Development and Evaluation of an Infection Model for *Stemphylium vesicarium* on Pear Based on Temperature and Wetness Duration

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ABSTRACT

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Nonwounded fruits of pear cultivar Passe Crassane and plants of cultivar Conference were inoculated with conidia of *Stemphylium vesicarium* at temperatures ranging from 5 to 30°C and wetness durations from 0 to 24 h. Plants and fruit were maintained for expression of symptoms of brown spot at 20°C and 80% relative humidity. Polynomial regression models using the $\log_{10}$ transformation of disease severity as the dependent variable were developed for fruit and leaf infection, and described well the effects of temperature (T) and duration of wetness (W). The coefficients of determination for the fruit and leaf infection models were 0.88 and 0.92, respectively. Optimal conditions for infection were >24 h of continuous wetness at 22.6°C for fruits and 21.1°C for leaves. The leaf model was evaluated in 40 field trials in Spain and Italy under a wide range of wetness duration periods and temperature conditions, and was validated in relation to its capacity to predict infection periods. The coefficient of determination for the regression of observed disease severity on the predicted disease severity was 0.83 ($P = 0.0001$) and the slope was not different from 1 ($P = 0.44$), but the intercept was –0.08, indicating that the model overestimated infection risk. However, the leaf model under-predicted disease when high relative humidity periods with low wetness duration occurs. The leaf and fruit models also were relatively accurate in identifying likely infection periods in the field. During 2 years of a study in a commercial orchard, the leaf model accurately predicted six, and indicated broadly the other four, of ten infection periods.

Brown spot of pear, caused by *Stemphylium vesicarium* (Wallr.) E. Simmons (4), is an important fungal disease of pear (*Pyrus communis* L.) in the temperate fruit-growing regions of southern Europe: the Po River Valley (Italy), Catalunya (Spain), and Bouches du Rhone (France) (3,12,16). These areas produce about 9.2×10^8 kg of pears annually on 5×10^4 ha (1).

The control of brown spot of pear is based on protectant sprays of fungicides applied, at 7- to 15-day intervals depending on the type of fungicide, from fruit set to preharvest (10 to 25 applications) regardless of the risk of infection (12,16). The high number of fungicide applications needed to maintain acceptable levels of disease has stimulated research oriented to eliminate unnecessary sprays. Recently it has been shown that fungicides scheduled by FAST, a forecast model originally devised for timing fungicide sprays for control of *Alternaria solani* on tomato (10), limited the development of brown spot of pear to the same level achieved with a commonly followed 7-day commercial schedule, but with 28% fewer applications (11).

For increasing forecast accuracy and realizing potentially greater reductions in spray applications, the model should be adapted specifically to the biology of *S. vesicarium*. Several aspects of the biology of *S. vesicarium* have been studied, including the effect of temperature and relative humidity (RH) on mycelial growth and conidial germination of Italian (13), French (3), and Spanish isolates (11). However, the environmental conditions that influence infection of pear, specifically temperature and wetness, have been elucidated only partially (3,11).

The objectives of our study were to quantify the effect of wetness duration and temperature on the severity of infections by *S. vesicarium* on pear fruit and leaves, and to develop and validate a regression model of potential applicability in orchards to predict disease infection periods.

MATERIALS AND METHODS

Plant and fruit material. Pear plants of cultivar Conference of the CAV clone (Agromillora Catalana S.A., Barcelona, Spain), were used for inoculations. Plants were 3 years old, about 1 m high and were grown in 20-cm-diameter plastic containers filled with BVU potting mix (Prodeasa, Girona, Spain). Plants were maintained in the greenhouse and fertilized once a month with a solution of 200 ppm of 20-10-20 N-P-K. Pear fruit of the cultivar Passe Crassane were collected 2 weeks before harvest from a commercial orchard in Girona (Spain). Standard insecticide sprays were applied to the trees in the orchard and to the plants in the greenhouse. Nonsystemic fungicides also were applied until 3 weeks before the experiments were begun.

Inoculum production. A virulent strain of *S. vesicarium*, EPS26, isolated from a necrotic lesion on fruit of Passe Crassane was used for inoculations. Cultures were grown on V8 agar pre-

pared by combining, per 1 liter of distilled water, 100 g of V8 juice, 3 g of $CaCO_3$ and 20 g of agar. The pH was adjusted to 7. Inoculated petri dishes were incubated at 20°C under a 12-h photoperiod of fluorescent light. Conidial suspensions were prepared from 8- to 10-day-old cultures by gently rubbing the agar surface with a sterile cotton swab wet with a solution of 0.05% Tween 20 in distilled water. The suspension was shaken vigorously with a vortex mixer to break up conidia from mycelium and filtered through two layers of cheesecloth. Spore concentration was assessed with a hemacytometer and adjusted to 2×10^5 conidia per ml. Conidia germination was assessed after 2 h of incubation at 20°C in the hemacytometer chamber. Suspensions with less than 90% germination of conidia were discarded.

