Environmental Influence on the Infection of Wheat by *Mycosphaerella graminicola*

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ABSTRACT

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Mycosphaerella graminicola (anamorph, *Septoria tritici*), the causal agent of Septoria tritici blotch on wheat, can cause a significant yield loss for most commercially grown wheat cultivars and in many wheat-growing areas. Environmental factors which affect infection and disease development such as light intensity, temperature, and moisture have been studied extensively. Yet, reports on specific effects of temperature and leaf wetness period on the infection process are limited. The objectives of this study were to quantify the effect of temperature and duration of leaf wetness and their interaction during the infection process on major disease components, to study the dynamic characteristics of these disease components, and to derive a mathematical expression for each disease component, as a function of infection temperature and leaf wetness period. Results showed that temperature is a critical determinant of wheat infection by *M. graminicola* and that the optimum temperature for the infection process does not depend on leaf wetness period. Furthermore, the initial conditions of temperature and leaf wetness period following inoculation not only affect the rate of disease development but also the asymptotic level of disease severity that ultimately may be achieved. Except for lesion length, the maximum rate of increase in all disease components responds linearly to changes in leaf wetness period, and quadratically to changes in temperature. The rate of lesion length expansion is mainly influenced by postinfection conditions. Of the four components of Septoria tritici blotch of wheat studied, lesion unit (number of average-sized lesions) per square centimeter is the most sensitive indicator of a successful infection.

Mycosphaerella graminicola (Fuckel) Schroeter (anamorph: *Septoria tritici*), the causal agent of Septoria tritici blotch on wheat, is a pathogen of worldwide importance (6,22) and causes a significant yield loss in many wheat-growing areas (6,7,16,18). Studies show that certain weather patterns such as wet weather and moderate temperature favor disease development (4,10,13, 18,20). Moisture in the form of rain or dew is required for successful infection (2,8,14) and plays an essential role in all stages of the infection cycle involving the anamorph (5,8,11,13–15). In the laboratory, conidial germination occurs on moist leaves within 12 h and penetration occurs only after 24 h (14). Royle et al (18) and Holmes and Colhoun (15) also reported that 12 h of leaf wetness is sufficient to allow infection to take place while Renfro and Young (17) found that 15 h of leaf wetness is minimal for infection. Hess and Shaner (12) and Shipton et al (21) reported that disease severity increases as the postinoculation moist period increases.

Previous investigators recognized the close association between moisture and infection and subsequent pathogen development in the host plant. Research into the specific effect of temperature and its interaction with moisture on the infection process, however, has been limited (15,17,23). Therefore, the objectives of this study were to quantify the effect of temperature and duration of leaf wetness and their interaction during the infection process on several disease components, study the dynamic characteristics of these disease components, and develop a mathematical relationship between each disease component and the environmental factors of leaf wetness period and infection temperature.

MATERIALS AND METHODS

Plant material. Spring wheat cultivar Anza, susceptible to *M. graminicola*, was used in this study. Anza was chosen because preliminary studies showed that the leaves of this cultivar could endure long periods of wetness without becoming chlorotic or senescing prematurely under the conditions employed herein. Twenty 15-cm-diameter pots, each containing four plants, were used for each temperature experiment. Each experiment was repeated three times. The plants were grown under greenhouse conditions and were moved to growth chambers one week before inoculation.

Inoculum preparation. *M. graminicola* isolate DRT, a single pycnidiospore isolate obtained from field-infected Anza wheat near Davis, CA, was used in all experiments. Preliminary studies with isolate DRT under controlled environment conditions confirmed that it was pathogenic on a range of cultivars including Anza. Inoculum was increased on a solid medium containing 9 g of yeast extract, 9 g of malt extract, 9 g of sucrose, and 12 g of agar in 900 ml of distilled water. The cultures, incubated 6–8 days in a 12-h photoperiod at 20 C, grew as the anamorph (*S. tritici*) and displayed yeastlike growth by formation of blastospores without development of pycnidia. Blastospores were washed from the agar surface and collected in a glass of distilled water. The inoculum concentration was adjusted with a hemacytometer to 10^6 spores per milliliter of water containing 1 ml of Tween 20 per liter. This inoculum concentration gave reproducible infection level based on preliminary studies of the effect of inoculum concentration on disease severity.

Infection chamber. A misting system was developed to provide a fine layer of moisture on the leaf surface required for infection. The major parts of this system included a 1/4J air atomizing nozzle consisting of fluid cap no. 2050 and air cap no. 73328 (Spraying Systems Co., Wheaton, IL), master timer, day timer, night timer, pressure regulator, liquid strainer, air filter, and two solenoid valves. The air atomizing nozzle was placed beneath the plants within the growth chamber to avoid direct spray on the plants, and the water was supplied to the nozzle under pressure. Air and deionized water were internally mixed to produce a completely atomized flat spray pattern. The spray nozzle was placed in such a way that the spray was aimed in the same direction as the airflow so that the smallest spray droplets would be carried by the air and deposited on the leaf surfaces. This technique maintained a uniform distribution of minute droplets on the leaf surfaces without runoff throughout the infection period.

Inoculation. Plants between growth stages 40 (flag leaves

emerged) to 59 (heads fully emerged) (25) were acclimated at 20 C and 12 h/day light intensity of 420 $\mu Em^{-2}s^{-1}$ in a growth chamber for one week before inoculation. The spore suspension was sprayed onto both adaxial and abaxial flag leaf surfaces using an atomizer until runoff occurred. After inoculation, plants were incubated in the infection chamber with periodic misting as previously described. After incubation periods of 15, 24, 48, 72, and 96 h, pots were transferred to the postinfection chamber and observed daily for lesion development. Typically, visible symptoms, consisting of vein limited necrotic area with pycnidia, appeared 9–12 days after inoculation.

