

A Simple Generic Infection Model for Foliar Fungal Plant Pathogens

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ABSTRACT

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In this study, a simple generic infection model was developed for predicting infection periods by fungal foliar pathogens. The model is designed primarily for use in forecasting pathogens that do not have extensive epidemiological data. Most existing infection models require a background epidemiological data set, usually including laboratory estimates of infection at multiple temperature and wetness combinations. The model developed in this study can use inputs based on subjective estimates of the cardinal temperatures and the wetness duration requirement. These inputs are available for many pathogens or may be estimated from related pathogens. The model uses a temperature response function which is scaled to the minimum and optimum values of the surface wetness duration requirement. The minimum wetness duration requirement (W_{\min}) is the number of hours required to produce 20% disease incidence or 5% disease severity on inoculated plant parts at a given temperature. The

model was validated with published data from 53 controlled laboratory studies, each with at least four combinations of temperature and wetness. Validation yielded an average correlation coefficient of 0.83 and a root mean square error of 4.9 h, but there was uncertainty about the value of the input parameters for some pathogens. The value of W_{\min} varied from 1 to 48 h and was relatively uniform for species in the genera *Cercospora*, *Alternaria*, and *Puccinia* but less so for species of *Phytophthora*, *Venturia*, and *Colletotrichum*. Operationally, infection models may use hourly or daily weather inputs. In the case of the former, information also is required to estimate the critical dry-period interruption value, defined as the duration of a dry period at relative humidities <95% that will result in a 50% reduction in disease compared with a continuous wetness period. Pathogens were classified into three groups based on their critical dry-period interruption value. The infection model is being used to create risk maps of exotic pests for the U.S. Department of Agriculture's Animal Plant Health and Inspection Service.

Additional keyword: risk assessment.

Disease forecast models can be classified as empirical models based upon statistical relationships between environmental variables and disease versus fundamental models based upon laboratory, greenhouse, or field experiments (42,45). The typical components of a fundamental, mechanistic model include sporulation, dispersal, infection, incubation, and integration (74, 89). For many foliar pathogens, the infection submodel is one of the most critical components for disease forecasting (45). This is because the infection process usually is limited by the duration of surface wetness or high humidity in most terrestrial environments.

Many infection models use regression equations, such as those based on polynomials (13,31,39,43,68,84), logistic equations (15, 47,68), and complex three-dimensional response surfaces (14,20, 26,32,36,49,67,72,93). Other infection models have been constructed using variations of the Analytis Beta function, including infection (5,30,50). However, only a few models have been constructed using temperature response equations (16,17,30) or wet degree-hours (60). These infection models are created from either laboratory or field observations of resulting disease intensity at multiple combinations of temperature and wetness (45). However, for many pathogens, especially those from overseas, such data sets may not exist. Instead, the data available for many of these less-studied pathogens may be limited to growth studies in culture, simple correlations of disease observations in the field with

environmental variables, or inferences made from closely related organisms. Consequently, a generic model that can predict infection based only upon estimates of the three cardinal temperatures and a surface wetness duration requirement could be helpful for modeling pathogens for which extensive epidemiological data are unavailable.

In this study, a generic model was developed to estimate infection from an organism's cardinal temperatures and surface wetness duration requirement. The model is based upon a temperature response function (90,97) which is scaled to the surface wetness duration requirement. Predictions from the model were statistically compared with disease observations from 53 published studies of infection under controlled environmental conditions. Interruptions to wetness are also important for estimating infection from hourly weather data; therefore, a separate analysis examined influence of the duration and timing of such interruptions. The overall objective was to develop a simple, generic model for infection by foliar fungal pathogens for use in exotic disease forecast systems.

MATERIALS AND METHODS

Theory. The model estimates the wetness duration required to achieve a critical disease intensity at a given temperature. The critical disease threshold is defined here operationally as 20% disease incidence or 5% disease severity on an infected plant part at nonlimiting inoculum concentration. The threshold is chosen to enable the model predictions for each study to be compared uniformly rather than as an indicator of disease incidence in the field. The wetness duration requirement ($W_{(T)}$) for the critical disease threshold at temperature T is estimated from a temperature

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response function ($f_{(T)}$) and the minimum value of the wetness duration requirement (W_{\min}):

$$W_{(T)} = W_{\min}/f_{(T)} \leq W_{\max} \quad (1)$$

where $W_{(T)}$ = wetness duration requirement (in hours) for the critical disease threshold at temperature T , W_{\min} = the minimum value of the wetness duration requirement for the critical disease threshold at any temperature, and $f_{(T)}$ = temperature response function (97). Thus, the model estimates a simple temperature-wetness response for each pathogen with the interaction between temperature and wetness ignored; the implications of these assumptions are discussed later. The parameter W_{\max} provides an upper boundary on the value of $W_{(T)}$ because wetness is not always a rate-limiting factor. For pathogens that require high relative humidity rather than free moisture, the wetness requirement may also be defined as the number of hours above a relative humidity threshold. The model uses the temperature response function of Yin et al. (95,97), which is a simplified and improved version of the rice clock model (33). The function uses a pathogen's cardinal temperatures to estimate the shape parameter and the temperature response:

