PROGRESS AND SPREAD
OF BEAN RUST

BY

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"Omnes observationes nostrae propter
instrumentorum sensuumque imperfectionem
non senti nisi aproximationes ad
veritatem."

Gauss, 1809
cited by Hunt (52)

To
my father, who has gone,
my mother,
my wife, and
our baby, who is coming.

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PROGRESS AND SPREAD
OF BEAN RUST

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Rust (Uromyces phaseoli [Pers.] Wint. = U. appendiculatus [Pers.] Unger) is highly destructive to beans (Phaseolus vulgaris L.). Reddish-brown pustules, typically found on leaves, rupture the epidermis, exposing urediospores.

Progress (increase in time) and spread (increase in distance) of bean rust were studied from three source types (area, line, and point) with three assessment types (incidence of diseased plants [INCPL], incidence of diseased leaves [INCLF], and severity [SEV]) in experiments in spring and fall.

From three models of disease progress (logistic, Gompertz, and monomolecular), the best fit was obtained with the Gompertz model.

Zellner's procedure was used to estimate and compare epidemic rates (k) and initial disease (y_0), with the linearized Gompertz model. Rates based on INCPL were faster than rates for INCLF and SEV. For the three source types, fastest rates were observed in epidemics originating from point sources. The rates, in general, did not differ between spring and fall epidemics, or between west and east directions. The y_0 's were different among all treatments. As distance from the source increased, y_0 's decreased. The k 's in most cases did not vary with distance.

From seven nonlinear models of disease gradients, the best fit was observed with the three-parameter models of Hoerl and Lambert and coworkers. Zellner's procedure was used to estimate and compare gradient parameters with the linearized two-parameter model of Kiyosawa and Shiyomi.

As predicted by Gregory, the weaker the source strength, the steeper the gradient. However, no difference between the estimated slopes of all source types was detected in the last assessments. Intercepts were often larger in the area source. Gradients based on assessment types were successively steeper from INCPL to INCLF to SEV. Gradients west from the source were flatter than those to the east. Gradient slopes flattened over time. The flattening probably occurred when the maximum asymptote of disease was reached.

Epidemics should be viewed as changes in disease with time and space; hence isopathetic rates were estimated. Isopathetic rates were faster for epidemics initiated from

point sources. Jeger's tridimensional models were also tested. The model proposed for polycyclic diseases gave a good fit.

INTRODUCTION

Although several early contributions in epidemiology are notable (146), the first book of Vanderplank (132) was a major stimulus to the development of botanical epidemiology. As a result, in the last 20 years we have witnessed a spectacular expansion in epidemiological concepts and techniques. Moreover, the popularity of microcomputers has facilitated the modeling of epidemiological phenomena. Two phenomena constitute the core of any epidemic: progress (disease increase in time) and spread (disease increase in space), sensu Jeger (60). A thorough understanding of both progress and spread is essential to monitor and control epidemics of plant diseases.

The fitting of curves to a time series of observations is a popular activity in biology (53). Also, over the past two decades, modeling of disease progress has become an integral part of the quantitative description of plant diseases (126). Amongst the descriptive models, three are used most commonly: monomolecular, logistic, and Gompertz. The monomolecular curve rises to the asymptote at a decreasing rate, and there is no inflection point. The logistic curve is sigmoid and symmetrical about $y=0.5$ (y = disease proportion), the inflection point. The Gompertz is also sigmoid but asymmetrical about $y=0.37$, the inflection point (13, 128).

If the appropriateness of a model to describe disease progress and the biological reasoning for the use of the model are known, a better understanding of epidemic development and the rationalization of control strategies will follow. Researchers commonly use the logistic model and its transformation, as recommended by Vanderplank (132), to fit disease progress data, even though other models and transformations might provide a better statistical fit (13, 102). The use and misuse of models and transformations have induced the warning words of Kranz (77): "do not apply any transformation blindly" (p. 22).

Whatever the transformation, the linear regression of disease proportions versus time yields two parameters: the initial disease and the disease progress rate, which are useful for comparative epidemiology. The suitability of a sound statistical technique to compare these parameters is needed in botanical epidemiology.

One of the most authoritative workers in disease spread has been P. H. Gregory. According to Gregory (43), "the intensity of a plant disease over a field sometimes shows a definite trend, increasing or decreasing in a particular direction, in such a way that when the number of infections per unit area of ground surface or plant tissue is plotted against distance, a sloping line is obtained" (p. 189). This line characterizes the disease gradient. The theory of gradients has been extensively studied for pollutants (48, 118), pollen (104, 144), insects and animals (137, 144), and diseases and spores of plant pathogens (43, 44, 144).

Since Gregory's initial treatment of gradients (41, 43, 45), many workers have analyzed disease gradients in various pathosystems. Therefore, gradients of disease incited by bacteria (39), viruses (2, 38), and, most extensively, by fungi (43, 144) have been researched. However, several debatable points still endure in the theory of gradients. Gregory (41, 43, 45) has pointed out that the strength of the inoculum source determines the gradient. Thus, gradients are successively steeper in area, line, and point sources. This principle has been reported by other authors (56, 81, 142), who did not provide experimental evidence. Therefore, a good approach would be to compare the effect of the three sources of inoculum on the gradients. Gregory (43) and other authors (18, 75, 111) have proposed that gradients in the field flatten with time. The flattening was not observed by Berger and Luke (14) and MacKenzie (86). An interesting result was found by Danos et al. (25), in which disease gradients became steeper with time. This opposing result may have been from the latter authors' basing their disease assessments on disease incidence rather than severity. Several models to characterize gradients have been proposed. Therefore, the study of gradients by utilizing different models and assessments of disease intensity in the field is needed.

Disease can be sometimes a focus expanding over time and distance. The spatial and temporal factors interact, and their interaction determines the nature of the growth of epidemics. The main limitation of the gradient models is that they are static in time. Similarly, the main limitation

of progress models is that they lack the spatial component very important for understanding focal development (60, 62). According to Vanderplank (133), the integration of spatial concepts into temporal disease progress has been difficult. Therefore, more research on models that merge progress and spread is needed. In the epidemic simulators EPIMUL (70) and EPIDEMIC (119), disease growth in time and space is considered, but these simulators are of limited use (60). A time-space approach was considered by Berger and Luke (14), when they developed the concept of isopathic rate. A similar approach was used by Minogue and Fry (93, 94). A good contribution was made by Jeger (60) with the tridimensional models that merge progress and spread. The testing of these models is required if we are to understand epidemics completely.

