

Influence of Temperature and Wetness Duration on Infection of Immature Strawberry Fruit by *Phytophthora cactorum*

G. G. Grove, L. V. Madden, M. A. Ellis, and A. F. Schmitthenner

Graduate research associate, assistant professor, associate professor, and professor, respectively, Department of Plant Pathology, Ohio State University, Ohio Agricultural Research and Development Center, Wooster 44691.

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ABSTRACT

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Strawberry fruits (cultivar Midway) inoculated with a sporangial suspension (400/ml) of *Phytophthora cactorum* were used to determine the effects of wetness duration and temperature on infection level. Infection increased with increased wetness duration (0-5 hr) at all temperatures tested (6-30 C). For each wetness duration, infection increased up to the

optimum temperature (21 C) and then declined. At temperatures between 17 and 25 C, >1 hr of wetness resulted in >80% infection. A multiple-regression, logistic model accurately described infection as a function of wetness duration and temperature. The model was validated under natural field conditions.

Additional key words: disease forecasting, *Fragaria* × *ananassa*, quantitative epidemiology.

Leather rot, caused by *Phytophthora cactorum* (Leb. & Cohn) Schroet., is a serious fruit rot disease of the cultivated strawberry (*Fragaria* × *ananassa* Duch.) (6,16). First reported by Rose in 1924 in the southern United States (16), the disease has since been mentioned only occasionally (25-27).

Severe epidemics of leather rot have occurred in the Midwest, particularly in Ohio during 1980 and 1981, with several growers reporting up to 40% fruit losses due to leather rot. Above-average rainfall was generally associated with disease outbreaks (6,16,25).

In addition to describing the symptoms and causal organism of leather rot, Rose (16,17) correlated disease severity with rainfall. He found leather rot to be much more prevalent in areas that had received large amounts of precipitation or had poor drainage. Other than these observations, no other information is available on the epidemiology of this disease. The initial observations of Rose (16,17) and the epidemics experienced in Ohio in recent years suggested that moisture and/or rainfall were important epidemiological factors favoring development of strawberry leather rot.

The purposes of this study were to determine the duration of fruit wetness at specific temperatures necessary for infection of strawberry fruit by *P. cactorum* and to develop a model to predict levels of fruit infection under both controlled and field conditions.

MATERIALS AND METHODS

All inoculations were performed with cultures of *P. cactorum* freshly isolated from infected strawberry fruit (cultivar Midway) on pentachloronitrobenzene-benomyl-neomycin sulfate-chloramphenicol (PBNC) medium (20). For sporangial production, mycelia from the edges of 3-day-old cultures were transferred to lima bean agar (20). Cultures were incubated for 7 days at 22 C in continuous light at 2.7 W/m². Sporangia were washed from the surface of plates by adding 20 ml of sterile distilled water and gently swirling for 1 min. All inoculations were made with sporangial

suspensions adjusted to 400 per milliliter in sterile distilled water after counting with a hemocytometer. Suspensions were kept at 5 C for no longer than 30 min to delay germination. All inoculations were made by applying 1 ml of suspension to each fruit with an atomizer.

Controlled environment studies. Strawberry plants (cultivar Midway) were grown to reproductive maturity in a mixture of peat, sand, and steam-disinfested loam (1:1:1, v/v). Immature (green) fruit on each plant were tagged and 5-18 fruit per plant were inoculated with *P. cactorum* as previously described. To induce infection, inoculated plants were kept continuously wet at constant temperature in a controlled environment chamber (Environmental Growth Chambers [EGC], Chagrin Falls, OH) containing a Herrmidifier mister (Herrmidifier Co., Lancaster, PA) inside a 1-m³ clear plastic chamber. All tests were conducted in darkness. Two plants were removed at 1-, 2-, 3-, and 4-hr intervals and placed in a second chamber (EGC), at the same temperature as the first, for drying. Inoculations for wetness durations of less than 1 hr were performed by inoculating plants in the drying chamber. Temperature and fruit wetness in both chambers were continuously monitored with thermistors (Fenwall Electronics, Ashland, MA) and printed-circuit leaf wetness sensors (Wong Labs, Cincinnati, OH) connected to a microprocessor-controlled Datalogger (model CR-21; Campbell Scientific, Logan, UT). Leaf wetness sensors in the drying chamber were gently misted with an atomizer upon transfer of inoculated plants from the wetness chamber. After a 24-hr drying period, inoculated plants were removed and incubated in a third growth chamber (EGC) at 22 C with a 14-hr photoperiod at 99 W/m². By visual examination, fruit were classified as infected or uninfected 72 hr later. Isolations were made on the PBNC medium to verify the presence of *P. cactorum*. Tests were performed at nine constant temperatures between 6 and 30 C. The experiment was repeated once. The order of temperatures tested was random.