$$f_{(T)} = \left(\frac{T_{\max} - T}{T_{\max} - T_{\text{opt}}} \right) \left(\frac{T - T_{\min}}{T_{\text{opt}} - T_{\min}} \right)^{(T_{\text{opt}} - T_{\min}) / (T_{\max} - T_{\text{opt}})} \quad (2)$$

if $T_{\min} \leq T \leq T_{\max}$ and 0 otherwise, where T = mean temperature ($^{\circ}\text{C}$) during wetness period, T_{\min} = minimum temperature for infection, T_{\max} = maximum temperature for infection, and T_{opt} = optimum temperature for infection. The advantages of the Yin function compared with other growth functions include the fact that the function has only three parameters (T_{\min} , T_{opt} , and T_{\max}) and each parameter has a clear biological meaning (95). The function gives a smooth curve as opposed to a series of lines with abrupt changes between them. The function combines the advantages of several equations: an exponential response at low temperatures, a positive linear response at intermediate temperatures, a parabola response at optimum temperatures, and a negative response at high temperatures. The model has been validated with data sets of crop growth (95). In developing the model, other growth functions were examined and the Wang and Engel (90) response function also was found to be suitable. A comparison between the Wang and Engel (90) and Yin (97) function showed that the results were almost identical (data not shown); therefore, only the results for the Yin function are presented.

Infection models commonly are run from daily or hourly temperature and leaf wetness data. With hourly data, it is necessary to know how many dry hours may interrupt a wet period without terminating the infection process. The additivity of two interrupted wet periods is determined by the critical dry-period interruption value (D_{50}). Consider the case of two wet periods, W_1 and W_2 , separated by a dry period D . The sum of the surface wetting periods (W_{sum}) is given as $W_{\text{sum}} = W_1 + W_2$ if $D < D_{50}$ or $W_{\text{sum}} = W_1, W_2$ if $D > D_{50}$.

The parameter D_{50} is defined as the duration of a dry period at relative humidities $<95\%$ that will result in a 50% reduction in disease compared with a continuous wetness period. The value of D_{50} is sensitive to the time when the dry period occurs.

Experimental data. Data from 53 published studies of the temperature and moisture response for plant pathogens was utilized. The studies include a variety of crop and pasture plants. These studies were identified primarily by searching in the Commonwealth Agricultural Bureau literature database (CABI, Cambridge, MA) using the keyword search "infection and temperature and wetness". Other studies were identified from the references contained in these papers. And all were made under controlled environmental conditions. In the experiments, either whole plants or plant parts were inoculated with a defined spore concentration, specified by either volume or area (Table 1). The

plants then were incubated at different temperatures in moist conditions by enclosure with plastic bags or by placement in dew chambers. To be included, each study had to have the critical disease threshold determined at four temperatures. Only those studies that used constant temperatures were included. After different wetness durations, the plants were withdrawn, dried, or allowed to dry, then placed in a growth chamber that was not subject to further wetness or high humidity. After sufficient time had elapsed, the intensity of disease on plants was assessed. Disease assessments were made in a variety of ways and included incidence, severity, categories, ratings, and lesion counts (Table 1).

For each study, the duration of wetness required to achieve either 5% disease severity or 20% disease incidence (W_{\min}) was recorded by reading values from the original graphs or tables. A severity value of 25% was used for *Ascochyta rabiei* because the slope of the response at 5% was zero (84). Studies that used lesion counts were difficult to compare because the counts could be made per square centimeter, per leaf, or per plant. Where ratings or lesions counts were used, the point of inflection of the disease intensity curve was chosen as a representative W_{\min} . Likewise for studies using severe or light designations (7,76,78), the category corresponding to light was used. Because of its general importance, a compilation of studies was used in the case of *Venturia inaequalis* (78).

Similar to W_{\min} , the values of the model parameters T_{\min} , T_{opt} , T_{\max} , W_{\min} , and W_{\max} were visually estimated solely from the data contained in the individual studies. If there was no apparent lower limit for infection in the data, T_{\min} was estimated from the host's development threshold. If data from the study showed that infection occurred below the host's development threshold, T_{\min} was set at 5°C lower than the lowest tested temperature, but not lower than 1°C . If there was no upper temperature limit on infection, T_{\max} was set at 35°C . The observed value of W_{\max} was used in the model; however, because this parameter may be unknown for some pathogens the relationship between W_{\min} and W_{\max} was statistically examined by linear regression analysis.

Parameter estimates were entered and model predictions for $W_{(T)}$ based on equation 1 and 2 were calculated in MS Excel (Microsoft, Redmond, WA). The observations from the original studies and model predictions of $W_{(T)}$ were compared using Pearson's correlation coefficient (54) and the root mean square error (RMS) (75). These statistical tests all were calculated manually in Excel. In order to make the statistical analysis more sensitive, observations where the critical disease threshold was not reached were not included. The value of RMS was influenced by W_{\min} ; therefore, a standardized RMS (SRMS) was estimated by dividing RMS by W_{\min} .