To test some of the previous concepts was the objective of this research, which was conducted under field conditions. The pathosystem Uromyces phaseoli-Phaseolus vulgaris was chosen. Rust has been among the most destructive diseases of common bean in the United States in recent years (123). Yields of pinto bean were reduced up to 78% in a epidemic in Michigan in 1981 (6, 122). Besides the importance of the disease, the symptomatology of rust is very typical, and not easily mistaken for that of any other leaf spot of beans, which facilitates disease assessments in the field.

Three models of disease progress and seven models of disease gradients were compared for fit, as well as the respective linear transformations. A system of linear regressions for both progress and gradients was developed, and the parameters were estimated and compared using Zellner's procedure (149). The comparisons were based on three types of assessments (disease severity, incidence of diseased plants, and incidence of diseased leaves), that originated from three types of inoculum sources (area, line, and point), in two seasons (spring and fall). In the fall, comparisons were also made between two directions (east and west from the source). Comparisons among disease progress rates over distance from the source and comparisons among gradient slopes over time were also conducted. A tridimensional approach to describe epidemics was tested. Isopathic rates of bean rust were calculated, and the models of Jeger (60) to merge progress and spread were fitted to the data. The trends of the parameters estimated by Jeger's model were compared to the trends of the parameters estimated separately in the models for progress and spread.

LITERATURE REVIEW

Bean Rust

Bean rust, incited by Uromyces phaseoli (Pers.) Wint. (= U. appendiculatus [Pers.] Unger), was first reported from Germany in 1795 (148). Since then, it has been reported in every continent in the world (148). Bean rust causes one of the most important production problems in many areas of Latin America, including Brazil (20, 148). Baker et al. (6) and Stavely (122, 123) report that rust has been among the most destructive diseases of common bean in recent years in the USA, where losses up to 78% occurred in pinto beans in 1981. Large yield losses were reported along the coast of California, in Colorado, Louisiana, Washington, New Jersey, Massachusetts, and Virginia in the late 1920's (129). Losses in certain Florida counties ranged from 40 to 80% in 1938, and hundreds of acres in Florida were never harvested (129).

The fungus may infect leaves, pods, and, rarely, stems and branches. Initial infection may occur on the upper or lower leaf surface, but symptoms usually appear first on the lower surface, as minute, whitish, slightly raised spots 5 or 6 days after inoculation. These spots enlarge to form mature reddish-brown pustules that rupture the epidermis and may attain a diameter of 1-2 mm, 10-12 days after inoculation. Usually a yellow halo is formed around the pustules (148).

The infection frequency, measured by the number of pustles/cm², of bean rust decreases with leaf age (136). Thus, as the leaf ages, it becomes more resistant (136, 150). Also the appressorial formation of U. phaseoli decreases considerably on adult leaves (136).

Growth Curves

Many biological processes are basically nonlinear in nature, and should be described as such in empirical modeling (98). Therefore, the fitting of curves to a time series of observations on the growth of organisms became a popular activity in biology (53). This is usually done by nonlinear regression. The attraction to nonlinear models is attributed to their apparent ability to describe a complete process, to cope with asymptotic trends, and to estimate parameters with a meaningful interpretation (109). The limitations of nonlinear models are that some data sets are not fitted adequately and the absence of a well-known statistical theory to guide their interpretation (109). The Marquardt method for nonlinear estimation is a method which appears to work well in many circumstances (27).

A mathematical model developed from the understanding of the biological mechanism should be utilized whenever feasible. Experimenters often seem obsessed with the idea of finding the best-fitting model (92). In many situations, comparisons of models is difficult, because the meaning of "best fitting" is not very well defined. For models with an equal number of parameters, a simple comparison of the

residual sum of squares can be used (92). Hunt (52) states that "it is inescapable that the more complicated the mathematical function (that is, the more parameters it contains), the more closely it will fit any given series of experimental observations (that is, the smaller will be the deviations between the fitted function and the original data" (p. 247). Kvalseth (79) suggests the $R^2 = 1 - (SSR/SST)$ (where SSR = sum of squares of residuals and SST = corrected total sum of squares) as ideal to compare the fit of nonlinear models. However, Ross (109) reports that it is not very useful to quote R^2 or the percentage variance accounted for by the model. The R^2 is usually very close to 1 in a curve fitting application. Montgomery (95) states that R^2 will always increase if a new variable is included in a model. However, this increase does not necessarily mean that the new model is superior to the old one. Unless the SSR in the new model is reduced by an amount equal to the original MSE (error mean square), the new model will have a larger MSE than the old one, because of the loss of one degree of freedom (95). Thus, the new model will be actually worse than the old one. According to Madden (89), one of the most important ways to choose an acceptable model is to plot the residuals (y observed - y predicted) versus the independent variable. If this plot consists of a random scatter of points, there is no systematic deviation of the data points from the model under consideration. Therefore the model chosen is an acceptable one.

Disease Progress

Epidemics are defined as a change of disease with time (89). As reported by Madden (89), epidemics were quantified early by Barratt in 1945, by Large in 1952, and by Schmitt in 1959. However, the greatest advancement in the analysis of epidemics occurred with the publication of Vanderplank's 1963 book (132). As a result, in the last 20 years, we have witnessed a spectacular expansion in the use of modeling and simulation techniques in the study of plant disease epidemics (120, 126). This approach, based largely on the concepts and mathematics developed by Vanderplank, has greatly enhanced our capacity to use epidemiological principles in the control of plant diseases (120).

The quantification of epidemiological parameters added accuracy and objectivity to epidemic analysis (40). The quantification of disease progress over time is desirable for many reasons (89), one of which is the comparative use of estimates of the parameters of simple models for practical disease management (59).

Disease Progress Models

A knowledge of the appropriateness of certain models to describe disease progress and the biological explanation for the suitability of these models will lead to a better understanding of epidemic development. This should enhance our ability to forecast diseases and, ultimately, contribute to the development of better management strategies (19).

