Effects of Temperature and Leaf Wetness Duration on the Infection of Celery by *Septoria apiicola*

D. Mathieu and A. C. Kushalappa

Graduate student and associate professor, respectively, Department of Plant Science, Macdonald Campus of McGill University, 21,111 Lakeshore Road, Ste-Anne-de-Bellevue, Québec, Canada H9X 3V9.
This research was funded by the Conseil des recherches en pêche et agroalimentaire du Québec. The scholarship awarded to the first author by the Province of Quebec Fonds pour la formation de chercheurs et l'aide à la recherche was greatly appreciated.
Accepted for publication 24 May 1993.

ABSTRACT

Mathieu, D., and Kushalappa, A. C. 1993. Effects of temperature and leaf wetness duration on the infection of celery by *Septoria apiicola*. Phytopathology 83:1036-1040.

The effect of temperature during various leaf wetness periods on the infection of celery (*Apium graveolens*) by *Septoria apiicola* was studied by inoculating plants with a suspension of 20,000 conidia per milliliter and incubating at five temperatures (10, 15, 20, 25, and 30 C) and for five wetness periods (12, 24, 48, 72, and 96 h). The number of lesions increased with temperature up to 25 C and then diminished. At each temperature, lesions increased in number with increasing leaf wetness duration, except at 25 and 30 C at which fewer lesions appeared after 72 and 48 h of wetness, respectively. The highest number of lesions was recorded at 25 C for 72 h and the lowest at 10 and 30 C. The angular transformation of the proportion of the maximum number of lesions for each repetition was related to centered temperature and leaf wetness duration by a weighted least squares polynomial regression, (R^2-*adj.* [adjusted] = 74.8%). Cluster analysis was used to divide the response surface into four disease-severity values. The shortest time interval from inoculation to the appearance of first lesions occurred under optimal conditions of temperature and leaf wetness duration.

Additional keywords: infection model.

Septoria blight, caused by *Septoria apiicola* Speg., is a serious disease affecting leaves and petioles of celery, *Apium graveolens* L. var. *dulce* (Mill.) Pers. Lesions are dotted with black pycnidia extruding masses of pycnidiospores in cirrhi that protect them against desiccation and inhibit germination until diluted by rain (8,14). Disease spreads from foci characteristic of splash dispersal. Heavy precipitation promotes disease increase (1,24). Favorable conditions for disease development are prolonged as the maturing crop canopy covers row spaces, reducing aeration and increasing the time required for plants to dry. At that time, the movement of machinery and workers in the fields constitutes an important source of secondary spread (13). Significant losses may occur in wet years when blight incidence and severity is such that portions of fields may be left unharvested. Indirect losses occur as the removal of diseased parts increases labor costs. In addition, the relatively long incubation period of this disease (1) means that lesions continue to appear in storage further increasing losses.

Disease-management recommendations for the nursery include hot-water seed treatments with thiram (18); the use of seed that is 3 yr old, the point at which the conidia in seedborne pycnidia are no longer considered viable (4); soil sterilization; and weekly fungicide applications from seed germination until transplantation. It also is recommended that growers water in the morning and provide good ventilation (6). In the field, farmers are advised to apply fungicides at weekly intervals from transplant recovery until harvest (6,19) to protect their crops, which are valued at $4.4 million for the 343 ha harvested in Québec (25).

Increasingly, health, environmental, and economic concerns are motivating research into disease-forecasting systems to improve the efficiency of disease control and reduce the number of pesticide applications. Given the importance of weather factors on disease development (11), plant disease-forecasting models are often based on fundamental studies of the effects of leaf wetness duration and temperature on disease severity (17). Earlier work with *S. apiicola* showed that a minimum of 12 h at 100% relative humidity (RH) and temperatures ranging from 5 to 25 C were required for spore germination on water agar. Optimal conditions occurred between 20 and 22.5 C (23). Another study indicated that a minimum of 36 h at no less than 94.5% RH was required for leaf infection, and temperatures exceeding 30 C were inhibitory (24). Berger (1), who studied the epidemiology of celery late blight under field conditions, reported high rates of infection in association with periods of heavy rainfall and average monthly temperatures below 25 C. To date, no reports have been published on the influence of temperature and leaf wetness duration for the full range of conditions conducive to Septoria blight epidemics.

