A Simulation Model to Describe Epidemics of Rust of Phaseolus Beans I. Development of the Model and Sensitivity Analysis

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ABSTRACT

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A simulation model was developed for bean rust epidemics based on infections that occurred in daily cohorts of leaf growth. The most sensitive function in the model was the daily correction of the basic infection rate for the amount of healthy host tissue on each cohort that was available for infection. Components of the basic infection equation, i.e., maximum basic infection rate (R_{max}) and parameters for environmental favorability (F) and the rate of the decreased susceptibility of leaves with age (A), also were sensitive factors. A greater pustular area on day 70 occurred when the rate of host growth was slower than standard, and a lower pustular area occurred when the rate of host growth was faster than standard. The maximum leaf area per plant was a relatively insensitive parameter. The total pustular area (y_{70}) was greatest when an epidemic was initiated at the highest level ($f_0 = 0.01$) of initial infection, on the earliest day of infection [$i(f_0) = 2$], and at maximum environmental favorability ($F = 1.0$). With epidemic rates (k_G) calculated with the Gompertz transformation, the fastest rates also occurred at $f_0 = 0.01$, $i(f_0) = 2$, and $F = 1.0$. In contrast, with epidemic rates (r) calculated with the logistic transformation, the fastest rates occurred with epidemics begun at the lowest level ($f_0 = 0.00001$) of initial infection. The logistic transformation of the low levels of y_{50-70} caused inflated values of r. The model was verified by simulating an epidemic using temperatures and hours of leaf wetness that were recorded during an actual season. The curve for the increase in pustular area simulated by the model was representative of the progress of rust observed in the natural epidemic.

Rust (*Uromyces appendiculatus* (Pers.) Unger) is an important yield-reducing disease of beans (*Phaseolus vulgaris* L.) worldwide (14). Bean yields have been reduced by more than 50% during rust epidemics in certain years (14). The bean rust fungus has many physiologic races, and several races usually are found in collections of urediniospores from the same field (20). Although germ plasm of *P. vulgaris* varies greatly in reaction to rust pathotypes, very few accessions of bean are resistant to all pathotypes that occur in a given geographic area (20). Consequently, control practices in addition to the use of resistant cultivars are widely used. Historically, growers prefer to manage the disease on familiar, long-used cultivars rather than to switch to another cultivar with some resistance to rust but with unknown agronomic requirements and with possible reduced preference by consumers.

Various aspects of the epidemiology of bean rust have been studied (2,14–17), and much of this information can be used to develop a comprehensive model of the pathosystem. To examine the many intricacies of the intensification of rust on beans, a model to simulate the progress of the disease is needed. The development of an epidemic simulator for bean rust could lead to applications with numerous practical and experimental objectives: i) prediction of the hazard of bean rust for various geographic sites and crop seasons; ii) forecasting the progress and final intensity of bean rust; iii) evaluation of strategies for control of the disease; iv) aid in interpretation of the progress and intensity of rust during various epochs of weather and applications of control measures; v) aid in interpretation of differences in resistance among cultivars or in aggressiveness among races of the pathogen; vi) estimation of the time course and leaf area involved in latent infections; vii) determination of the effect of the "virtual" (4) latent infections on infection efficiency of later arriving urediniospores; viii) determination of the effect of the virtual latent infections on the net photosynthetic rate of parasitized tissue; and ix) determination of the relationship of crop yield to duration of healthy leaf area and to light absorption (23). In this paper, we describe the development of a simulation model for epidemics of bean rust (termed FERRUGEM, which is Portuguese for rust) and the sensitivity analysis of its components.

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MATERIALS AND METHODS

Host growth and defoliation. In each of three seasons, 15 to 20 plants each of bean cvs. Rosinha and Carioca were tagged (15). The day of appearance, periodic estimates of area, and day of defoliation were recorded for each leaf. The plotted curves for the increase in total leaf area over time were sigmoidal for the six crops (2 cultivars × 3 seasons); however, the maximum estimated leaf areas were vastly different among crops. The average leaf area per plant for Rosinha and Carioca during average, wet, and dry seasons were 2,500, 3,350, and 1,650 cm^2 and 1,950, 2,650, and 1,500 cm^2, respectively. A modified Weibull function (10) was fitted to the data ($R^2 = 0.99$), and this equation was used to generate the leaf area in the simulation model. The leaf area on

the day of plant emergence (L_1) averaged 3 cm^2 per plant. In the model, leaf area was calculated on subsequent days as:

$$L_i = L_{max}\{1 - \exp[-0.0232i)^{4.27}]\} + L_1$$

where i was the time in days, L_{max} was the maximum accumulated leaf area for the season, 0.0232 was the rate of foliation, and 4.27 was the Weibull shape parameter. The daily increment of new leaf area was calculated simply as $dL_i = L_i - L_{i-1}$. This characterization of host growth as daily cohorts (dL_i) based on leaf age allowed the use of the daily cohort model (6) as the structure of the simulator for bean rust epidemics. The parameters and variables used in the model are defined in Table 1.

