Infection Rates in Three Pathosystem Epidemics Initiated With Reduced Disease Severities

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ABSTRACT

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The progress of bean rust (caused by *Uromyces phaseoli*), early leafspot (caused by *Cercospora arachidicola*) of peanut, and grey blight (caused by *Botrytis cinerea*) of begonia was monitored in epidemics initiated with high, medium, and low levels of inoculum. Polycyclic grey blight and bean rust epidemics developed in replicated greenhouse chambers equipped to simulate field weather conditions and bean rust and early leafspot epidemics were studied in buffered field plots. The Gompertz transformation of the disease proportions consistently provided a better statistical fit than did the logistic transformation when both were tested by simple linear regression. Polynomial models were inferior to the simple linear regression models. When the disease proportions were transformed with the logistic equation, rapid, nonlinear disease increases aberrantly were projected for initial epidemic stages. Regardless of the transformation, model, pathosystem, or experimental location, the calculated rates of disease increase were greatest for epidemics starting from the lowest initial disease severity in 73% of all cases. Low initial disease apparently was compensated for by accelerated rates of disease increase. Thus, sanitation measures in the management of compound interest diseases may be less effective than previously theorized.

Additional key words: Gompertz and logistic transformations, curve fitting, sanitation.

The effect of reducing initial disease severity (y_0, sensu Vanderplank [20]) on the rate of disease increase and subsequent severity of compound interest diseases has not been critically quantified. Kucharek (12) reported that reduction of initial inoculum of peanut leafspot by crop rotation resulted in 88–93% less disease at midseason but he made no inferences about effects of the treatments on rates of disease increase. Roelfs et al (17) reported higher disease severities, earlier maturity, and lighter kernel weights with higher initial wheat stem and leaf rust inoculum densities. The apparent infection rates were nearly identical with high and low inoculum densities for five of the cultivars, while higher rates of disease increase with low inoculum density occurred in other instances. There is no field evidence to show that the rate of epidemic progress with sanitation is the same as epidemic progress in comparable crops without sanitation (3). Both Berger (3, and *unpublished*) and Shrum (18) found that infection rates were partially dependent upon y_0 in simulations of epidemics.

The purpose of this research was to determine if the rate of disease progress was independent of the initial severity of disease. Different amounts of initial inoculum for three pathosystems (sensu Robinson [16]) were used to examine the problem. Portions of this work have been previously published (15).

MATERIALS AND METHODS

Greenhouse experiments. Two pathosystems, *Uromyces phaseoli* (Pers.) Wint. var. *typica* Pers. on *Phaselous vulgaris* L. and *Botrytis cinerea* Pers. ex Fr. on *Begonia semperflorens* Link and Otto, with three different initial amounts of disease and three replications were each tested twice in greenhouse chambers.

Ten growth chambers were constructed with the dimensions, 56 × 43 × 56 cm. To simulate air movement in the field and to facilitate spore dispersal, a Rotorod spore sampler (Ted Brown Associates, 26338 Esperanza Drive, Los Altos, CA 94022) fitted with a 10.2-cm-diameter fan blade was placed at the center of each chamber.

One chamber housed four to eight test plants and a hygrothermograph which continuously monitored relative humidity and temperature. The uninoculated test plants in this chamber also served as indicators of spore influx and cross-contamination among chambers. The other nine chambers each contained six to eight test plants. Chambers were arranged on greenhouse benches in a randomized complete block design.

The chambers were slightly opened and the air circulation was activated at 1000 hours to simulate field conditions of drying plants. At night (1700 hours) the chambers were closed and the air circulation was turned off to encourage condensation on the inner walls of the chambers and dew on the leaf surfaces. Supplemental mist was applied daily to leaf surfaces and chamber walls at 1700 hours to ensure high levels of relative humidity.

Field experiments. Bean rust and peanut leafspot epidemics were also established in the field. Peanut (*Arachis hypogea* 'Florunner') plots were square and consisted of two parallel 1-m-long rows. Plots were established on 17 April 1979 in a field of hybrid field corn in Gainesville, Florida. Bean (cultivar Bountiful) plots measured 1 m on a side. Plots consisted of three parallel rows. These were established on 18 August 1979 at the University of Florida's Horticultural Unit at Gainesville. Plots were separated by six rows of double-seeded sweet corn. A randomized block design with four treatments (three initial disease severities and an uninoculated control) and three replications was used in all field experiments.