This study was conducted to quantify the relationship between infection and leaf wetness duration at various temperatures as a preliminary step in the development of a management system for Septoria blight of celery.

MATERIALS AND METHODS

Production of plants. Three-year-old celery seeds of the cultivar Florida-683 obtained from a commercial grower were sown in groups of 50 in 12.5-cm pots filled with a substrate composed of organic soil (27–30% organic matter), peat moss, and perlite (4:1:1, v/v) supplemented with horticultural lime. The pots were enclosed in a plastic bag to maintain high RH and placed in a growth cabinet set at 20 C. Four-week-old seedlings were transplanted individually into 13-cm pots containing the same substrate moistened with a solution of 400 ppm phosphorus at 10-52-10 (N-P-K). Fertilizer was applied in the irrigation water at a rate of 100 ppm nitrogen at 15-15-17 (N-P-K). Foliar applications of 8% calcium chloride were made at the three-leaf stage to suppress black heart. Plants were placed in a growth cabinet with a 14-h photoperiod provided by a mixture of fluorescent and incandescent light fixtures yielding 250 $\mu Em^{-2} s^{-1}$. Temperature was maintained at 22 ± 2 C during the light period and at 18 ± 2 C in the dark. The RH ranged from 45 to 60% during the light period and increased to 60 to 80% during the dark period. The wide range in RH was due to variations in the plant canopy at different growth stages as well as to the removal and replacement of plants used in experiments. RH was monitored with a model 594 hygrothermograph (Bendix, Baltimore) located among plants. Aphid control in the growth chamber

was achieved with biweekly applications of pirimor, orthene, and Safer's soap (Safer's Ltd., Toronto) in rotation.

Inoculum production. An isolate of *S. apiicola* obtained from a commercial field south of Montreal was cultured on 15% celery-juice agar supplemented with 0.4% potato-dextrose broth (PDB). The celery juice was prepared by grinding 50 g of fresh celery leaves in 500 ml of water for 2 min in a blender set at high speed. The mixture was strained, and the juice was boiled and strained again to remove coagulated material. Agar (7.5 g) and PDB (2 g) were added, and the volume was adjusted to 500 ml by adding distilled water before autoclaving. Fresh cultures were maintained by regular isolations from diseased plants. These were incubated 6 days in the dark at 20 C, after which they were exposed for 8 days to a 12-h light regime provided by cool-white fluorescent (Sylvania F15T12/CW, Sylvania Consumer Lighting, Danvers, MA) light bulbs yielding 210 $\mu Em^{-2}\ s^{-1}$, and near-UV (GE 15T8-BLB, General Electric Co., Fairfield, CT) lamps to promote sporulation (5). At the end of the period, cultures were either used immediately for inoculations, or subcultures were made.

Inoculum was prepared by flooding culture plates with a 5-ml solution of 0.01% Tween 80 in distilled water and dislodging cirrhi by gentle syringe outflow. The mean of 12 spore counts, measured with a haemacytometer, was used as a basis for dilutions to obtain 20,000 spores per milliliter.

Treatments and inoculations. In this experiment, which was repeated once, five leaf wetness periods were tested (12, 24, 38, 72, and 96 h) each at five temperatures (10, 15, 20, 25, and 30 C). Because there were fewer mist chambers available than there were temperatures being tested, the experiment was conducted over time, resulting in one inoculation per temperature and five wetness periods. Temperature treatments were randomized within each repetition.