Infection chamber conditions. Misting periodicity to maintain free moisture on leaves was predetermined empirically for each experimental temperature (10, 15, 20, and 25 C) because temperature and light influence the rate of condensation and evaporation of moisture on the leaf surface. The time required to produce a film of moisture on the leaf surface and the actual drying time of the leaf surface were determined for both day and night conditions. The average numbers of mistings required for 10, 15, 20, and 25 C were 3, 4, 8, and 12, respectively. This standardized periodic misting method prevented the leaf surface from being too wet, which would dilute the inoculum, or too dry, which could interfere with spore germination. The light intensity was maintained constant at 555 $\mu Em^{-2}s^{-1}$ for 12 h/day for all experiments.

Postinfection chamber conditions. The growth chambers were maintained at a constant 20 C, and at 75% relative humidity in a 12 h/day photoperiod with light intensity, 420 $\mu Em^{-2}s^{-1}$ during the postinfection period preceding symptom development.

Measurement of flag leaf area. The length and breadth (at six points) of the leaf blade were measured once on the first day of visible symptoms, and the data were used to calculate leaf area.

Disease assessment. To facilitate disease assessment, a permanent ink was used to mark the adaxial surface of each leaf so as to divide the blade into three sections. The following measurements were taken every 3–5 days from the time the first lesion appeared until complete senescence of the leaf. Keys or diagrams were not used.

Lesion length and width. The length of each sampled lesion was measured with a reticle metric scale. If there were more than 10 distinct lesions, then only 10 lesions were sampled from each section. If there were fewer than 10 distinct lesions, all were ex-

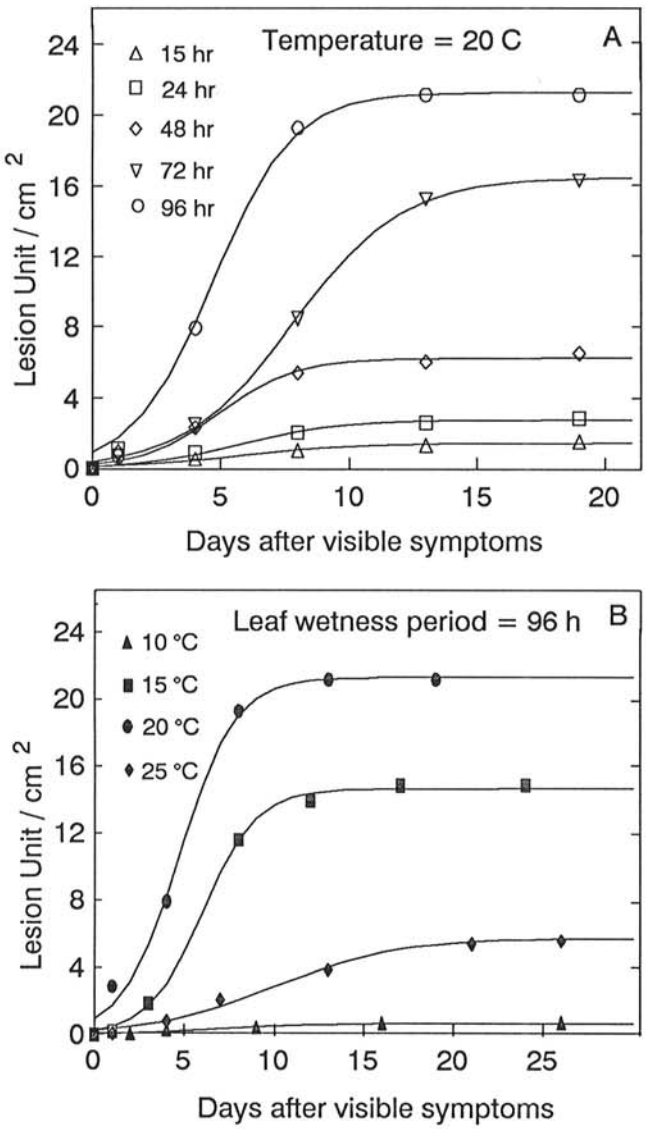

Fig. 1. The increase of lesion unit with time **A**, at fixed infection temperature (20 C) and different leaf wetness periods and, **B**, at fixed leaf wetness period (96 h) and different infection temperatures. The symbols on the graphs indicate different leaf wetness periods and different infection temperatures for **A** and **B**, respectively.

