Development of an Infection Efficiency Model for *Plasmopara viticola* on American Grape Based on Temperature and Duration of Leaf Wetness

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ABSTRACT

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The infection efficiency of *Plasmopara viticola* was determined for the American grape, *Vitis lambrusca* 'Catawba.' Leaves on potted vines were inoculated with the fungus and exposed to a range of wetness durations (1–15 hr) at each of six fixed temperatures (5–30 C) in a growth chamber. The Richards function was then used for describing the results. The maximum infection efficiency obtained at each temperature, a measure of the asymptote parameter of the Richards model, was derived as a function of temperature using a second-order polynomial; the models fit to the pooled and averaged data described 84 and 96%, respectively, of the variation in this parameter. An optimum maximum efficiency of 0.07–0.08 occurred at 15–20 C, whereas little or no disease occurred at the 5 and 30 C extremes. Similarly, the rate parameter of the Richards model also could be expressed as a quadratic function of temperature; the models fit to the pooled and averaged data explained 72 and 82%, respectively, of the variation in the rate. Values of this parameter ranged from 0 at 3.8 and 30 C to 0.36 at 16.9 C. After substituting the polynomials for the asymptote and rate parameters in the linearized version of the Richards function, the model described 73% (pooled data) and 84% (averaged data) of the variation in infection efficiency. At 15 and 20 C, the efficiency rapidly increased from approximately 0 after 2 hr wetness to 0.06 after 4–5 hr; subsequent increase was gradual until a maximum of 0.08 was reached at 15 hr and 15 C. At 10 and 25 C, the initial increase required approximately 8–10 hr of wetness before leveling off at an efficiency of 0.05–0.06.

The infection process by which a fungal plant pathogen initiates disease can be divided into three distinct phases: germination, penetration, and colonization (10). In downy mildew of grape, caused by *Plasmopara viticola* (Berk. & Curt) Bel. and de Toni, the germination subphase consists of sporangia germination, zoospore swarming, and zoospore germination (7). Although each of these infection processes can be independently studied, infection efficiency, defined as the ratio between number of lesions produced and number of infection units (zoospores), is a measure of their combined effect. In epidemic development, infection efficiency is an important component of the corrected basic infection rate (19).

Early studies on downy mildew of grape examined the effects of environmental conditions on the germination of *P. viticola* sporangia (13,14,17) and on various phases of the infection cycle (8). Recent research has indicated that the infection process occurs if the product of the temperature and duration of wetness reaches a specific constant (1). Given these two abiotic factors, our objective was to develop a mathematical model that would allow us to quantitatively describe infection efficiency. Because the altered physiology of detached leaves may influence the infection process (6), our research was performed on whole plants. Also, the experiments were conducted on the American grape *Vitis lambrusca* 'Catawba' because it is commonly grown in Ohio and many states in the Northeast. Previously, most research involving *P. viticola* has been performed on the more susceptible cultivars of the European grape, *V. vinifera*, which also is commonly grown in California. Preliminary research on the methodology used for estimating the infection efficiency has been recently reported (12).

MATERIALS AND METHODS

Inoculation and treatments. One-year-old Catawba vines obtained from a commercial nursery were grown in a greenhouse in 15-cm pots. The vines were planted in a soilless medium of peat moss, perlite, and vermiculite (Pro-Mix BX, Premier Brands, Inc., New Rochelle, NY), pruned to a single shoot, and fertilized twice a month (Peters Peat-Lite Special, 15-16-17, W. R. Grace & Co., Fogelsville, PA). After approximately seven to 10 leaves had been produced on each vine, the second, third, and fourth youngest leaves were tagged; the first or youngest leaf was not used because it lacks functional stomates, a necessary prerequisite for infection (5,15). Video images of these leaves were recorded on VHS tape and later played back on an image analyzer (Imageplus, Dapple Systems, Inc., Sunnyvale, CA) to estimate leaf area.

Another set of vines, grown in the same fashion, were used to produce inoculum. At 8–12 days after inoculation, those vines exhibiting chlorotic lesions were placed in a dark mist chamber at 20 C for a minimum of 12 hr. Sporangia were then harvested from these leaves by washing the lesions with cold (4 C) distilled water containing a surfactant (1 drop of Tween 40/ L). Their germination was examined in vitro to determine the proportion of viable spores.

The abaxial surface of each tagged leaf was then inoculated by atomizing it at 69 KPa pressure with a suspension (100/ml) of *P. viticola* sporangia. Three spray passes were made across each leaf, each pass consisting of a set of parallel, overlapping strokes. This technique produced a uniform distribution of minute droplets on the leaf surface without runoff. The resulting sporangia density, as previously reported (12), was estimated to be approximately 1.5 sporangia/cm^2.