As determined by the wetness-sensing grids, the combined mean drying time for both experiments was 33 min. Actual drying time for each temperature/wetness inoculation ranged from 15-50 min. The specific drying time for each inoculation was added to the preassigned wetness periods to give a total time of wetness duration.

Field studies. Field studies were performed during the 1983 growing season in order to validate a regression model developed

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from the EGC data. Midway strawberries were also used in field studies. Attached, immature fruit were used in all inoculations. Fifty immature, attached fruit were tagged in each plot. Twenty-five were inoculated with a sporangial suspension of *P. cactorum* as previously described and the remaining 25 served as uninoculated controls. Inoculations were made during natural wetness (dew or rainfall) periods or during wetness periods induced by gently misting the fruit (with an atomizer) to runoff. At times, plants were covered with 1.5 × 1.5 × 1 m, wood-framed, clear plastic chambers to prolong wetness periods. Chambers were used only at night to prevent significant alteration of radiation and temperature. Wetness, temperature, and relative humidity (Phys-Chem sensor; Phys-Chemical Research Corporation, New York, NY) were continuously monitored throughout the experiment with the CR-21 datalogger. Sensors were placed immediately adjacent (within 5 cm) to inoculated fruit in the plant canopy. At least 1 hr after the conclusion of wetness periods (as determined by the wetness sensors), both inoculated and uninoculated fruit were harvested, placed on metal screens, and incubated for 24 hr at 22 C with a 14-hr photoperiod at 99 W/m². Fruit and screens were then transferred to 5-L plastic containers containing 100 ml of sterile distilled water; containers were then covered with plastic wrap, sealed with tape, and incubated for an additional 48 hr at 22 C in a 14-hr photoperiod at 99 W/m². Numbers of infected and uninfected fruit from both the inoculated and uninoculated treatments were visually determined 72 hr after fruit harvest. Isolations from all fruits were made on the PBNC medium to verify the presence of *P. cactorum*.

Data analyses. Regression analysis was used to determine the effect of temperature (*T*) and wetness duration (*W*) on the proportion of strawberry fruit infected (*Y*) by *P. cactorum*. Analysis was based on the controlled environment studies. Properties of the regression had to include: an optimum relationship between *Y* and *T*, in which *Y* increases to a maximum and then declines; and a positive (monotonically increasing) relationship between *Y* and *W*, with the provisions that predicted infection cannot be less than 0.0 or greater than 1.0, regardless of

the value of *W*. A logistic model was chosen of the form

$$Y = 1 / (1 + \exp(-f(T, W))) \quad (1)$$

in which \exp is e (2.718) raised to a specified power, and $f(T, W)$ is an arbitrary function of *T* and *W*. To handle cases when *Y* would normally equal 0 or 1, a slightly biased estimator of *Y* used: $(I + 0.5) / (N + 1)$, in which *I* is the number of infected fruit and *N* is the number inoculated. Equation 1 can be transformed to

$$\ln(Y/[1-Y]) = f(T, W) \quad (2)$$

in which $\ln(Y/[1-Y])$ is the logit of *Y*. Linear terms were tested for making up $f(T, W)$. The terms evaluated were: *W*, *T*, $W * T$, T^2 , $W * T^2$, T^3 , and $W * T^3$, in which "*" represents multiplication. All possible combinations of these terms were evaluated for significance of the estimated parameters, coefficient of determination, and pattern of residuals (12,15). The regression analysis was performed on the data for each growth chamber experiment separately, and then on the combined data. An *F*-test was conducted to determine if the results from the two experiments were significantly different (15).