The curve for defoliation that followed the natural senescence of leaves was sigmoidal over time. This response, beginning 20 days after plant emergence, also was fit well (R^2 = 0.99) by the Weibull function as $D_i = L_{max}(1 - \exp\{-[0.018(i - 20)]^{4.3}\})$, where

TABLE 1. Parameters and state and rate variables used in the model for simulation of rust epidemics on Phaseolus bean

Term	Description
Parameters	
L_{max}	Maximum accumulated leaf area (cm^2 per plant)
p_1	Minimum length of latent period (days)
$p_1 \cdot v \ldots p_n \cdot v$	Variable latency; where v = proportion (dimensionless) of new infections that occurred on day $i - p_n$
R_{max}	Maximum basic infection rate (dimensionless)
f_0	Proportion (dimensionless) of leaf area with initial infection
$i(f_0)$	Day of initial infection
F_i	Proportional (dimensionless) favorability of environment for infection ($0 < F < 1$) on day i
$T_{min(i)}$	Minimum temperature (degrees Celsius) on day i
$T_{mean(i)}$	Mean temperature (degrees Celsius) on day i
W_i	Duration (hours) of leaf wetness on day i
A_j	Effect (dimensionless) of age on susceptibility of cohort j to infection ($0 < A < 1$)
a	Scaling parameter (dimensionless) in the negative-power equation for the basic infection rate versus proportion of infected area
b	Exponent (dimensionless) in the negative-power equation for the basic infection rate versus proportion of infected area
k_L	Rate parameter (dimensionless) in the Weibull function for foliation
c_L	Shape parameter (dimensionless) in the Weibull function for foliation
k_D	Rate parameter (dimensionless) in the Weibull function for defoliation
c_D	Shape parameter (dimensionless) in the Weibull function for defoliation
B	Scaling parameter (dimensionless) of the logistic function; $B = (1 - y_0)/y_0$
State variables	
i	(days)
j	Age of cohort (days)
dL_{ij}	Leaf area (cm^2 per plant) newly appeared on day i; i.e., the leaf cohort
L_i	Leaf area (cm^2 per plant) summed over days 1 to i; ΣdL_{ij}
dD_i	Leaf area (cm^2 per plant) defoliated on day i
D_i	Defoliated leaf area (cm^2 per plant) summed over days 1 to I; ΣdD_i
dI_{ij}	Infected leaf area(cm^2 per cohort) that occurred on day i in cohort j
I_{ij}	Infected leaf area (cm^2 per cohort) summed for days 1 to i for cohort j; ΣdI_{ij}
N_i	Infected leaf area (cm^2 per plant) summed for days 1 to i; ΣI_{ij}
Y_i	Total area (cm^2 per plant) of pustules on day i
y_i	Proportion (dimensionless) of total leaf area with pustules ($0 < y < 1$) on day i
Rate variables	
R_{ij}	Basic infection rate (dimensionless) on day i for cohort j
r	Epidemic rate (logits per day)
k_G	Epidemic rate (gompits per day)

D_i was the accumulating leaf area that has been defoliated, L_{max} was as above, 0.018 was the defoliating rate, and 4.3 was the value of the shape parameter for the function. The daily increment of leaf removal (dD_i) by defoliation was calculated as $dD_i = D_i - D_{i-1}$. A special routine was written to remove the cohorts sequentially in the order of their appearance. The acceleration of leaf fall associated with disease was not included in the model because of the incomplete quantification of this response.

Infection and pustular area. The maximum rate of infection on a given proportion of healthy tissue was assumed to occur only at optimum environmental favorability and maximum host susceptibility. A function for the daily environmental favorability ($0.0 < F_i < 1.0$) was derived from the fit of a beta-Richards equation to the response surface of the length of leaf wetness periods and temperature on disease efficiency as described earlier (2,14) (Fig. 1). This relationship was estimated as

$$F_i = ([0.0058(T_{min(i)} - 8)^{1.481}]\,[(29 - T_{min(i)})^{2.93}]\,\{[1 - \exp(-0.02095W_i)]^{5.509}\})\,0.004423$$

R^2 = 0.995, where $T_{min(i)}$ was the daily minimum temperature (degrees Celsius) and W_i was daily hours of leaf wetness. With this function, the maximum favorability occurred at 15°C and 24 h of leaf wetness, and no infection occurred when the minimum temperature was <8°C or >29°C. Disease efficiency was defined as the ratio of the number of pustules that developed one latent period after inoculation at given environmental conditions to the number of pustules that developed after inoculation at the optimum conditions.

Bean leaf tissue decreases in susceptibility with age at a rate of about 2.5% per day after leaf emergence (16). A function for this effect of leaf age on susceptibility ($0.0 < A_j < 1.0$) was derived as $A_j = (1 - 0.025_{j-1})$; where j was age (in days) of a cohort on day i.

The basic infection rate equation of van der Plank (22) [$dy_t/dt = Ry_{t-p}\,(1 - y_t)$] for the proportion of infected host tissue, as modified for host area (6), was used as the driving equation for the rust model. The basic infection rate, R, was a very sensitive parameter (22) and warranted special consideration. A negative power function was used to characterize the suspected relationship (6) of the decreasing basic infection rate with the decreasing proportion of healthy tissue of the cohort. For a given cohort on a