After inoculation, the plants were placed in a mist chamber located within a larger lighted growth chamber (130 μE m^{-2} s^{-1}) kept at a specified temperature. A humidifier (Herrmidifier Company, Inc., Lancaster, PA) maintained leaf surfaces at or near the free-water saturation point. Runoff from the mist occurred on only the upper leaf surface and therefore did not affect the inoculum droplets on the abaxial side. At approximately 2, 5, 8, 11, and 14 hr after inoculation, five plants were removed and allowed to dry in another lighted chamber (330 μE m^{-2} s^{-1}) at the same temperature but at ambient humidity (40–80% RH). Their drying time in this second chamber was estimated using a leaf wetness sensor attached to a CR21 micrologger (Campbell Scientific, Inc., Logan, UT); the sensor was misted when plants were placed in the chamber. The drying period ended when sensor voltage was < 0.3 mV (1.9 mV when wet). At this time the leaves were mostly dry, although an occasional droplet could be found. Actual drying times varied from 30 min to 4.5 hr, depending on temperature and ambient relative humidity. The temperature was monitored by placing a thermistor, also attached to the micrologger, in the center of each chamber at foliage height. After the leaves had dried, the plants were moved to a greenhouse maintained at approximately 25 ± 5 C. The total duration of wetness for each set of plants was the time spent in the "wet" chamber plus the drying time.

The above procedure was performed for chamber temperatures of 5, 10, 15, 20, 25, and 30 C; at 15, 20, and 25 C, an additional 1-hr wetness duration was examined. Thus, the experiment consisted of a total of 33 treatment combinations of temperature and leaf wetness. The entire experiment was conducted three times and the order in which the temperature treatments were performed within each replicate was random.

Estimation of infection efficiency. Infection efficiency (*IE*), defined as the number of lesions produced per zoospore, was estimated for each plant by using the following equation:

$$IE = (les)/(7 \text{ zoospores/sporangium}) \times (100 \text{ sporangia/ml}) \times (\text{ml inoculum})$$

in which *les* = total number of lesions on all three leaves on each plant at 8 days after inoculation, and ml inoculum = $0.016 \times$ total area of those three leaves at the time of inoculation. The number of lesions was obtained by directly counting the chlorotic spots on the leaves; keys or standard diagrams were not used. The constant 0.016 was determined in a previous study (12), which showed that the volume of inoculum applied was directly proportional to leaf area. Also, earlier experimentation had shown that an average of 7 zoospores are produced per sporangium (12).

Because leaf area was obtained, the severity of disease, measured as the number of lesions per square centimeter, also could be readily calculated. However, disease severity was not examined statistically because it was directly proportional to *IE*. Severity values can be obtained by multiplying *IE* by 11.2, which is the product of $7 \times 100 \times 0.016$.

Model development. The procedure used for development of a model to describe our results consisted of several interrelated steps. First, an equation for predicting the upper asymptote of IE (k) was derived by regressing the maximum *IE* obtained at each temperature against a second-order polynomial of temperature (T):

$$k = b_0 + b_1 * T + b_2 * T^2 \quad (1)$$

in which b_0, b_1, and b_2 are regression coefficients. Regressions were performed separately for each replicate (full model), the pooled data (reduced model), and the data averaged over all replicates.

In the second step, the linearized, no-intercept version of the Richards function (18) was fitted to the data for each temperature:

$$\ln[(IE/k)^{1-m} - 1] = -r * W \quad (2)$$

in which k = estimated asymptote from equation 1, m = shape parameter, r = rate parameter, and W = (duration of leaf wetness) − 1. The minimum wetness duration, 1 hr, was subtracted from the time period so that the model would begin at the origin. Equivalent results could have been achieved by adding an intercept term to the equation and not subtracting 1 hr from the time period. Also, a range of values of m were tried to achieve the best possible fit. The rate parameter estimates were then regressed against temperature by fitting a quadratic equation similar to that used to predict k:

$$r = b_0 + b_1 * T + b_2 * T^2 \quad (3)$$

Because the purpose of this step was to determine the general form of the relationship between the rate and temperature, only the averaged and pooled data were analyzed.

The final step, modeling *IE* as a function of T and W, consisted of substituting the rate parameter function (equation 3) for r in the Richards model (equation 2):

$$\ln[(IE/k)^{1-m} - 1] = b_1 * W + b_2 * W * T + b_3 * W * T^2 \quad (4)$$

The coefficients of this function were estimated for each replicate, pooled data, and the data obtained by averaging over all replicates. A standard F-test was performed to determine if the results for each replication were similar enough to allow pooling of the data (15), i.e., to determine if the full model was different from the reduced. Because some *IE*s were greater than the value of k predicted by the quadratic functions, the logarithm of a negative number, an undefined quantity, occurred. To avoid this situation, 0.01 and 0.035 were added to the predicted ks (equation 1) derived for the averaged and reduced models, respectively. The quadratic function developed from the pooled data also was used for predicting k for each of the separate replicate models.

In fitting the averaged data, each *IE* value was the mean of 15 observations: three replicates × five plants per replicate. In the full and reduced models, each *IE* value was an average over the five plants in that particular treatment combination. Thus, each replication model had a total of 33 observations (five temperatures × six wetness durations + three 1-hr wetness treatments), and the reduced model consisted of 99 observations (all three replicates combined). All regression analyses were performed by the Regress procedure of PRODAS (2).