In addition to equation 1, two other models were evaluated. The first was an extension of Schrödter's (21) sine-model of the form

$$Y = \sin^2(f(T, W)) \quad (3)$$

which can be transformed to

$$\arcsin(\sqrt{[Y]}) = f(T, W). \quad (4)$$

In these two equations, \sin^2 is the square of the trigonometric sine function and \arcsin is the inverse sine function. All possible combinations of the temperature and wetness terms described above were evaluated as $f(T, W)$.

The second model was a generalization of Analytis' (1) "Beta" model of the form

$$Y = p t^m (1 - t)^n W^q \quad (5)$$

in which *p*, *m*, *n*, and *q* are parameters, and $t = (T - T_{\min}) / (T_{\max} - T_{\min})$. The maximum (T_{\max}) and minimum (T_{\min}) temperature were not known precisely, but were assigned 5 and 34 C, respectively. Other values did not significantly alter the fit of the model. Equation 5 can be transformed to

$$\ln(Y) = \ln(p) + m \ln(t) + n \ln(1-t) + q \ln(W). \quad (6)$$

RESULTS

In general, there was an increase in infection with increase in *W* at all *T* (Fig. 1). At 6 C, the maximum fruit wetness duration (4 hr in mist chamber plus drying time) resulted in very low infection (average of 8.3% for the two tests). Infection levels were even less at shorter wetness durations. At 9 C, 84.6% infection was obtained at the longest wetness duration. At 12 C, 78.4% infection resulted from wetness durations between 2 and 3 hr, and almost 100% infection between 4 and 5 hr. At 15 C, between 2 and 3 hr of wetness resulted in 100% infection. An average infection level greater than 80% was obtained at wetness durations exceeding 1 hr at 17–25 C, and greater than 20% was obtained at 20–25 C for wetness durations less than 1 hr. Infection at 30 C, the maximum temperature studied, required between 2 and 3 hr of wetness to reach levels of 80% or more. Infection was negligible at durations less than 1 hr at this temperature.

Data analyses. The best logistic model representing the controlled environment data for both tests was of the form

$$\ln(Y/[1-Y]) = b_0 + b_1 T + b_2 W * T + b_3 T^3 + b_4 W * T^3 \quad (7)$$

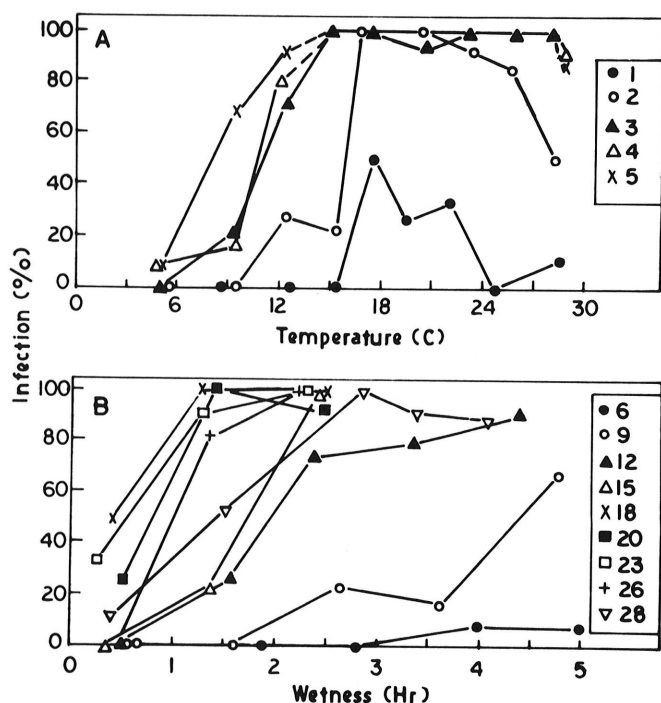


Fig. 1. Infection of immature strawberry fruit by *Phytophthora cactorum* at different **A**, temperatures and **B**, wetness durations for test two. Curves represent the levels of infection **A**, at temperatures between 6 and 30 C with different wetness durations and **B**, at wetness durations between 0 and 5 hr at different temperatures. Wetness duration labels in **A** are rounded to the next highest integer.

in which the b_s are the unknown parameters estimated from the data. Estimated parameters for both tests, and for the combined data, are presented in Table 1. An F -test indicated there was no significant difference in results between the two tests ($P > 0.50$). Therefore, we only presented the means for test two (Fig. 1). All estimated parameters in the model were significant ($P < 0.05$). The residuals had a random pattern and were normally distributed (15). The coefficient of determination was fairly high ($R^2 = 0.75$ for the combined data). The coefficient of determination adjusted for degrees of freedom (R_a^2), which can be considerably lower than R^2 if unnecessary or redundant terms are in the model, was almost as large as R^2 . Both of the coefficients are based on goodness of fit between the observed and predicted logits. We untransformed the predicted logits and determined the goodness of fit between the observed and predicted Y_s (R^{*2}) and found that the coefficient was fairly high ($R^{*2} = 0.85$ for combined data).