Inoculations. Before inoculation, the fruits were gently washed with a mild detergent solution, surface disinfested for 30 s in 0.1% sodium hypochlorite, and rinsed three times in deionized water. Fruits were inoculated by immersion for 1 min in a conidial suspension that was maintained at 4°C to prevent germination prior to inoculation. Plants were inoculated to incipient runoff by spraying the conidial suspension with a compressed air atomizer operated at 2 kPa. Two spray passes were made across each plant, one to wet the adaxial leaf face and another to wet the abaxial face. Noninoculated checks were also processed using a sterile Tween 20 distilled water solution.

Controlled environment studies. Immediately after inoculation fruits were placed in moist chambers made of transparent plastic bags with the inside sprayed with water. The moist chambers were placed at 5, 10, 15, 20, 25, and 30°C under continuous darkness in incubators (Hotcold, Selecta, Spain). Ten fruit were removed from incubators at periodic intervals ranging from 0 to 24 h depending on the temperature studied. Ten noninoculated fruit were also used as a nontreated control and were removed after 24 h. A total of 35 temperature and surface wetness combinations were tested. The experiment was performed twice.

Similarly inoculated plants were placed in a moist chamber consisting of a polyethylene plastic bag in which the inner side was sprayed with water to maintain leaf wetness. Wetness was continuous within the moist chambers and was monitored in the noninoculated checks. Plants were placed into a controlled-environment chamber (model PGR15, Conviron, Winnipeg, Manitoba, Canada) maintained at constant temperature and 90% RH in darkness. Temperatures of 5, 10, 15, 20, 25, and 30°C were maintained. Five plants were removed from the incubator chamber at postinoculation times from 0 to 24 h, depending on the temperature studied. A total of 33 temperature and surface wetness combinations were tested. The experiment was repeated once.

After removal from moist chambers, plants and fruits were placed in a drying room equipped with an air-conditioning system, maintained at the same temperature as during the wetting period, and exposed to an air stream supplied by an electric fan until they were dry. Drying times were <60 min. Although this additional wetting probably had some limited effect on infections, its distribution among leaves was not uniform and was considered of low quality. Therefore, residual wetting times during the drying process were not taken into account.

Upon drying, plants and fruits were incubated in a controlled-environment chamber maintained at 20±2°C, 80±5% RH and 16 h light, 8 h dark photoperiod (180 $\mu E\ m^{-2}\ s^{-1}$), until symptom development. Relative humidity was controlled by a steam humidifier model SD-103 equipped with an RH probe model SHWOOP (Carel, Padova, Italy). These conditions of incubation for symptom expression were chosen because preliminary experiments indicated there were no infections at 80% RH, and because the average temperature of wetness periods in orchards from June to September ranged from 16 to 23°C (mean 20°C). Care was taken to avoid wetting leaves and fruit during the incubation period.

Temperature, RH, and wetness duration were monitored in the 24 h moist chambers and in the symptom expression chamber by a temperature and humidity probe model SKH103, a leaf wetness sensor model 237 and a temperature probe model 107 interfaced to a CR-10 microprocessor datalogger (Campbell Scientific Ltd., Leicester, UK).

Disease severity was recorded 10 days after inoculation by visually determining the number of lesions per fruit or leaf. For plants, only the 10 to 15 leaves nearest the shoot tip on the current-season growth were used for disease assessment because young leaves are more susceptible to *S. vesicarium* than old leaves. The first three leaves resulting from new shoot growth during the incubation period were not considered.

Regression analysis was used to estimate parameters for the effect of temperature (T) and wetness duration (W) on disease severity (S). The following model was tested: $\log_{10}(S) = f(T, W)$, in which S is the number of lesions per fruit or leaf and $f(T,W)$ is a linear function of the terms W, T, WT, T^2, W^2, T^2W, TW^2, T^2W^2, T^3, W^3, T^3W, TW^3. Data were fitted to the model by means of the REG procedure of the Statistical Analysis System (13) using the backward method. Six criteria were used to evaluate the model: (i) randomness and normality of residuals; (ii) agreement between predicted and observed transformed values measured as the coefficient of determination (R^2); (iii) coefficient of determination adjusted for degrees of freedom (R_a^2); (iv) goodness of fit between back-transformed and observed values (R^{*2}); (v) significance of the estimated parameters; and (vi) standard deviation around the regression line (E) (18).

Comparison of regression equations was performed with an standard F test by comparing the residual sum of squares and degrees of freedom for error for each model (17).

Field studies and model validation. To validate results from studies with controlled-environment chambers, field studies were performed during 1989, 1990, 1993, and 1994 growing seasons in experimental and commercial orchards in Catalunya near Girona (Spain) and during 1994 in Emilia-Romagna near Bologna (Italy).