The monomolecular, logistic, and Gompertz models are usually described in fitting growth data (8, 27, 89, 101). The monomolecular or simple interest model has been used to describe a monomolecular chemical reaction of the first order, cell expansion or response of crops to nutrients, etc. (89, 105). The absolute rate of disease increase is proportional to the amount of healthy tissue. The fastest rate occurs at the beginning of the epidemic and the rate declines as the disease level approaches a maximum. Thus, the monomolecular curve rises to the asymptote at a decreasing rate; there is no inflection point (101).

According to Madden (89), the logistic model is the most important in botanical epidemiology, not necessarily because of its appropriateness, but because of the frequency of its use today. The model originally was used by Verhulst in 1838 to describe human population growth. Vanderplank in 1960 (131) proposed this model to describe the progression of polycyclic diseases. The rate of disease increase is proportional to the level of both diseased and healthy tissues (89). The logistic model is expressed mathematically as $y = y_{\max} / (1 + b \cdot \exp(-kt))$, where 'y' is disease intensity at any time 't', ' $y_{\max}$ ' is the asymptotic maximum disease intensity, 'b' is an initial position parameter, and 'k' is the rate of disease increase (101). The logistic curve is sigmoid and symmetrical about the inflection point (8, 101). The logistic and monomolecular models are very similar during the last phases of polycyclic epidemics (89).

Analytis, cited by Berger (13), found that disease-progress curves of apple scab fit poorly with the logistic model, and better fits were obtained with the models of von Bertalanffy, Mitscherlich, and Gompertz. The Gompertz model dates back to the early 1800s (89). The mathematician, B. Gompertz, derived this model to develop actuarial tables (5), and the model was very useful in animal population studies, to describe the increased frequency of deaths per unit of time with increasing age of test animals (89, 101). The Gompertz model was used to summarize disease progression by Berger and Mishoe (15). According to Tipton (128), the Gompertz curve is sigmoid and asymmetrical about the inflection point. This model may be more appropriate than the logistic when the maximal rate of disease increase occurs early (101). The Gompertz model provided a better statistical fit than did the logistic model for all 113 disease progress curves of 9 pathosystems (13). Stewart (125) observed that in crop growth studies, the Gompertz was also better than the logistic. For seed germination, the Gompertz model gave a better fit of the response than the monomolecular and logistic models (128).

According to Skylakakis (120), the difference between the logistic and Gompertz models is the rate at which disease progress is slowed down as disease-free susceptible sites in the host become fewer. Thus, in the logistic model it is assumed that the slow-down effect of reduced healthy tissue availability increases linearly with the amount of disease present and in the Gompertz model "it is assumed that the

slow-down effect increases with disease present, but that the magnitude of the effect becomes smaller as more and more of the available healthy tissue becomes occupied" (p. 119).

Other models have recently been used to describe disease progress. Fleming and Holling (33) developed a model which is better than the logistic and Gompertz to describe epidemics of cereal rusts. The number of parameters, five, may cause the model to be difficult to use. The same problem may occur with the Weibull, although Thal et al. (126) describe the Weibull as a flexible model that may offer an improved fit to disease progression data compared with the more commonly used two-parameter models.

Transformations

The linearization of disease progress curves is essential to determine epidemic speed, to project future disease, and to estimate initial disease (13). Plaut (101) reports that many researchers have proposed transformations and models with possible applications to all pathosystems. Some of these proposed models are based only upon the effective linearization, or fitting of curves, to a few pathosystems (49, 133). Since the monomolecular and logistic equations yield respectively linear relations of $\log_e(1/(1-y))$ and $\log_e(y/(1-y))$ with time, a common approach to define a disease as simple or compound interest has been to compare observed disease progress data suitably transformed against these two linear relations (120). Kranz (77) has warned "do not apply a transformation model blindly to any disease.

check its suitability first by verification of the underlying distribution" (p. 22). The criterion in the selection of a transformation must be that the biological phenomenon which gives rise to the data is interpreted accurately (68). Evidence is accumulating that there is no universal transformation applicable to all disease-progress curves (101). Therefore correct analysis is dependent upon the underlying distribution of the data, the growth function of the epidemic, as well as the adequate linearization of the disease-progress curve (101, 102).

"The logistically transformed disease-progress curves are frequently characterized by steep slopes at $y < 0.05$, linearization for the range $0.05 < y < 0.6$, and values that fall below the general slopes when $y > 0.6$. Berger drew attention to the rapid initial increase of logistically transformed curves and Zadoks noted the common occurrence of values below the line at upper levels of 'y'" (13, p. 716). Both logistic and Gompertz transformed values are approximately equal during high disease values ($y > 0.75$) (101). Berger and Mishoe (15) and Plaut (101) obtained better statistical fit to simple linear regression with the Gompertz transformation compared with the logistic for disease progress of certain epidemics. Rapid initial increases in disease were minimized with the Gompertz equation (85).

Rates Comparison

A very important consequence of linearizing disease-progress curves is the comparison of rates. In botanical epidemiology, disease-progress rates have been compared by Rouse et al. (110), who found that as ' y_0 ' increases, the disease-progress rate decreases. Plaut and Berger (102) also found fastest rates with lowest initial inoculum.

From ecological theory, Levin et al. (83) report that local increase in population density decreases the growth rates of individuals that remain close to their parental sites. Examples of this trend were observed by Kingsolver et al. (73) who observed that fastest progress rates of wheat rust occurred in the most distant points from the inoculum source. Lim (84) also reports that, in soybean downy mildew, the slope for disease progress at 27.4 m from the source was steeper than at 3 and 12.2 m. However, Emge and Shrum (29) found in gradient plots of wheat rusts that disease-increase rates were not significantly different with increasing distance from the inoculum source, but the occurrence of infections was progressively delayed. This delay was attributed to the dilution of inoculum levels with distance, and the problems associated with the detection of extremely low levels of disease. In addition, disease progress of plots upwind from the source was compared with disease progress in plots downwind from the source (29). The rate of disease increase was the same in both directions, but the level of initial infection was lower and delayed in the upwind direction (similar to the effect of increasing

distance from the source). In oat rust, Luke and Berger (85) found no difference between rates at 1 and 2 m from the source.