Standard cultural practices, including adequate fertilization, were used. Plots were irrigated when necessary and received 2 cm of water per irrigation.

Inoculation procedures. *Bean rust (greenhouse).* Urediniospores were collected from at least four different bean cultivars and stored under negative pressure at 8 C.

Two-week-old Bountiful bean plants in 10-cm-diameter pots were wetted to runoff by using a carbon dioxide-powered chromatography sprayer (Universal Multimister, Professional Packaging Corp., Chicago, IL 60614), with tap water containing a few drops of Triton X-100 (E. I. du Pont de Nemours & Co., Wilmington, DE 19899). Urediniospores in a spore-talc mix (15 g of talc per plot or chamber) were then dusted evenly onto the abaxial surface of bean leaflets by hand. The weight of urediniospores in the mixture was adjusted to obtain three levels of inoculum (Table 1).

Bean rust (field). The field inoculation for bean rust was identical to the greenhouse procedure except the bean plants were 30 days old at the time of inoculation (18 September 1979; day 0). Plots were wetted thoroughly with overhead irrigation (2 cm). All inoculations were completed in the early evening to ensure optimum temperatures and sufficient moisture for spore

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germination on the leaf surfaces for a minimum of 16 hr.

Grey blight of begonia. Begonia plants were obtained from a commercial nursery in Apopka, FL. Plants were maintained for several days under the optimum greenhouse conditions for grey blight disease development (9) to ensure that the plants were disease-free before inoculation. A mixture of four begonia cultivars (Whiskey, Gin, Vodka, and Scarlanda) served as host plants in the first experiment. To minimize variability between plants, only cultivar Whiskey was used in the second experiment.

A mixture of three isolates of *B. cinerea*, one from *Philodendron* sp. and two from *Begonia* sp. was used in all experiments. Isolates were maintained on acidified potato-dextrose agar (APDA) slants at 10 C.

The inoculation method described by Hyre (9) was followed to obtain conidia on 4-mm-diameter leaf disks for inoculation. Plants were inoculated by placing one, three, or five disks with abundant Botrytis conidia on intact leaves to obtain different initial disease severities (see Table 1). Plants were misted, enclosed in plastic bags, and incubated at 20 C under 12-hr diurnal fluorescent light cycles for 5 days. The bags were removed, and plants placed in greenhouse growth chambers for the start of the epidemic.

Early leafspot of peanut. A mixture of 10 isolates of *Cercospora arachidicola* selected from fields in Gainsville and Marianna, FL, was used. Isolates were maintained at 10 C on APDA slants. Inoculum was produced on APDA plates incubated at 25 C under continuous fluorescent lights for 7–10 days. The plates were flooded with tap water, and a camel's-hair brush was stroked gently over the surface of the fungal colonies to release the conidia. The conidial suspension was filtered through four layers of cheesecloth and diluted with tap water containing a few drops of Triton X-100 to predetermined concentrations (Table 1).

Immediately preceding inoculation, field plots were sprinkler irrigated (2 cm) to ensure a period of adequate moisture for infection. Fifty milliliters of inoculum per plot was sprayed evenly onto 45-day-old peanut plants on 8 June 1979 (day 0) using a carbon dioxide-powered chromatography sprayer. All inoculations were completed at dusk to ensure optimum temperatures and 100% RH for a minimum of 16 hr.

Phytopathometry. The proportion of visible disease (y_v) including necrosis and associated chlorosis, and the proportion of defoliation (d) due to disease were estimated with the Horsfall-Barratt rating scale (8) modified to obtain more accurate intraclass ratings (5). The proportion of total disease (y_t) was calculated by the equation (14):

$$y_t = ((1-d) \times y_v) + d. \quad (1)$$

Field assessment keys including that of James (10) for common leafspot of alfalfa provided a standard reference for estimating visible disease.

During initial stages of bean rust and early leafspot epidemics, the total number of lesions per chamber or per field plot (P_t) were counted. The total proportion of disease at any time (y_t) was calculated by the equation:

$$y_t = (P_t \times C/100\ L_t). \quad (2)$$

The constant (C, in square millimeters) for each pathosystem was calculated by averaging 100 randomly selected lesion areas which were measured with an ocular micrometer (Bausch and Lomb Incorporated, Rochester, NY 14602). The constant was equal to 12.6 mm^2 for bean rust and 7.07 mm^2 for peanut leafspot. The total leaf area (L_t, in square centimeters) per chamber or per plot was determined from standard area diagrams. The 100 is included in equation (2) to transform leaf area units from square centimeters to square millimeters.