Inspection of the model (equation 7) revealed a linear and cubic relationship between logits and T . There was also an interaction of W and T as well as W and T^3 . This indicates that the response to T is not consistent for all wetness periods. Predicted values of $\ln(Y/[1-Y])$, and therefore Y , were calculated for temperatures between 1 and 5 hr, based on the parameters for the combined data. Bell-shaped curves were produced when Y was plotted versus T (Fig. 2). The curve spread out as W increased and the T at the maximum infection level shifted slightly to the left. There was a monotonic increase in Y with increase in W at all T (Fig. 3); the shape of the curves varied with the value of T . Equation 7 was rearranged to

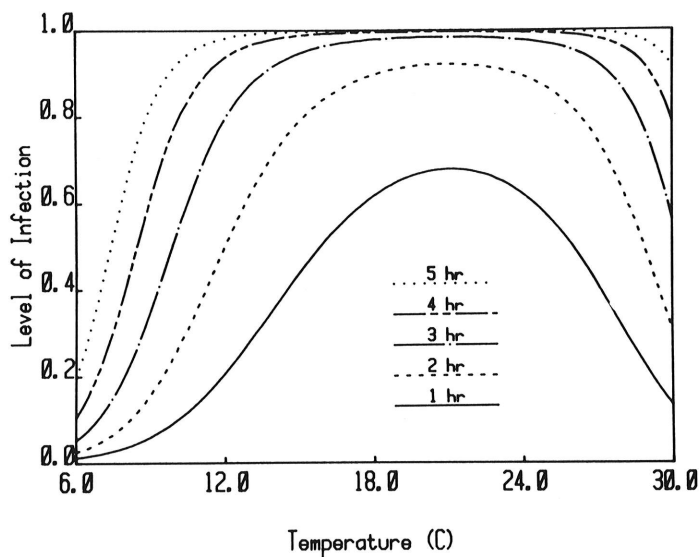


Fig. 2. Effect of temperature on predicted infection of immature strawberry fruit by *Phytophthora cactorum* at wetness durations between 1 and 5 hr. Curves were generated by using equation 7 with the parameters listed in Table 1 for the combined tests.

TABLE 1. Estimated parameters of equation 7 for temperature (T) and wetness duration (W) effects on infection of immature strawberry fruit by *Phytophthora cactorum*, together with the coefficient of determination (R^2), R^2 adjusted for degrees of freedom (R_a^2), coefficient of determination for untransformed infection levels (R^{*2}), and the standard error about the regression curve (S)

	Estimated parameters ^y					R^2	R_a^2	R^{*2}	S
	b_0	b_1	b_2	b_3	b_4				
Test 1	-8.32 ^z (1.0)	0.525 (0.093)	0.132 (0.024)	-0.41×10^{-3} (0.11×10^{-3})	0.10×10^{-3} (0.35×10^{-4})	0.75	0.72	0.84	1.39
Test 2	-7.60 (0.925)	0.452 (0.089)	0.122 (0.023)	-0.30×10^{-3} (0.11×10^{-3})	-0.10×10^{-3} (0.37×10^{-4})	0.76	0.73	0.86	1.25
Combined tests	-8.00 (0.667)	0.493 (0.062)	0.127 (0.016)	-0.36×10^{-3} (0.72×10^{-4})	-0.10×10^{-3} (0.24×10^{-4})	0.75	0.74	0.85	1.28

^yEstimated parameters for equation 7 corresponding to the intercept (b_0), T (b_1), $W \cdot T$ (b_2), T^3 (b_3), and $W \cdot T^3$ (b_4). Numbers in parentheses under the parameters correspond to their standard deviations.