In some of the previous comparisons, the authors did not specify the test they used to compare the parameters, and in others cases they used the Duncan's multiple range test. The comparison of parameters obtained in the linear regression is a statistical problem. As described by Williams (141), the fitting of a straight line to a set of observations on two variables is a mere application of the method of least squares. When several regression lines are fit to several data sets, sometimes the researcher wants to determine whether or not the slopes of the lines differ. Sokal and Rohlf (121), Seber (116), Edwards (28), Freund and Minton (35), and Allen and Cady (1) describe tests to compare parameters of regression lines, using a t-test, a special F-test, or covariance analysis. These tests are suitable when the regression lines are uncorrelated. When the regression lines are correlated, as when comparing the progress rates at 0.3 and 8.5 m from the inoculum source, Khuri (72) mentions Zellner's procedure. As Zellner (149) reports, "given a set of regression equations, we consider the problem of estimating regression coefficients efficiently" (p. 348). In Zellner's procedure (149), regression coefficients of all equations are estimated simultaneously by applying Aitken's generalized least-squares. This is more efficient than an

equation-by-equation application of least squares when the equations are correlated (149).

Disease Gradients

According to Gregory (43), "the intensity of a plant disease over a field sometimes shows a definite trend, increasing or decreasing in a particular direction, in such a way that when the number of infections per unit area of ground tissue is plotted against distance travelled, they yield a sloping line" (p. 189). The slope of this line characterizes the disease gradient.

Probably one of the first to recognize a disease gradient was Windt in 1806, cited by Gregory (41), who observed that rust on rye was severe near barberry bushes which are now known to be the alternate host of the fungus: "These effects are striking and desolating in the distance of 12 paces. I have also perceived them visibly at the distance of 50, 100 and 150 paces, and a final attack at above 1000 paces" (p. 26).

Nowadays, gradient theory is widely developed in diseases caused by fungi (32, 43, 44, 130, 144), bacteria (39), and viruses (2, 38, 58, 127); and the theory is applied to detect local sources of inoculum (26, 39, 106), for general sanitation (10, 69, 78, 45, 140), and to study the effect of interplot interference (64). Also, gradients have been also considered in other fields of science, as dispersal of pollutants (48, 100, 118), dispersal of pollen (104, 144), and insects (137, 144).

Different kinds of gradients are recognized: environmental, dispersal (deposition), infection, and disease (43). The disease gradient results from a deposition gradient of infective propagules (99). According to Gregory (44), from the knowledge of deposition gradients, it is impossible to predict the level of infection of a plant at a given distance from the inoculum source; however, there is a possibility of predicting an upper limit. Waggoner (140) states that the pattern of disease in a field is "the footprint of the cloud of propagules that has flown over, and the sharp decrease in concentration with distance that is reflected in the footprint is striking" (p. 456). Dispersal gradients have been extensively studied (31, 41, 42, 46, 124, 144) and modeled (3, 4, 82, 108, 115). However in both dispersal and disease gradients, many questions remain to be answered. Kranz (77) reports that "while some evidence suggests that mathematical models fit observed deposition gradients well, much less is known about infection gradients. For obvious reasons in most cases it is not possible to deduce the latter from the former" (p. 33).

Disease Gradient Models

If we graph distances from the source (d) in the x-axis and disease intensity (y) in the y-axis, a hollow curve, decreasing rapidly near the source, then flattening out and becoming nearly parallel with the y-axis, will be produced (43). The first question in the study of gradients is how to represent these changes of disease over distance, i.e., which

is an "ideal" model to represent gradients. Gregory (43) used a model, previously cited by Wilson and Baker (142, 143), in which $y = a/d^b$ ('a' is disease intensity at the source and 'b' is gradient slope). A major problem in the Gregory model is that it estimates the lesion numbers to be infinite at the source, which is not real (74). Therefore, Kiyosawa and Shiyomi (74) described a model, previously reported by Brownlee for insect flights and cited by Bateman (7), in which $y = b \cdot \exp(-a \cdot d)$. When these two models were compared, Kiyosawa and Shiyomi (74) found their model better than Gregory model to describe gradients of several epidemics. The Kiyosawa and Shiyomi model was also better than the Gregory's model to describe the gradients of fusiform rust, based on the number of sori/cm² of oak leaves (114). Other pathosystems in which the model of Kiyosawa and Shiyomi gave a good description of gradients were maize dwarf mosaic virus (75) and potato late blight (99). Gregory's model was found to be superior for peach mildew (69) and for severity of rice blast disease (87). However, neither of these two models described the observed gradients completely (88). It has been reported (80, 88) that the model of Gregory overestimates disease intensity close to the source, and the Kiyosawa and Shiyomi model underestimates this parameter.

Lambert et al. (80) describe a general gradient model, which has been reported by Bateman (7) for insects, in which $y = a \cdot \exp(-b \cdot d^n)$. The 'n' allows variation in shape and indicates any data set's relative position of best fit in the

continuum of curves (80). A model described by Daniel and Wood (23), the Hoerl's special function, also has a third parameter: $y = a \cdot d^b \cdot \exp(n \cdot d)$. Other models are described by Daniel and Wood (23): $y = d / (a \cdot d - b)$ and $y = a \cdot \exp(b/d)$, by Bateman (7): $y = a \cdot \exp(-b \cdot d) / d$, and by Prunty (103): $y = a \cdot (-d \cdot (d^c + 1)^{1/c})$. More recently, Mundt and Leonard (96) described a modified Gregory's model, $y = a(x+c)^{-b}$, in which the 'c' parameter estimates a finite number of lesions at the source.

Linearization

As suggested by Gregory (43), a simple method is needed to compare disease gradients. Usually to facilitate these comparisons the gradient data are transformed before linear regressions are fitted. Therefore, for Gregory's model, the $\log(y)$ vs. $\log(d)$ transformation is used, as done by Rowe and Powelson (111) in *Cercospora* footrot of wheat and by Imhoff et al (54) in bean rust. The linearization of Kiyosawa and Shiyomi's is $\log_e(y)$ vs. 'd' untransformed, as done by Knorr et al. (76) in alfalfa mosaic virus. When regressions of both transformations were compared, sometimes the $\log(y)$ vs. $\log(d)$ gave the better fit (36), and sometimes the $\log_e(y)$ vs. 'd' was better (114). A common fact in the transformation choice is that, instead of being based on the model, it was just a researcher's choice (36, 54, 76, 111).