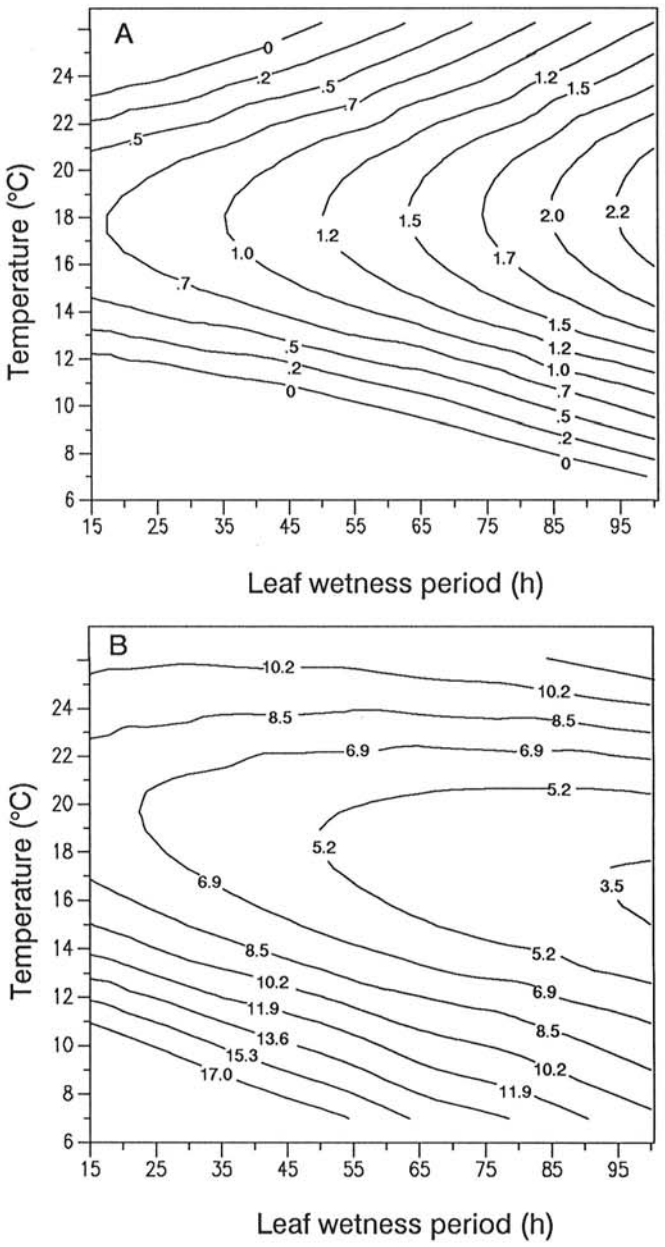

Fig. 2. Contour projection of **A**, maximum rate of lesion unit (lesions per square centimeter per day) and, **B**, time (days) required to reach the maximum rate of lesion unit as a function of temperature and leaf wetness period.

amined. The growth of *M. graminicola* within the leaf blade is usually restricted by vascular tissues (14), and lesions grow longitudinally between veins on the flag leaf. The lesion width is equal to the average distance between two adjacent veins (0.557 mm for spring wheat cv. Anza) multiplied by the number of veins involved.

Infected area. At an early stage of lesion development, the infected area was estimated from the combined area of all distinct lesions. When blotches formed, the length and width of each blotch (coalesced lesions) on each section were measured. The sum of the areas of all blotches and all distinct lesions was used as an estimate of the infected area. The percentage of infected area is called disease severity.

Lesion unit. At the early stage, all lesions were distinct and easy to count. But over time, the lesions coalesced to form blotches, and it became impossible to count individually or directly measure the individual expansion rate. In this study we adopted a measure of lesion development, namely lesion unit. We defined lesion unit as the value that results when the infected area is divided by the average lesion size at the time that the lesions could be measured individually. Lesion unit provided a scoring system which measured lesion area in terms of units of average lesion size. This scoring system proved useful in assessing the rate of lesion expansion in terms of nonoverlapping numerical units for modeling purposes. Necrotic lesions which developed pycnidia, defined as lesion units, did not exhibit marginal chlorosis and were typical of field symptoms observed at Davis, CA. Lesion unit per square centimeter is the total score of lesion units on a leaf divided by the leaf area.

Senescent area. Senescent tissues (natural senescence and pathogen-induced senescence) were visually distinguished from necrotic infected tissues due to the absence of pycnidia within the dead area. The length and width of each senescent area without pycnidia on each section was measured. The total area of senescence was the sum of individual senescent areas. The percentage of senescence is the percentage of senescent area on a leaf.

Data analysis. The four disease components defined in this paper are lesion unit per square centimeter, disease severity, senescence, and lesion length. The mean values of the disease components over time were fitted by a first-order logistic equation for each treatment condition:

$$Y = \alpha_0/(1 + \alpha_1 \exp(-\alpha_2 t))$$

where Y represents a disease component, t represents time (days) after symptoms become visible, and α_0, α_1, and α_2 are coefficients of the response function. These coefficients were estimated by a nonlinear estimation procedure (19) and were used to estimate additional rate and response characteristics. These characteristics include the maximum rate (MR), days to the maximum rate (DMR), the maximum response (MY), and days to the maximum response (DMY). They are calculated as follows: $MR = \alpha_0\alpha_2/4$; $DMR = \ln(\alpha_1)/\alpha_2$; $MY = 0.95(\alpha_0)$; and DMY is the time when $Y = 0.95(\alpha_0)$.

The effects of temperature and leaf wetness period on these characteristics were estimated by the fitted response function,

$$P = \beta_0 + \beta_1 T + \beta_2 W + \beta_{11} T^2 + \beta_{22} W^2 + \beta_{12} TW,$$

where P represents a characteristics of a disease component, T is temperature (C), W is leaf wetness period (h), and βs are regression coefficients.

The correlation analysis was performed to examine the relationships among the characteristics within and between disease components. A linear plateau model (1) was used to illustrate the relationship of the maximum lesion length and the disease severity. In this case, the R^2 was calculated as one minus the sum of the residual sum of squares of the two parts divided by the total sum of squares.