Interrupted wet periods. The literature was searched for studies which investigated the effect of the interruption of continuous wetness to determine D_{50} for a selection of pathogens. In each study, the following information was recorded: the time of initial wetness prior to the interruption, the duration of tested dry interruptions, the relative humidity during the interruption, and the incidence or severity of disease for the interrupted and continuous wetness. The duration of the dry period for which disease was reduced by 50% was estimated as the value of D_{50} . To be included in the data set, the initial wet period had to be lower than the estimated W_{\min} for that pathogen.

RESULTS

Approximately 90% of the pathogens had a value of $W_{\min} < 20$ h and the average value of W_{\min} was 9 h. In contrast, the average value of W_{\max} was 30 h, and 90% of pathogens had a value of $W_{\max} < 75$ h. W_{\max} could be estimated from W_{\min} based on the equation $W_{\max} = 3.8 + 3.0 W_{\min}$ ($r = 0.71$, RMS = 6.0 h, $n = 64$ studies).

There was some uniformity in W_{\min} among pathogens in the same genus (Table 2). For example, three species of *Cercospora* had a W_{\min} close to 24 h. Six species of *Alternaria* had a W_{\min} of ≈ 6 to 8 h and five species of *Puccinia* had a W_{\min} of 5 to 7 h. There were larger differences between the two *Venturia* and *Colletotrichum* spp., as well as among the *Phytophthora* spp. However, some of these differences may be due to the difference between infection requirements for fruit and leaf tissue.

In general, the model was able to predict $W_{(T)}$ successfully compared with the original observations in most cases (Table 2). The average and median values of r were 0.83 and 0.94, respectively. The RMS and the SRMS were 4.8 h and 0.6, respec-

tively. The model fitted the data quite well for many organisms, such as *V. inaequalis* (Fig. 1A) and *Pseudoperonospora cubensis* (Fig. 1B). In some cases, the model might fit poorly when there was uncertainty about the values of the parameters, particularly T_{\min} or W_{\min} , as explained above. Examples of this problem include *Sclerotinia sclerotiorum* (Fig. 1C), *Pyrenophora teres*, and *Mycosphaerella pinodes*. Some of the larger RMS values can be explained by the way the studies were conducted. For example, the selection of the wetness durations to be tested (Table 1) was made by the individual researchers. Pathogens with a $W_{\min} \geq 24$ h tended to have larger errors, because the tested wetness durations usually had low temporal resolution (e.g., 24 h or greater). The fit

TABLE 1. Methodology of published studies relating fungal infection to temperature and wetness duration