Other transformations are described by Danos et al. (25): $\text{gompit}(y)$ vs. $\log(d)$, Berger and Luke (14): $\text{logit}(y)$ vs.

$\log(d)$, and Minogue and Fry (94): $\text{logit}(y)$ vs. 'd' untransformed).

Flattening

A debatable point in gradient theory is the flattening of gradients, which is measured by the increase of the slope 'b' over time. According to Gregory (43, 44) the primary gradient is steeper than gradients in which secondary spread has occurred. Secondary spread flattens a primary gradient. Gradients of bean rust (54) and of other pathosystems (18, 84, 94) have been observed to flatten over time. Zadoks and Schein (147) also report that the secondary gradient is flatter; 'b' is smaller (in absolute value) and has a higher average value 'a' than does a primary gradient.

It is difficult to determine which part of gradient flattening is due to the enlargement of the primary focus, which to secondary spread, and which to background contamination (147). Kampmeijer and Zadoks (70) report that flattening around a point source must be ascribed to invasion of inoculum from natural outside sources into the experimental area. In EPIMUL, an epidemic simulator, spore influx from outside the plot was not considered; so flattening did not occur normally in the model (70). Lim (84), Waggoner (138), and Mahindapala (90) concluded that flattening was due to secondary spread within the crop. Cammack (18) observed that the flattening was caused by secondary spread and background contamination. Kiyosawa and Shiyomi (74) found flattening and a smaller increase of

lesions on plants near the source, which they suggested may be due to the limitation of healthy tissue. The hypothesis of limitation of healthy tissue is shared by Rowe and Powelson (111) and Knoke et al. (75).

Although MacKenzie (86) and Vanderplank (132) report that one would expect such a flattening of the disease gradient, MacKenzie (86) found no statistical indication of the flattening, although the disease levels in the subplot adjacent to the inoculum source exceeded 50% for the susceptible varieties. Similarly, Johnson and Powelson (67) did not observe gradient flattening with *Botrytis* blight of beans. Luke and Berger (85), using the $\text{logit}(y)$ vs. $\text{log}(d)$ transformation, did not observe the flattening, even when final disease severities were high. Danos et al. (25), in a study on the spread of citrus canker, using the $\text{gompit}(y)$ vs. $\text{log}(d)$ transformation, observed the steepening of slopes with time. Berger and Luke (14) believe that the flattening was more an aberration of the log-log transformation than a real fact. Although Cammack (18) sees the flattening as real, he believes it is exaggerated by the log-log scale.

Sources

In gradient experiments, usually the inoculum sources are recognized as area, line, and point. The size and shape of these sources are important factors to be considered when studying disease gradients. According to Gregory (44), real sources in the field that correspond approximately to the ideal sources would be a single plant (point), a hedge

(line), a ground crop (area), and an orchard (volume). The dimensions of the source must be treated as relative to their distances; thus, a field would be regarded as a point source when considered from distances many times its own width. Zadoks and Schein (147) state that a point source ideally has a diameter smaller than 1% of the gradient length. A barberry hedge and windbreak represent line sources. An early and severely infected field or region can serve as an area source, as do oversized point sources in some experiments (147). As stated by Gregory (43), "other sources can be regarded as made up of close assemblage of point sources which are not additively effective, because they receive and lose some of each others disseminules" (p. 194).

According to Gregory (43), the strength of a source affects the amount of disease it causes, altering the constant 'a'. This parameter is successively higher in point, line, and area sources (147). Therefore, 'a' is a measure of the different source strengths (36).

The shape of the source affects the gradient and its coefficient 'b' (43, 44). A principle theorized by Gregory (43, 44, 47) is that other things being equal, gradients from point sources are steeper than those from line, which are steeper than gradients from area sources. Other researchers (56, 81, 142) have repeated this principle, without obtaining real experimental evidence.

Assessments

One aspect not usually considered when gradients are studied and compared is how disease assessment type affects the trends. Jeger et al. (63) found that to describe incidence, the best regression equation involved distance transformed to $\log_e$ and for severity the best regression involved distance untransformed. They also concluded that incidence gradients were dependent, in part, on distance from the plot centres whereas severity gradients were not, being dependent only on the proportion of area diseased and the proportion of area healthy at a given distance. However, the authors did not compare the incidence and severity gradients.

Direction

The effect of the direction of assessment is a factor that should be understood better when gradients are analyzed. According to Paysour and Fry (99), disease gradients are often asymmetrical around the source due to such factors as strong prevailing winds. Vanderplank (132) points out that there is considerable evidence that wind can strongly alter the spread in distance. One reason why wind affects the spread in different directions is that more inoculum leaves the source in one direction than in another. Examples of wind direction affecting the gradients are found in the literature (97, 73, 138).

According to Gregory (43), in examples of wind-dispersed pathogens, the gradient in the supposed-downwind direction of a point source is often not very different from the gradient

around the source, because wind direction fluctuates during dispersal. Values for 'b' downwind are nearly all higher than for the mean of 'b' at all wind directions, but the difference may be small. The difference between the two values will depend on the constancy of the wind direction during the experiment (43). Emge and Shrum (29) observed that the effect of prevailing westerly winds was obvious because spread of inoculum was most rapid eastward and southward. Although steeper gradients were expected for the upwind direction, the upwind and downwind gradients did not differ significantly.

Comparison Between Disease-Progress Rate and Gradient Slope

Researchers have been trying to compare mathematically the concepts of disease progress and disease spread, and MacKenzie (86) concluded that it was difficult to merge gregorian and vanderplankian models through regression techniques. Therefore, the relative rates of disease spread probably are independent of the rate of disease increase (86). MacKenzie (86) pointed out that the intercept and not the slope of the disease gradient was associated with a reduced rate of spread of slow rusting of wheat. He made no interpretation as to the source of variation of 'a'. According to Danos (24), in the linearized disease gradients, the rate of increase of 'a' vs. time is determined by the rate 'k' of disease increase in time.