RESULTS

Lesion unit per square centimeter of leaf area. The lesion developmental curves of various leaf wetness periods under a fixed temperature of 20 C are shown in Figure 1A and of various temperatures with a fixed 96-h leaf wetness treatment in Figure 1B. The R^2 values of the fitted curves are greater than 0.97 in all cases. In general, lesion development increased with increasing leaf wetness period, but quadratically with increased infection temperature. The developmental rate of the lesion unit and the time to reach the maximum rate with respect to the temperature and humidity conditions are presented in Figures 2A and 2B, respectively. The maximum developmental rate, 2.2 units per day per square centimeter, which occurred at about 5 days after the first symptom, appeared under the conditions of 20 C and 96 h of leaf wetness. The maximum number of lesion units was 20.3 per square centimeter.

TABLE 1. Response surface coefficients for the parameters of disease components

Equation number	Parameters[a]	Response surface coefficients						
		β_0	β_1	β_2	β_{11}	β_{22}	β_{12}	R^2
Lesion unit per square centimeter								
1	MRLU	−5.8432	0.7266	0.0021	−0.0206	0.0001	0.0004	0.69
2	DMRLU	55.2912	−4.4902	−0.2831	0.1080	0.0005	0.0095	0.87
3	MLU	−31.7505	3.9459	0.0011	−0.1116	0.0004	0.0042	0.72
4	DMLU	109.2672	−9.1689	−0.4546	0.2340	0.0011	0.0122	0.91
Disease severity								
5	MRDS	−21.601	2.6498	0.0165	−0.0747	0.0001	0.0031	0.69
6	DMRDS	52.7072	−4.1812	−0.1278	0.116	0.0006	−0.0008	0.82
7	MDS	−118.4820	13.4271	0.5544	−0.3467	−0.0039	0.0178	0.89
8	DMDS	92.4982	−7.5394	−0.1299	0.214	0.0006	−0.0040	0.84
Lesion length								
9	MRLL	0.6742	0.0351	−0.0069	−0.001	0.0001	0.0002	0.20
10	DMRLL	13.6295	−0.9257	0.1302	0.036	−0.0001	−0.0077	0.38
11	MLL	34.2036	−2.5710	0.0030	0.0787	−0.0002	−0.0044	0.74
12	DMLL	48.0882	−3.6668	0.2605	0.118	−0.0007	−0.0137	0.44
Senescence								
13	MRS	−2.2068	0.2985	−0.0080	−0.0078	0.0001	0.0066	0.75
14	DMRS	28.1382	−1.4650	−0.1231	0.034	−0.0003	0.0070	0.23
15	MS	−0.7320	−0.1146	0.0605	0.0193	−0.0010	0.0120	0.91
16	DMS	76.5449	−5.3858	−0.1523	0.142	−0.0009	0.0104	0.38

[a] Keys to acronyms: M = maximum; D = days to maximum; R = rate of development; LU = lesion unit; DS = disease severity; LL = lesion length; and S = senescence.

The contour projection (Fig. 2A) of the response surface function of the maximum developmental rate of the lesion unit (equation 1 in Table 1) shows that the optimum temperature for maximum rates ranged between 16 and 20 C for all wetness periods. It also shows that the temperature range at which infection occurs increases when the leaf wetness period is prolonged. For example, when the wetness period was 25 h, the effective temperature range was 13–23 C; but when the wetness period was greater than 80 h, the effective temperature range increased to 9–25 C. The number of days required to reach maximum rates decreased linearly with increasing wetness period if the infection temperature was less than 21 C, and tended to increase linearly with increased wetness period if the infection temperature was above 22 C. The contour map (Fig. 2B) also shows that the number of days required to reach the maximum rate was more responsive to the infection temperature than to the wetness period.

Disease severity. A minimum of 30% disease severity developed when wetness periods were extended beyond 48 h and the temperature ranged between 15 and 25 C. Under the low temperature (10 C), disease did not develop to more than 11% severity at any level of wetness treatment. The disease development curves under different leaf wetness periods, but a fixed temperature of 20 C, and different temperature treatments, but a fixed wetness of 96 h, are shown in Figure 3. The effects of infection temperature and wetness period on the maximum rate of disease severity development and the time required to reach the maximum rate of disease severity development are shown in Figure 4. The R^2 values of these response functions were 0.69 and 0.82, respectively (equations 5 and 6 in Table 1). Generally the maximum rate exhibited a positive linear response to the increased length in wetness period but a quadratic response to the infection temperature. The development rate of disease severity reached the maximum at 18 C and ranged from 3.0 to 9.7% per day depending on the leaf wetness period. The optimum temperature range for the maximum rate was between 16 and 20 C. The time required to reach the maximum rate of disease development exhibited a negative linear response with the leaf wetness period and a quadratic relationship with the infection temperature. The optimum condition for the disease developmental rate was 18 C and 90 h, and the minimum time was 6.7 days after the symptoms appeared.

Lesion length. The dynamic expansion of lesion length under a fixed temperature of 20 C and a leaf wetness of 96 h are shown in Figure 5A and B. The estimated maximum lesion length decreased with increased wetness period at all temperatures except 10 C, where lesion length maintained at about 15 mm in all wetness periods. Within each temperature treatment, the maximum lesion