^z b_0 is the value of $\ln(Y/(1-Y))$ when $T = 0$ and $W = 0$; value of Y at these conditions is equal to $1/(1+\exp(-b_0))$.

determine the hours of wetness required for a specific level of infection (eg, 50%) at temperatures between 6 and 30 C (Fig. 4). For $Y = 0.50$, more than 6 hr were required at 6 C, ~2 hr were required at 12 C, and <1 hr was required at 21 C (Fig. 4). The same type of curve is produced for other levels of Y . It was impossible to get values of $Y < 0.25$ around the optimum temperature (~21 C).

Neither the sine nor Beta models fit the data as well as the logistic. The same temperature and wetness terms were significant with the sine and logistic models. For the combined data, the sine model (equation 3) had an R^{*2} equal to 0.75. The Beta model (equation 5) had an even poorer fit to the data with $R^{*2} = 0.70$.

For field validation, the logistic equation, with the coefficients for the growth chamber study, was used to predict infection level. Hours of wetness and the average temperature during the wetness period in the field were used in the prediction equation. Forty-seven inoculations were made. Wetness duration ranged from 0 to 15 hr after inoculations; average temperatures ranged from 13.2 to 33.5 C. The observed Y_s from the field inoculations (I) were regressed on the predicted Y_s from the growth chamber prediction model (P) (see Fig. 5). Each datum point represents the observed infection level in relation to the predicted level. An unbiased prediction model should have a slope of one and an intercept of zero for the regression. The estimated equation was

$$I = -0.09 + 0.996(P). \quad (8)$$

The intercept of -0.09 was not significantly different from 0, and

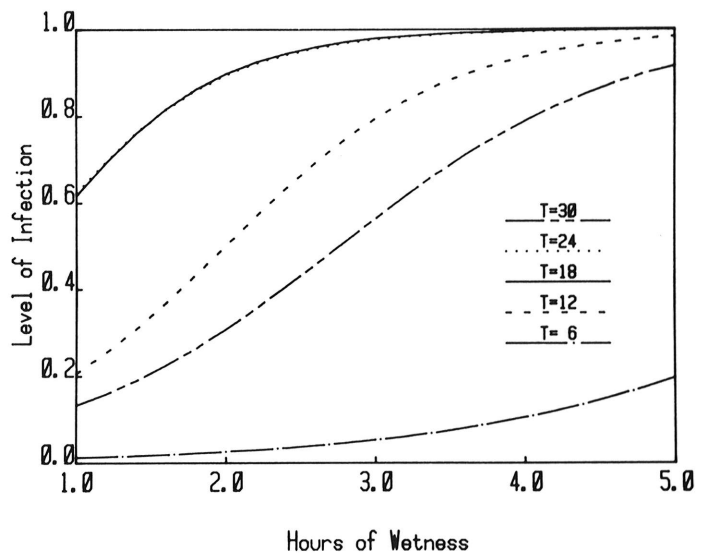


Fig. 3. Effect of wetness duration on predicted infection of immature strawberry fruit by *Phytophthora cactorum* at temperatures of 6, 12, 18, 24, and 30 C. Curves were generated using equation 7 with parameters listed in Table 1 for the combined tests. Prediction curves overlapped for temperatures of 18 and 24 C.

the slope of 0.996 was not significantly different from 1.0 ($P > 0.25$). The coefficient of determination equaled 0.83 for the equation. The correlation between observed and predicted values was 0.91.

DISCUSSION

Results clearly indicate that wetness period (W) and temperature (T) are significant factors influencing the infection (Y) of immature strawberry fruit by *P. cactorum* and that equation 7 is a valid model for predicting the level of infection at different values of W and T . Coefficients of determination (R^2) of 0.75, 0.76, and 0.75 for tests one, two, and the combined tests, respectively, indicate that a relatively high proportion of the variability of infection levels experienced in the test are accounted for by the components of the model. Coefficients of determination adjusted for degrees of freedom (R_a^2) were also relatively high, indicating the importance of various terms in the model. Low values of R_a^2 relative to R^2 indicate redundancy, ie, that some components of the model may not be necessary (10,12,15). This clearly was not the case with our results. Kranz (10) recommends obtaining R^2 values for both the transformed and untransformed values, as R^2 values obtained for transformed values measure the explained variability of the transformed variable. R^2 values obtained for untransformed Y values (R^{*2}) in this study were also high, 0.85 for the combined tests. The logistic model with the environmental terms in equation 7 provided the best fit of the controlled environment infection data. The generalizations of the sine and Beta models were clearly inferior to the logistic. The Beta model might be improved with different estimates of T_{min} and T_{max} . However, we have no reason to believe that T_{min} and T_{max} are constant for different levels of W .