Standard area diagrams were prepared for peanut and for bean by measuring pressed detached leaves with a leaf area meter (Li-Cor, Inc., Lambda Instrument Corporation, Lincoln, NE 68504). Leaf size classes were established according to the Weber-Fechner law (8).

The total leaf area of each plot was determined each time disease was assessed. The number of leaves in each of the size classes was calculated from counts on three to five randomly selected plants per plot. The mean value of leaf area on these plants was multiplied by the number of plants per plot.

Early leafspot of peanut was assessed approximately weekly beginning 18 June 1979 (day 16). Grey blight of begonia was assessed approximately biweekly beginning 25 February 1979 (day 0) in experiment one, and 30 April 1979 (day 0) in experiment two. Bean rust was assessed approximately biweekly beginning 25 September 1979 (day 7) in the field and 14 June 1979 (day 10) in the greenhouse.

Analyses. The total proportion of disease (y_t) at any assessment time was calculated as the mean value of three replications. The values of y_t were transformed by using the logistic transformation (20) of the form:

$$\text{logit } y = \ln (y/(1-y)) \quad (3)$$

and the Gompertz transformation (6,11) of the form:

$$\text{gompit } y = -\ln(-\ln(y)) \quad (4)$$

to linearize the disease proportions. Unweighted, multilevel polynomial models of the second degree and higher, and weighted and unweighted simple linear regression models were applied to transformed and nontransformed values. The criteria for evaluation of the goodness of fit to the model were the correlation coefficient (r), the standardized residual sum of squares from regression analysis, and a lack-of-fit test (19). Slope parameters (the infection rates [k]) of the calculated regression lines for each transformation and pathosystem were compared by using Duncan's multiple range test ($P = 0.05$).

All data were analyzed by using the Statistical Analysis System (SAS Inst. Inc., P.O. Box 10066, Raleigh, NC 27605) (1) package on the Amdahl 470 V/6-11 with OS/MVS Release 3.8. Computing was done at the facilities of the Northeast Regional Data Center of the State University System of Florida, located on the campus of the University of Florida in Gainesville.

RESULTS

Plant growth. Total leaflet area for each assessment date was

TABLE 1. Initial inoculum (II) and initial disease (y_o) for three pathosystem epidemics[a]

Initial disease severity class	Pathosystem[b]									
	Bean rust				Begonia grey blight				Peanut leafspot	
	Greenhouse		Field		Exp. 1		Exp. 2		Field	
	II (mg)[c]	y_o	II (mg)[c]	y_o	II (disks)[c]	y_o	II (disks)[c]	y_o	II[d]	y_o
High	5.00	0.082	5.00	0.0012	5	0.11	10	0.045	750,000	0.02
Medium	1.00	0.006	1.00	0.00023	3	0.035	5	0.024	75,000	0.0009
Low	0.025	0.002	0.025	0.0000034	1	0.007	1	0.006	7,500	0.0001

[a] Values are the average of three replicates.

[b] *Uromyces phaseoli* on *Phaseolus vulgaris*, *Botrytis cinerea* on *Begonia semperflorens*, and *Cercospora arachidicola* on *Arachis hypogaea*.

[c] Weight of urediniospores applied per greenhouse chamber or field plot. "Disks" are 4-mm-diameter disks from abundantly sporulating Botrytis-infected leaves that were placed on intact leaves (9).

[d] Number of spores applied per field plot.

compared by using Duncan's multiple range test ($P=0.05$) for each pathosystem. Total leaflet area was not significantly different among any of the disease treatments in any pathosystem in the greenhouse or the field. There were significant differences, however, among locations and experiments for bean and begonia plants. Regardless of treatment, bean plants were significantly larger in the field than in the greenhouse. Begonia plants were significantly larger in the second than in the first experimental epidemic.

Analysis of disease progress curves. Nontransformed disease progress curves were approximately J-shaped for all pathosystem epidemics. Polynomial models fitted to nontransformed disease progress curves retained much of the original curve shape. However, a minimum fourth degree polynomial model was necessary for reasonably high correlation coefficients ($r>0.70$).

To facilitate analysis of curve parameters and to linearize the data, logarithmic transformations were utilized. Both the logistic (equation 3) and Gompertz (equation 4) transformations linearized the disease progress curves, however the accuracy of fit differed significantly.