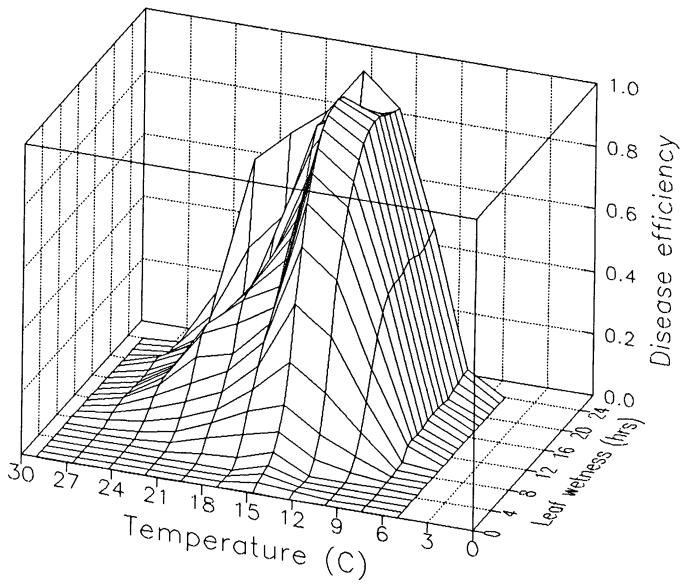

Fig. 1. Response surface for disease efficiency of *Uromyces appendiculatus* on *Phaseolus vulgaris* based on the favorability (F) of daily minimum temperature (T_{min}) and hours of leaf wetness (W). The surface was described as $F = ([0.0058(T_{min} - 8)^{1.481}][(29 - T_{min})^{2.93}]\{[1 - \exp(-0.2095W)]^{5.509}\})$ 0.004423, $R^2 = 0.995$.

given day, R_{max} was multiplied by both the F_i and A_j values for that day to obtain the corrected basic infection rate for the cohort. The equation for this corrected rate was $R_{ij} = 0.05\ R_{max}\ F_i\ A_j(I_{ij}/dL_{ij})^{-0.5}$, where R_{ij} was the unique infection rate on day i for cohort of age j; R_{max}, F_i, and A_j were as described above; I_{ij} was the area of the cohort that was already infected; and dL_{ij} was the total area of the cohort.

Initial estimates of R_{max} for the rust-susceptible cultivar Rosinha were obtained from the rates of progress during the first few cycles of disease in natural epidemics. These estimates were made by solving $R = r\exp(pr)$ (22), where r was the average logistic rate during the early part of the epidemic and p was the length of the latent period. Estimates of R_{max} also were obtained by comparing the rate of progress of bean rust to the progress of other pathosystems in which R_{max} was estimated from the initial waves of disease (7). These estimates of R_{max} were in the range of 6 to 8, and a final value of $R_{max} = 12$ was determined in calibration of the model.

The infection rate, R_{ij}, was inserted into the basic infection rate equation for leaf area to determine the area of new infections for each day. The infection equation was $dI_{ij} = R_{ij}\ Y_i(dL_{ij}/L_i)(1 - I_{ij}/dL_{ij})$, where dI_{ij} was the area of new infections on cohorts of age j on day i, Y_i was the total pustular area of a plant on day i that provided inoculum to be partitioned to the cohort depending on the proportional size of the cohort determined as (dL_{ij}/L_i), and $(1 - I_{ij}/dL_{ij})$ was the correction factor for tissue already infected on the cohort. Thus, to obtain simulated curves of the increase in area of pustules that mimicked actual curves, a correction factor for a state variable (amount of healthy area remaining on a cohort) and a correction for the rate variable (R_{ij}) were needed. The pustular area on which inoculum was no longer being produced was not removed from Y_{ij} because the effects of such removal were inconsequential in a sensitivity analysis of the model. The simulations typically were run for 70 days or less, so the variables R_{ij} and I_{ij} each were handled in a 70 × 70 matrix of dimensioned memory.

Variable latency. The length of time from host penetration to the first sporulating pustule is affected largely by temperature (17). In this model, we used the U-shaped function for the effect of temperature versus time for the latent period of bean rust (2, 17). The function was rescaled to obtain the relative rate of latent period duration with a maximum rate of 1 at the optimum temperature (Fig. 2). This latter curve was described by a polynomial equation:

$$1/p(T) = -0.19966 + 0.04888T - 0.00392T^2 + 0.00019T^3 - 0.0000035T^4$$

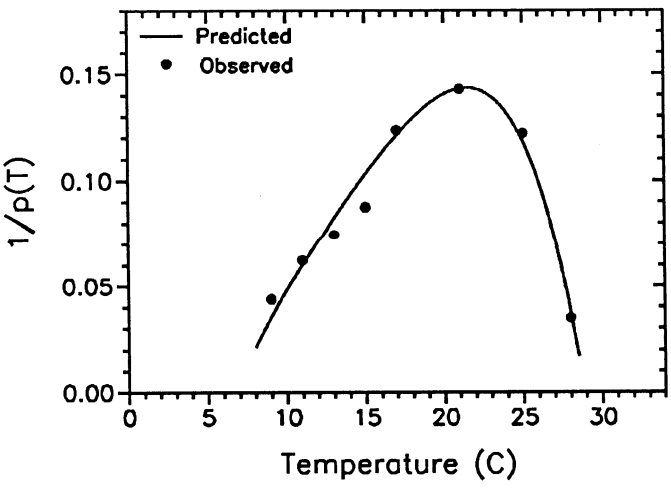

Fig. 2. The relative rate $[1/p(T)]$ of latent period duration versus temperature for *Uromyces appendiculatus* on *Phaseolus vulgaris* estimated as $[1/p(T)] = -0.19966 + 0.04888T - 0.00392T^2 + 0.00019T^3 - 0.0000035T^4$, $R^2 = 0.993$; where T = daily mean temperature (degrees Celsius).