The effect of temperature was most pronounced at short wetness durations, eg, 1 hr wetness at 6 C resulted in a much lower infection level than 1 hr at 21 C. At the short wetness durations, optimum infection occurred over a relatively short T range, whereas at long wetness periods, high infection occurred over a wide range. The interaction of W with T and T^3 in the model (equation 7) accounted for the broadening of the optimum infection interval.

Field validation resulted in linear equation 8 with intercept and slope not significantly different from 0 and 1, respectively, indicating that the growth chamber derived model produced unbiased predictors of field infection. The coefficient of determination ($R^2 = 0.83$) was acceptable (15). The model obtained in this study accurately predicted the levels of strawberry fruit infection at different wetness durations and temperatures in the

field. Several wetness/temperature combinations recorded in the field experiments resulted in high predicted infection levels; however, observed infection for these periods was substantially lower (see data points at right-hand side of Fig. 5). Perhaps sensor dryness failed to correspond to actual fruit dryness during these periods or the wetness duration cannot be extrapolated this far beyond the maximum of 5 hr used in the growth chamber.

The wetness period required for the infection of unripe strawberry fruit by *P. cactorum* was exceedingly short over a broad range of temperatures. Substantial levels of infection (>80%) could result from a 2-hr wetness period between 17 and 25 C; the optimum temperature for infection was ~21 C. Progressively longer wetness periods were required to produce correspondingly high levels of infection as the temperature was increased or decreased away from the optimum. No temperature maxima and minima were found, although the longest wetness periods at the lowest temperature tested failed to result in infections >20%.

The importance of wetness and temperature on the epidemiology of several diseases caused by airborne (*P. infestans*, *P. phaseoli*) and/or water splashed (*P. palmivora*, *P. citrophthora*, *P. lateralis*, *P. capsici*, and *P. syringae*) *Phytophthora* spp. is well documented in the literature (4,8,18,23,24). Infection of potato leaves (*Solanum tuberosum* L.) by *P. infestans* can result from 6–8 hr wetness periods at 15 C; infection can result from 12–24 hr periods at 12 and 24 C (4,18). Schlub (19) has reported infection of bell pepper (*Capsicum sativum* L.) by *P. capsici* under various temperatures and wetness periods. Periods of at least 24 hr are necessary for >50% infection at 15 or 31 C, while 4 hr at 27 C results in 60% infection. Clearly, an interaction between temperature and wetness periods, similar to that revealed in *P. cactorum*, exists in the two forementioned examples. Gerlach et al (7) reported that infection of *Pieris japonica* (Thunb.) D. Don by zoospores of *P. citrophthora* requires inoculum exposures of 15, 2, and 4 hr at 12, 24, and 32 C, respectively. Infection of papaya (*Carica papaya* L.) fruit by *P. palmivora* can occur after a 15-min exposure to a zoospore suspension (5,8). Hunter and Kunimoto (8) and Dao (5) reported that surface sterilization of fruits after several hours of exposure to a zoospore suspension failed to prevent infection. As expected, the interaction between temperature and wetness, as well as temperature optima, are different for each of these examples, and the relative effect of each is unique to each host:pathogen relationship. However, no *Phytophthora* sp. appears to require shorter wetness periods for infection than *P. cactorum* and *P. palmivora*.