Pathogen	Host ^b	Reference	Temp. range (°C) ^a		Tested wetness durations (h)	Disease assessment	
			Min.	Max.		Value ^c	Units ^d
<i>Albugo occidentalis</i>	Spinach	81	6	28	0,3,6,12,36,48,60,72,84	0.05	Severity
<i>Alternaria brassicae</i>	Oilseed rape	38	6	25	2,4,6,8,12,24	0.20	Incidence
<i>Alternaria cucumerina</i>	Muskmelon	31	12	30	2,4,8,12,16,24	0.05	Severity
<i>Alternaria mali</i>	Apple	32	4	36	2,4,6,12,18,24,36,48	0.02	Severity
<i>Alternaria porri</i>	Onion	80	5	25	2,4,8,12,16,20,24	2.00	Lesions per plant
<i>Alternaria</i> sp.	Mineola tangelo	18	17	32	4,8,12,24,36	5.00	Lesions per leaf
<i>Ascochyta rabiei</i>	Chickpea	84	5	30	3,6,12,24,48,96	0.25	Severity
<i>Bipolaris oryzae</i>	Rice	59	10	36	10,12,14,...,20,24,28,36	1.00	Lesions/cm ²
<i>Botryosphaeria dothidea</i>	Apple fruit	58	8	28	2,4,8,12,16,20,24,36,48	0.20	Incidence
<i>Botryosphaeria obtusa</i>	Apple fruit	7	8	32	2,4,8,12,16,...,44	Light	Category
<i>Botrytis cinerea</i>	Grape	56	12	30	4,8,12,16,20	0.20	Incidence
<i>Botrytis cinerea</i>	Strawberry flowers	15	5	30	2,4,5,8,10,15,16,20,24	0.2	Incidence
<i>Botrytis cinerea</i>	Grape flowers	56	5	30	1,2,4,6,8,10,12,18,24	0.20	Incidence
<i>Botrytis squamosa</i>	Onion	82	6	28	6,9,12,15,18,21,24,48	0.20	Incidence of sites
<i>Bremia lactucae</i>	Lettuce	67	5	30	2,4,6,12,24	0.10	Severity
<i>Cercospora arachidicola</i>	Peanut	93	18	30	12, 24,48,...,96 ^e	1.00	Lesions per leaf
<i>Cercospora carotae</i>	Carrot	20	16	32	12, 24,48,72,96	0.20	Proportion ^f
<i>Cercosporidium personatum</i>	Peanut	17	13	30	4,8,12,16	ND	Lesion density
<i>Coccomyces hiemalis</i>	<i>Prunus</i> sp.	28	8	28	4–70, undefined	14.0	Rating
<i>Colletotrichum acutatum</i>	Strawberry fruit	92	6	30	0.5,1,2,4,6,8,24,36,48, 51	0.20	Incidence
<i>Colletotrichum orbiculare</i>	Watermelon	53	12	30	2,4,8,12,16,24	0.20	Incidence
<i>Didymella arachidicola</i>	Peanut	79	15	35	24,48,72,..., 184	0.05	Severity
<i>Diplocarpon earlianum</i>	Strawberry	98	10	30	6,12,18,...,36	1.0	Lesions/cm ²
<i>Guignardia bidwellii</i>	Grape	76	10	32	0.5,1,1.5, ..., 48	Light	Category
<i>Gymnosporangium juniperi-virginianae</i>	Apple	4	2	24	Variable	Any	Infection
<i>Leptosphaeria maculans</i>	Oilseed rape	12	8	24	4,8,16,20,24,30,48,72	0.20	Incidence
<i>Melampsora medusae</i>	Poplar	50	8	31	1,2,3,...,24	0.20	Incidence
<i>Monilinia fructicola</i>	<i>Prunus</i> fruit	13	15	30	6,9,12,15,18	0.20	Incidence
<i>Mycosphaerella pinodes</i>	Pea	61	5	25	2,4,6,8,24,48,72	1.00	Severity rating
<i>Phakopsora pachyrhizi</i>	Soybean	48	6	30	1,2,3,..., 12	0.20	Rating
<i>Phytophthora cactorum</i>	Apple fruit	36	6	30	1,2,3,4,5	0.20	Incidence
<i>Phytophthora cactorum</i>	Strawberry fruit	37	5	28	3,6,12,24	0.20	Incidence
<i>Phytophthora infestans</i>	Potato	62	5	30	2,5,8,11,14	1.00	Rating
<i>Plasmopara viticola</i>	Grape	43	5	28	2,6,12,24	0.20	Incidence
<i>Pseudoperonospora cubensis</i>	Cucumber	23	10	30	ND	1.00	Rating
<i>Puccinia arachidis</i>	Peanut	16	5	27	1,2,4,6,12,24	0.20	Rating
<i>Puccinia menthae</i>	Peppermint	27	10	30	6,12,24,36,48	0.05	Severity
<i>Puccinia psidii</i>	Eucalyptus	63	5	25	1,2,4,6,8,16,25	4.2.	Lesions/cm ²
<i>Puccinia recondita</i>	Wheat	85	5	18	1,2,...,12,16,20,24	0.20	Proportion ^f
<i>Puccinia striiformis</i>	Wheat	24	5	25	1,2,4,6,8,16,24	0.20	Incidence
<i>Puccinia striiformis</i>	Wheat	85	4	20	6,10,16,24	0.20	Proportion ^f
<i>Pyrenopeziza brassicae</i>	Oilseed rape	34	10	25	Hourly	0.20	Incidence
<i>Pyrenophora teres</i>	Barley	11	4	18	Variable	ND	Presence or absence
<i>Pyrenophora teres</i>	Barley	72	10	25	1,2,4,6,9,12,24,36,48	0.05	Lesions per spore
<i>Rhynchosporium secalis</i>	Barley	94	7	30	1,2,3, ...,14,19,24,29,34,44	20.0	Rating
<i>Rhynchosporium secalis</i>	Barley	65	10	30	24,48,72,...,216	0.05	Severity
<i>Sclerotinia sclerotiorum</i>	Beans	91	15	30	6,12,18,24,36	0.05	Severity
<i>Septoria glycines</i>	Soybean	70	1	26	ND	0.01	Severity
<i>Venturia inaequalis</i>	Apple	78	7	24	7,9,11,... 29	Light	Category
<i>Venturia pirina</i>	Pear	77	4	26	7,8,9,...17,23,27,28,29	0.2	Incidence
<i>Venturia pirina</i>	Pear	87	5	25	12,24,36,48	0.2	Incidence
<i>Wilsonomyces carpophilus</i>	Almond	71	50.0	Lesions per plant

^a Tested temperature range (°C); Min. = minimum and Max. = maximum.

^b Foliage unless otherwise noted.

^c ND = not defined or not clear from text.

^d Threshold disease intensity value at which observations in the study were classified as infected.

^e Wet periods in 12-h increments per 24 h.

^f Proportion of maximum lesion number.

for *Microcyclus ulei* was very poor ($r = -0.3$, $RMS = 4.5$), but it was unclear whether this was due to experimental or model uncertainty. The fit for *Didymella arachidicola* was also poor ($r = -0.1$, $RMS = 55.5$); however, when the predictions are viewed graphically (Fig. 1D), it becomes apparent that the model follows the trend in the observations well, and that error is due to the

asymptotical uncertainty as T approaches T_{max} . The fit for *A. rabiei* was poor ($r = 0.1$, $RMS = 19.2$), partly because W_{min} occurs near T_{max} rather than T_{opt} , but also because there is a flat response between 5 and 10°C. Poor fits also may be observed because temperature or moisture levels in the experiment were not well controlled.