$R^2 = 0.993$, where $1/p(T)$ was the daily rate of latent period duration, and T was the daily mean temperature (degrees Celsius) in the range of $7 < T < 30$. The shortest latent period occurred at 21°C. In the simulations, the daily latent-period rates were summed over time for each day until a value of 1.0 was attained to determine the day of appearance of new infections (8). A 4-day period of variable latency was incorporated into the model for the differential appearance of pustules after the initial latency as 10% on day p_1, and 50%, 30%, and 10% on the following three days (p_2, p_3, and p_4).

Initiation of the simulated epidemic. To initiate an epidemic, the first infections were distributed among the present cohorts based on the size of the individual cohorts as a proportion of the total leaf area on the day the epidemic began. The environmental favorability for infection (F) was either set as a parameter or daily weather variables ($T_{min(i)}$ and W_i) were used. An integration interval of 1 day was used in the simulations.

Model output, verification, and calibration. The variables monitored in model output were: daily and accumulated growing and defoliating host leaf areas, daily and accumulated infected and pustular areas, the proportion of area with pustules, the proportion of pustular area transformed with logits (22) or gompits (5), and the area under the disease progress curve (AUDPC). For verification of the model, the values and trends of the variables monitored in the model output were compared to the values and trends of an epidemic of rust monitored on the cultivar Rosinha during July 1990. For calibration, adjustments were made only to the parameters of R_{max} and day $[i(f_0)]$ and level (f_0) of initial infection.

Sensitivity analysis. Three approaches were used to conduct the sensitivity analysis after calibration of the model. First, with the parameters of the model set at typical values, each of the parameters were varied individually ±10 and ±20% while the rest of the parameters were kept constant, as in a similar procedure by Bourgeois (9) for a simulator of peanut leafspot epidemics. Second, four alternative functions were evaluated that describe the dependence of the basic infection rate on the proportion of infected area in each cohort (I_{ij}/dL_{ij}). The four alternative functions were: i) the negative-power equation described above; ii) the nearly proportional decrease in basic infection rate (R_{ij}) with the decrease in proportion of healthy area (Fig. 5.3 in [22]), modeled as $R_{ij} = F_i\ A_j\{r + [(R_{max} - r)(1 - I_{ij}/dL_{ij})]\}$, where $r = 0.3984$; iii) a

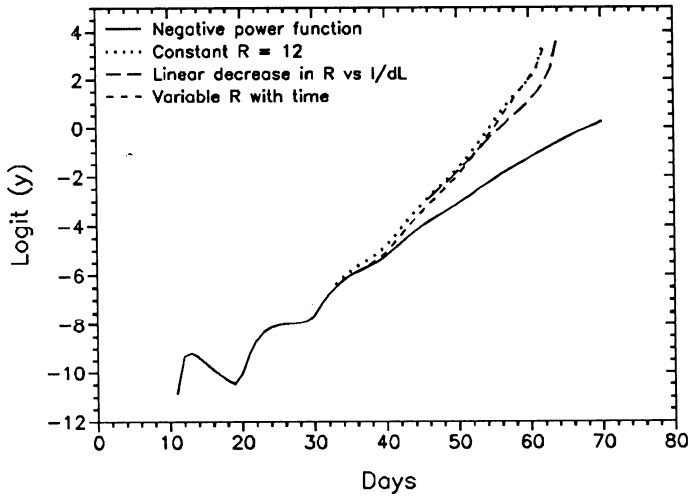

Fig. 3. Comparison of four functions to describe the relationship of the basic infection rate (R) of *Uromyces appendiculatus* on the proportion of *Phaseolus vulgaris* tissue already infected (I_{ij}/dL_{ij}) expressed as the simulated increase in pustular area. The functions were: i) a negative-power function (the base equation used in the model); ii) a constant basic infection rate (21,24); iii) a linear decrease in the basic infection rate (Fig. 5.3 in [22]); and iv) a variable basic infection rate with time (11).

variable basic infection rate as a function of time (with an assumed constant epidemic rate [r]) (11), modeled as $R_{ij} = F_i A_j r/\exp(-pr) f_{(j)}$, where $r = 0.3984$, $f_{(j)} = [1 + B\exp(-r_j)]/[\exp(-pr) + B\exp(r_j)]$, and $B = (1 - f_0)/f_0$; and iv) a constant basic infection rate, $R_{ij} = R_{max}$ (21,24). With simulations using the four functions, the following parameters were set: $R_{max} = 12.0$, $F = 0.8$, $p = 8$, $f_0 = 0.0007$, and $i(f_0) = 4$. Third, the interactions among the level of initial infected area (f_0), the day of initial infection [$i(f_0)$], and the environmental favorability (F) were examined. The ranges of values used were $0.00001 < f_0 < 0.01$, $2 < i(f_0) < 40$, and $0.0 < F < 1.0$. For this latter analysis, two parameters at a time were varied over the possible ranges of values, and the resulting responses of the model were expressed as three-dimensional surfaces.