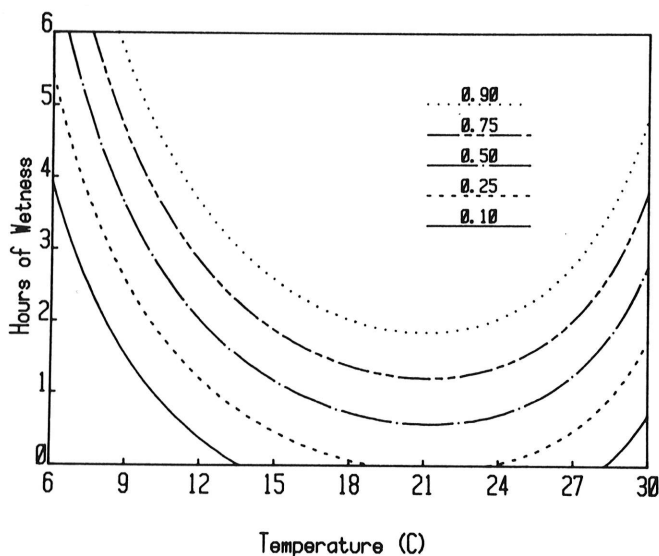


Fig. 4. Combinations of temperature and wetness durations necessary to predict 10, 25, 50, 75, and 90% infection of immature strawberry fruit by *Phytophthora cactorum*. Curves were generated by using equation 7 with the parameters listed in Table 1 for the combined tests.

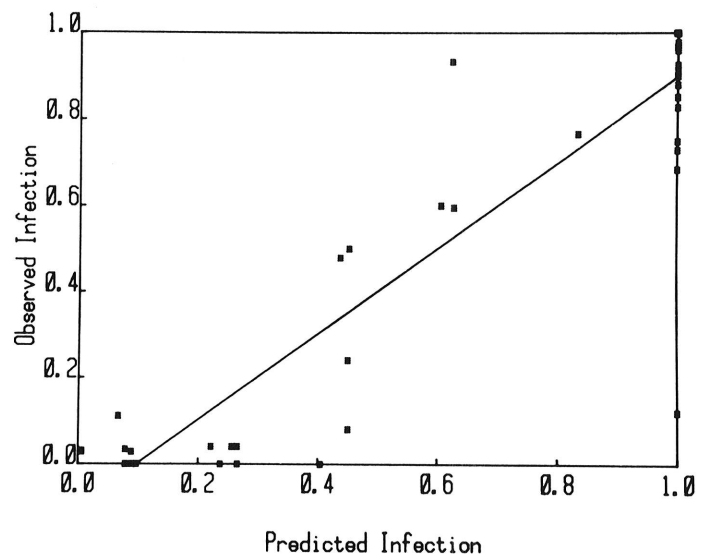


Fig. 5. Observed proportion of field-inoculated strawberry fruit infected by *Phytophthora cactorum* versus the predicted proportion of infected fruit based on equation 7 with the parameters listed in Table 1 for the combined tests ($R^2 = 0.83$). Temperatures and wetness durations input to the equations were measured in the field.

Environmental parameters, such as wetness duration and temperature, conducive to infection by pathogenic fungi have been determined for several diseases (2,3,5,13,14,22). Microcomputer-based disease forecasting systems have been developed utilizing environmental parameters similar to those described here (9,11). Unfortunately, wetness periods and temperatures falling within the optimal range found for strawberry infection occur frequently during the Ohio strawberry season (G. G. Grove, L. V. Madden, and M. A. Ellis, *unpublished*). Dew periods of much greater than 1 hr are quite common and the mean temperature falls well within the optimal range for infection. Other components of the disease cycle are more limiting to the development of leather rot epidemics than infection. Wetness durations longer than reported here for infection are necessary for the production of many sporangia (*unpublished*). Additionally, investigations indicate that caducous sporangia that form on the surface of infected strawberry fruit are readily dispersed by water drops (*unpublished*). We believe that water splash from rain or irrigation is necessary for the development of epidemics. Research in progress concerning conditions favoring sporulation and pathogen dispersal will contribute to our knowledge of this disease.