According to Vanderplank (135), other things being equal, the infection rate itself flattens or steepens the disease gradient. Vanderplank (134) also stated that "the gradient of disease at the epidemic's front is determined by the infection rate. The greater the horizontal resistance, the lower the infection rate, the steeper the gradient, and the more compact the epidemic's front will be" (p. 135). However, this trend was not observed by MacKenzie (86) nor by Luke and Berger (85), who concluded that Vanderplank's idea was a fallacy.

Tridimensional Description of Epidemics

Gradient models are limited because they are static in time; progress models are also limited because they lack a spatial component (60). Vanderplank (133) indicates that the integration of spatial concepts into temporal disease-progress models has been difficult. Although there are tridimensional models that describe the diffusion of gases (48) and pollutants (71), there have been no analytical models to describe progress and spread of diseases (147). Some epidemic simulators do this (70, 119), but they are of limited value (60).

If we are to understand the nature of epidemics, we must consider the interaction of spatial and temporal factors (94). An important start in this direction was made by Berger and Luke (14), when they calculated the rates of isopathic movement of oat crown rust. These authors calculated the rate of disease spread in space from plotted

logit lines for disease observed at 1.22 and 4.88 m from the plot centers at four time periods. The interlinear distance (3.66 m) was divided by the number of days needed for disease at 1.22 m to reach the same severity at 4.88 m. According to Berger and Luke (14), "the outward spread of disease from infection foci can be considered moving annuli of disease of equal intensity (isopaths). The outward movement of rust isopaths was calculated to give a measure of rust movement over distance in time. The rate of movement of isopaths would be assumed to be nearly constant if environmental conditions were stable and favorable, and if no spurious disease spread occurred" (p. 1200). The concepts of isopaths was extended by Minogue and Fry (93, 94); their parameter 'v' is equivalent to the rate of isopathic movement.

A more recent approach was taken by Jeger (60), with models to merge the concept of progress (increase in time) and spread (increase in distance). From eight models to describe epidemics of *Septoria* blight of wheat, Jeger (60) found three to be more applicable. The classical polycyclic diseases correspond to the model $y = 1/(1+a*\exp(bx+ct))$, in which secondary inoculum rapidly outnumbers the inoculum originated from the primary source. This model gave a good fit to disease severity of *Septoria* blight (60). The classical monocyclic diseases correspond to the model $y = 1-a*x^b*\exp(-ct)$, in which the primary source produces the inoculum throughout the epidemics. Disease incidence was best explained by this model; although disease was polycyclic, the increase in diseased leaves was influenced by

the nearness to the focal center. The model $y = 1 - ax^b \exp(-ct)$ is likely to fit the early stages of a polycyclic epidemic, when there is a large source strength. Jeger (60) proposed the first two models to be used for diseases which are strictly polycyclic or monocyclic, respectively. When polycyclic epidemics are initiated by artificial inoculation and the spread is local rather than general, the third model may be appropriate.

Field Experimentation

Ideally, small plot experiments should represent field situations (64). Researchers have observed that, in such conditions, the representational error due to interplot interference in experiments with air-dispersed pathogens may be important. However, according to James et al. (56, 57), to reduce substantially the effect of the representational error, the experiment would have to occupy such a large space that it could not fit in a normal experimental area. A good approach is to separate one field from another. Fleming et al. (34) report that the distance separating two fields must be big enough to inhibit dispersal between them and, at the same time, be small enough to allow essentially equal environmental conditions. Wider separation would bring problems, particularly in variations in soil type. Thus, the compromise between plot separation, to reduce interference, and small area, to reduce variability, needs solution (65). Jenkyn et al. (66) state that much of the interplot interference can be avoided by separating plots with guard

plots with similar dimensions to the experimental plots. Bowen et al. (16) compared corn and wheat as guard plots in experiments with leaf rust of wheat. They found that the type of guard crop had no effect on disease development in the associated plots or on negative interference between plots.

The size and shape of fields can also have effects on the interference. Although Vanderplank (132) recommends large fields, Waggoner (139) found that the probability of infection in one field by inoculum from another decreases as the area increases. Fleming et al. (34) also found that decreasing and elongating field shape can retard disease progress.

MATERIALS AND METHODS

Area Description

This research was conducted under field conditions at the Horticultural Farm of the Institute of Food and Agricultural Sciences, University of Florida, Gainesville, FL. The experimental area was surrounded on the north by pine trees and grass, on the east by a soybean planting, on the south by tomato and strawberry experiments, and on the west by a road and a turf experimental area.

The closest bean planting, an experimental area for plant breeding, was about 1 Km NE and surrounded by pine plantations. In 1984, low incidence of rust was found in this breeding area (Dr. Bassett, personal communication). Therefore, interplot interference caused by incoming inoculum was considered to be minimal.

Two experiments were conducted successively in the spring and fall of 1984. The bean cultivar Sprite, considered very susceptible to bean rust (11), was planted west to east in both seasons. Although the overall design was basically the same, there were some differences between spring and fall experiments.

Spring Experiments

After the normal preparation of the land, fumigant (Telone II, 20.7 l/ha) was applied with broadcast chisels, 0.31 m apart. On the same day, fertilization was applied (363 kg of 10-10-10, and 91 kg of 15-0-15). Seventeen days later, the total area of 55 by 94 m was divided in ten plots of 12 x 20 m, which were separated from each other by a border of 6 m. A border of 4 m surrounding the plots was also delineated. The preplanting herbicide, Eptam 7-E, was applied (0.55 kg a.i./ha) and incorporated in the area of the ten plots (the interplot strips did not receive herbicide). One day later, millet cv. Pearl HyI (Production Associates, Albany, GA 31707) was planted in the border areas, with a planter drill, with 13 holes spaced 0.18 m apart. The medium density of planting was used, and 11 kg seeds were planted.

Each of the plots was divided into three subplots of 12 x 6.6 m, which were planted respectively with beans, zinnias, and sunflowers. Bean cv. Sprite Green Pod (Northrup King Co., Columbus, MS 39701) was planted twenty days after the millet. The bean subplot was divided into 12 rows, 0.55 m apart. In each row, holes were made 2.5 cm deep, 0.2 m apart, and three seeds were deposited in each hole. In the extreme west of the subplot, 1.5m were left unplanted. The herbicide Roundup (0.01 l/l water) and nitrate fertilizer were applied post-emergence. Overhead irrigation, equivalent to 0.025 m, was applied to the area. Fifteen days after planting, the bean plants in the holes were thinned, leaving one plant/hole.