The sensitivity of the model was evaluated by changes in the: i) final proportion of pustular area (usually y_{70}); ii) logistic (or Gompertz) rate of increase in pustular area from days 50 to 70; and iii) AUDPC. These parameters are usually considered important indicators of the status of the epidemic (12,22). We observed that the percentage of changes in the AUDPC values was highly correlated ($r = 0.999$) with the percentage of changes in y_{70}. Also, for 36 of 48 simulations, the epidemic rate (r_{50-70}) varied by <8%. Consequently, only the changes in final proportion of pustular area from the sensitivity analysis of individual parameters are presented.

RESULTS

Sensitivity analysis. The functions to describe the influence of the decreasing proportion of healthy tissue on the basic infection rate were the most sensitive terms in the simulation model for bean rust. The proposed relationship by van der Plank (Fig. 5.3 in [22]), the variable basic infection rate with time (11), and the constant basic infection rate (21,24) caused more than a 340% increase in y_{55} over that obtained with the negative-power equation ($y_{55} = 0.436$, 0.673, 0.555, and 0.108, respectively) (Fig. 3). The first three functions also led to an unrealistic upturn in the progress curve at logit values greater than 0.0 ($y = 0.5$). The parameters and variables that pertained to the basic infection equation were sensitive terms (Table 2). The ±20% change in the a and b parameters of the negative-power function for adjustments to the basic infection rate affected y_{70} from 16 to 28%. A ±20% change in the two modifying variables (F and rate of A) for the basic infection rate affected y_{70} by about 30% for F and by about 9% for rate of A.

The sensitivity of the parameters for foliation and defoliation varied considerably (Table 2). Parameters with relative insensitivity were the maximum leaf area, L_{max}, and the shape of the Weibull curve for foliation, c_L. A ±20% change in either of these two parameters caused less than an 5.2% change in y_{70}. A slow rate of foliation (–20% of k_L) led to 14.3% more pustular area, whereas a fast rate of foliation (+20% of k_L) led to 18.6% less pustular area by day 70. The response to changes in the parameters of the defoliation function were minimal, as the greatest increase in defoliation occurred rather late in the simulated epidemics.

The sensitivity of the three two-way interactions of initial level of infected area, day of initial infection, and environmental favorability were examined. The highest levels of final pustular area, y_{70}, occurred at the highest level of initial infection ($f_0 = 0.01$), the earliest day of infection [$i(f_0) = 2$], and maximum environmental favorability ($F = 1.0$). The three response surfaces (Fig. 4A–C) were fitted (1) to an interactive equation as $y_{70} = A + BX + CY + DXY + EX^2 + FY^2 + GX^2Y + HXY^2$, where A–H were the eight regression coefficients, and X and Y were the two interacting parameters. The regression coefficients, A–H, were, respectively, for i) environmental favorability versus log (initial infected area): 0.0587, 1.7928, 0.0819, 0.4695, 0.2918, 0.0154, –0.0271, and 0.0125, $R^2 = 0.99$; ii) day of initial infection versus log (initial infected area): 2.1935, –0.0405, 0.6715, –0.0078, 2.297×10^{-5}, 0.054, -3.624×10^{-5}, and -4.1×10^{-4}, $R^2 = 0.993$; and iii) environmental favorability versus day of initial infection: –0.0425, 0.3975, 0.0035, –0.0211, 0.5671, -6.833×10^{-5}, –0.016, and 3.516×10^{-4}, $R^2 = 0.99$. All coefficients were significant in a t test at $P < 0.05$.

The logistic epidemic rates (r_{50-70}) were calculated for the three two-way interactions given above. When low final proportions of pustular area ($y_{70} < 0.015$) occurred, small differences in the proportions of pustular area caused great differences in logistic rates, as noted earlier (13). Therefore, epidemic rates (k_G) for days 50 to 70 were calculated with the Gompertz transformation (5). The three response surfaces of k_{50-70} (Fig. 4D–F) were fitted to the interactive equation given above for y_{70}. The regression coefficients, A–H, in the response of k_{50-70} were, respectively, for i) environmental favorability versus log (initial infected area): –0.0216, 0.1613, –0.0243, 0.0919, 0.3028, –0.004, 0.0753, and 0.0172, $R^2 = 0.986$; ii) day of initial infection versus log (initial infected area): 0.3452, –0.0111, 0.1158, –0.0038, 9.47×10^{-5}, 0.0116, 2.1336×10^{-5}, and –0.0003, $R^2 = 0.978$; and iii) environmental favorability versus day of initial infection: 0.0052, 0.0884, 0.0008, –0.0021, 0.0382, -2.289×10^{-5}, –0.0027, and 6.803×10^{-5}, $R^2 = 0.951$. All coefficients were significant in a t test at $P < 0.05$. In general, the epidemic rates became faster when the level of initial infection was higher, when the day of initial infection was earlier, and when environmental favorability increased.