At each temperature, 40 plants were inoculated, eight plants per temperature–wetness combination. Two leaves per plant were tagged and inoculated with an artist airbrush (Badger-350, Franklin Park, IL) operated at 100 kPa until close to runoff. Inoculated plants were placed randomly in a mist chamber assembled from Plexiglas sheets and positioned within a model 15E growth cabinet (Conviron, Winnipeg, Manitoba, Canada). Intermittent mist was provided by a timer-operated cold-mist humidifier located at the center of the mist chamber and above the plant canopy. At the end of each wetness period, eight plants were removed and dried with an electrical fan. Drying time averaged 20 min, verified visually with a hand-held lens. After removal from the mist chamber, plants were placed in the growth cabinet at 20 C to allow symptom development. Lesions were counted every 3 days from day 9 to day 27 after inoculation. In the first repetition, the fourth and fifth true leaves of 10-wk-old plants were inoculated, whereas in the second repetition, the fifth and sixth leaves of 12-wk-old plants were used, so the developmental stage of the inoculated leaves would be the same in both repetitions.

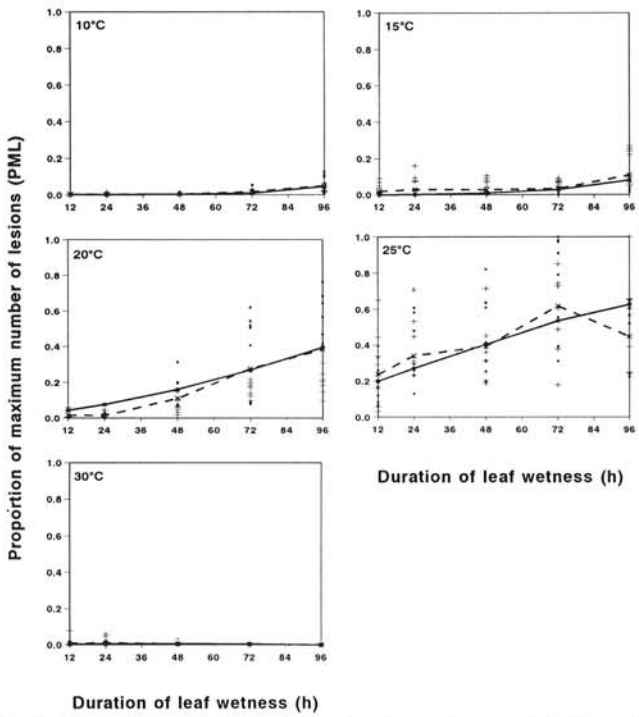

Fig. 1. Proportion of the maximum number of lesions (*PML*) at 10, 15, 20, 25, and 30 C for five leaf wetness periods. Repetition 1 observations: '·'; repetition 2 observations: '+'; line joining pooled observation means: '---'; predicted *PML*: '—' obtained by back-transformation of predicted values, $PML = \sin^2 f(TW)$, in which $f(TW)$ is the solution of the weighted least squares regression (R^2-*adj.* = 74.8%; Table 2 contains the equation).

Because temperatures were tested over time, their effect was confounded with that of inoculum. To minimize confounding, percent spore germination was determined by microscopic examination of three celery-juice agar plates with inoculum atomized onto them at intervals during inoculation and incubated in the dark for 48 h at 20 C. Spores were considered germinated if germ tubes of at least one-third the spore length were clearly visible. To ensure uniform spore viability over all temperatures, only the results from inoculations with ≥95% spore germination were included in the analyses.

Data analysis and selection of the regression model. Temperature and leaf wetness duration were related to infection by means of multiple linear regression, using SAS PROC REG (22). Infection was expressed as a proportion of the maximum number of lesions (*PML*) observed in each repetition and defined as follows:

$$PML_k = no.\ lesions\ W_{ik}T_{jk} / Max.\ no.\ lesions_k \qquad (1)$$

in which W_i = wetness, $i = 1, 2,...5$, T_j = temperature, $j = 1, 2,...5$, and $k = 1, 2$ stands for repetition.

