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Disease progression in wheat lines and cultivars differing in levels of resistance to common root rot

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The expression of resistance to common root rot in spring wheat was studied by observing the disease development on resistant and susceptible cultivars and lines throughout the growing seasons of 1984-86. Disease incidence and severity on subcrown internodes increased rapidly 25-30 days after seeding, but disease development occurred more slowly in resistant than in susceptible cultivars. The average disease incidence in resistant and susceptible lines over all three years was 51.5% and 78.2%, respectively. The average disease severity of the resistant and susceptible lines over all three years was 3.8% and 30.6%, respectively. Changes in disease incidence were affected by moisture-related parameters when moisture was limiting and by temperature-related parameters when moisture was not a limiting factor. Changes in disease severity were most affected by temperature-related parameters.

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L'expression de la résistance au piétin commun a été étudiée pendant trois saisons de croissance chez des cultivars et des lignées de blé de printemps résistants ou sensibles à la maladie. Vingt-cinq à 30 jours après le semis, on a observé une augmentation rapide de la fréquence et de la gravité de la maladie sur les entre-noeuds sous le collet; l'évolution de la maladie était cependant plus lente chez les cultivars résistants que chez les cultivars sensibles. La fréquence de la maladie a atteint une valeur moyenne de 51,5% chez les lignées résistantes et 78,2% chez les lignées sensibles. La gravité de la maladie s'est élevée en moyenne à 3,8% chez les lignées résistantes et 30,6% chez les lignées sensibles. La fréquence de la maladie a varié en fonction des paramètres pluviométriques en période de sécheresse et en fonction des paramètres liés à la température en l'absence de stress hydrique. La gravité de la maladie a été affectée principalement par les paramètres liés à la température.

Resistance in spring wheat to common root rot, caused primarily by *Cochliobolus sativus* (Ito & Kurib.) Drechsl. ex Dastur, anamorph *Bipolaris sorokiniana* (Sacc. in Sorok.) Shoem., synonym *Helminthosporium sativum* Pamm., King and Bakke has been demonstrated in several studies (Sallans & Tinline 1965, 1969; Stack 1982, Bailey et al. 1988). Bailey et al. (1988) reported that the heritability of common root rot resistance ranged from moderately low to moderately high in the progeny of crosses between resistant and susceptible lines. Therefore, a considerable proportion of the variance may be attributed to environmental factors. Casual field observations suggest disease severity increases with soil temperature and low water-holding-capacity of the soil (Machacek 1943, Sallans & Ledingham 1943). The correlation between common root rot ratings and June-July rainfall was -0.63 and between common root rot ratings and air temperature, 0.55 (Sallans 1948). Previous studies have not examined the differences in disease progression of resistant and susceptible cultivars (Verma et al. 1974, Stack 1982, Kidambi et al. 1985). Stack (1982) did examine the changes in susceptibility at different stages of plant development but concluded that no effects could be shown. The goal of the present study was to determine the time at which resistance was expressed during plant development and to examine the effects of different environmental conditions on disease progression in resistant and susceptible spring wheat lines and cultivars.

Materials and methods

The experiments were conducted in a root rot disease nursery under dryland conditions at the Agriculture Canada Experimental Farm, Saskatoon, Saskatchewan, in 1984 and 1985. In 1986, the experiment was grown under irrigation on land previously cropped to wheat and barley for at least 4 years at the University of Saskatchewan, Saskatoon. Although this site was not part of a disease nursery, soil samples taken at the beginning of the season indicated an equivalent number of *C. sativus* conidia per gram of soil to that found in the disease nurseries. The experimental design was a split plot, with time of sampling as the mainplot factor and wheat lines as the subplot factor. Treatments were replicated four times in 1984 and six times in 1985 and 1986. The common root rot resistant lines H-159 and H-105, the intermediate cultivar Neepawa, and the susceptible line H-186 were planted in 1984 and 1985. The cultivar Leader was also included in 1985 as another susceptible check. In 1986, only three cultivars were planted (H-159, Neepawa, and Leader) to reduce the size of the experiment.

Each plot consisted of 100 seeds, sown 6 cm deep in a 2-m row. The rows were spaced 35 cm apart. The plots were seeded on May 22 in 1984, May 22 in 1985, and May 20 in 1986.