RESULTS

In general, the incidence of disease with respect to temperature and duration of wetness resembled observations on the *IE*. At temperatures of 15–25 C, incidence increased rapidly during the first 4 hr of wetness to an upper asymptote of approximately 80% of the leaves infected (Fig. 1A). A similar rapid increase in *IE* occurred for this same temperature range; however, the increase did not begin until after 2 hr of wetness and the final *IE* attained appeared temperature dependent within the 15–25 C range (Fig. 1B). Nevertheless, both incidence and *IE* were lowest at the temperature extremes of 5 and 30 C.

Estimation of *k* and *r* parameters. The maximum *IE* obtained at each temperature, a measure of the upper asymptote k, was highest at 15 and 20 C and lowest at 5 and 30 C (Figs. 1B and 2). When the maximum *IE* was regressed against temperature using a quadratic equation (equation 1), the models fit to each replication, reduced model, and model fit to the averaged data all had relatively high coefficients of determination (Table 1). However, the intercept parameter, b_0, for replicate 1 was not statistically different from zero; the same parameter for the replicate 2 model was significant at $P < 0.05$. Nevertheless, an F-test comparing the full and reduced models (16) produced an F-value of 1.68 with $P = 0.23$. Hence, the parameters of the separate replication models were not statistically different overall, allowing the data to be pooled. Using the parameters of the quadratic equation derived from the averaged data, the predicted value of k reached a maximum *IE* of 0.083 at

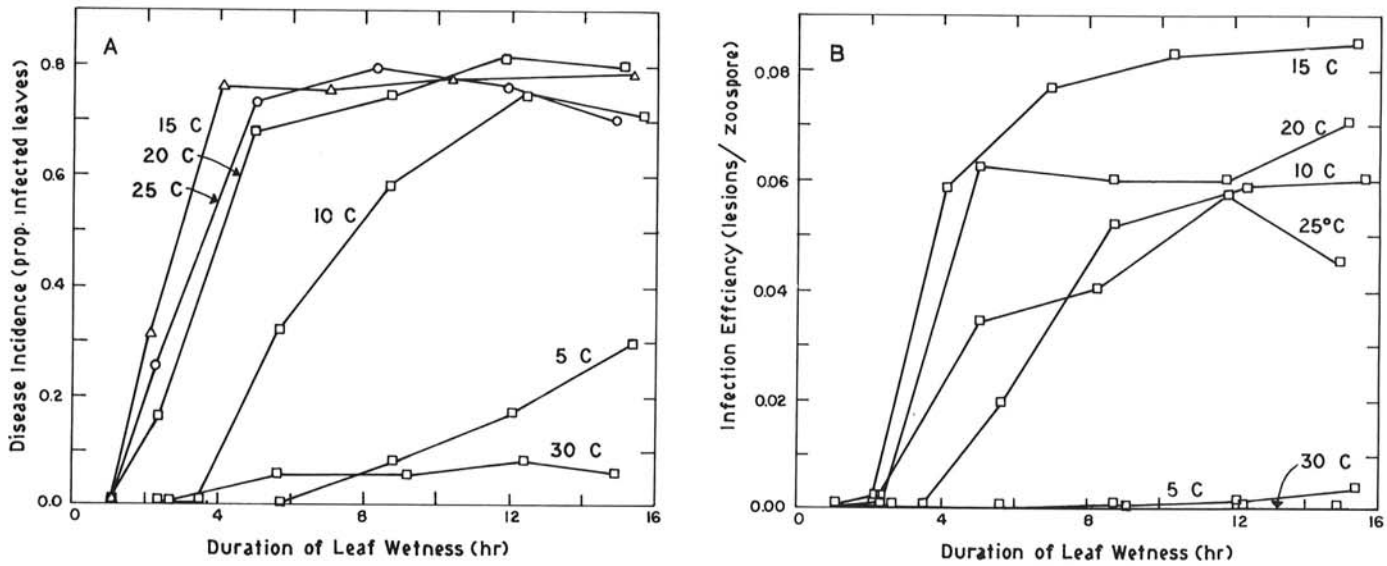

Fig. 1. Disease incidence **(A)** and infection efficiency **(B)** of *Plasmopara viticola* as a function of duration of leaf wetness for temperatures ranging from 5 to 30 C. Each point was an average of observations made on 15 plants (3 experimental replications, 5 plants/replication).