Angular, logistic, and Gompertz transformations were used to stabilize the variance. However, the angular transformation of *PML* was retained for its better coefficient of determination and random pattern of residuals. It is defined as

$$Y' = \arcsin\sqrt{PML} \qquad (2)$$

in which Y' is expressed in radians.

The first step in model selection was done by the ordinary least squares method using Mallow's criterion *Cp* (12). Analysis of residuals was performed with the SAS UNIVARIATE procedure (22). Removal of outliers was based on indications from the box plots and stem-and-leaf diagrams of jackknife residuals (in SAS, studentized residuals with the current observation were deleted [22]).

Predictor variables were centered as a first measure to reduce colinearity. Diagnostic statistics such as the variance inflation factor, eigenvalues of the correlation matrix, their associated condition number, and variance proportions were used to detect near-colinearities and to delete troublesome predictors (12).

A weighted least squares regression was conducted to address a problem of increasing variances as the mean *PML* increased (7,9). The most suitable weight in this case was the inverse of the squared standard error of residuals from the previous least squares regression.

Predicted values from the weighted least squares model were back-transformed to *PML*, and observed values were regressed on predicted values to verify the adequacy of the model.

Cluster analysis was then used to categorize predicted *PML* into disease-severity values. Clustering was done according to the unweighted pair-group method using arithmetic averages, UPGMA (7,21), SAS's METHOD = AVERAGE.

RESULTS

In general, the number of lesions increased with increasing duration of leaf wetness, except at 25 and 30 C at which fewer

lesions developed after 72 and 48 h, respectively. For all leaf wetness periods, lesions were more numerous at 25 C; the highest number occurred at 25 C/72 h and the lowest at 10 C/12 h and 30 C/96 h of leaf wetness. The trends generally were similar in the two repetitions, except for the following: At 15 C, there generally were more lesions in the second repetition than in the first, and at 20 C, the number of lesions generally was higher in the first repetition (Fig. 1). The data was quite variable, particularly at temperatures of 20 and 25 C.

The time at which the first lesions on each leaf appeared varied according to temperature and leaf wetness conditions during the infection process (Table 1). For example, under optimal conditions of 25 C/72 h, it took 12 days for the first lesions to appear on all samples from both repetitions. In all other cases, first lesions appeared 12–24 days postinoculation. For that reason, the lesion count on day 24 was used in the analyses.

The equation for the final weighted least squares regression was

$$Y' = b_0 + b_1T + b_2W + b_3TW + b_4T^2W + b_5T^3 + b_6T^3W + b_7T^4 + b_8TW^4 \quad (3)$$

in which Y' was the angular transformation of PML (equation 1).

Homogeneity of variances of data from the two repetitions was tested before pooling results by fitting the selected model to both sets of data ($F_{186,189} = 1.04$; $P > 0.25$). Estimates of all parameters included in the pooled model were significant at the 0.01 level, and their values were intermediate between those of the first and second repetitions (Table 2). The model accounted for 74.9% of the total variation.

The intercept of the regression of observed values on back-transformed predicted values was not significantly different from zero ($b_0 = 0.004$; $P = 0.559$), with a slope of 1.014 ($P = 0.0001$), indicating that the model adequately described the data. Solution of the regression equation for increments of 6 h of wetness and 1 C produced a three-dimensional response surface (Fig. 2). Predicted values were back-transformed as follows:

$$PML = \sin^2 f(TW) \quad (4)$$

The resulting PML was plotted as a function of leaf wetness and temperature.

Equation 3 underestimated infection at 25 C/24 h and 25 C/72 h of wetness, whereas overestimations occurred at 20 C/48 h and 25 C/96 h of wetness.