Model verification. The verification of the model for bean rust was accomplished by simulating an epidemic using the weather variables monitored during a crop season of cv. Rosinha during 1990 at Piracicaba, SP, Brazil. The daily favorability values (F_i)

TABLE 2. Sensitivity analysis of the primary parameters of the simulator for rust epidemics on Phaseolus bean expressed as proportion of leaf area with pustules on day 70 (y_{70}) and percent change (±) from the proportion with base values[a]

Parameter[b]	Base value	Percent change in parameter							
		–20		–10		+10		+20	
R_{max}	12.0	0.391	(–30.2)	0.474	(–15.4)	0.649	(+15.9)	0.740	(+32.1)
F	0.8	0.391	(–30.2)	0.474	(–15.4)	0.649	(+15.9)	0.740	(+32.1)
Rate of A	0.025	0.611	(+9.1)	0.586	(+4.6)	0.533	(–4.8)	0.506	(–9.6)
a (R)	0.05	0.453	(–19.1)	0.509	(–9.1)	0.608	(+8.6)	0.651	(+16.2)
b (R)	0.5	0.405	(–27.7)	0.488	(–12.9)	0.620	(+10.7)	0.672	(+20.0)
L_{max}	2,500.00	0.589	(+5.2)	0.574	(+2.5)	0.548	(–2.1)	0.537	(–4.1)
k_L	0.0232	0.640	(+14.3)	0.617	(+10.2)	0.501	(–10.5)	0.456	(–18.6)
c_L	4.27	0.583	(+4.1)	0.569	(+1.6)	0.549	(–2.0)	0.536	(–4.3)
k_D	0.018	0.587	(+4.8)	0.577	(+3.0)	0.536	(–4.3)	0.503	(–10.2)
c_D	4.3	0.534	(–4.6)	0.549	(–2.0)	0.569	(+1.6)	0.575	(+2.7)

[a] Proportion of leaf area with pustules for the base values of parameters was 0.560. The percent change in y_{70} for the various parameters was calculated as $(y_{70} - 0.56)/0.56$. All simulations were begun with an initial infected area of 0.07% on day 4.

[b] R_{max} = maximum basic infection rate; F = environmental favorability for infection ($0 < F < 1$); rate of A = daily decrease in susceptibility of leaves due to age; a and b were used to determine the daily infection rate ($R = a\ R_{max}(I/dL)^{-b}$); L_{max} = maximum leaf area; k_L = Weibull rate of foliation; c_L = shape of Weibull curve for foliation; k_D = Weibull rate of defoliation; and c_D = shape of Weibull curve for defoliation.

were calculated with the equation given above from the hours of leaf wetness and the daily minimum temperatures monitored in the field (Fig. 5B). The minimum latent periods (p_1) were calculated from the equation for $1/p(T)$ given above. These values were: days 1 to 10, $p_1 = 7$; days 11 to 18, $p_1 = 8$; days 19 to 32, $p_1 = 7$; days 33 to 61, $p_1 = 10$; and days 62 to 70, $p_1 = 12$. The epidemic was initiated with a proportion of infected area (f_0 = 0.000037) on day 7. The value of f_0 was obtained in calibration to achieve a proportion of pustular area (y_{14} = 0.000013) that was approximate to the proportion estimated in the field at the first assessment on day 14. The proportions of pustular area (logit scale) for the simulated epidemic were plotted against the mean proportions (logit scale) assessed on 20 plants in the natural epidemic (Fig. 5A). The increase in pustular area predicted by the model was quite representative of the increase in pustular area assessed in the natural epidemic.

DISCUSSION

A disease-progress model based on infections that occurred on daily cohorts of leaves (6) was expanded to arrive at an epidemic simulator for bean rust. Disease efficiency, length of latent period, and the pattern of variable latency were determined from monocyclic experiments conducted in growth chambers (2,16). The decline in disease efficiency as leaves age (A) was a refinement to the model. It is unreasonable to assume that the rate of decline