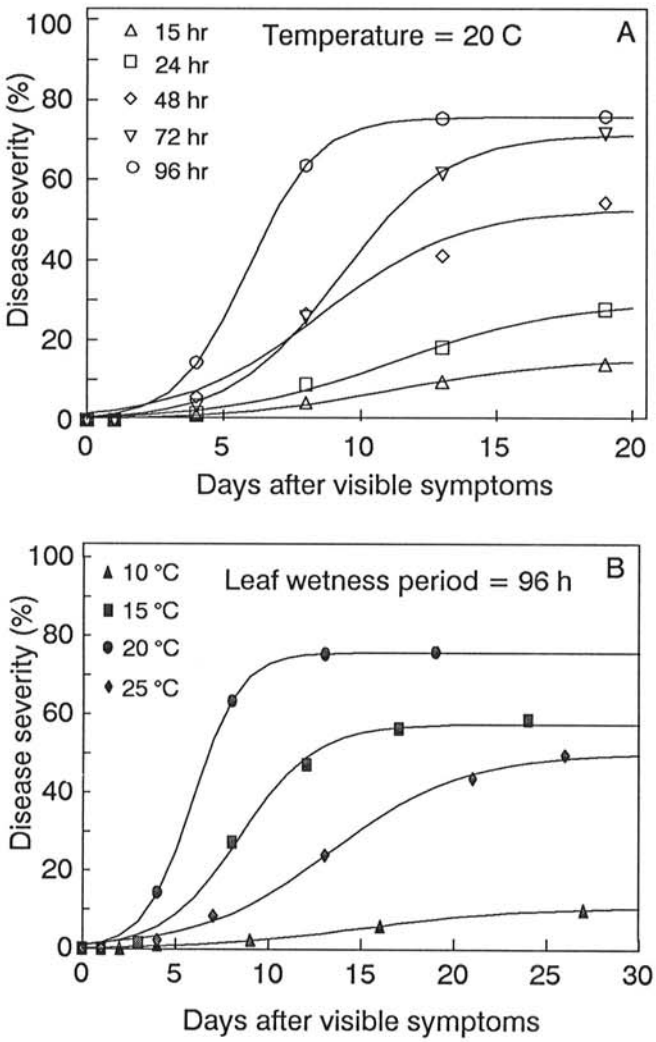

Fig. 3. Progress of Septoria tritici leaf blotch of wheat **A**, at fixed infection temperature (20 C) and different leaf wetness periods and, **B**, at fixed leaf wetness period (96 h) and different infection temperatures. The symbols on the graphs indicate different leaf wetness periods and different infection temperatures for **A** and **B**, respectively.

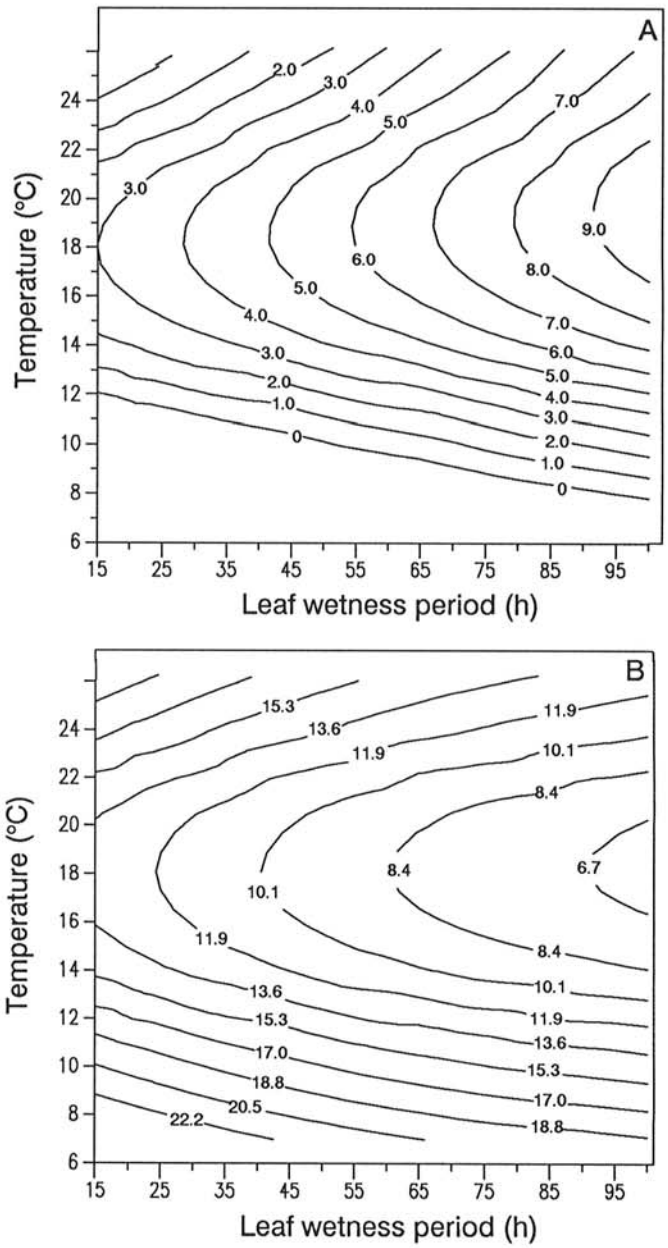

Fig. 4. Contour projection of **A**, the maximum developmental rate of severity (increase in disease severity per day) and, **B**, time (days) required to reach the maximum developmental rate of disease severity as a function of temperature and leaf wetness period.

length showed a similar response over a wide range of wetness periods (15–48 h). There was no specific trend for the maximum rate of lesion length expansion across all treatments as judged from the low R^2 values of the fitted response surface models (equations 9, 10, and 12 in Table 1). The maximum rate of lesion length expansion, the time required to reach the maximum rate of lesion length expansion, and the time required to reach the maximum lesion length showed no significant response to the treatment combinations of the infection temperature and the leaf wetness period. The maximum lesion length, however, showed a significant response to temperature and leaf wetness period ($R^2 = 0.74$, equation 11 in Table 1).