Cluster analysis of the back-transformed predicted values yielded five clusters with the tree cut at $R^2 = 92\%$. Figure 3 shows an overhead view of the original three-dimensional response surface on which the clusters appear as contour lines. These lines join at points at which changes occur to the next lower disease-severity value. A given disease-severity value should be interpreted as affecting the points on the line plus the area between it and the next lower disease-severity value line. The fifth cluster, the area delineated by the top of the graph and the fine contour line (Fig. 3), was assigned a disease-severity value of four because predicted values above that line were overestimated by the model (Fig. 1).

TABLE 1. Effects of temperature and leaf wetness duration during the infection period on the number of days between inoculation and appearance of the first lesions of *Septoria apiicola* on celery

Temp. (C)	Wetness duration (h)				
	12	24	48	72	96
10	15–18[z]	15–24	15–24	12–21	12–24
15	15–18	12–24	12–24	12–14	12–15
20	12–21	12–24	12–15	12	12
25	12–18	12–15	12	12	12
30	15–21	15–24	15–24	15–24	21–24

[z] The range represents the period during which first lesions appeared on the 16 plants sampled.

DISCUSSION

In this experiment, maximum infection of celery by *S. apiicola* occurred at 25 C/72 h of leaf wetness. In an earlier experiment carried out at 15–17 C and 94% RH, Sheridan (24) also reported the highest number of lesions at 72 h, and none at 30 h or less. His observations were taken 14 days after inoculation. Our results, though, show that lesions may take as long as 15–24 days to appear under less favorable infection conditions, indicating that Sheridan may have underestimated the duration of the incubation

TABLE 2. Parameter estimates of the regression equation describing the effects of temperature and leaf wetness duration on the proportion of maximum number of lesions, including coefficients of determination for transformed values (R^2 unadjusted and R^2_a adjusted for degrees of freedom [df]) and untransformed values (R^{*2} adjusted for df)

Parameter	Parameter estimates/(SE)		
	Rep. 1[w]	Rep. 2[x]	Pooled data[y]
Error df	189	186	384
SS error	178.437	181.843	392.917
F value	101.347	60.267	147.146
R^2	0.811	0.722	0.754
R^2_a	0.803	0.702	0.749
R^{*2}			0.707
b_0[z] (int.)	0.41632	0.43683	0.42673
	(0.01491)	(0.01536)	(0.01104)
b_1 (T)	0.09390	0.06272	0.07868
	(0.00483)	(0.00502)	(0.00359)
b_2 (W)	0.00669	0.00432	0.00552
	(0.00055)	(0.00056)	(0.00040)
b_3 (TW)	0.00049	0.00026	0.00037
	(0.00015)	(0.00015)	(0.00011)
b_4 (T^2W)	-6.06×10^{-5}	-3.76×10^{-5}	-4.92×10^{-5}
	(7.9×10^{-6})	(8.0×10^{-6})	(5.8×10^{-6})
b_5 (T^3)	-9.41×10^{-4}	-6.03×10^{-4}	-7.76×10^{-4})
	(5.04×10^{-5})	(5.21×10^{-5})	(1.16×10^{-6})
b_6 (T^3W)	-5.96×10^{-6}	-3.35×10^{-6}	-4.65×10^{-6})
	(1.58×10^{-6})	(1.61×10^{-6})	(1.16×10^{-6})
b_7 (T^4)	-3.47×10^{-5}	-3.65×10^{-5})	-3.56×10^{-5}
	(2.3×10^{-6})	(2.3×10^{-6})	(1.66×10^{-6})
b_8 (TW^4)	-2.32×10^{-9}	-1.74×10^{-9}	-2.05×10^{-9}
	(0.000000)	(0.000000)	(0.000000)

[w] Significant at the 0.001 level for all parameters except TW^4 in which $Prob > |T| = 0.0175$.

[x] Significant at the 0.001 level for all parameters except TW, T^3W, and TW^4 in which $Prob > |T| = 0.0841$, 0.0391, and 0.0802, respectively.