LITERATURE CITED

1. Analytis, S. 1977. Über die relation zwischen biologischer entwicklung und temperatur phytopathogen pilzen. *Phytopathol. Z.* 90:64-76.
2. Bashi, E., Rotem, J., and Putter, J. 1973. Effect of wetting duration, and of other environmental factors, on the development of *Stemphylium botryosum* f. sp. *lycopersici* on tomatoes. *Phytoparasitica* 1:87-94.
3. Castor, L. L., Ayers, J. E., and Nelson, R. R. 1977. Controlled-environment studies of the epidemiology of yellow leaf blight of corn. *Phytopathology* 67:85-90.
4. Crosier, W. 1934. Studies in the biology of *Phytophthora infestans* (Mont.) De Bary. N.Y. (Cornell) Agric. Exp. Stn. Mem. 155. 40 pp.
5. Dao, S. Y. A. C. 1964. The influence of temperature and inoculum level on disease development in *Carica papaya* L. caused by *Phytophthora palmivora* Dast. M.S. thesis, University of Hawaii, Honolulu. 43 pp.
6. Ellis, M. A., and Grove, G. G. 1983. Leather rot in Ohio strawberries. *Plant Dis.* 67:549.
7. Gerlach, W. W. P., Hoitink, H. A. J., and Schmitthenner, A. F. 1976. *Phytophthora citrophthora* on *Pieris japonica*: Infection, sporulation, dissemination. *Phytopathology* 66:302-308.
8. Hunter, J. E., and Kunimoto, R. K. 1974. Dispersal of *Phytophthora palmivora* by wind-blown rain. *Phytopathology* 64:202-206.
9. Jones, A. L., Lillevik, S. L., Fisher, P. D., and Stebbins, T. C. 1980. A microcomputer-based instrument to predict primary apple scab infection periods. *Plant Dis.* 64:69-72.
10. Kranz, J. 1974. Epidemics of Plant Diseases: Mathematical Analysis and Modeling. Springer-Verlag. Berlin, Heidelberg, and New York. 170 pp.
11. Krause, R. A., Massie, L. B., and Hyre, R. A. 1975. Blitecast: A computerized forecast of potato late blight. *Plant Dis. Rep.* 59:95-98.
12. Madden, L. V. 1983. Measuring and modeling crop losses at the field level. *Phytopathology* 73:1591-1593.
13. Mills, W. D. 1944. Efficient use of sulphur dusts and sprays during rain to control apple scab. NY Agric. Exp. Stn. (Ithaca) Ext. Bull. 630. 4 pp.
14. Mills, W. D., and Laplante, A. A. 1951. Diseases and insects in the orchard. Pages 21-27 in: NY Agric. Exp. Stn. (Ithaca) Ext. Bull. 711.
15. Neter, J., and Wasserman, W. 1974. Applied Linear Statistical Models. Richard D. Irwin, Inc. Homewood, IL. 842 pp.
16. Rose, D. H. 1924. Leather rot of strawberries. *J. Agric. Res.* 28:357-376.
17. Rose, D. H. 1926. Relation of strawberry fruit rots to weather conditions in the field. *Phytopathology* 16:229-232.
18. Rotem, J., Cohen, Y., and Putter, J. 1971. Relativity of limiting and optimum inoculum loads, wetting durations, and temperatures for infection by *Phytophthora infestans*. *Phytopathology* 61:275-278.
19. Schlub, R. L. 1983. Epidemiology of *Phytophthora capsici* on bell pepper. *J. Agric. Sci. (Camb.)* 100:7-11.
20. Schmitthenner, A. F. 1973. Isolation and identification methods for *Phytophthora* and *Pythium*. Pub. in Woody Ornamental Dis. Workshop. University of Missouri, Columbia. 128 pp.
21. Schrödter, H. 1965. Methodisches zur bearbeitung phytometeoropathologischer untersuchungen, dargestellt am beispiel der temperaturrelation. *Phytopathol. Z.* 53:154-156.
22. Spotts, R. A. 1977. Effect of leaf wetness duration and temperature on the infectivity of *Guignardia bidwellii* on grape leaves. *Phytopathology* 67:1378-1381.
23. Trione, E. J., and Roth, L. F. 1957. Aerial infection of *Chaemaesypris* by *Phytophthora lateralis*. *Plant Dis. Rep.* 41:211-215.
24. Upstone, M. E. 1978. *Phytophthora syringae* fruit rot of apples. *Plant Pathol.* 27:24-30.
25. Wilhelm, S. 1961. Diseases of strawberry, a guide for the commercial grower. Calif. Agric. Ext. Serv. Circ. 494. 26 pp.
26. Wright, W. R., Beraha, L., and Smith, M. A. 1964. Leather rot in Louisiana strawberries on the Chicago market in 1964. *Plant Dis. Rep.* 48:747-748.
27. Wright, W. R., Beraha, L., and Smith, M. A. 1966. Leather rot on California strawberries. *Plant Dis. Rep.* 50:283-287.