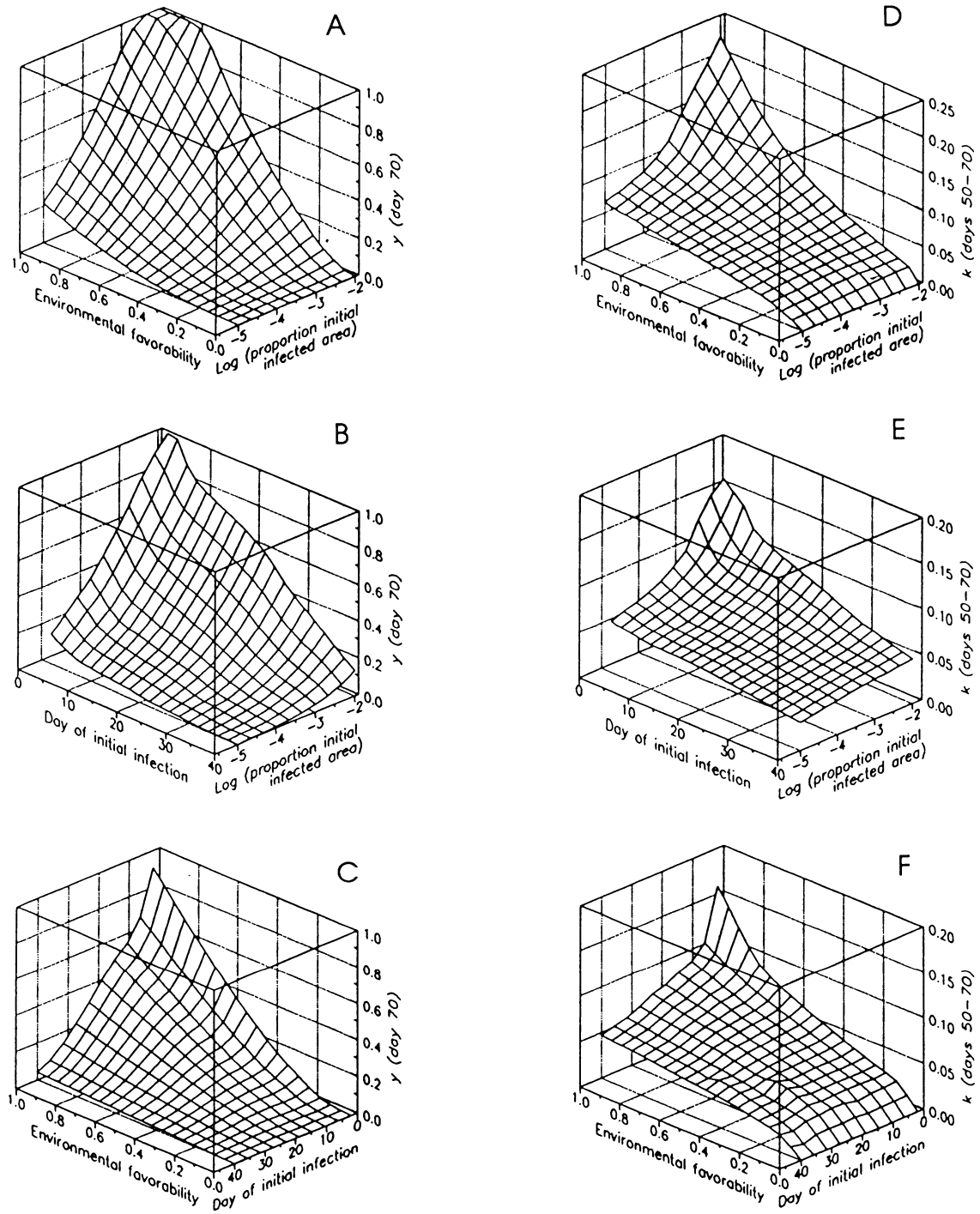

Fig. 4. *Uromyces appendiculatus* on *Phaseolus vulgaris*. Sensitivity analysis of three two-way interactions **A–C,** on final proportion of pustular area (y_{70}) of simulated disease and **D–F,** on the Gompertz epidemic rate (k_G) for days 50–70. The equations to describe the response surfaces are given in the text.

due to aging (2.5% day^{-1} [13]) would be constant over the entire life of a leaf, but the exact characterization of this relationship may not be too important since this rate was not a particularly sensitive parameter (Table 2). Natural epidemics of rust in beans in the field were carefully monitored for numbers and areas of leaves and numbers and sizes of pustules, which gave rise to the functions for host growth and defoliation, pustular area, and estimates of R_{max}, and the choice of a function to handle the relationship between the basic infection rate and the proportion of infected leaf area.

U. appendiculatus is very responsive to both temperature and the length of the leaf wetness period, as shown in the rather steep slopes of the response for disease efficiency (Fig. 1). Logically, it would be preferable to use hourly temperatures during the period of leaf wetness (usually nighttime periods) rather than the daily minimum temperature to determine favorability for infection. However, in a sensitivity analysis with the daily minimum temperature ±2°C (data not shown), only minimal changes in the average favorability values occurred. A model to forecast the occurrence of rust should be easily derived from the surface in Figure 1, perhaps as a simple modification of the disease-efficiency equation. The daily environmental favorability values, F_i, were determined from this disease-efficiency equation applied to the hours of leaf wetness and minimum temperatures recorded in the field. The responsiveness of the pathogen to environment was incorporated into the simulation model simply as a multiplicative adjustment by F_i to the basic infection rate, like the method used earlier by Sall (19) in a model for grape powdery mildew. This technique must have provided sufficient realism of the natural system since the simulated increase in pustular area was a very satisfactory characterization of the progress of rust in the field (Fig. 5A).

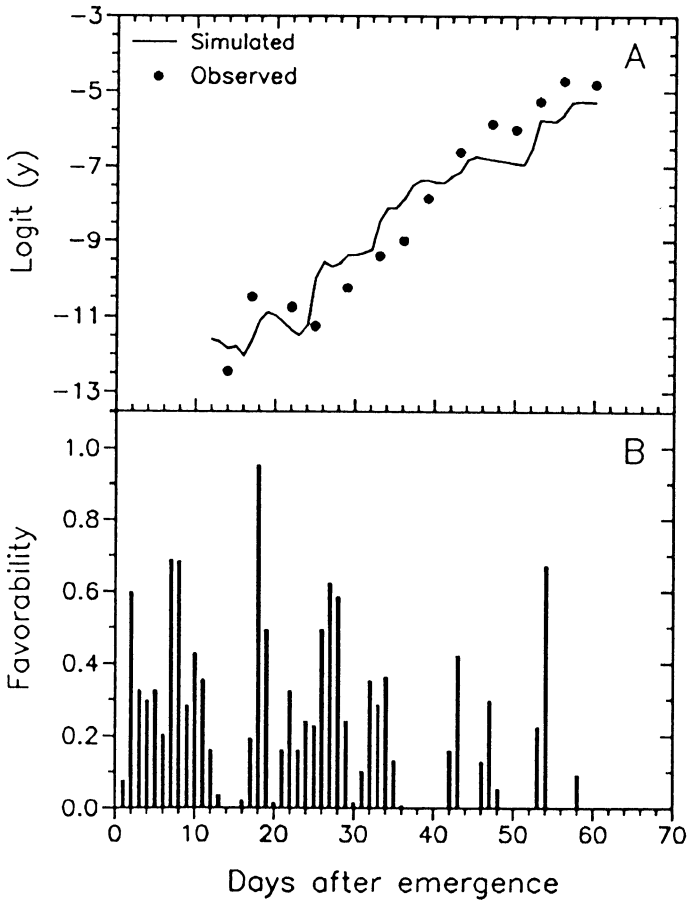

Fig. 5. Verification of the simulator for *Uromyces appendiculatus* on *Phaseolus vulgaris*. **A,** Pustular areas estimated in the field during 1990 and the progress curve for pustular area simulated with the model. **B,** The daily favorability values (F) for infection during the 1990 season at Piracicaba, Brazil.