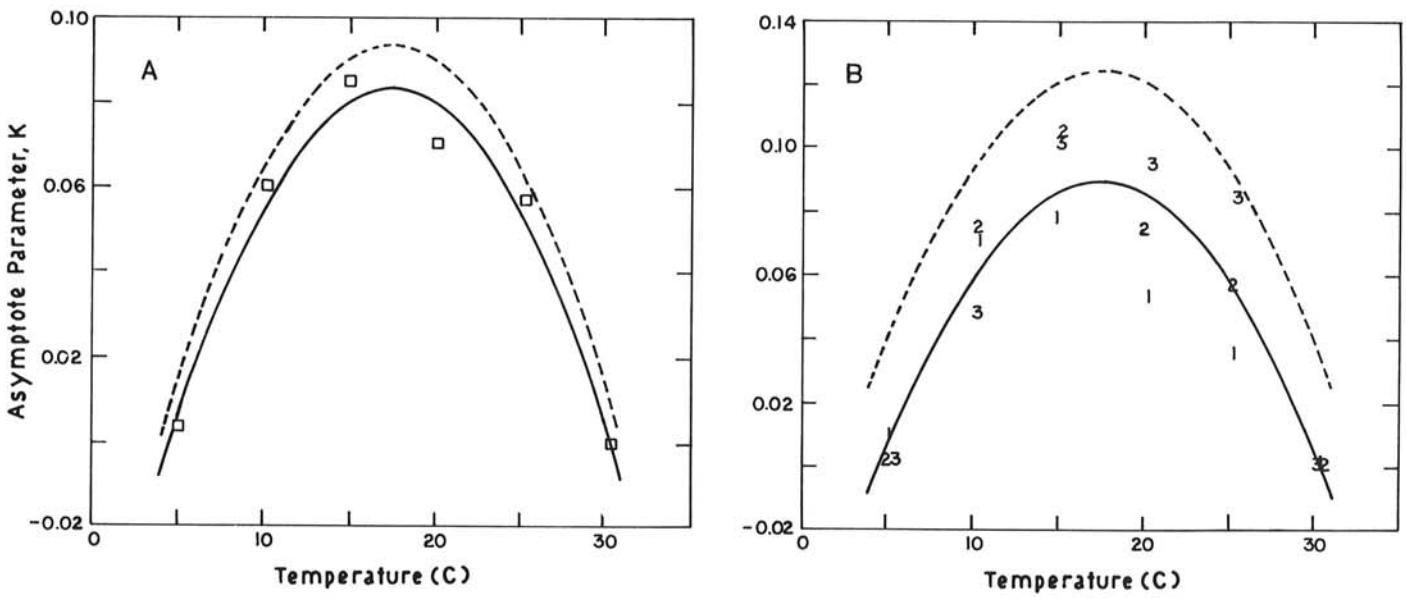

Fig. 2. The relationship between the maximum infection efficiency (k) and temperature for the averaged data **(A)** and pooled data **(B)** of the three replications. In A, the dashed line represents the predicted value of $k + 0.01$; in B, it is $k + 0.035$. The numbers 1–3 plotted in B refer to the experimental replications.

TABLE 1. Polynomial regression of the maximum $IE(k)$ on temperature[a]

Model	dF error	SS error	R_a^2	F P-value	Estimate/ P-value b_0	b_1	b_2
Replicate 1	3	0.00057	0.81	0.04	−0.039	0.013	−0.00040
					0.21	0.02	0.02
Replicate 2	3	0.00057	0.89	0.02	−0.077	0.020	−0.00057
					0.05	<0.01	<0.01
Replicate 3	3	0.00058	0.91	0.01	−0.114	0.024	−0.00066
					0.02	<0.01	<0.01
Reduced	15	0.0036	0.84	<0.01	−0.076	0.019	−0.00054
					<0.01	<0.01	<0.01
Average	3	0.00015	0.96	<0.01	−0.071	0.018	−0.00050
					0.01	<0.01	<0.01

[a]See text, equation 1.

17.5 C and 0 at 4.6 and 30.4 C.

When fitting the Richards model to the data for each temperature (equation 2), all of the estimated coefficients (r parameters), except those at 30 C, were significant at $P < 0.01$. Values of the shape parameter (m) near zero, which produce a monomolecular-type function, provided good fits to the data. This form of the function, however, did not account for the initial delay in the increase in IE observed at 15 to 25 C. Consequently, values of m near 1, which produces a Gompertz-type curve, were examined and found to give equally good fits. A final value of $m = 1.2$ was chosen because it provided the best overall fit across the different temperatures.

In the second step of model development, the rates from equation 2 were regressed against temperature. The quadratic function (equation 3) described 72 and 82% of the variation in the r parameter for the pooled and averaged data, respectively (Table 2). The model fit to the averaged data, however, had an insignificant intercept parameter. Given the parameters of the quadratic equation derived from the averaged data, the predicted value for r reached a maximum of 0.36 at 16.9 C and 0 at 3.8 and 30 C (Fig. 3A). When a single outlier, defined as having a standardized residual > 2, was removed from the pooled data set (Fig. 3B), the

reduced model described an additional 10% of the variation in the rate parameter (Table 2).

Infection efficiency model. All of the regression functions derived by fitting the Richards model (equation 4) to the data had highly significant F-values (Table 3). The model fit to the reduced or pooled data accounted for 73% of the variation in the infection efficiency. Although all but one of the parameter estimates for the replication models were significantly different from zero, corresponding estimates in each model were quite different. Furthermore, the estimated parameters increased in absolute value from the first to the third replication. An F-test comparing the full and reduced models produced an F-value of 4.26, which was significant at $P < 0.01$. Thus, the estimates for at least one parameter were statistically different among the three full models. However, when F-tests were conducted for each possible pair of the three models, replicates two and three had statistically similar estimates ($P = 0.28$), whereas the estimates of replicate one were different from replicate two ($P = 0.03$) and replicate three ($P = 0.001$).