TABLE 2. Infection model parameters and statistical comparison between model predictions and observations based on published studies relating fungal infection to temperature and wetness duration

Pathogen	Host ^a	Ref. ^b	Ref. T_{min} ^c	T_{min} ^d	T_{max} ^e	T_{opt} ^f	W_{min} ^g	W_{max} ^h	Obs ⁱ	r ^j	RMS ^k	SRMS ^l
<i>Albugo occidentalis</i>	Spinach	81	...	6	28	16	3	12	12	0.87	2.8	0.9
<i>Alternaria brassicae</i>	Oilseed rape	38	6	2.6	35	18	6	22	9	0.96	4.0	0.7
<i>Alternaria cucumerina</i>	Muskmelon	31	...	12	25	19	8	24	6	0.98	1.6	0.2
<i>Alternaria mali</i>	Apple	32	...	1	35	23	5	40	16	0.88	5.2	1.0
<i>Alternaria porri</i>	Onion	80	...	1	35	23	8	24	5	1.00	0.7	0.1
<i>Alternaria</i> sp.	Mineola tangelo	18	...	9.4	35	25	8	16	5	0.90	1.3	0.2
<i>Ascochyta rabiei</i>	Chick pea	84	...	1	35	25	12	48	6	0.10	19.2	1.6
<i>Bipolaris oryzae</i>	Rice	59	25	8	35	27.5	10	24	6	0.78	5.0	0.5
<i>Botryosphaeria dothidea</i>	Apple fruit	58	...	8	35	28	8	19	6	0.95	1.6	0.2
<i>Botryosphaeria obtuse</i>	Apple fruit	7	...	1	35	26	5	40	7	0.97	3.2	0.6
<i>Botrytis cinerea</i>	Grape	56	57	10	35	20	4	10	11	0.94	0.8	0.2
<i>Botrytis cinerea</i>	Strawberry flower	15	...	5	35	25	8	18	7	0.13	5.0	0.6
<i>Botrytis cinerea</i>	Grape flower	56	57	1	34	25	1	12	6	0.99	0.6	0.6
<i>Botrytis squamosa</i>	Onion	82	...	1	28	18	15	24	8	0.50	4.7	0.3
<i>Bremia lactucae</i>	Lettuce	67	...	1	25	15	4	10	6	0.98	0.8	0.2
<i>Cercospora arachidicola</i>	Peanut	93	6	13.3	35	24	24	48	5	0.72	8.9	0.4
<i>Cercospora carotae</i>	Carrot	20	...	11	32	24	28	96	5	0.98	16.5	0.6
<i>Cercosporidium personatum</i>	Peanut	17	6	8	35	20	16	33	6	0.33	6.0	0.4
<i>Coccomyces hiemalis</i>	Prunus sp.	28	29	4	30	18	5	30	11	0.96	7.8	1.6
<i>Colletotrichum acutatum</i>	Strawberry fruit	92	...	7	35	27.5	6	36	6	0.93	4.4	0.7
<i>Colletotrichum orbiculare</i>	Watermelon	53	...	7	30	24	2	16	7	0.69	5.6	2.8
<i>Didymella arachidicola</i>	Peanut	79	6	13.3	35	18.5	24	210	5	-0.10	55.5	2.3
<i>Diplocarpon earlianum</i>	Strawberry	98	51	2.9	35	22.5	12	18	5	0.53	3.2	0.3
<i>Guignardia bidwellii</i>	Grape	76	...	7	35	27	6	24	10	0.74	5.1	0.9
<i>Gymnosporangium juniperi-virginianae</i>	Apple	4	...	1	35	21	2	24	12	0.99	1.1	0.5
<i>Leptosphaeria maculans</i>	Oilseed rape	12	6	2.6	35	18.5	7	18	5	0.81	4.8	0.7
<i>Melampsora medusae</i>	Poplar	50	...	12	28	20.5	5	12	10	0.96	0.7	0.1
<i>Monilinia fructicola</i>	Prunus fruit	13	44	10	35	20	10	16	5	0.96	0.9	0.1
<i>Mycosphaerella pinodes</i>	Pea	61	6	1.4	35	20	6	72	6	1.00	21.9	3.7
<i>Phakopsora pachyrhizi</i>	Soybean	48	6	10	28	23	8	12	6	0.86	1.3	0.2
<i>Phytophthora cactorum</i>	Apple fruit	36	...	1	35	25	2	5	6	0.97	0.4	0.2
<i>Phytophthora cactorum</i>	Strawberry fruit	37	...	6	35	20.5	1	3	8	0.85	0.6	0.6
<i>Phytophthora infestans</i>	Potato	62	19	1	28	15	6	12	6	0.53	3.2	0.5
<i>Plasmopara viticola</i>	Grape	43	...	1	30	20	2	14	6	0.99	0.6	0.3
<i>Pseudoperonospora cubensis</i>	Cucumber	23	1	1	28	20	2	12	6	0.98	0.7	0.4
<i>Puccinia arachidis</i>	Peanut	16	6	5	35	25	5	25	5	0.82	5.2	1.0
<i>Puccinia menthae</i>	Peppermint	27	...	5	35	15	6	12	5	0.87	1.6	0.3
<i>Puccinia psidii</i>	Eucalyptus	63	52	1	30	21.5	6	24	5	0.98	3.9	0.6
<i>Puccinia recondita</i>	Wheat	85	6	2.6	30	25	5	16	6	0.61	5.4	1.1
<i>Puccinia striiformis</i>	Wheat	24	6	2.6	18	8.5	5	8	6	0.99	0.2	0.0
<i>Pyrenopeziza brassicae</i>	Oilseed rape	34	6	2.6	24	16	6	24	7	0.90	3.6	0.6
<i>Pyrenophora teres</i>	Barley	11	6	2.6	35	23	3	6	4	0.95	0.4	0.1
<i>Pyrenophora teres</i>	Barley	72	6	2.6	35	18	5	48	4	1.00	11.9	2.4
<i>Rhynchosporium secalis</i>	Barley	94	6	2.6	30	22.5	12	48	4	0.98	4.2	0.4
<i>Rhynchosporium secalis</i>	Barley	65	6	2.6	30	22.5	6	19	6	0.94	2.3	0.4
<i>Sclerotinia sclerotiorum</i>	Bean	91	...	1	30	25	48	144	5	0.88	24.0	0.5
<i>Septoria glycines</i>	Soybean	70	6	10	35	25	6	18	4	0.83	4.2	0.7
<i>Venturia inaequalis</i>	Apple	78	...	1	35	20	6	40.5	26	0.65	2.7	0.5
<i>Venturia pirina</i>	Pear	77	...	1	35	22	10	25	7	0.98	1.3	0.1
<i>Venturia pirina</i>	Pear	87	...	1	35	20	10	30	7	0.99	1.5	0.1
<i>Wilsonomyces carpophilus</i>	Almond	71	...	5	35	25	12	48	9	0.92	6.6	0.6