Since there were great differences in scale (0.018 to 2500.0) among the parameters used in the model, judicious care should be used in making interpretations of the percentage of changes in the sensitivity values given in Table 2. With the sensitivity analysis, several responses were obtained that warrant further investigation. The most sensitive response was with the shape of the curve for the decreasing basic infection rate with the decreasing area of healthy tissue available for infection. The problems associated with the characterization of this relationship were noted earlier (6). Unfortunately, frequently the most sensitive parameter in a model is the one that has not been defined with accuracy. Such was the case for these functions. Despite intensive investigation (R Wannigen and R. D. Berger, *unpublished data*), the actual nature of the relationship of the basic infection rate versus proportion of healthy tissue for this pathosystem remains conjecture supported only by circumstantial evidence (6). Nonetheless, persistence to define this response seems a worthwhile goal.

Interesting effects for the rate of host growth on disease progress were obtained. The total pustular area was reduced when host growth was faster (a higher value of k_L), which supports the commonly accepted concept that growers experience fewer and milder disease problems if they can keep a crop growing well. In addition, final disease severity was affected only minimally by changes in the maximum plant size. In the examination of the influence of host growth on disease severity in natural epidemics, it must be kept in mind that manipulation of host growth will affect the microclimate and, hence, also affect disease efficiency.

To interpret the effect of the levels of initial infection on the sensitivity of the model, the proportions of pustular area were transformed with the Gompertz transformation (5) to calculate the epidemic rates (k_G). The epidemic rates (k_G) increased with each increase in the initial infected area (Fig. 4D and E). When these same values were transformed instead with the logistic transformation (22), the epidemic rates (r) increased with the decrease in initial infected area, as reported earlier (18). Consequently, when one is evaluating, for example, the effect of sanitation of initial inoculum on an epidemic, it is important to determine whether the effect on epidemic rate is a true representation of the biological relationship or an aberration due to the selected mathematical transformation.

The sensitivity analyses of the three two-way interactions of environmental favorability, level of initial infection, and day of initial infection (Fig. 4A–F) can be used to formulate recommendations to reduce the intensity of rust. Practices, such as host resistance, application of fungicides, and choice of time of planting to avoid favorable environment during the first few weeks of the crop season, would delay the time of initial infection. Even though favorable environment might occur later in the season, there would not be sufficient time for the cycling generations of rust to reach a level of intensity that could cause significant damage. Practices such as isolation of fields, applications of low doses of fungicides, and use of partially resistant cultivars would lower the initial intensity of rust, which would lead to a lower final severity.

The intensity of rust in natural epidemics was assessed with considerable accuracy because the epidemics were monitored by counting and sizing the pustules on each leaf of the marked plants on each date of assessment. Therefore, the model for epidemics of bean rust was based solely on the increase in pustular area; the other manifestations of the syndrome (halos around pustules, leaf yellowing, green islands, necrosis, and defoliation) were not included in the model. Under certain conditions of environment and crop culture, large, distinct yellow halos form around the pustules. These halos may be from 5 to >15 times the area of the pustule. With the substantial amount of leaf area sometimes involved in halos, the inclusion of this aspect in the simulator is needed to characterize the full manifestations of the disease.

The maximum pustular area in the current model was assumed

to be 100% of the total leaf area. Under inoculative conditions in which the infection sites on the leaf surface are saturated with urediniospores on a single day of infection, more than 150 pustules per cm^2 might form, and the leaf surface would become essentially covered with pustules. After inoculation with low doses of spores, a few, scattered large pustules develop. Under some conditions, annuli of secondary pustules later develop around these large pustules, but the maximum pustular area would still be less than 100% of the total leaf area.

Beginning about 3 days after infection by *U. appendiculatus*, a "virtual" lesion (4) zone exists around each site of infection. Spores that arrive later in the zone of the virtual lesion or on the haloed area around pustules will not infect successfully, and this occurrence further decreases the efficiency of the spores. Although this decrease in efficiency is incorporated in the shape of the function curve for the calculation of the basic infection rate, the y_{max} for pustular area probably should be less than 1.0.

The acceleration of leaf fall associated with disease was not included in the model because of the incomplete quantification of this response. Once this relationship is defined, the accelerated defoliation by disease can be handled easily in the model by the appropriate adjustment to the rate parameter of the Weibull function for defoliation.

The inclusion of a module for yield loss in the simulator for bean rust would be an important advancement. The development of such a module would require careful definition of the contribution (23) to plant stress of the virtual lesion during latency, the haloed area, and the accelerated defoliation by disease. Some progress has already been made on the change in color with age (3) and on the decline in the net photosynthetic rate for bean leaves with and without rust (M. Iamauti and R. Berger, *unpublished data*). With the inclusion of the enhancements mentioned above, a simulation model for this pathosystem would be more complete, and the model would have even more utility.

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