The differences between the replicate models also are evident by comparing their predicted rates and infection efficiencies. The replicate one model estimated a maximum rate of 0.17, whereas the other models predicted a value of 0.29 (Fig. 4). Although this difference in rates translated into an IE difference of approximately 0.04 lesions/zoospore (Fig. 5), all three models nevertheless had similar optimum temperatures for their maximum rates and infection efficiencies. Furthermore, the

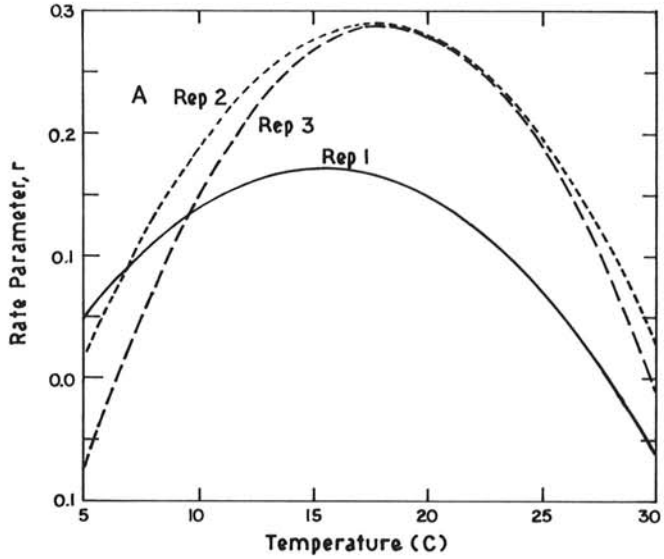

Fig. 4. Relationship between the predicted rate parameter (r) of the Richards function and temperature for each of the replicate models. The predictions were obtained by substituting the parameters from Table 3 into equation 3.

TABLE 2. Polynomial regression of the rate parameter of the Richards model (r) on temperature

Model	dF error	SS error	R_a^2	F P-value	Estimate/ P-value b_0	b_1	b_2
Reduced	15	0.062	0.72	<0.01	−0.24	0.056	−0.0016
					<0.01	<0.01	<0.01
Reduced[b]	14	0.035	0.82	<0.01	−0.22	0.054	−0.0016
					<0.01	<0.01	<0.01
Average	3	0.014	0.82	0.04	−0.24	0.071	−0.0021
					0.15	0.02	0.02

[a] See text, equation 3.
[b] Single outlier removed; see Figure 3B.

TABLE 3. Estimation of the infection efficiency of *Plasmopara viticola* as a function of temperature and leaf wetness by fitting the Richards model[a]

Model	dF error	SS error	R_a^2	F P-value	b_1	Estimate/ P-value b_2	b_3
Replicate 1	30	11.41	0.78	<0.01	−0.91	0.034	−0.0011
					0.07	<0.01	<0.01
Replicate 2	30	20.24	0.74	<0.01	−0.24	0.060	−0.0017
					<0.01	<0.01	<0.01
Replicate 3	30	18.66	0.79	<0.01	−0.40	0.076	−0.0021
					<0.01	<0.01	<0.01
Reduced	96	64.63	0.73	<0.01	−0.22	0.053	−0.0015
					<0.01	<0.01	<0.01
Average	30	22.61	0.84	<0.01	−0.24	0.070	−0.0021
					<0.01	<0.01	<0.01

[a] See test, equation 4.