Senescence. The development of senescence over time for a fixed temperature of 20 C and a fixed leaf wetness period of 96 h are shown in Figure 6A and B. The estimated maximum amount of senescence increased with increasing leaf wetness period at all infection temperatures. The shortest time to reach the maximum rate of senescence (7 days) and the maximum senescence (14 days) was found at 20 C and 96 h. Higher temperatures and longer periods of leaf wetness tended to increase the maximum rate of senescence (Fig. 7). The maximum rate was 2% per day. The maximum rate and maximum response of the tissue to senescence could be described adequately by the response surface

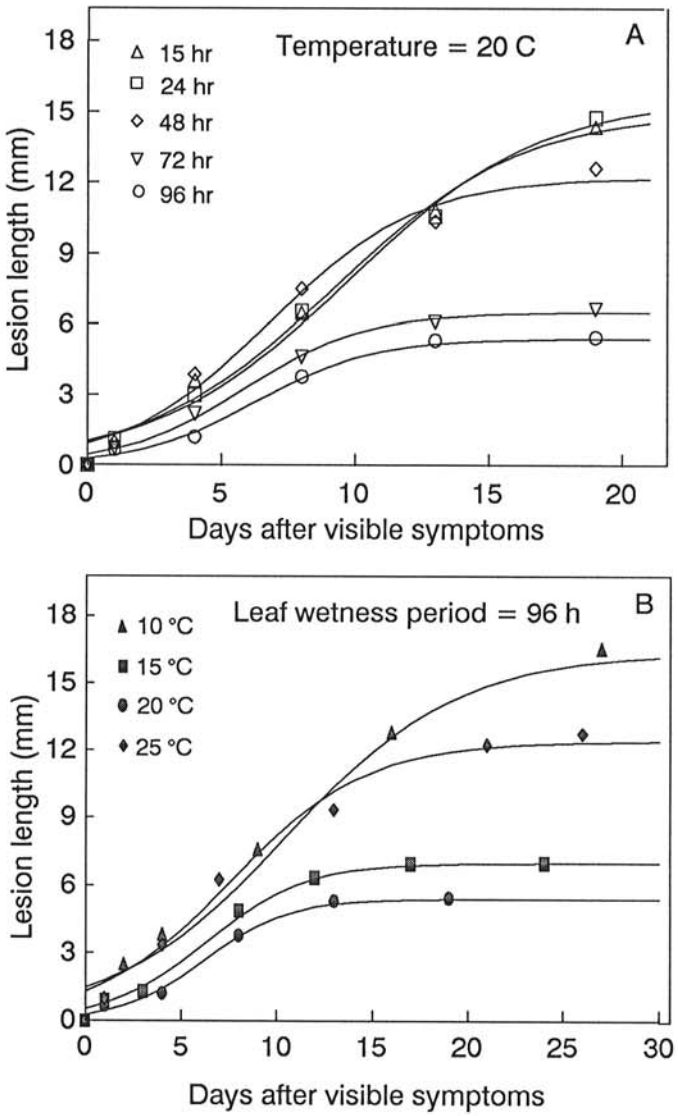

Fig. 5. Expansion of lesion length on the flag leaf of spring wheat Anza **A**, at fixed temperature (20 C) and different leaf wetness periods and, **B**, at fixed leaf wetness period (96 h) and different infection temperatures. The symbols on the graphs indicate different leaf wetness periods and different infection temperatures for **A** and **B**, respectively.

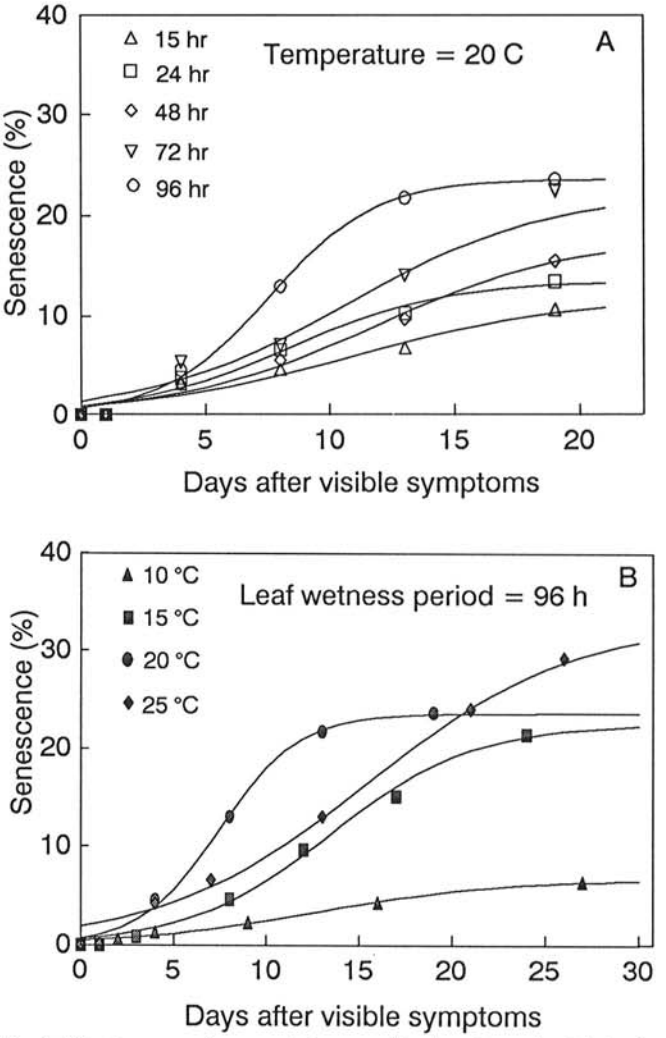

Fig. 6. The increase of senesced tissues with time **A**, at fixed infection temperature (20 C) and different leaf wetness periods and, **B**, at fixed leaf wetness period (96 h) and different infection temperatures. The symbols on the graphs indicate different leaf wetness periods and different infection temperatures for **A** and **B**, respectively.