^a Foliage unless otherwise noted.

^b Reference to temperature-wetness combination study.

^c Reference for estimation of T_{min} from crop development.

^d T_{min} = minimum temperature for infection (°C).

^e T_{max} = maximum temperature for infection (°C).

^f T_{opt} = optimum temperature for infection (°C).

^g W_{min} = minimum value of the wetness duration requirement for infection (h).

^h W_{max} = optimum value of the wetness duration requirement (h).

ⁱ Number of temperature/wetness combinations included as observations.

^j r = Pearson's correlation coefficient (54).

^k RMS = root mean square error (75).

^l SRMS = standardized root mean square/ W_{min} .

Pathogens varied in their ability to tolerate interruptions of wetness (Table 3). Some pathogens were highly sensitive; for example, even a 1- to 2-h interruption dramatically reduced the level of infection for many *Puccinia* spp. For most *Puccinia* spp., the timing of the interruption was critical. For *Puccinia recondita* and *P. striiformis*, an interruption to wetness during the first 2 to 4 h of wetness greatly reduced the level of disease from ≈ 70 to $\approx 10\%$; however, interruptions after 4 h had a much smaller influence (85). Other plant pathogens, such as *Alternaria linicola* (88), also are sensitive to the time of the wetness interruption. Most plant pathogens were sensitive to wetness interruptions between 4 and 20 h, whereas a few, such as *Cercospora carotae* and *V. inaequalis*, were insensitive to wetness interruptions of <24 h.

DISCUSSION

In this study, we adapted a temperature response function to create a generic infection model and validated it with a large experimental data set. The generic model has several advantages compared with other approaches published in the literature. The main advantage is that the generic model can be used with subjective estimates of cardinal temperatures (T_{\min} , T_{opt} , and T_{\max}) and wetness requirements for infection (W_{\min} and W_{\max}). A limitation with many of the published models is that they require extensive amounts of biological data for parameter estimation. Duthie (26) estimated that 20 to 30 combinations of wetness duration and temperature are likely to be needed for parameter estimation in his infection response model. From the literature search made as part of this study, we estimate that there are fewer than 100 pathogens with a published infection response using controlled temperature and wetness combinations. This compares with the remaining thousands of economically important plant pathogens,

whose parameters must be estimated from other studies or field observations.

The temperature parameters are likely to be easier to estimate than the moisture requirement. Cardinal temperatures commonly are described in the literature. For example, Togashi (83) de-

TABLE 3. Classification of fungal foliar pathogens based on their ability to withstand interruptions to wetting during infection based on published studies relating infection to temperature and wetness duration

Sensitivity to dry interruption	D_{50}^a	Species	Reference		
Sensitive	1–2 h	<i>Puccinia recondita</i>	85		
		<i>Puccinia striiformis</i>	85		
		<i>Pyrenophora tritici-repentis</i>	66		
		<i>Alternaria brassicae</i>	55		
		<i>Alternaria linicola</i>	88		
		<i>Alternaria porri</i>	80		
Moderate	4–20 h	<i>Ascochyta rabiei</i>	84		
		<i>Bipolaris oryzae</i>	59		
		<i>Botryosphaeria obtusa</i>	8		
		<i>Botrytis squamosa</i>	3		
		<i>Cercospora kikuchii</i>	68		
		<i>Coccomyces hiemalis</i>	28		
		<i>Stagonospora nodorum</i>	41		
		<i>Uromyces phaseoli</i>	9		
		<i>Venturia pirina</i>	86		
		Insensitive	≥ 24 h	<i>Cercospora carotae</i>	21
				<i>Mycosphaerella graminicola</i>	73
				<i>Stemphylium botryosum</i>	9
<i>Venturia inaequalis</i>	10,86				

^a D_{50} is defined as the duration of a dry period at relative humidities of $<95\%$ that will result in a 50% reduction in infection compared with a continuous wetness period.