[y] All parameters significant at the 0.0001 level except TW and TW^4 in which $Prob > |T| = 0.0006$ and 0.0043, respectively.

[z] b_0 is the value of arcsin $\sqrt{Y}$ when T and $W = 0$.

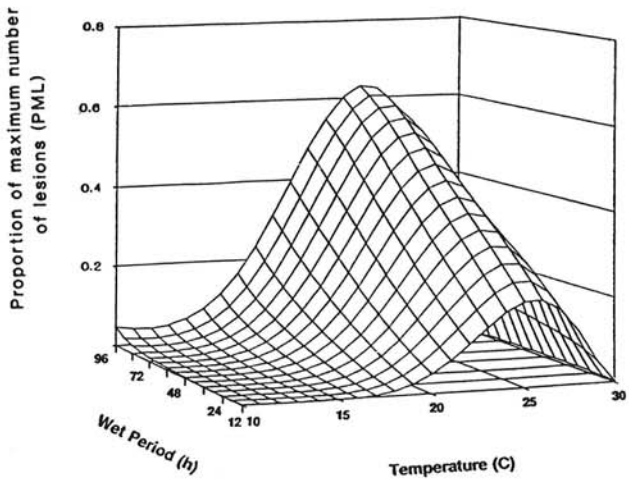

Fig. 2. Proportion of maximum number of lesions (PML) as a function of temperature and leaf wetness duration. Proportions are back-transformed predicted values, $PML = \sin^2 f(TW)$, in which $f(TW)$ is the solution of the weighted least squares regression (Table 2 contains the equation).

period for shorter exposures, hence the lack of infection.

Overall, the model developed using weighted least squares regression adequately described infection as a function of temperature and leaf wetness. Many terms were required for proper representation of the relationships under study. For example, T^4 and TW^4 were necessary to predict the declining trend in *PML* as the temperature approached 30 C and leaf wetness increased past 48 h.

The sine function has no biological cause-and-effect implications but can reliably describe the growth of pathogens in response to temperature, provided cardinal temperatures are tested (2). Being intrinsically linear (3), the sine function is amenable to regression analysis and has been used in similar studies to describe interactions (10).

The variability of the data at optimal temperatures could undermine the usefulness of the model, but this was partly compensated for by clustering predicted values into four severity classes corresponding to very low, low, moderate, and severe infection. This procedure has been used previously to facilitate field application of a complex model (26,27).

The regression model slightly underestimated infection at certain combinations of temperature and leaf wetness with little effect on resulting disease-severity values, except at 15 C/96 h of leaf wetness, when a disease-severity value of two may have been more appropriate. Similarly, predicted infection at 25 C/24 h of leaf wetness was assigned a disease-severity value of three when four may have been safer. Finally, underestimation at 25 C/72 h of leaf wetness and overestimation at 25 C/96 h of leaf wetness have no serious implications, because all fall within disease-severity value four, corresponding to the highest probability of infection (>0.32). Field testing will point out desirable corrections.

Based on the presence of moderate infection at 20 and 25 C for 12 h of leaf wetness, wetness periods of 0, 3, and 6 h should have been tested to cover the entire infectious range of *S. apiicola*. Nonetheless, judging from the shape of the response surface, it may be assumed that infection would continue to decline under 12 h of wetness. A graphic approach could be taken to assign untested combinations to likely disease-severity values to be confirmed and corrected by field experimentation.

Some of the unexpected observations, such as the decline in the number of lesions with increasing leaf wetness at 30 C, could be interpreted as a progressive loss of spore viability as the time required for host penetration increased beyond a certain limit.