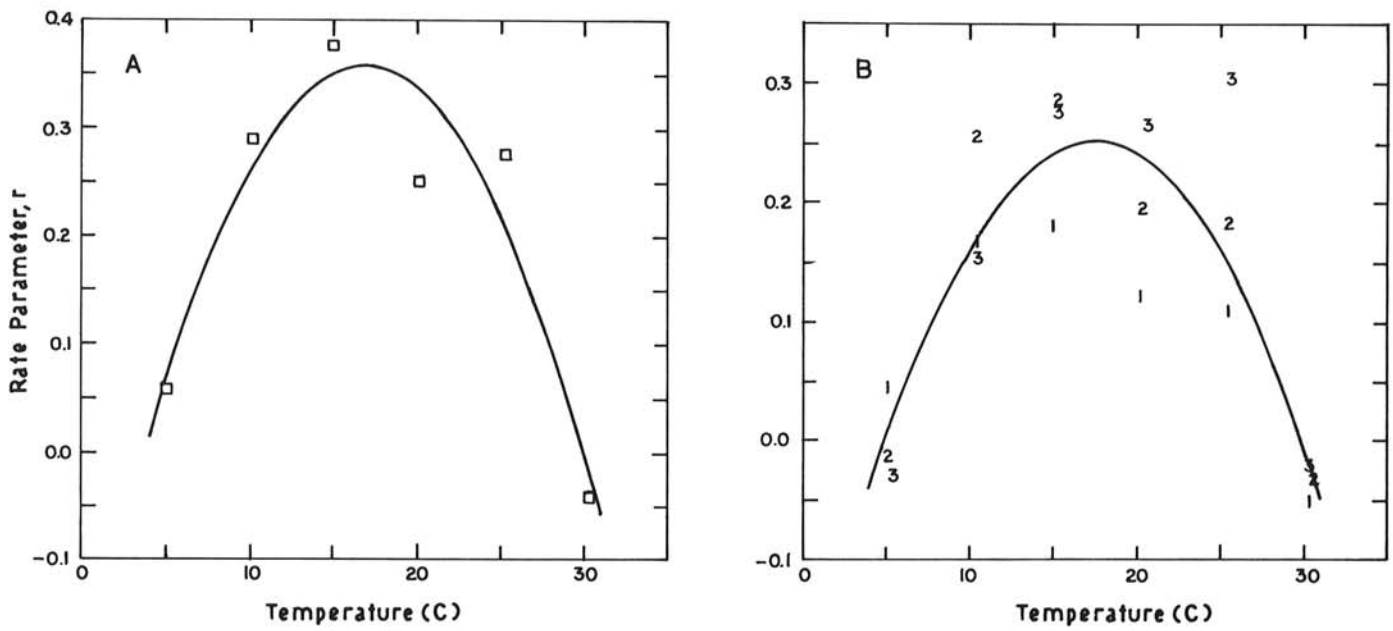

Fig. 3. Relationship between the rate parameter (r) of the Richards function and temperature for the averaged data **(A)** and pooled data **(B)** of the three replications. Values of r were generated by fitting the Richards function to the data for each temperature. The numbers 1–3 plotted in B refer to the experimental replications. When the observation for replication three at 25 C was removed from the pooled data set, the quadratic function described an additional 10% of the variation in r (see Table 2).

difference between the efficiencies decreased as the duration of wetness increased; for example, at 16 hr a difference of 0.3 lesions/zoospore occurred.

Instead of pooling the data from each replicate, an alternative was to first average over all replicates and then fit the model. Given this approach, the model described 84% of the variation in the infection efficiency (Table 3). The nonlinear relationship between IE and W and T was:

$$IE = k * (1 + e^{-\rho})^{1/(1-m)} \quad (5)$$

in which $k = -0.071 + 0.018*T - 0.0005*T^2 + 0.01$, $\rho = r*W = -0.24*W + 0.070*W*T - 0.0021*W*T^2$, and m = 1.2. The back-transformed predictions from this equation were highly correlated (0.91) with the observed infection efficiencies. Because three variables were involved, the equation described a three-dimensional response surface (Fig. 6). Although the model closely predicted the IE at 5 and 30 C, it underestimated the IE at 10 to 25 C during the intermediate durations of wetness (Fig. 7). An increase in the coefficient of the WT term from 0.07 to 0.08 provided a better fit during these intermediate durations, particularly at 10 and 15 C. However, the model now tended to overpredict at 20 and 30 C (Fig. 7, dashed lines). Equation 5 was actually a compromise: within the 10 to 25 C temperature range, 11 and 12 data points were located above and below the predicted curve, respectively (Fig. 7).

DISCUSSION

Although earlier studies did not quantitatively examine the relationship between temperature, wetness duration, and infection efficiency, their observations are nevertheless useful for comparison. Muller and Sleumer (14), Ravaz (17), and Maier (13) showed that the germination of sporangia occurred in less than 2 hr in the optimum temperature range of 15–24 C; much longer incubation periods were necessary as the temperature approached the 5 or 30 C temperature extremes. Our results revealed a similar optimum range of 15–20 C for infection efficiency. However, the efficiency remained low after 2 hr of leaf wetness at temperatures in this range. This discrepancy may be due to the additional time necessary for zoospore swarming and encystment, which have been reported to take an additional 1–1.5 hr (7). Nevertheless, enough zoospores are able to initiate infection during this short time interval. A recent study showed that 50% of the inoculated leaves of *V. vinifera* became infected at these temperatures with as little as 2 hr of leaf wetness (1). In our study on *V. lambrusca*, approximately 15–30% of the leaves became infected when exposed to the same conditions (Fig. 1A). In almost all cases, however, only a single lesion was observed on any given leaf.

When both the asymptote (k) and rate (r) parameters of the Richards equation were modeled as quadratic functions of temperature, the composite model provided a reasonably good fit to the data in each experimental replicate. Thus, the choice of model seemed appropriate. However, the absolute value of the parameter estimates increased for each successive replicate such that the predicted IE values were lowest for replicate one and highest for replicate three. Most variables, such as inoculum viability, plant age/maturity, observation time, or chamber conditions were held relatively constant or controlled and would not be expected to produce the observed pattern of increase. The conditions during greenhouse incubation, however, would have been different. The first replicate was performed during February, March, and April when temperatures were easily maintained near 25 C. Replicates two and three occurred in May, June, and July, a time when temperature fluctuations were greater. Warmer temperatures have been reported to lengthen the incubation period (14), but this would have resulted in lower, not higher, IE estimates for replicates two and three. Another possibility may be that the ability of the observer to detect and differentiate lesions on a leaf was "learned" during the course of the experiment; more lesions were observed as sensitivity increased. Nevertheless, the average IE obtained at 20 C, approximately 0.06, was the same as that observed in an earlier, independent experiment (12). Also, the maximum IEs (k) attained at each temperature were statistically similar for each of the replicates (Table 1, Fig. 2B).