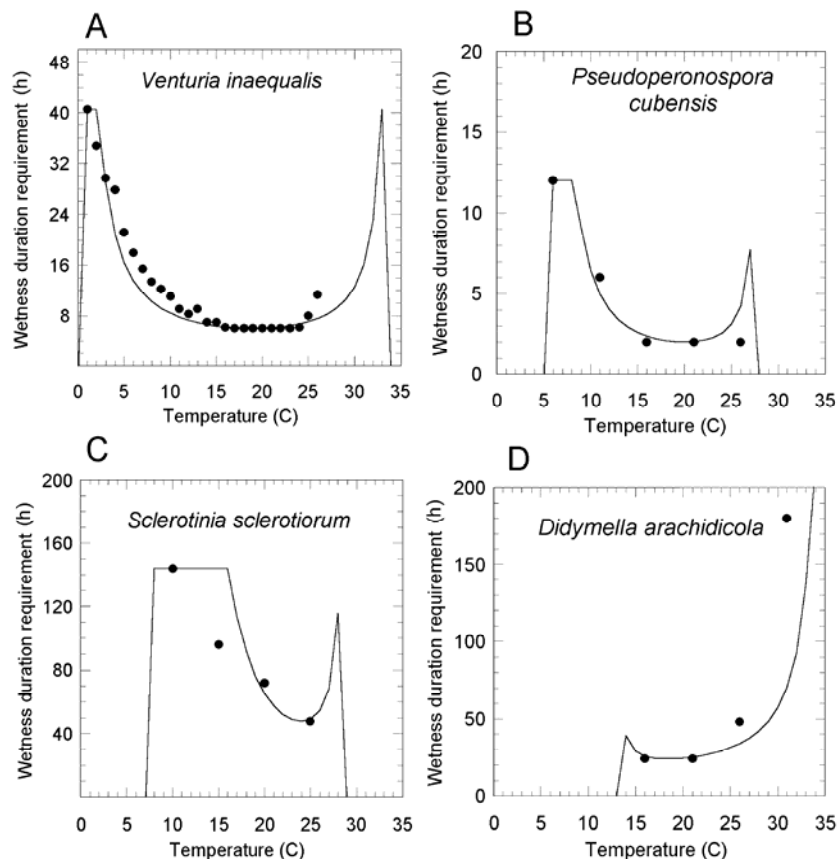


Fig. 1. Examples of goodness of fit of model predictions (solid lines) of wetness requirements at different temperatures compared with experimental observations (solid circles) for A, *Venturia inaequalis* (78), B, *Pseudoperonospora cubensis* (23), C, *Sclerotinia sclerotiorum* (91), and D, *Didymella arachidicola* (79). In each case, model predictions were produced with equations 1 and 2 (described in text) and the parameters in Table 2.

scribes cardinal temperatures for plant pathogens compiled from approximately 1,500 studies. The cardinal temperatures and wetness requirements are often described in crop compendia, such as those produced by the Commonwealth Agricultural Bureau or by The American Phytopathological Society. Although minimum wetness requirements are often stated in data sheets in these compendia, comparisons between related species have been hampered by the lack of a standard definition. We believe this study is one of the first to propose a standard definition of W_{\min} and compare its value for a wide cross-section of pathogens. It is likely that, in some studies, the wetness requirement was not determined at T_{opt} , and this could have led to an underestimation in W_{\min} . Likewise, there are instances where W_{\max} was not determined at T_{\min} or T_{\max} , leading to a similar underestimation. Although data to estimate W_{\max} is less commonly available, it can be estimated from W_{\min} using the regression equation developed in this study. There is a considerable amount of information in the literature regarding the humidity requirements for germination of fungal spores (96), information that may be used to estimate W_{\min} or D_{50} . Where there are little or no epidemiological data on a particular pathogen, such as is the case with many exotic pathogens, the parameters may be estimated first by comparison with related organisms.

A second advantage is that all five parameters in our infection model have a clear biological meaning. In contrast, many of the other infection models published in the literature, such as those based upon higher-order polynomials, are difficult to interpret in biological terms (95) or poorly describe biological processes (26). Other models, such as those proposed by Duthie (26), use complex parameters that describe the shape, scale, and symmetry of the response. These parameters usually are statistically derived and depend upon conditions unique to each experiment; therefore, they may not be directly compared from one study to another. As yet, a comparative fit of the Duthie model to multiple foliar parasites has not yet been published (26). Another practical advantage of our generic model in the present study is that the temperature response approach is widely recognized and used in many fields, including crop physiology and agricultural meteorology (90,95,97). Importantly, this may allow an infection model to be incorporated quickly into a crop model by using an existing temperature response equation.