The leaf infection model was evaluated during 1993 and 1994 with artificially inoculated pear plants. The method consisted of exposing inoculated plants to natural conditions daily for periods of 24 h starting at 0800 h. For each validation trial, five plants were inoculated in the field with a conidial suspension of 2×10^5 conidia per ml and occasionally another set was sprayed with deionized water. Wetness, rainfall, and temperature were monitored with electronic sensors connected to a CR-10 datalogger. Also on each date, a set of five plants (positive control), was inoculated in the laboratory and placed in a controlled-environment chamber at 22°C but for 24 h of continuous wetness. Both sets of plants, field-exposed and chamber-exposed, were dried as described above and then incubated in a controlled-environment chamber at 20±2°C, 80±5% RH and 16 h light, 8 h dark photoperiod (180 $\mu E\ m^{-2}\ s^{-1}$) until symptom development. Disease levels were recorded after 10 days by visually determining the number of lesions per leaf on the 10 to 15 youngest leaves nearest the shoot tip. A relative index of disease was calculated using the ratio of the disease level obtained under field conditions to the level obtained under optimum temperature and wetness duration in the laboratory. This ratio corrected for variability in inoculum and plant performance during the experiment. Predicted disease levels were determined by interpolating the results of controlled-environment studies with wetness duration and mean temperature of wetness periods observed in the field. A predicted disease index was then calculated using the ratio of the disease level predicted under field conditions to the disease level predicted at 22°C and 24 h wetness. Model performance was evaluated by regression of the observed on the predicted disease index. An unbiased result should give a linear relationship with an intercept of 0 and a slope of 1.

The leaf and fruit models were also used to identify likely in-

fection periods during the 1989 and 1990 growing seasons in a 11-year-old commercial orchard of cultivar Passe Crassane located near Girona. Temperature and RH were assessed with a 7-day recording hygrothermograph and duration of wetness was determined with a 7-day leaf-wetness recording meter (Jules Richard Instruments, Belgium). Rainfall was assessed with a 12-cm plastic rain gauge. Hours of surface wetness and mean air temperature during wetness periods recorded in the field were used to interpolate daily disease severity values based on the results of the controlled-environment study. Actual infection periods during the growing season were determined by a previously described method based on sequential omission of protective fungicide sprays (11).

A chart of risk of infection has been developed by combining data generated by equations 1 and 3. The data were scaled by dividing by the predicted maximum value. Then, the highest prediction risk value for each T and W combination was selected and the isopaths corresponding to 0.2, 0.4, and 0.7 relative risk index were plotted.

RESULTS

Duration of wetness and temperature effects on severity. No lesions were observed on any noninoculated fruits or leaves, whereas some lesions (mean of 0.8 lesions per fruit) were observed on some inoculated 0 h wetness fruit controls. Therefore these background levels of severity were subtracted from all fruit data. Disease severity ranged from 0 to 14.6 mean lesions per fruit and from 0 to 3.5 mean lesions per leaf (Figs. 1A and 2A). For both fruit and leaves, there was a rapid increase in severity with increased wetness duration at temperatures of 15, 20, 25, and 30°C. At 5°C, no significant ($P > 0.05$) increase of infection levels was observed with increased wetness duration. Maximum disease severity was found at 20 to 25°C with between 18 and 24

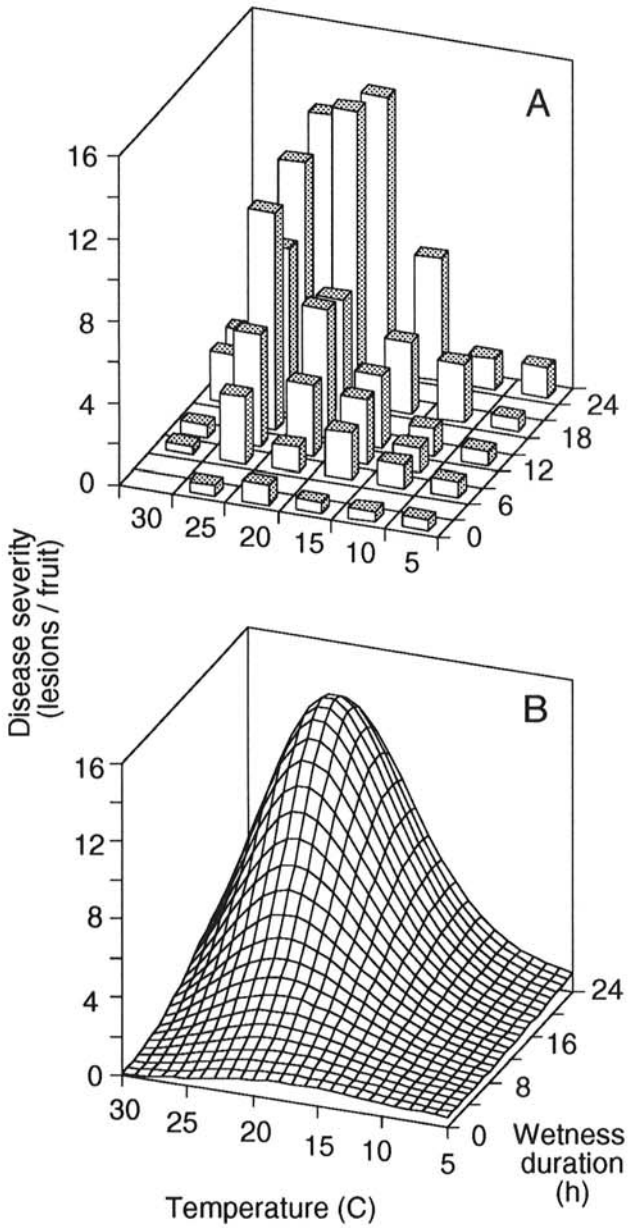

Fig. 1. A, Brown spot severities on fruit of pear cultivar Passe Crassane inoculated with conidia of *Stemphylium vesicarium* at different temperatures and wetness durations (mean of two experiments). **B**, Response surface based on the polynomial model predicting the number of lesions per fruit. Predicted lesion numbers were obtained by back transforming values generated by equation 1 (see text).