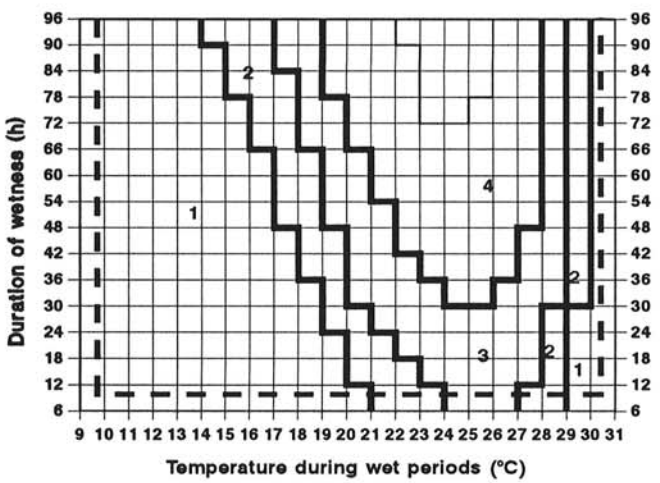

Fig. 3. Disease-severity values produced by cluster analysis of the predicted proportion of the maximum number of lesions (*PML*). Solutions are given for increments of 6 h of leaf wetness and a 1 C rise in temperature. Limit of predictions for the range of conditions tested: '---'. Disease-severity values 1–4 represent the following *PML*s: disease-severity value 1 $\leq$ 0.08 $<$ disease-severity value 2 $\leq$ 0.19 $<$ disease-severity value 3 $\leq$ 0.32 $<$ disease-severity value 4 $>$ 0.32. A fifth cluster delineated by the top of the graph and the fine contour line was assigned a disease-severity value of 4 (explained in text).

At 25 C, however, the lower number of lesions at 96 h of wetness was probably due to variability and removal of outliers.

Variability can be attributed to a number of causes that could be partially reduced by improved technique. Some of the avoidable causes include uneven spore distribution on the foliage brought about by manual operation of the airbrush. Among probable, but less avoidable causes, is variation in spore vigor from one inoculation to another. Differences in germ tube elongation were noted among experiments during the examination of culture plates to determine the percent germination. Genetic variation among plants cannot be overlooked either. Florida-683 is an open-pollinated cultivar with an expected out-crossing rate ranging from 50 and 90% (20), which may account for variations in disease susceptibility.

The disease-severity values established in this study should serve as a starting point in the development of a forecasting model to initiate fungicide applications. It could be based on the accumulation of daily severity values up to a predetermined threshold, as were other very valuable forecast models of similar, empirical origins (15,16,26). Once tested and validated, this model may be refined to determine when additional treatments are needed. However, its effectiveness is not expected to extend beyond canopy closure, at which point plants in infected fields are likely to require constant protection because of reduced evaporation.

Ultimately, disease-severity values should include the effects of interrupted leaf wetness and high RH. Under field conditions, leaf wetness periods of 72 h required for heaviest infection are not too common, but shorter wetness periods interspersed with intervals of high RH certainly are. Knowing that spores sprayed on celery leaves can germinate and cause infection at 94% RH under controlled conditions (23,24), it may be assumed that such intervals in the field could sustain ongoing infections by spores already present on leaf tissue. Finally, it may be interesting to test the expediency of varying the threshold for treatment, as a function of plant growth stage to account for the changing microclimate during the course of the growing season.

