of NDD in the winter by reducing adult-plant resistance and delaying wheat maturity, thus providing a maximum period for disease development.

NDD occur early in the growing season and therefore are more

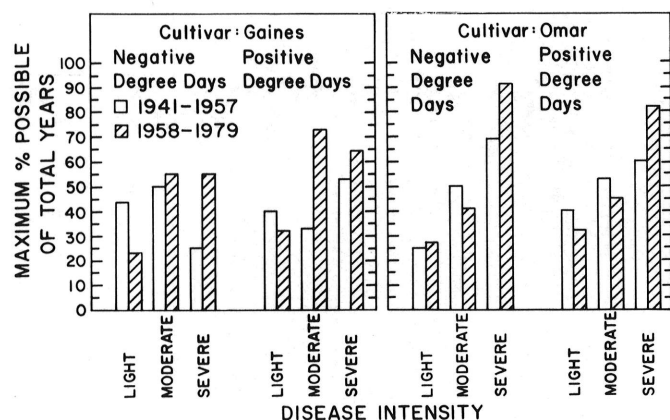


Fig. 6. Maximum percentage of years in 1941–1957 and 1958–1979 that could have light, moderate, and severe stripe rust on winter wheat cultivars Gaines and Omar. Data are based on the relationship between rust severity and the range of negative degree days in the winter and positive degree days in the spring in 1963–1979.

useful for forecasting subsequent disease development. Less than 500 NDD allow development of severe rust on Gaines. If  $\leq 500$  NDD accumulate, a grower might consider application of chemicals or replanting with an alternative crop. The decision might be modified depending upon whether the cultivar has temperature-sensitive resistance. PDD are less useful to the grower for making management decisions because total PDD are not known until July 1 when the most important phase of disease increase has already occurred.

Changes in NDD or PDD affect rust on Omar and Gaines in a similar way, but rust on Gaines is affected by fewer NDD and PDD (Table 2). Perhaps rust on Gaines is limited by fewer NDD because after low winter temperatures reduce the potential inoculum, the short spring period of susceptibility for Gaines does not allow sufficient time for rust to increase.

Nugaines has even greater temperature-sensitive resistance (4) than Gaines and spring temperatures need not be as high for expression of its resistance. The difference between rust intensity on Gaines and Nugaines was evident in most years. However, in 1975 and 1976 when spring temperatures were unusually low, rust intensities on both cultivars were similar (Fig. 1). In 1975 and 1976, cultivars that had greater temperature-sensitive resistance continued to have low rust intensities.

Data not presented here (R. F. Line, *unpublished*) indicate that disease intensity on temperature-sensitive cultivars is slightly higher in the small plots than in large plots or fields, but that the highly susceptible or highly resistant cultivars respond the same regardless of plot size. When fields of susceptible cultivars are in the region, disease intensities on fields of temperature-sensitive cultivars are similar to intensities in small plots. Therefore, our disease measurements on Gaines might be slightly higher than the disease intensity that actually occurred in some commercial fields, but in years with severe rust the differences are negligible. In 1976, rust was as severe in large fields of Nugaines as it was in small plots. Under those low spring temperatures, Nugaines was more susceptible and behaved more like Omar. In the future, the disease index scale can be adjusted if necessary by comparing disease measurements in small plots with those in commercial fields.

Developing models for other locations is often hindered by insufficient disease intensity data. We have data from other locations in the PNW which we plan to use to test the model developed for Pullman. Preliminary results from comparable regression equations for Walla Walla, WA, indicate that the slopes of the lines are similar to slopes for Pullman, which indicate that the models may have wider application.

Some studies by Line have been made and others are presently in

progress to determine the relationship of yield losses to rust intensity in various environments and in different years. Severe rust reduces yield by 20–50% (see Fig. 3), moderate rust reduces yield 10–20%, and light rust reduces yield less than 10% (R. F. Line, *unpublished*). If climatic conditions can be used to estimate rust intensity, then by using data on the relationship of losses to rust intensity we may be able to use climatic data to predict losses caused by rust.

The results support the earlier hypothesis that climatic variation has increased the frequency and severity of stripe rust epidemics in recent years (1), and they provide models for describing the relationship of disease intensity to temperature changes. The methods described in this paper should be applicable to studies of other diseases that have adequate data bases. Climatic variation is a normal phenomenon (6) and in the future, accurate long-term weather forecasts may become available; if the climate/disease relationship has been defined, phytopathologists could use the climatic forecast to predict disease occurrence.

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