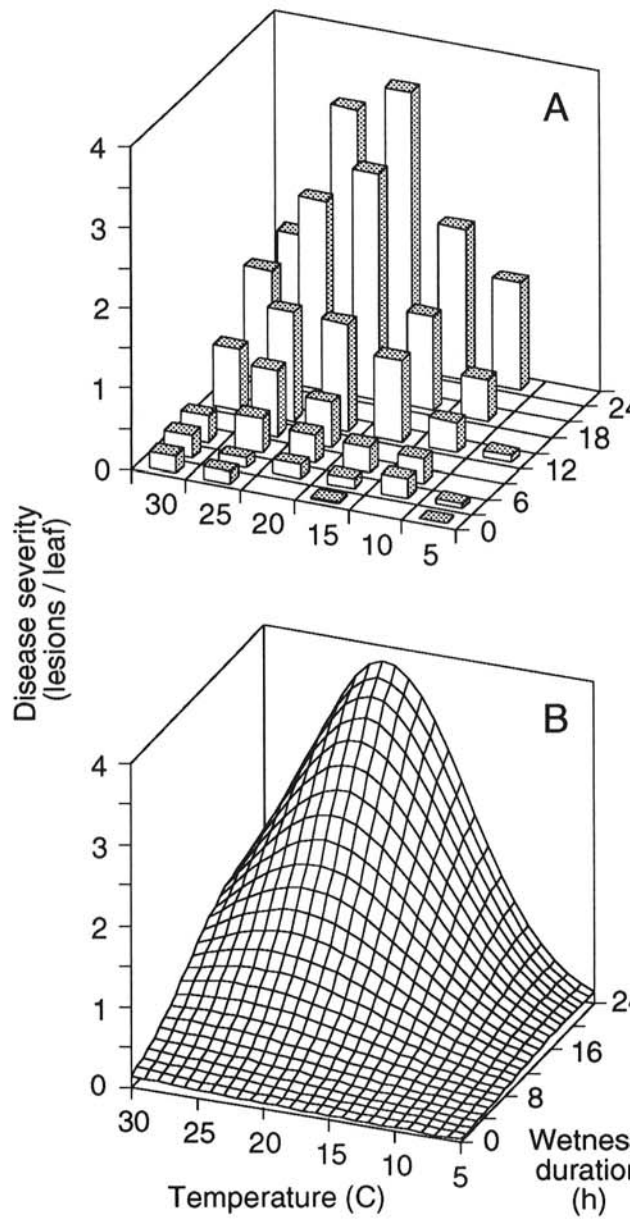

Fig. 2. A, Brown spot severities on leaves of plants of pear cultivar Conference inoculated with conidia of *Stemphylium vesicarium* at different temperatures and wetness durations. **B**, Response surface based on the polynomial model predicting the number of lesions per leaf. Predicted lesion numbers were obtained by back transforming values generated by equation 3 (see text).

h of wetness duration. A significant ($P < 0.05$) increase in disease above the background was detected only after 6 h of wetness at 10 to 30°C.

Model development. The analysis of fruit data sets for the two separate experiments performed in 1991 and 1992 yielded regression models that were not significantly different according to an F test ($F = 0.55$, $P > 0.25$), allowing the data to be pooled. The model fitted to the combined data was the following:

$$\log_{10}(S) = -0.38710366 + 0.00391111\,T^2 - 0.00015456\,T^3 + 0.00003139\,W^3 + 0.00034243\,T^2\,W - 0.00000696\,T^3\,W - 0.00000450\,T\,W^3 \quad (1)$$

Each estimated parameter was significant at $P < 0.05$ and the regression component of total variation was significant at $P < 0.01$. The coefficient of determination (R^2), R^2 adjusted for degrees of freedom (R_a^2), and the coefficient of determination for the back-transformed infection levels (R^{*2}) were 0.88, 0.86, and 0.89, respectively. The intercept of the regression of observed values on back-transformed predicted values was not significantly different from zero ($P = 0.063$) with a slope of 0.83 ($P = 0.0001$). Residuals had a random pattern and were normally distributed according to the Shapiro-Wilk test ($W > 0.99$, $P = 0.96$). Equation 1 described the data closely at 15, 20, and 30°C, but slightly overestimated disease severities at 10°C/18 h and at low wetness duration at 25°C.