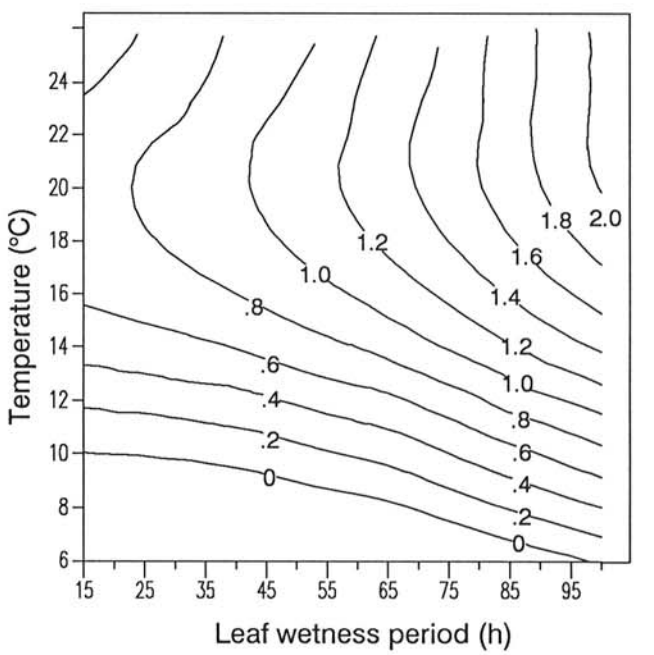

Fig. 7. Contour projection of the maximum rate of senescence as a function of temperature and leaf wetness period.

models (with the $R^2 = 0.75$ and $R^2 = 0.91$, respectively, for equations 13 and 15 in Table 1).

Relationships among disease components. The correlation coefficients among the disease characteristics within and between disease components are presented in Table 2. It was generally true that the number of days required to reach the maximum response of a disease component was negatively correlated with the maximum rate of the disease component development, but positively correlated with the number of days required to reach the maximum rate. In addition, the maximum response was positively correlated with the maximum developmental rate for lesion unit and senescence, but not for lesion length. Between the components, maximum lesion unit was positively correlated with maximum percent senescence but negatively correlated with maximum lesion length.

DISCUSSION

Temperature range and leaf wetness period are extremely critical for infection of wheat by *Septoria tritici* and subsequent disease development. The score of lesion unit provides an indicator of the degree of successful infection. Since lesions coalesced, a direct estimation of lesion number and size became difficult, if not impossible, particularly if assessment was made at a later stage of disease development (3). The measure of lesion unit utilized here is obviously a function of lesion number and thus provides an indirect estimate of lesion number if certain assumptions of uniform lesion formation and lesion size can be made. The lesion unit values recorded in our study exceeded the lesion numbers reported by previous investigators (8,14). But the definition of lesion size was not specified in these reports. Hess and Shaner (13) and Shaner and Finney (20) reported that, under field conditions, rainy days enhance spore germination and infection. These observations agree with our results which showed that lesion units increase with the length of the leaf wetness period under all temperature conditions. This suggests that lesion unit corresponds with spore germination, germ tube growth, or penetration rate, which are promoted by prolonged periods of free water on the leaf surfaces. Further studies are required to determine the exact relationship between lesion unit values and actual lesion numbers.

Weber (24) reported that the most favorable temperatures for spore germination in water were 22–24 C. We found that the optimum temperature for infection was 18 C for all leaf wetness periods. Successful infection, however, was obtained within the temperature range of 16–19 C when leaves were wet for 48 h or more. Based on the interaction between temperature and leaf wetness period, temperatures of 20–25 C promote infection that is consistent with Weber's finding. Temperatures ≥25 C, however, may suppress pathogen growth or the infection process when the leaf wetness period exceeds 48 h.

Renfro and Young (17) reported that infection failed to develop when the minimum temperature was 7 C or less during a two-day postinoculation period. In our study, the minimum temperature was 10 C. Although this temperature showed a reduction in lesion unit/cm^2 and disease severity, it did not inhibit infection completely.

Disease severity parameters generally showed responses similar to those of lesion unit parameters. Hess and Shaner (12) reported that disease severity on the flag leaves increased with an increase in the moist period from 24 through 96 h, which is consistent with the present investigation. The response surface analysis of the parameters of lesion unit/cm^2 and disease severity indicated that 69 to 91 percent of the variation in these parameters was accounted for by changes in temperature and leaf wetness period. This emphasizes the importance of these environmental conditions on the infection process and subsequent disease development.

M. graminicola causes a reduction in the photosynthetic area through destruction of leaf tissue and induction of premature senescence. Gaunt et al (9) stated that the senescence induced by the pathogen is an important aspect of the disease syndrome. We found that the developmental rate of senescence without pycnidia is linearly related to the rate of disease severity (Fig. 8). However, the distribution and location of lesions on the leaf surface may also play an important role in determining the rate of leaf senescence. For example, lesions formed at the base of the leaf caused the leaf to senesce at a faster rate, even when the number of lesions was small. Accordingly, the response surface functions showed no significant fit for both the time required to reach the maximum rate of senescence and the time required to reach maximum senescence.