At each sampling date, 40 plants were pulled from the center of a plot for the evaluation of common root rot symptoms. Due to the destructive nature of sampling, different plots were evaluated at each sampling time over the course of a season. In 1984, samples were collected seven times and in 1985 and 1986, 15 times. Samples were collected twice a week for the first month starting June 10 and either once a week (1985 and 1986) or once every two weeks (1984) to maturity. The Horsfall-Barratt grading system (Horsfall and Barratt 1945) was used to estimate disease severity and incidence. Disease severity was estimated as the area on the subcrown internode that was covered by black-brown lesions. The scores were transformed to corresponding disease severity ratings (%) (Redman et al. 1969). Disease incidence was expressed as the percentage of infected plants. In this study, an infected plant was considered to have a graded score greater than one. The mean disease severity (%) and the disease incidence (%) were calculated for each row.

Disease progress curves were developed for both disease severity and disease incidence. Disease incidence values recorded during the period of exponential growth were transformed using the formula $\log_e(1/1\text{-}x)$ (Van der Plank, 1963). The transformed values (Y) were fitted to the linear regression equation ($Y = A + BX$), using the number of days after seeding as the independent variable (X). The slope of the regression line was the average daily rate of visible infection. The slopes of the regressions for all cultivars were tested for significant differences by t-tests using the standard error of the difference of infection rates at $P = 0.05$.

Local weather data (daily precipitation, soil temperatures at 5-cm depth, and growing degree days above 5°C) were obtained from the Saskatchewan Research Council, Saskatoon, Saskatchewan. These measurements were recorded in an area approximately 4 kilometres from the experimental sites. Stepwise multiple regression procedures (SAS User's Guide, Version 5, 1985: Statistics, SAS Institute, Box 8000, Cary, NC 27511-8000) were used for exploratory model analysis to see which environmental variables were significantly related to changes in disease incidence and changes in disease severity. The environmental variables tested were change in total precipitation accumulated during one sample period minus the total accumulated in the following sample period (CPPTE), average precipitation between sampling dates (AVEPP), change in degree days (CDD), air temperature on sampling date (AT), change in soil temperature (CST), average soil temperature between sampling dates (AVEST), and actual soil temperature on the sampling date (ACTST). The multiple stepwise regression model was used with significant F-tests at $P = 0.01$ and $P = 0.10$. The lower significance level was used to guard against including any variables that do not contribute to the predictive power of the model, since the probability of rejecting a true null hypothesis is greater than 1% (SAS User's Guide, Version 5, 1985). The moderately larger significance level was used to identify which variables may be important but have a smaller predictive value to the model.

Results and discussion

Analysis of variance indicated that year, sampling date, cultivar, and most interactions were highly significant at $P = 0.01$ for both disease incidence and severity (Table 1). Separate analyses of variance for each year indicated that replicates were not significantly different in 1984 and 1985 for disease incidence but were highly significant in 1986, which was the result of the mean of one replicate being less than the means of the other replicates. In 1986, the cultivar × date interactions were not significantly different for disease incidence and severity, whereas in the other years these interactions were significant.

Table 1. Analysis of variance of disease incidence and disease severity for the progression of common root rot on wheat lines sampled on the same number of days after seeding in 1984, 1985, and 1986

Source	Degrees of freedom	Disease incidence mean square	Disease severity mean square
Year	2	8980.6 **	1737.3 **
Year × rep	13	587.1 **	56.3 NS
Date	5	28504.7 **	2466.1 **
Date × year	10	3134.9 **	728.9 **
Date × year × rep	65	403.8 **	46.0 **
Line	4	6226.2 **	1871.5 **
Line × date	20	720.7 **	349.4 **
Line × year	5	1091.3 **	226.3 **
Line × date × year	25	567.3 **	129.3 **
Error	232	223.1	27.9