The analysis of leaf infection data sets for the two separate experiments yielded regression models that were significantly different according to an F test ($F = 2.05$, $P = 0.05$). The results from both runs were not combined and are reported separately. Estimated parameters were significant at $P < 0.05$ and the regression coefficients for the models were significant at $P < 0.01$. The leaf infection model for the first run fitted to the following equation:

$$\log_{10}(S) = -0.28041180 - 0.31559679\,T + 0.10033443\,W + 0.02823408\,T^2 - 0.00199313\,W^2 - 0.00062564\,T^3 - 0.00003122\,T^2\,W \quad (2)$$

The R^2, R_a^2, and R^{*2} were 0.93, 0.90, and 0.73, respectively. The intercept of the regression of observed values on back-transformed predicted values was not significantly different from zero ($P = 0.20$) with a slope of 0.79 ($P = 0.0001$). Residuals had a random pattern and were normally distributed according to the Shapiro-Wilk test ($W > 0.98$, $P = 0.76$). The leaf infection model for the second run fitted to the following equation:

$$\log 10(S) = -1.70961637 + 0.02886124\,T + 0.04943329\,W + 0.00868240\,T\,W - 0.00236213\,W^2 - 0.00023788\,T^2\,W \quad (3)$$

The R^2, R_a^2 and R^{*2} were 0.92, 0.90, and 0.95, respectively. The intercept of the regression of observed values on back-transformed predicted values was not significantly different from zero ($P = 0.94$) with a slope of 1.00 ($P = 0.0001$). Residuals had a random pattern and were normally distributed according to the Shapiro-Wilk test ($W > 0.97$, $P = 0.51$). Equation 3 described the data very closely at 15, 20, 25, and 30°C but slightly overestimated disease severities at 5°C/12 h and produced underestimations at 10°C/24 h.

Equations 1 and 3 were used to generate response surfaces (Figs. 1B and 2B). In both surfaces, S increased monotonically with increases in W at all T and bell-shaped curves were predicted for the effect of temperature on S. Expected disease severity was maximal at 22.6°C for fruit infection and at 21.1°C for leaf infection after 24 h of wetness.

Model validation. The leaf infection model (equation 3), obtained from the controlled-environment study, was used to predict disease levels on inoculated plants exposed to field conditions in Girona (Spain) and Bologna (Italy). Average temperature observed during wetness periods in 24-h periods was used in the equation. For the total of 40 validation trials, daily wetness durations ranged from 0 to 24 h, and average temperatures ranged from 9.5 to 26.5°C. The observed disease severities in the field-exposed plants ranged from 0 to 11.9 mean lesions per leaf (Fig. 3).

The observed and predicted disease indices, calculated as described above were divided into three categories: low (0 to ≤0.33), medium (>0.33 to ≤0.66) and high disease potential (>0.66). Among the exposure periods, 31 were identified as low disease potential events, 5 as medium, and 4 as high. The model predictions agreed on 30 occasions (75%) corresponding to 26 events observed as low disease potential, two observed as me-

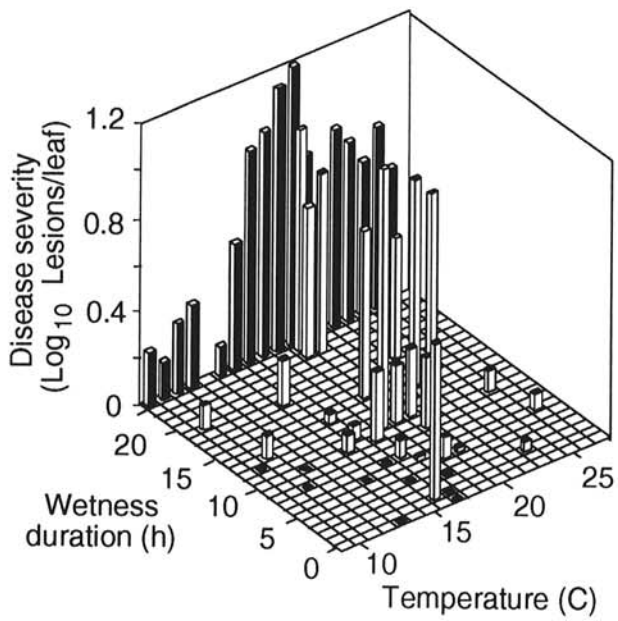

Fig. 3. Mean brown spot severity on leaves of plants of pear cultivar Conference inoculated with conidia of *Stemphylium vesicarium* and exposed for 24-h periods in the field or in growth chambers in relation to wetness duration and average temperature during the wetness period. Data correspond to natural conditions (open columns) in the orchard and to artificial wetting periods in controlled environment conditions (closed columns).