Prediction of disease incidence or severity from environmental data is an alternative to predicting infection efficiency. Blaeser and Weltzien (1), who studied the effects of temperature and duration of leaf wetness on infection of *V. vinifera*, showed that the product of these two variables must be, on the average, 49.7 to obtain infection in at least 50% of the inoculated leaves. They observed this relationship to be valid within the temperature range of 6 to 25 C. Thus, at 10, 15, 20, and 25 C, wetness durations necessary for 50% incidence are approximately 5, 3.3, 2.5, and 2 hr, respectively. On *V. lambrusca*, we observed somewhat lower levels of disease for similar infection conditions. At 10, 15, 20, and 25 C with wetness durations of 5.6, 2.1, 2.3, and 2.2 hr, we obtained 32, 31, 16, and 25% of the leaves infected; these levels correspond to temperature-duration products of 56, 31, 46, and 55, respectively. At 5 C, 1 degree below their studied range, we obtained only 29% of the leaves infected after 15.4 hr of leaf wetness, whereas the relationship predicts 50% infection after 9.9 hr. Also, at the extreme temperature of 30 C, our highest incidence was 8% after

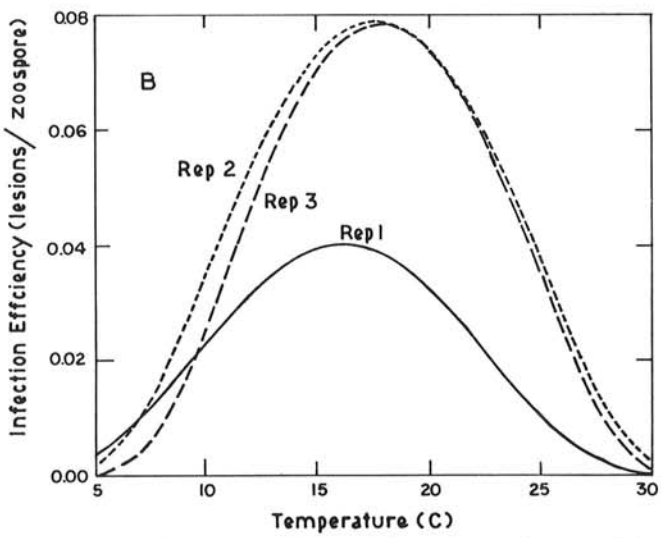

Fig. 5. Infection efficiency as a function of temperature for each of the replicate models. The wetness duration was held constant at 8 hr. Predictions were obtained by using the parameters given in Table 3.

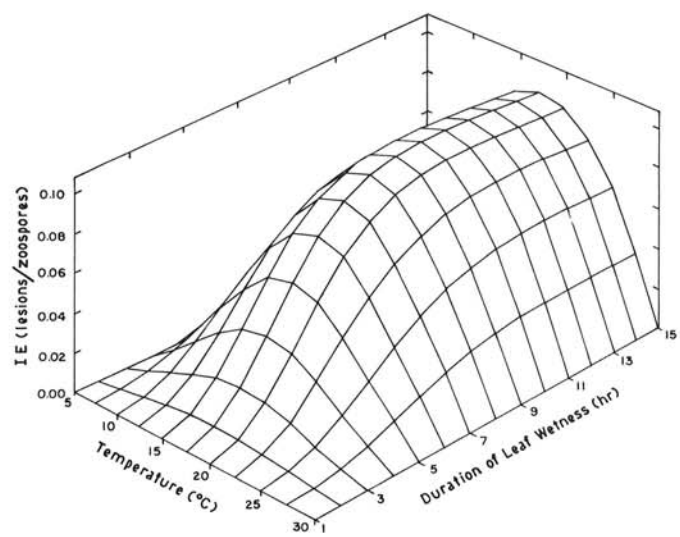

Fig. 6. Three-dimensional representation of the infection efficiency as a function of temperature and duration of leaf wetness. The nonlinear form of the model derived from the averaged data was used to calculate the predicted values (see text, equation 5). The duration of wetness is the observed time less 1 hr.