Extremely rapid rates of disease increase were observed during initial disease stages ($y_t<0.10$) for early leafspot of peanut and grey blight of begonia epidemics when plotted with the logistic transformation. Similar problems with logistically transformed data have been previously reported (2,4). Regression lines calculated for the epidemics did not include both the initial increase, and the progress of disease when $y_t>0.10$. While correlation coefficients for the single total regression line remained high ($r>0.80$) (Tables 2–4), the lack-of-fit parameter was poor.

The Gompertz equation fitted to simple linear regression analysis was usually the better model as determined by correlation coefficients, standard residual sum of squares, and the lack-of-fit test (Tables 2–4). High initial slopes in transformed disease progress curves when $y_t<0.10$, a recurrent problem for all pathosystem epidemics analyzed with logistic methods, were minimized with the Gompertz transformation. Thus, disease progress for all pathosystems in all locations was explicable by a single linear relationship (Tables 2–4).

Epidemic progress. *Bean rust.* Disease increased more rapidily in the field than in the greenhouse regardless of initial severity. In both greenhouse and field experiments, rates of disease increase calculated from the Gompertz transformed values were higher for epidemics that started from medium than from high or low disease severities. These differences, however, were not statistically significant ($P=0.05$) (Table 2, Fig. 1). Rates of disease increase calculated from logistic-transformed data were significantly ($P=0.05$) higher for epidemics that started with low rather than high initial disease severity for both greenhouse and field experiments. In the greenhouse, rates of increase from medium initial disease severity also were significantly greater than those for high initial severity.

Grey blight of begonia. Correlation coefficients were greater for the Gompertz regression models than for the logistic models (Table 3) except for the medium inoculum level in experiment one. In experiment one, rates of disease increase calculated from either the Gompertz or logistic transformation were significantly higher for epidemics from low than from high initial disease severity (Table 3, Fig. 2). In the second experiment there were no significant differences among rates of disease increase, although rates from medium levels were slightly higher than others.

Early leafspot of peanut. Disease increased more rapidly from the low and medium initial disease severities than from the high level regardless of the transformation (Table 4, Fig. 3). The ranking of relative rates of disease progress by using the Gompertz model were the same as those calculated with the logistic model when $y_t>0.10$ only (Table 4).

TABLE 2. Linear coefficients from regression analysis of epidemics of *Uromyces phaseoli* on *Phaseolus vulgaris* initiated by three disease severities

		Greenhouse			Field		
Transformation[a]	Initial disease proportion	Regression[b] term (*a*)	(*b*)	Correlation coefficient (*r*)	Regression[c] term (*a*)	(*b*)	Correlation coefficient (*r*)
Logistic	0.082	0.041a[z]	−3.16	0.93	0.145a	−7.45	0.94
	0.006	0.102b	−5.66	0.95	0.180a	−9.19	0.95
	0.002	0.108b	−5.70	0.94	0.251b	−14.56	0.97
Gompertz	0.082	0.017a	−1.19	0.90	0.047a	−2.35	0.95
	0.006	0.037a	−2.00	0.98	0.055a	−2.68	0.98
	0.002	0.029a	−2.08	0.96	0.045a	−3.08	0.96

[a] Logistic transformation = $\ln(y/(1-y))$; Gompertz transformation = $-\ln(-\ln(y))$.
[b] Model $y=at+b+\epsilon$ in which a = slope of the regression line and is the infection rate (k); t = transformed disease proportion; b = intercept; ϵ standard error term.
[c] The values for each transformation in each column followed by the same letter are not significantly different ($P=0.05$) according to Duncan's multiple range test.

TABLE 3. Linear coefficients from regression analysis of epidemics of *Botrytis cinerea* on *Begonia semperflorens* initiated by three disease severities

		Experiment 1				Experiment 2		
Transformation[x]	Initial disease proportion	Regression[y] term (*a*)	(*b*)	Correlation coefficient (*r*)	Initial disease proportion	Regression[y] term (*a*)	(*b*)	Correlation coefficient (*r*)
Logistic	0.11	−0.005a[z]	−1.94	−0.17	0.045	0.105a	−2.36	0.77
	0.035	0.095b	−3.59	0.99	0.024	0.195a	−3.40	0.97
	0.007	0.185c	−4.72	0.97	0.006	0.192a	−4.20	0.83
Gompertz	0.11	−0.003a	−0.72	−0.18	0.045	0.047a	−0.865	0.80
	0.035	0.030b	−1.29	0.98	0.024	0.088a	−1.29	0.99
	0.007	0.057b	−1.58	0.99	0.006	0.064a	−1.40	0.86

[x] Logistic transformation = $\ln(y/(1-y))$; Gompertz transformation = $-\ln(-\ln(y))$.
[y] Model $y=at+b+\epsilon$ in which a = slope of the regression line and is the infection rate (k); t = transformed disease proportion; b=intercept; ϵ = standard error term.
[z] The values for each transformation in each column followed by the same letter are not significantly different ($P=0.05$) according to Duncan's multiple range test.