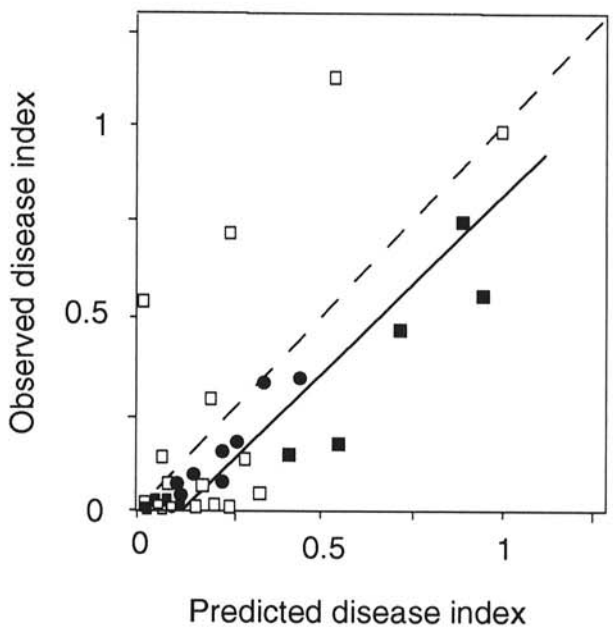

Fig. 4. Observed brown spot severity index on pear leaves based on equation 3 (see text), versus the predicted disease severity index of field-inoculated pear plants infected by *Stemphylium vesicarium*. Data correspond to 1993 (□) and 1994 (■) trials at Girona and to 1994 (●) at Bologna. Dotted line corresponds to a fitted line with an intercept of 0 and a slope of 1.

dium and on two observed as high infection risk. Two observed medium disease potential events were predicted as high and two observed high were predicted as medium. One observed medium disease potential event was predicted as low and five observed low were predicted as medium.

Model performance was also evaluated by regression of the observed disease potential index on the predicted disease potential index obtained from field-exposed and chamber-exposed plants (Fig. 4). The intercept was –0.18 ($P = 0.012$), the slope was 1.49 ($P = 0.0001$) and the R^2 value was 0.54 ($P = 0.0001$). When the data were examined in relation to the RH and wetness duration, three outliers that were clearly underestimated by the model were identified as high RH periods (>80% RH) but of low wetness duration. When these data were not included, the intercept was –0.08 ($P = 0.0001$), the slope was 0.93 ($P= 0.0001$) and the coefficient of determination was 0.83 ($P= 0.0001$). The intercept was significantly different from 0 ($P< 0.01$) and the slope was not significantly different from 1 ($P = 0.44$). Therefore, the model for predicting disease severity on leaves based on duration of wetness and average temperature of wetness periods is unbiased but tends to overestimate the potential for disease development.

In another evaluation approach, the fruit (equation 1) and leaf (equation 3) models were used to identify likely infection periods in a commercial orchard during 1989 and 1990. During the 1989 growing season daily wetness durations ranged from 0 to 24 h with average temperature from 12 to 25°C. Only 61% of the wetness periods were longer than 10 h and only 38% of these periods occurred at temperatures between 20 and 25°C. During the 1990 growing season daily wetness durations ranged from 0 to 24 h with an average temperature from 9 to 25°C. Eighty-three percent of the wetness periods were longer than 10 h and only 43% of these periods occurred at temperatures between 20 and 25°C.

Daily wetness duration and mean temperature of wetness periods were used to calculate a daily disease index for leaves and fruit. Predicted disease potential indices were compared with the results previously published on the observed infection periods in the orchard during 1989 and 1990 as determined by the method of sequential omission of fungicide sprays (12). During the 1989 experiment the disease index surpassed the threshold of 0.33 disease potential units on 21 occasions for fruit and 50 for leaves. However, these medium to high disease potential days were not uniformly distributed through the growing season, but clustered at particular times. Both models identified three major periods of disease development that were in agreement with the observed infection periods in the orchard during June, from mid-August to mid-September, and in late September (Fig. 5). During the 1990 experiment the disease index surpassed the threshold on 20 occasions for fruit and 57 for leaves. Only observed disease incidence on leaves reached values above the background level that permitted comparison with predicted infection risk levels (Fig. 6). The leaf model predicted seven main periods of potential disease development corresponding to late May, late June, mid-July, early August, late August early September, and a broad series of peaks in late September. In the orchard, seven leaf infection periods were actually observed after sequential omission of fungicide sprays.

DISCUSSION

Temperature and wetness duration were important environmental factors affecting fruit and leaf infection of pear by *S. vesicarium*. Equations 1 and 3 described disease severity well under the controlled conditions used in this study. The coefficients of determination (R^2) obtained indicate that a significant proportion of variability in disease severity was accounted for by the independent variables. The regression equations accounted for 89% and 95% of the variability of data for fruit and leaf infection, respectively. The relationship between disease severity and temperature gave bell-shaped curves, whereas with wetness duration sigmoid asymptotic curves were obtained.

The optimum temperature for brown spot of pear development was between 20 and 25°C. Maximum infection was observed at 22.6°C on Passe Crassane fruit and at 21.1°C on Conference leaves. These correspond to optimum temperatures for conidial germination (23°C) and for radial growth of mycelium (21°C) previously reported for six Spanish strains of *S. vesicarium*, including strain EPS26 used in this study for model development (11). Also, maximum disease levels on inoculated plants exposed to field conditions in Spain and Italy were observed in the range of 17 to 24°C. However, the optimum temperature for leaf infection of cultivar Alexandrine by a French isolate of *S. vesicarium* was between 25 and 30°C, and was in agreement with the optimum temperature for conidial germination (28°C) for the same isolate (3).