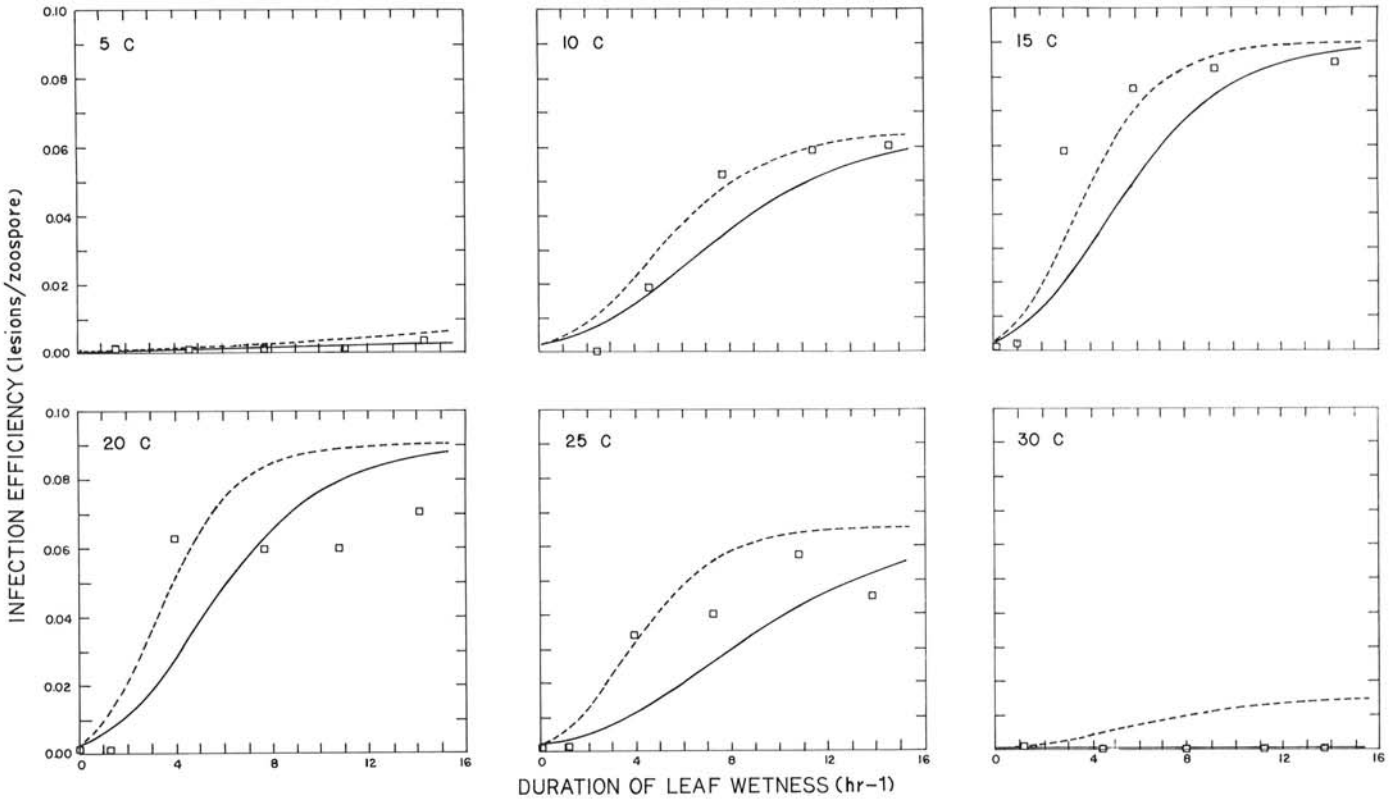

Fig. 7. Prediction of the infection efficiency of *Plasmopara viticola* for various combinations of temperature and duration of leaf wetness. The solid line represents the Richards function fit to the averaged data (see text, equation 5). If the coefficient of the *WT* term of the model is increased by 0.01, the predicted values increase, as shown by the dashed line. The plotted points were averages of observations made on all three replications (15 plants). The abscissa is the duration of wetness less 1 hr.

12.3 hr of wetness, which produced a temperature-duration product of 374. Obviously, the relationship is invalid for high temperatures because the infection efficiency is decreasing while the temperature-duration product is increasing. We suspect that the lower incidences were due to the higher degree of resistance present in *V. lambrusca*.

The procedure used for finding the mathematical function for the response surface was only one of several possible approaches (9). The untransformed *IE* observations could have been directly regressed against a second-order or even third-order model of temperature and duration of wetness. A model for predicting infection periods of *Coccomyces hiemalis* on sour cherry was developed in this manner (4). This empirical approach, however, excludes the possibility of using a growth function, such as the Richards function, in which parameters have biological meaning. Another possibility was to directly estimate the parameters of the model using nonlinear regression. This method avoids the necessity of first expressing the parameters as some function of an independent variable; however, it may be very difficult to determine the functional form of the model. Imhoff et al (11), for example, were unable to find a relationship between Richards model parameter values and temperature for predicting bean rust urediospore germination; this could have been due to a high degree of intercorrelation among parameter estimates, particularly with the Richards function (3). Our results showed a clear relationship between the rate and asymptote parameters and temperature. Consequently, a multiple stage approach was considered reasonable, especially because it would allow us to indirectly evaluate the effect of temperature on infection efficiency through its effects on the model parameters.

Two factors must be considered when implementing the model for prediction purposes. First, because the r and k parameters were expressed as second-order polynomials, the potential exists for spurious results if the model is applied outside the examined range of temperature. Second, because a constant was added to the k parameter during model development, the model may tend to overpredict the amount of infection. In terms of application, this type of bias, however, is much more desirable than the converse.

The infection process is no doubt one of the primary phenomena in the epidemiology of grape downy mildew. Given our model, the duration of leaf wetness can be viewed as the factor that allows infection to take place, whereas the temperature determines the rapidity and extent of that infection. We chose infection efficiency as the dependent variable, as opposed to disease incidence or severity, because it can be directly coupled to quantitative predictions of sporulation. For example, based on environmental conditions, a sporulation model could estimate the number of sporangia produced per unit area of lesion. Then, during a subsequent infection period, the efficiency or proportion of those spores capable of initiating disease could be easily calculated. Any loss of viability of the sporangia during the interim between the sporulation and infection periods could be accounted for by a survival model. The quantitative nature of such a forecasting system should, theoretically, allow for more accurate predictions than a similar, but qualitative, system. However, if this were not the case, the quantitative models would nevertheless be useful for simulation purposes.

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