**significant at $P = 0.01$

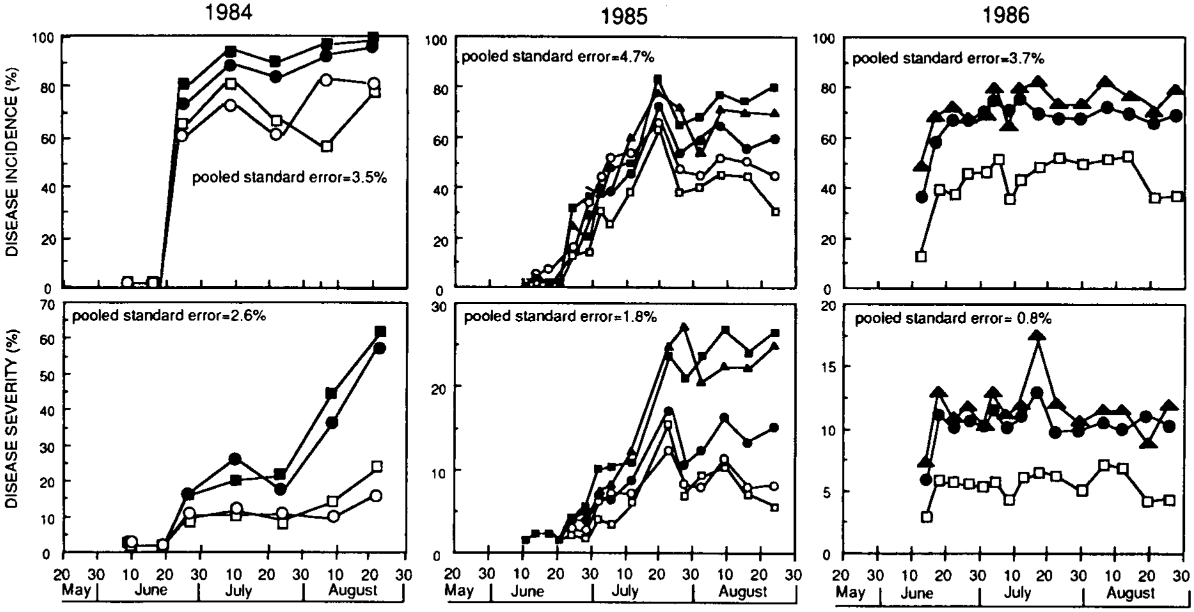

Figure 1. Disease incidence and disease severity of common root rot resistant (○ H-105 □ H-159), intermediate (● NEEPAWA), and susceptible (■ H-186 ▲ LEADER) lines in 1984, 1985, and 1986.

In 1984 and 1985, disease symptoms did not become apparent until the third sampling date, i.e. approximately 4 weeks after seeding (Fig. 1). In 1986, symptoms were already apparent at the first sampling date, June 17, 28 days after seeding. The average disease incidence in the resistant lines was lower than that for the susceptible lines in each year (Fig. 1). This difference became apparent in late June-early July and increased to a maximum by late July or August. The average disease incidence in resistant and susceptible lines over all three years was 51.5% and 78.2%, respectively. Similarly, the average disease severity of resistant lines over all three years (3.8%) was lower than that for susceptible lines (30.6%). Generally, the difference between resistant and susceptible lines was at its maximum level by mid- to late season.

In 1984, an increase in disease severity during July and August occurred two weeks later in the resistant lines than in the susceptible lines (Fig. 1). Subsequently, the curves for resistant and susceptible cultivars were parallel. Changes in disease severity were probably a function of lesion expansion, since disease incidence did not change. However, the role of sequential infection by late-germinating conidia or of reinfection by secondary conidia is not clear.

The shapes of the disease progress curves were similar to those observed by others (Verma et al. 1974, Stack 1980, Kidambi et al. 1985). However, all the curves in this study could be described equally as well by the simple or compound interest transformations (Van der Plank 1963). Therefore no interpretation of the infection process as manifested by the shape of these curves was attempted.

The average infection rates based on disease incidence values for each year were 8.3% in 1984, 3.5% in 1985, and 4.3% in 1986 (Table 2). Except for the comparison of the average daily infection rates of H-105 and Neepawa in 1984, there were no significant differences among the resistant and susceptible lines tested, despite the tendency of the resistant lines H-159 and H-105 to have lower infection rates. The coefficients of correlation (R^2) were low in 1986. This high variability about the regression line probably reflects the fact that different personnel were responsible for making disease ratings compared to the previous two years.