TABLE 4. Linear coefficients from regression analysis of epidemics of *Cercospora arachidicola* on *Arachis hypogaea* in the field initiated by three disease severities

Transformation[x]	Initial disease proportion	Regression term[y] (a)	(b)	Correlation coefficient (r)
Logistic	0.02	0.052a[z]	−4.79	0.85
	0.0009	0.079ab	−7.25	0.89
	0.0001	0.120b	−9.86	0.91
Gompertz	0.02	0.019a	−1.67	0.89
	0.0009	0.021ab	−2.10	0.91
	0.0001	0.028b	−2.57	0.96

[x]Logistic transformation = $\ln(y/(1-y))$; Gompertz transformation = $-\ln(-\ln(y))$.

[y]Model $y = ab + b + \epsilon$ in which a = slope of the regression line and is the infection rate (k); t = transformed disease proportion; b = intercept; and ϵ = standard error term.

[z]The values for each transformation in each column followed by the same letter are not significantly different ($P = 0.05$) according to Duncan's multiple range test.

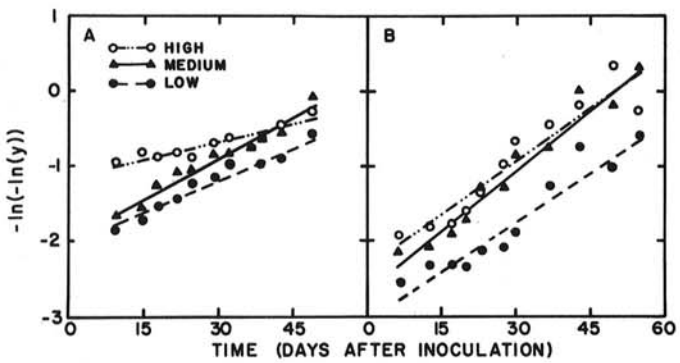

Fig. 1. Epidemic progress of rust caused by *Uromyces phaseoli* on *Phaseolus vulgaris*. **A,** Greenhouse epidemics initiated from low, medium, or high disease severities (see Table 1). **B,** Field epidemics. Values are the average of three replications, transformed by the Gompertz equation ($Y = -\ln(-\ln(y))$), and analyzed by least squares regression.

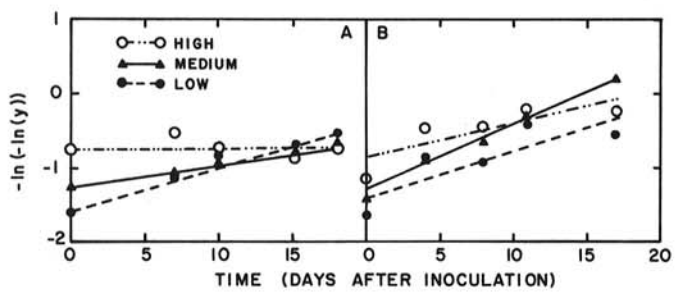

Fig. 2. Epidemic progress of grey blight caused by *Botrytis cinerea* on *Begonia semperflorens* in the greenhouse. In two experiments, A and B, epidemics were initiated from low, medium, or high disease severities (see Table 1). Values are the average of three replications, transformed with the Gompertz equation ($Y = -\ln(-\ln(y))$), and analyzed by least squares regression.

Control plots. Bean and peanut plants in uninoculated field plots and bean and begonia plants in uninoculated greenhouse chambers did not become diseased during the experiments. We therefore assumed that no spore movement occurred between plants in different treatments and that rates of disease increase in inoculated treatments were truly independent of each other.