LITERATURE CITED

1. Berger, R. D. 1970. Epiphytology of celery late blight. Fla. State Hortic. Soc. Proc. 83:208-212
2. Butt, D. J., and Royle, D. J. 1990. Multiple regression analysis in the epidemiology of plant diseases. Pages 82-85 in: Epidemics of Plant Diseases. Mathematical Analysis and Modeling. J. Kranz, ed. 2nd ed. Springer-Verlag, New York.
3. Campbell, C. L., and Madden, L. V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York. 135 pp.
4. Cochran, L. C. 1932. A Study of two Septoria leaf spots of celery. Phytopathology 22:791-812.
5. Cooke, B. M. and Jones, D. G. 1970. The effect of near-ultraviolet irradiation and agar medium on the sporulation of *Septoria nodorum* and *S. tritici*. Trans. Br. Mycol. Soc. 54:221-226.
6. CPVQ (Conseil des productions végétales du Québec). 1987. Légumes, Protection AGDEX 250-605. Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec. 112 pp.
7. Dillon, W. R., and Goldstein, M. 1984. Multivariate Analysis, Methods and Applications. John Wiley & Sons. New York. 587 pp.
8. Fitt, B. D. L., McCartney, H. A., and Walklate, P. J. 1989. The role of rain in dispersal of pathogen inoculum. Annu. Rev. Phytopathol. 27:241-270.
9. Freund, R. J., and Littell, R. C. 1986. SAS System for Regression. SAS Institute Inc., Cary, NC.
10. Grove, G. G., Madden, L. V., Ellis, M. A., and Schmitthenner, A. F. 1985. Influence of temperature and wetness duration on infection of immature strawberry fruit by *Phytophthora cactorum*. Phytopathology 75:165-169.
11. Jones, A. L. 1986. Role of wet periods in predicting foliar diseases. Pages 87-100 in: Plant Disease Epidemiology, Population Dynamics and Management. K. J. Leonard and W. E. Fry, eds. Macmillan Publishing Co., New York.
12. Kleinbaum, D. G., Kupper, L. L., and Muller, K. E. 1988. Applied Regression Analysis and Other Multivariable Methods. 2nd ed. PWS-KENT Publishing Co., Boston. 718 pp.
13. Linn, M. B. 1939. Dissemination of celery blight pathogens on the clothing of farm laborers. Phytopathology 29:553-554.

14. Louis, I., and Cooke, R. C. 1985. Conidial matrix and spore germination in some plant pathogens. Trans. Br. Mycol. Soc. 84:661-667.
15. MacKenzie, D. R. 1981. Scheduling fungicide applications for potato late blight with BLITECAST. Plant Dis. 65:394-399.
16. Madden, L., Pennypacker, S. P., and MacNab, A. A. 1978. FAST, a forecast system for *Alternaria solani* on tomato. Phytopathology 68:1354-1358.
17. Madden, L. V., and Ellis, M. A. 1988. How to develop plant disease forecasters. Pages 191-207 in: Experimental Techniques in Plant Disease Epidemiology. J. Kranz, and J. Rotem, eds. Springer-Verlag, New York.
18. Maude, R. B. 1970. The control of *Septoria* on celery seed. Ann. Appl. Biol. 65:249-254.
19. Ontario Ministry of Agriculture and Food. 1988. Vegetable Production Recommendations. OMAF Publ. 363. 78 pp.
20. Orton, T. J., and Arus, P. 1984. Outcrossing in celery (*Apium graveolens*). Euphytica 33:471-480.
21. Romesburg, H. C. 1984. Cluster Analysis for Researchers. Lifetime Learning Publications, Belmont, CA. 334 pp.
22. SAS Institute. 1988. SAS Guide for Personal Computers: Statistics. Version 6. SAS Institute, Cary, NC. 1028 pp.
23. Sheridan, J. E. 1968. Conditions for germination of pycnidiospores of *Septoria apiicola* Speg. N.Z. J. Bot. 6:315-322.
24. Sheridan, J. E. 1968. Conditions for infection of celery by *Septoria apiicola*. Plant Dis. Rep. 52:142-145.
25. Statistics Canada. 1991. Fruit and Vegetable Production. Catalogue 22-003 (May):18-19.
26. Sutton, J. C., James, T. D. W., and Rowell, P. M. 1986. Botcast: A forecasting system to time the initial fungicide spray for managing Botrytis leaf blight of onions. Agric. Ecosystems Environ. 18:123-143.
27. Sutton, J. C., Rowell, P. M., and James, T. D. W. 1984. Effects of leaf wax, wetness duration and temperature on infection of onion leaves by *Botrytis squamosa*. Phytoprotection 65:65-68.