More senescence developed at 25 C than at 20 C even though disease severity was much higher at 20 C and the time required to reach maximum senescence is longer at 25 C. The mechanism

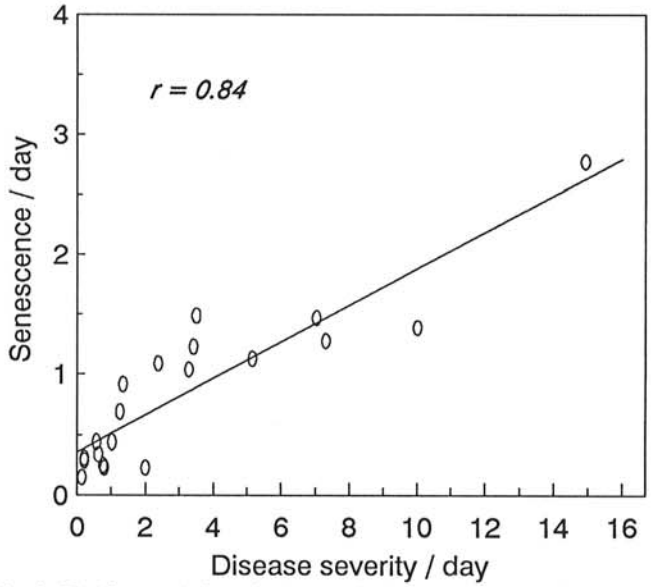

Fig. 8. The linear relationship (correlation) between the maximum rate of senescence and the maximum rate of disease development.

TABLE 2. Correlation coefficients between parameters of disease components

	MRLU[a]	DMRLU	MLU	DMLU	MRLL	DMRLL	MLL	DMLL	MRS	DMRS	MS	DMS
MRLU	1.00											
DMRLU	−0.57**[b]	1.00										
MLU	0.98**	−0.55**	1.00									
DMLU	−0.64**	0.97**	−0.62**	1.00								
MRLL	0.08	−0.14	0.06	−0.13	1.00							
DMRLL	−0.73**	0.19	−0.72**	0.25	−0.47**	1.00						
MLL	−0.96**	0.51*	−0.95**	0.60**	−0.01	0.76**	1.00					
DMLL	−0.79**	0.35	−0.78**	0.42	−0.55**	0.95**	0.79**	1.00				
MRS	0.83**	−0.55**	0.87**	−0.58**	0.29	−0.67**	−0.75**	−0.73**	1.00			
DMRS	−0.37	0.39	−0.33	0.33	−0.26	0.31	0.27	0.33	−0.39	1.00		
MS	0.46*	−0.35	0.57**	−0.36	0.14	−0.45*	−0.44*	−0.44*	0.77**	0.02	1.00	
DMS	−0.53*	0.52*	−0.48*	0.50*	−0.21	0.37	0.45*	0.42	−0.51*	0.92**	−0.03	1.00

[a] Acronyms are: M = Maximum; D = Days to maximum; R = Rate of development; LU = Lesion Unit; LL = Lesion Length; S = Senescence.
[b] Significant at * = 0.05 and ** = 0.01 levels.

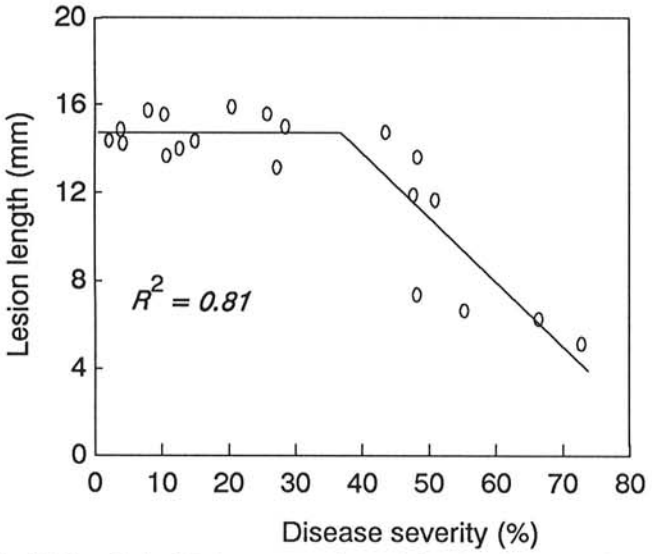

Fig. 9. The relationship between maximum lesion length and maximum disease severity. The average lesion length is 14.7 mm when disease severity is less than 37% and is linearly decreasing with an increasing disease severity. The linear equation is $Y = 25.4 - 0.29X$, where Y is the lesion length and X is the percent disease severity.

of this discrepancy is not clear but has implications for genetic management of disease severity. The relative importance of pathogen and host factors in regulating the response of plant tissue to temperature needs to be determined in future studies.

This study shows that maximum lesion length is mainly influenced by the duration of lesion expansion and the amount of nonsenescent leaf area. The duration of lesion length expansion is dependent on the related disease severity associated with the number of initial lesions. However, the maximum lesion length is not affected until disease severity exceeds 37% of the leaf area as indicated by the linear plateau model in Figure 9.

No consistent effect of infection conditions on the maximum rate of lesion length expansion could be detected. The maximum lesion length was not significantly correlated with the maximum rate of lesion length. This is because the rate of lesion length expansion was primarily influenced by the postinfection conditions which were kept constant throughout the study. The postinfection conditions were kept constant in order to analyze the specific infection conditions and assess the functional relationships between the induced disease components.

In this study, we have identified discrete environmental conditions for creating different but consistent levels of disease to assess genetic and biological factors influencing disease severity. In addition, the established quantitative relationships between the environmental factors and the disease components of the infection process provide the necessary information to develop disease forecasting and management models.

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