Fall Experiments

In the fall season, the size of the experimental area was increased to 95 x 70 m.

The herbicide glyphosate (Roundup, Monsanto Company of Agricultural Products, St Louis, MO 63167) at 0.36 kg a.i./l was sprayed on the remaining plants from the spring experiment. Twelve days later, all plant material was burned, and the soil turned over mechanically. Seven days later, the fertilizers (181 kg 10-10-10, 136 kg 15-0-15, and 45 kg magnesium sulfate) were applied. The area was then divided into 12 plots, 19.5 x 7.3 m, with a border of 9.8 m. Two days later, the herbicide Eptam 6-E at 0.55/kg/ha was applied to the plots. The next day, pearl millet was planted (9.18 kg/ha) in the border areas. Seven days later, PCNB (Terrachlor, 24% pentachloronitrobenzene, Olin Corporation, Agricultural Division, Little Rock, AR 72205) at 2.8 l/34 l was applied to the plot area. Ten days later, the rows and holes for planting were prepared as in the spring. The holes were sprayed with PCNB 0.08 l/l water. On the next day, bean seeds were planted, 3/hole. When the seedlings started to emerge, one week later, benomyl (Benlate at 40 g/l) was sprayed on them. The sprayings, at 7-day intervals, proceeded until 10 days before inoculation. Fifteen days after the planting, chlorpyrifos (Lorsban 40%, Dow Chemical Co., Midland, MI 48640) at 20.6% a.i., was sprayed to control lesser-cornstalk borer. At the same day, the plants in the holes were thinned, leaving one plant/hole. The weed control and irrigation were conducted as in the spring.

Inoculation

In both seasons, bean plants for the inoculum sources were artificially inoculated.

In the spring, plants were grown in paper cups in the greenhouse, and inoculated when they were 15 days old. In the fall, the plants were inoculated in the field, 30 days after planting.

The inoculum was prepared according to Stavely (122, 123) and Baker et al (6): approximately 0.03 g of urediospores were placed in 50 ml of 0.001% Tween-20 in tap water in a 250 ml Erlenmeyer flask. A magnetic stirring bar was added and the mixture was stirred at top speed on a stirrer for 3 minutes, while another 50 ml were added of the Tween-water suspension to wet and disperse the spores. Urediospore concentration was determined with a hemacytometer and adjusted to 5×10^4 urediospores/ml (20). The spore suspension was sprayed on the upper and lower portions of the leaf, using a crown spray tool (No. 8011- Crown Industrial Products Co., Hebron, IL 60034). The propellant container and a 237 ml jar for the urediospore suspension were attached to a sprayer head containing the sprayer nozzle. A fine spray was released by depressing a valve on the sprayer head. To provide a standard inoculation, the valve was depressed just twice, for 2-3 sec, about 10 cm above each leaf surface.

In the spring, after inoculation, the plants were placed in a dew chamber (Percival Manufacturing Inc., Boone IA 50036) at >90% RH and 20 C, for one day (55). The plants were kept in the greenhouse until transplanted to the field,

8 days after the inoculation. Care was taken to transplant only plants whose pustules were not opened, in which only yellow flecks could be seen. As some plants died, additional inoculated plants were transplanted one week later.

In the fall, the inoculum, prepared in the laboratory, was transported to the field in a cooler. The inoculation proceeded early in the evening (1800 hr). The methodology was the same as in the spring. To ensure optimum temperature and sufficient moisture for spore germination on the leaf surfaces (17, 55, 101), the plants were covered with moistened plastic bags, which were removed early (0800 hs) on the next day.

After the transplanting of inoculated plants in the spring and the field inoculation in the fall, all agricultural practices were avoided. Only the post-emergence control of weeds was done when strictly needed, with an apparatus which did not touch the bean plants.

Sources of Inoculum

The sources of inoculum in spring and fall were the transplanted inoculated plants and the inoculated plants in situ, respectively.

Three sources of inoculum, sensu Gregory (43, 44), were utilized: area, line, and point. The area source had a total of 48 infected plants, arranged as four adjacent inoculated plants in all the rows/plot. The line source had 12 infected plants, arranged as one inoculated plant in each row/plot.

The point source had two infected plants, positioned as one inoculated plant in each of the two middle rows/plot.

In both spring and fall, three plots were used with the area source, three with the line, and two with the point source (one plot was lost). One plot, which had no inoculated plants, was used to monitor movement of inoculum in the plot area.

In the spring, the sources were located on the extreme west of their respective plot, and disease assessments were made to the east direction of the source.

In the fall the sources were located in the middle of the plots, and disease assessments were made in both west and east directions of the sources.

As suggested (30), the amount of disease on all source plants was approximately equal at the beginning (30). These plants remained in the plots for the duration of the epidemic.

Disease Assessments

Because latent periods of bean rust range from 8 to 10 days (54, 55), primary gradients were always observed at about 20 to 23 days after inoculation. The first day that symptoms appeared was considered day 1. Although Berger (12) recommends assessments at every $1/2$ latent period to quantify the course of epidemics, the frequent physical disturbance to the diseased plants could significantly alter spore dispersal (86). Therefore, disease was assessed weekly. In the spring, disease was assessed on days 1, 8, 15, 22, and 29 at

0.3, 0.7, 1.5, 2.7, 4.3, 6.3, and 8.5 m from the sources. In the fall, the assessments were on days 1, 8, 15, 22, 29, and 36 at 0.3, 0.7, 1.5, 2.7, 4.9, and 8.5 m in two directions from the sources. In both seasons, 10 to 20 plants were assessed at each distance, on each date. To reduce variation between readings, each sample was marked so that the same plants were evaluated on each assessment date (29, 91).