The weather conditions were different in each of the three years. The accumulation of degree days above 5°C was greater in 1984 than in 1986 and 1985 (Fig. 2). The air temperature in 1985 was lower than in the other two years, whereas the air temperatures in 1984 and 1986 were similar until the latter part of July, when the 1984 temperatures were much higher. Soil temperatures on June 1 at 5-cm depths were 13°C, 13°C, and 21.5°C in 1984, 1985, and 1986 (Fig. 3). In 1984, soil temperature peaked at 28.5°C on July 28. In 1985, it peaked at 26.0°C on July 11. The soil temperature was lower during the latter part of the summer in 1985 than in

Table 2. The average daily infection rates (regression value = b) and the coefficient of correlation (R^2) of the regression of $\log_e(1/1\text{-}x)$ on disease incidence values for the resistant and susceptible lines tested in 1984, 1985, and 1986

	1984			1985			1986		
Line	Infection rate (b)	S.E.	R^2	Infection rate (b)	S.E.	R^2	Infection rate (b)	S.E.	R^2
H-159	0.074	0.010	0.83	0.032	0.004	0.55	0.027	0.007	0.35
H-105	0.055	0.015	0.54	0.036	0.004	0.60	NT		
H-186	0.093	0.021	0.70	0.040	0.005	0.60	NT		
Leader	NT			0.029	0.003	0.65	0.038	0.011	0.48
Neepawa	0.109	0.009	0.93	0.040	0.004	0.70	0.043	0.017	0.19
Pooled degrees of freedom		22			104			56	

S.E. = standard error.

NT = cultivars or lines not tested in those years.

1984. In 1986, soil temperature remained lower and relatively constant throughout the summer, with no clear peak.

The experiments were grown under dryland conditions in 1984 and 1985. Most of the 1984 precipitation was in June (Fig. 3). In 1985, the spring was quite dry but heavy rains occurred in mid July. In 1986, the experiment was grown on land that received overhead irrigation water (25 mm/day) on June 17, July 2, and July 11. Precipitation was more regular throughout June and July, and in the first week of August than in previous years.

Not all the environmental variables measured with stepwise regression had significant effects on the changes in incidence and severity of disease, nor were the same environmental variables significant in every year (Table 3). In 1984 and 1985, the environmental variables that accounted for the greatest variation in change in disease incidence

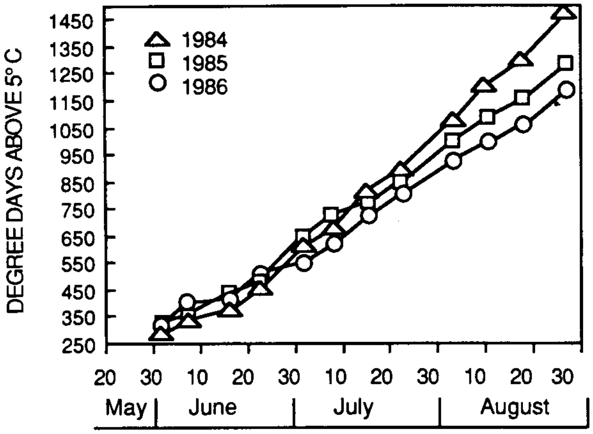

Figure 2. Growing degree days above 5° C accumulated between May 30 and August 25 in 1984, 1985, and 1986.