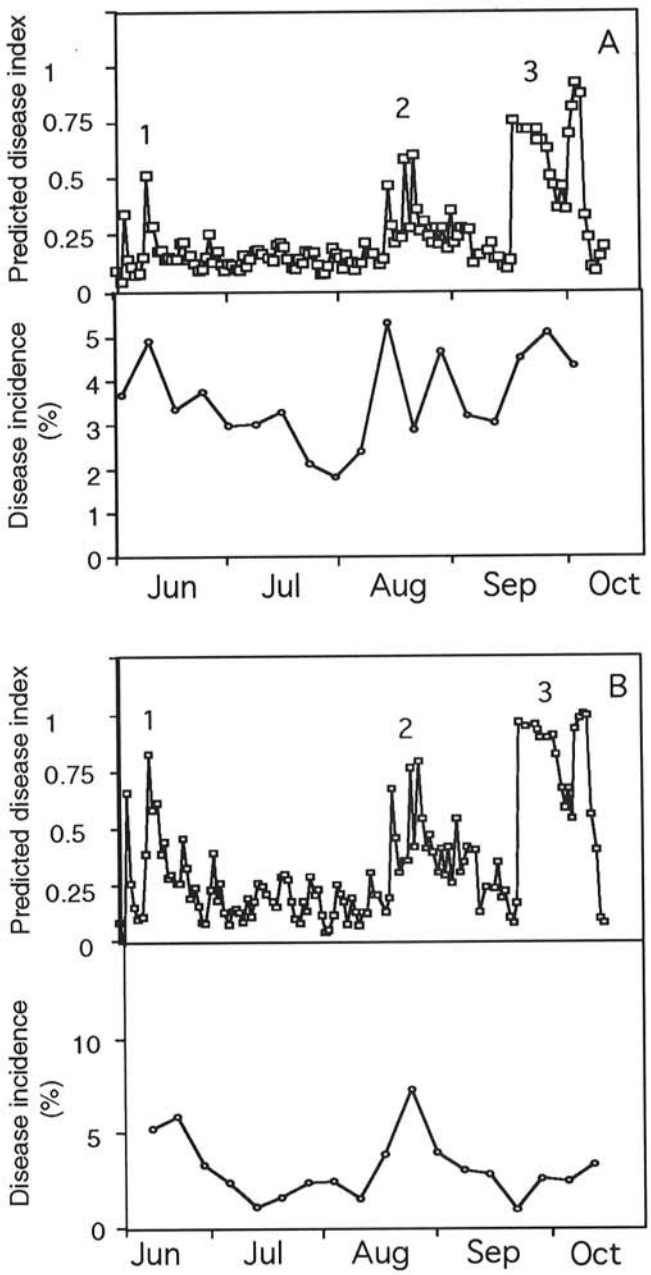

Fig. 5. Predicted brown spot severity index for each day on **A,** pear fruit and **B,** leaves according to equations 1 and 3 (see text) in relation to the disease incidence on trees of cultivar Passe Crassane left unprotected by fungicides for 4-week periods during the 1989 growing season. Numbers inside the graphs show main potential infection periods.

Under controlled-environment conditions, infection occurred after more than 6 h of wetness at the optimum temperature. Similarly, significant disease levels on inoculated plants of cv. Conference exposed to orchard conditions were only observed after wetting periods of 6 to 10 h at temperatures of 17 to 24°C. During 1989 and 1990, the periods of severe fruit infection in experimental orchards of cv. Passe Crassane, as determined by sequential omission of fungicide sprays, were observed at temperatures of 15 to 23°C and continuous daily wetness of 10 to 24 h (11).

The leaf model developed from controlled-environment studies and based on wetness duration and average temperature of wetness periods predicted leaf infection periods under field conditions with moderate accuracy. In 30 of 40 field trials the model predictions agreed with actual disease observations. If the low disease potential is arbitrarily considered as without risk of infection, then of the ten trials in which the model disagreed with the observations, 9 were not serious failures from a crop management point of view since they would only result in unnecessary sprays. Only in one case did a false negative occur, when an actual infection period was observed that was predicted by the model to be a period of low disease potential. The tendency of the model to slightly overestimate infections could be explained by 1) discontinuous wetting in the field resulting in a total wetness time of less infection efficiency than the equivalent at continuous wetness, or 2) loss of inoculum due to rain washoff from leaf surfaces. Additionally, the three outliers corresponding to underestimations at medium and high disease levels were observed during periods of high RH, which were not taken into account by the model, although they likely have an effect on infection.

The leaf and fruit models also closely identified likely infection periods in the field by using daily wetness duration and average temperature of wetness period. During the two growing seasons in a commercial orchard, over a total of 10 actual leaf infection periods, the leaf model timed accurately six and indicated broadly the other four infection periods. For unknown reasons, the fruit model predicted significant infections for fruit in 1990 and actual infections were not significant and remained below the background level during the growing season. Both models confirmed that periods of highest disease potential occur near the end of the growing season, from mid-September to harvest.

The results of our research with *S. vesicarium* in controlled-environment studies and in the field confirm the findings of many studies that have investigated the role of wetness periods in fungal diseases, concluding that the duration of wetness required for disease development depends on temperature (2,5,7,8,9,14,15, 18). The model described here for *S. vesicarium* on pear is similar in form to that used by Grove and Boal (7) for *Phytophthora cactorum* infection on pear, and also for the taxonomically related fungus *Alternaria mali* on apple (6).