DISCUSSION

Vanderplank (20) considered that the effectiveness of sanitation should be linearly related to the delay in reaching any given level of disease. This delay (Δt) is the additional time required to reach a given severity in a crop with sanitation (y_{os}) as compared to the same crop without sanitation (y_o) and can be calculated by:

$$\Delta t = \ln(y_o/y_{os})/k \quad (5)$$

in which k is equal to the slope of the regression line (Vanderplank's r value). The correction factor ($1-y$) does not appear in the calculation of the time delay (Δt) between epidemic progress curves (Eq. 5) "unless [y_o] and [y_{os}] are themselves outside this [the logarithmic] stage" of the epidemic (20, page 127). To calculate the time delay between two disease progress curves for any value of y greater than approximately 0.05, the correction factor must be included in the sanitation ratio. The equation for this calculation is:

$$\Delta t = (\text{logit } y - \text{logit } y_s)/k \quad (6)$$

in which the logit of disease severity without sanitation (y) and with sanitation (y_s) are included regardless of initial disease severity. If the Gompertz transformation is used, the equivalent calculation of the time delay from sanitation is given by:

$$\Delta t = (\text{gompit } y - \text{gompit } y_s)/k \quad (7)$$

Vanderplank considered some factors that could limit plant disease development when he first discussed sanitation theory (20). He was cautious in recommending sanitation as a disease control strategy, particularly for diseases with high infection rates and for epidemics of long duration. The inverse relationship between initial disease and rate of disease progress was observed in most all cases regardless of transformation. Most epidemics begun at low initial disease severity eventually (sometimes quickly) reached the same disease severity as corresponding epidemics begun at high severity. Therefore, we believe that this observed phenomenon is not a transformational artifact, but instead a biological relationship that requires other explanations. Limiting factors (eg, available susceptible host tissue, spore interactions, increased host resistance, etc.) may contribute to reduced rates of epidemic progress at high disease severities. The existence of limiting factors has been commonly accepted for biological systems (7,13).

We applied several transformations and models to the disease proportions. The logistic transformation did not always provide appropriate linearization. The logistic transformation could not be accurately fit to epidemic curves of grey blight of begonia and early leafspot of peanut, particularly during initial epidemic stages ($y < 0.10$). Kranz (11) cautioned against the universal application of the logistic transformation to epidemic progress curves, primarily because the goodness-of-fit statistics associated with the analysis are often poor.

The Gompertz transformation fit to a simple regression model best linearized the experimental data for all pathosystems while minimizing standard residual error terms. The Gompertz transformation was particularly effective for linearizing very rapid

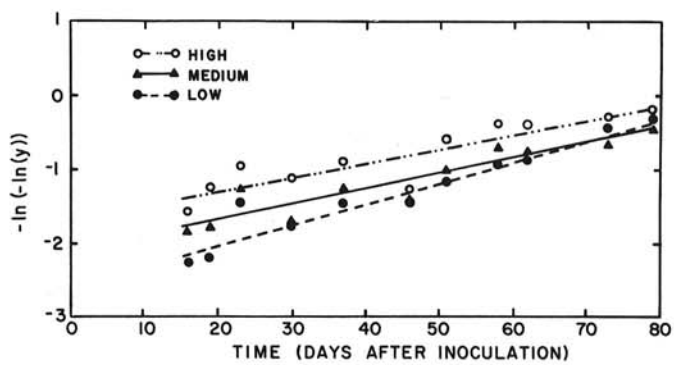

Fig. 3. Epidemic progress of leafspot caused by *Cercospora arachidicola* on *Arachis hypogaea* in the field. Epidemics were initiated by low, medium, or high disease severities (see Table 1). Values are the average of three replications, transformed with the Gompertz equation ($Y = -\ln(-\ln(y))$), and analyzed by least squares regression.

nonlinear increases during initial epidemic stages. High nonlinear rates of disease increase have been previously reported (2–4). The Gompertz transformation is suggested for accurate analysis for epidemics of broad ranges of severity and it has been used in this context (4,6). For 73% of the total 45 epidemic curves of our three pathosystems analyzed by this model, increasing amounts of initial disease were correlated with decreasing rates of disease progress, but differences were not always significant.

Epidemic progress was monitored for three pathosystems in greenhouse and small plots only. The epidemics in small plots and greenhouse chambers may not necessarily progress similarly to those in the large fields or for all pathosystems. The reduction in initial disease, which was used to simulate the effect of sanitation, may not be identical to benefits derived from actual sanitation measures. The initial disease severities in our experiments differed significantly, but the differences may not be biologically significant. Additional experimentation is needed to test whether these factors are important, to establish the role of initial disease in influencing the rate of disease progress in other pathosystems, and to reevaluate the wisdom of employing sanitation measures against compound interest diseases.

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