Because the model we propose is simple and generic, it is important to point out some limitations and uncertainties associated with its use. The model uses a simple temperature-wetness response for each pathogen and ignores the interaction between temperature and wetness which has been demonstrated in many studies (13,15,18,35,43,53). Although the interaction term is ignored, our results show that the model was able to accurately predict the temperature-wetness response curve for the disease intensity threshold selected in the study. Most likely, the uncertainties associated with the interaction term are likely to be smaller than those associated with estimating the appropriate disease intensity threshold and other model parameters, including the temperature and wetness parameters. The interaction term is likely to be more important when deriving a response surface; however, for practical forecasting needs, a simple threshold is likely to be more useful and widely used. Another consideration is that the critical disease intensity threshold is proposed mainly as a tool to enable a comparison of $W_{(T)}$ for different pathogens. This study makes no attempt to define a relationship between the disease intensity threshold and crop damage.

This study is one of the first to demonstrate that a single temperature-driven equation can simulate the infection response for a range of plant pathogens. Model validation was not made with independent data and the validation was less rigorous than one in which the values of the input parameters are determined in separate experiments. However, it is worth noting that almost all the published models have not been validated with independent data, because most models represent statistical fits of disease intensity

observations for different temperature and wetness combinations. The parameters in these models do not have biological meaning; therefore, the creation of a generic model that can be validated with multiple pathogens has not been practical. We have shown that a generic model can estimate infection when the values of the parameters have been determined under controlled conditions. What is required is an independent validation of models using observations for multiple pathogens but varying the quality and quantity of data for parameter fitting. In such a test, we would compare the performance of the models using observations obtained from controlled infection studies (as collected in this study) with predictions made using parameter estimates obtained from field observations, closely related organisms, and other types of controlled experiments, such as germination or culture growth studies. Such a validation would determine the uncertainties associated with each input data type and the confidence with which pathogens can be modeled when data is scarce.

The value of the key parameter W_{\min} is dependent upon both the pathogen and the host. There was a high degree of similarity in W_{\min} for species of *Cercospora*, *Alternaria*, and *Puccinia* but less so for *Mycosphaerella*, *Septoria*, and *Colletotrichum* spp. Some of the differences are likely to be due to differences in experimental techniques. Although every effort was made to standardize the comparison of the published studies, different disease assessment techniques, in particular, made this difficult. Inoculum concentration may also be important, but most studies used relatively high inoculum concentrations that are likely to be nonlimiting. For example, disease severity following inoculation with *Phytophthora infestans* was relatively similar when potato leaves were inoculated with 135 to 1,200 sporangia/cm² (62). Other differences may be due to method of host penetration, whether direct through the cuticle or indirect via stomata. Host differences include cuticle thickness, tissue type, and whether fruit or leaves differ in host defense mechanisms.

Pathogens differed in their sensitivity to wetness interruption. Without data on the critical dry-period interruption value, it is difficult to use an infection model with hourly weather data. Environmental conditions, especially humidity during the wetness interruption, exert an influence on severity of infection. Dry interruptions at 50% relative humidity had a more pronounced effect than those at 75% to *Mycosphaerella graminicola* infection (72). Some plant pathogens require only high humidity and not wetness. Infection by *Cercospora carotae* and *C. kikuchii* occurs when relative humidity stays above 88% (22,69). Other studies of germination and germ tube elongation support the importance of high humidity during dry periods (40). Germ tube elongation of *C. arachidicola* on leaf surfaces continues at 94 to 98% relative humidity, is reduced at 53 to 85%, and is minimal at 30 to 40% (2). Interruptions of 10 h at 85% relative humidity with *Cercosporidium personatum* (17) increases disease severity. Infection by *M. fijiensis* (40) continues at high humidities even with no initial wetness. In contrast, other studies showed that the humidity during the dry period had little effect on *V. inaequalis* (10) or on *Botryosphaeria obtusa* (8). Caution is needed in interpreting these laboratory experiments, because the desiccation of spores is determined by evaporation rate rather than by humidity alone. There is a need for a study of interruption to wetness using a broad range of pathogens under controlled conditions where light intensity, wind speed, humidity, and temperature can be varied.

The infection model described is being used by scientists in the United States Department of Agriculture's Animal Plant Health Inspection Service for several applications (R. D. Magarey, unpublished data). Because the model is generic in nature, it can be adapted to exotic pathogens for which a minimum of biological data is available. For import commodity risk assessments, the model can be used to predict the potential risk of establishment of fungal pathogens using national climatological data sets. The

model also can be used to better target the survey, detection, and eradication of exotic pests based upon forecast or observed weather data (46,64). Eventually, the infection model could be included in a more complex model that describes other functions such as incubation, sporulation, and dispersal (89). It should be emphasized that the parameter values defined for each species in this study only represent the infection response observed in the particular study. Studies of the same pathogen were not combined, nor was there an attempt to define the infection parameters by reviewing other literature sources. To do so, it may be important to consider ecotypes that may have different values of infection parameters.

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