A chart for risk prediction has been developed by combining back-transformed values from fruit and leaf models and plotting four categories of disease potential (Fig. 7). The chart will be used in small scale commercial programs for scheduling fungicides to control brown spot of pear.

The importance of high RH and discontinuous wetting periods,

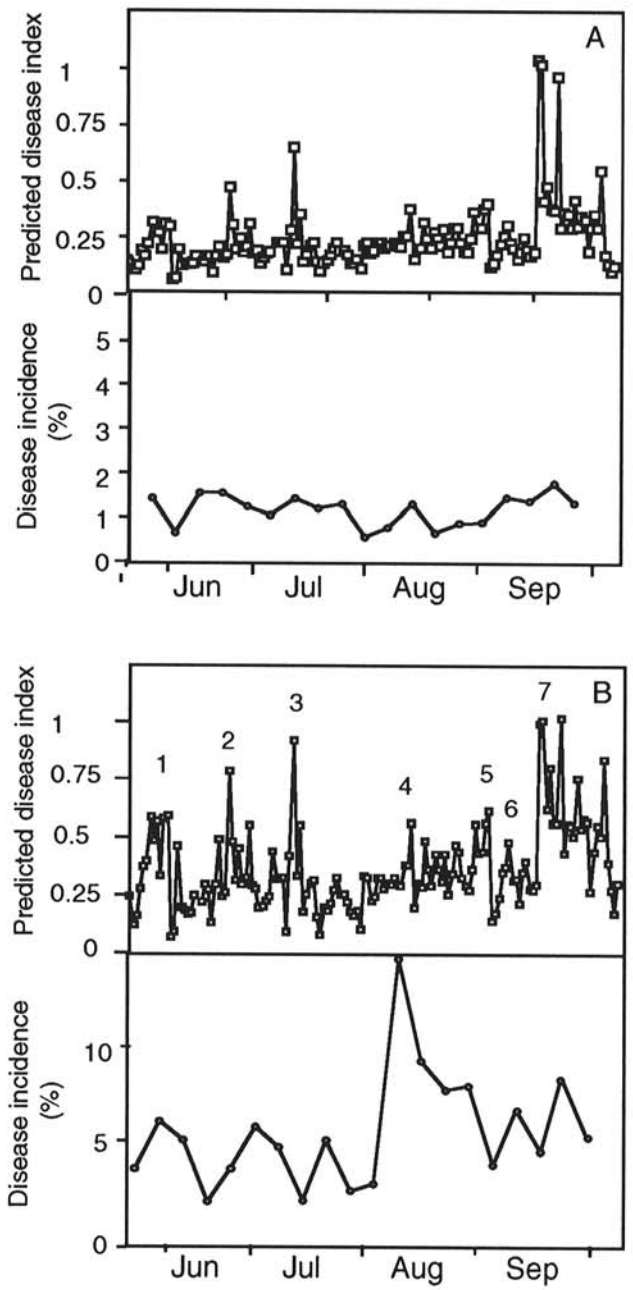

Fig. 6. Predicted brown spot severity index for each day on **A,** pear fruit and **B,** leaves according to equations 1 and 3 (see text) in relation to the disease incidence on trees of cultivar Passe Crassane left unprotected by fungicides for 4-wk periods during the 1990 growing season. Numbers inside the graphs show main potential infection periods.

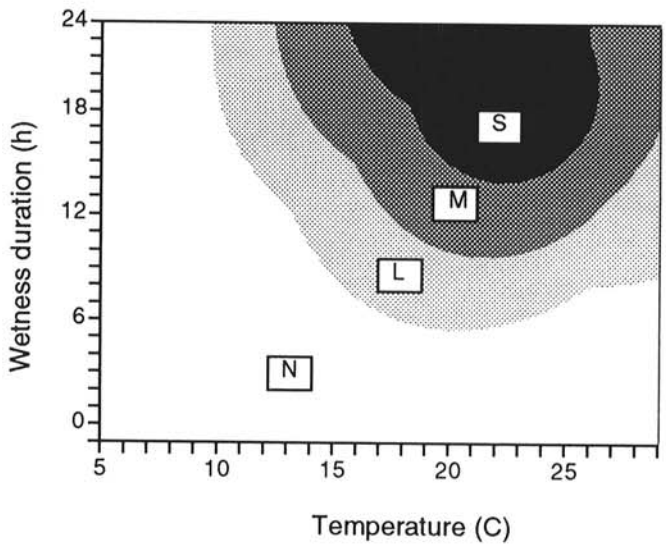

Fig. 7. Categories of potential brown spot severity as influenced by temperature and wetness duration assuming the presence of inoculum. Categories are based on the response surfaces (see Figs. 1 and 2) generated by equations 1 and 3 (see text), in which the isopaths separating the categories correspond to 0.2, 0.4, and 0.7 relative disease potential index. N = none; L = light; M = moderate; S = severe.

of host susceptibility and inoculum potential, should be studied and included to increase the predictive capacity of the model.

LITERATURE CITED

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