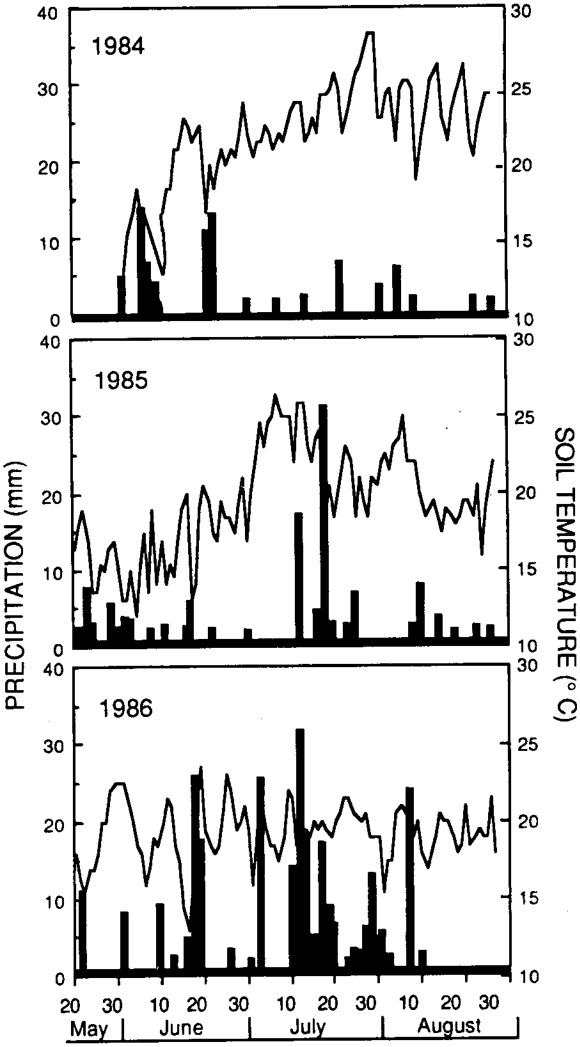

Figure 3. Precipitation (▮) and soil temperature (—) at 5 cm from May 20 to August 25 in 1984, 1985, and 1986.

Table 3. Multiple stepwise regression analysis of the dependent variables, change in incidence and change in severity on the independent environmental variables from 1984 to 1986

Change in incidence				Change in severity			
Independent variable	Regression value (b)	S.E.	Partial R^2	Independent variable	Regression value (b)	S.E.	Partial R^2
1984							
AVEPP	134.4 **	22.1	0.81	AT	1.7 **	0.6	0.35
CPPTE	-16.6 **	3.2	0.07				
CDD	0.3 **	0.1	0.06				
1985							
CPPTE	1.6 **	0.3	0.26	CDD	0.1 **	0.02	0.41
AVEPP	-17.8 **	2.7	0.04	CPPTE	0.4 **	0.1	0.14
AT	-1.4 *	0.5	0.05	AVEPP	-3.9 **	0.7	0.04
				CST	-0.01 *	0.01	0.03
1986							
ACTST	-4.1 **	0.8	0.23	ACTST	-1.2 **	0.2	0.28
AVEST	5.0 **	1.7	0.21	CPPTE	0.1 **	0.03	0.10
CST	-0.1 **	0.04	0.08	AT	-0.02**	0.01	0.09
CPPTE	0.2 *	0.04	0.04	CST	-0.03 **	0.01	0.09
				AVEPP	-0.2 *	0.1	0.04

*significant at $P = 0.10$, **significant at $P = 0.01$.
AVEPP = average precipitation between sampling dates, CPPTE = change in precipitation, CDD = change in degree days, AT = air temperature on sampling date, CST = change in soil temperature, AVEST = average soil temperature between sampling dates, ACTST = actual soil temperature on sampling date.

were moisture-related parameters (AVEPP $R^2 = 0.81$ and CPPTE $R^2 = 0.26$), whereas in 1986, they were temperature-related parameters (ACTST $R^2 = 0.23$ and AVEST $R^2 = 0.21$). Temperature-related parameters accounted for the greatest variation in changes in disease severity for all years (AT $R^2 = 0.35$, CDD $R^2 = 0.41$, and ACTST $R^2 = 0.28$).

Stepwise regression procedures showed that environmental factors affect the shape of the disease progress curves. It was not possible to conclude which of the factors were the most important over all three years. However, a trend was noted. In 1986, when the June-July precipitation was greater than in the previous two years, the common root rot ratings were low. The air temperature was higher in 1984 and 1985 than in 1986, and disease severity was also greater. These observations are similar to those of Sallans (1948). Changes in incidence (i.e. the infection process) were most affected by moisture-related conditions but when moisture was not a limiting factor, as in 1986 when the experiment was conducted under irrigation, soil temperature-related parameters were more significant. Changes in disease severity (i.e. lesion expansion) were most affected by temperature-related parameters. These observations indicate that moisture is the primary requirement for infection to occur. When moisture is available, soil temperature becomes critical for the infection process. Lesion expansion appears to be temperature dependent.

Resistance to common root rot is a heritable trait (Bailey et al. 1988). A consistent, distinct separation of disease ratings between resistant and susceptible genotypes is necessary for selection. Traditionally, the selection for common root rot resistance has been done at plant maturity. Stack (1981) reported a poor correlation ($r = 0.20$) between the disease on wheat seedlings and on adult plants under greenhouse and field conditions. The data from the present study support his conclusions. Resistant and susceptible lines could not be distinguished on the basis of disease incidence and severity during the first 30 days of plant growth, although after 30 days differences were observed readily. This is of practical importance since it allows a greater time-span for identifying resistant breeding lines than when selections are made only at maturity.

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