Three types of assessments were performed: incidence of diseased plants (INCPL), calculated as the proportion of diseased plants; incidence of diseased leaves (INCLF), calculated as the proportion of diseased leaves/plant, and disease severity (SEV), calculated as the proportion of diseased tissue/plant. In the early part of the epidemic, when the proportion of diseased tissue was low, the number of pustules per plant (Pt) were counted and the total leaf area estimated (Lt), by a standard leaf-area diagram (101). Thus, SEV was estimated by the equation : $SEV = (Pt * C / 100Lt)$ (9, 12, 101). The constant C was calculated from the average area of 100 randomly selected pustules, which were measured with an ocular micrometer (Bausch and Lomb Incorporated, Rochester, NY 14602). The constant was equal to 11.8 mm^2 . The 100 is included in the equation to transform leaf area units from cm^2 to mm^2 (101). Later in the epidemic, SEV was estimated by the Horsfall-Barratt scale (51), which is considered a natural grading system (50), and "its logarithmic basis on visual acuity provides for broad applicability, easy adaptation, and reasonable reproducibility among users" (12, p. 28).

Although yellow flecks appeared earlier, a leaf, and therefore a plant, was considered diseased only when a pustule was found. This procedure was followed to avoid mistaking rust-induced chlorotic flecks with those produced by other agents.

It is recommended (14) that experimenters should try to reduce the probable inadvertent spread of disease when they enter their plots. Thus, the noninoculated plots were always entered first for the assessments. The inoculated plots were entered from the side having the least amount of disease and sampling points were inspected in the order of expected increasing rust intensity. Exit was made by a fixed path. Additional care was taken to avoid brushing against the plants, to minimize inoculum spread by physical contact.

Statistical Analysis

Each treatment was replicated three times, except the point source that had only two replicates (one was lost). At each distance, 10 to 20 plants were sampled on each date. This number varied because some plants died in the course of the experiments. The INCLF and SEV were estimated as the average of the observed plants at each point. This average was used in the statistical analysis. The analysis was conducted using the Statistical Analysis System (SAS), 1982 version (112), 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.

Models of Disease Progress

Three models commonly used in growth description (27, 89, 128) were compared: the monomolecular, in which $y = y_{\max}[1 - a \exp(-k \cdot t)]$; the logistic, in which $y = y_{\max} / [1 + a \exp(-k \cdot t)]$; and the Gompertz, in which $y = y_{\max} \exp[-a \exp(-k \cdot t)]$. In each model, 'y' is disease proportion, 't' is time, and 'y_{max}' is the asymptote or theoretical maximum value for 'y'. The parameters 'a' and 'k' will have different values for the different curves (27, 128). The Marquardt technique, considered a good technique to fit nonlinear models (27), was used. The sums of squares of the residuals (SSR) and the R^2 , sensu Kvalseth (79): $R^2 = 1 - (\text{SSR}/\text{SST})$ (SST = corrected total sum of squares), were used to compare the fit of the models. However, the most important means to compare the models were by a ranking of the residuals (y-observed - y-predicted), by a program written in BASIC for the Apple II-Plus microcomputer.

Models of Disease Gradients

The fit of seven models were compared to describe gradient data. The models were A) the one described by Wilson and Baker, in 1946 (142, 143) and by Gregory in 1947 (44): $y = a \cdot d^{-b}$ (hereafter called the Gregory model); B) the model described by Brownlee in 1911, cited by Bateman (7), and by Kiyosawa and Shiyomi in 1972 (74): $y = a \exp(-b \cdot d)$ (hereafter referred as Kiyosawa and Shiyomi model); C) the model described by Bateman in 1947 (7) and by Lambert et al. in 1980 (80): $y = a \exp(-b \cdot d^c)$ (referred to as the model of

Lambert et al.); D) the model described by Prunty (103) and inverted for negative growth: $y = a*(-d*(d^c+1)^{1/c})$; E) the Hoerl's special function (23): $y = a*d^{-b}*exp(c*d)$; F) the model described by Bateman (7): $y = a*exp(-b*d)/d$; G) and the equation described by Daniel and Wood (23): $y = a*exp(-b/d)$. In each model, 'y' is a measure of disease intensity, 'd' is distance in meters from the source, 'a' is the disease intensity at the source, 'b' is the slope parameter, and 'c' is the shape parameter. The R^2 and SSR of all models were compared. The individual residuals of the Gregory, Kiyosawa and Shiyomi, Bateman, and Hoerl models were also studied.

Transformation Equations

For both disease progress and disease gradients, the linear regression of transformed data was also performed. The choice of which transformation to use was based on the fit of the nonlinear model (101). When two or more models gave similar fits, two or more transformations were tested.

For the disease-progress data, two transformed equations of disease intensity were compared:

logistic: $\log_e(y/(y_{\max}-y)) = \log_e(a/(y_{\max}-a))+k*t$ and
Gompertz: $-\log_e(-\log_e(y/y_{\max})) = -\log_e(-\log_e(a/y_{\max}))+k*t$.

For the gradient data, five equations were compared:

Gregory: $\log(y) = \log(a)-b*\log(d)$;
Kiyosawa and Shiyomi: $\log_e(y) = \log_e(a)-b*d$;
Lambert et al.: $\log_e(-\log_e(y/a)) = \log_e(b) + c*\log_e(d)$;
Hoerl: $\log_e(y) = \log_e(a)-b*\log_e(d)+c*d$; and
gompit transformation: $-\log_e(-\log_e(y)) = -\log_e(-\log_e(a))-b*d$.

The comparison of the fit of the linear regressions was based on the R^2 and on the CV (21). The test of the appropriateness of the chosen transformation was checked by plotting the residuals against the independent variable, either time or distance (1, 21, 89, 116).

Comparison of Slopes and Intercepts

The linear regressions of transformed disease intensity on time generates two parameters: the initial disease (intercept) and the disease progress rate (slope). The regression of transformed disease data on distance generates the disease at the source (intercept) and the gradient slope (slope).

The intercepts and slopes of the treatments were considered important parameters to compare. As correlation exists among regression lines, the "seemingly unrelated regressions procedure" developed by Zellner (149) was used in this research, to estimate and compare the slopes and intercepts of the disease progress lines from the three assessment types, three source types, two seasons, two positions in the fall, and the different distances from the source. The same procedure was used to estimate and compare the slopes and intercepts of the gradient lines from the three assessment types, three source types, two seasons, two positions in the fall, and the different assessment times. To implement this test the SYSREG (systems regression) procedure of SAS (113) was used.

Progress and Spread of Bean Rust

For the different assessment types, source types, seasons, and positions, two techniques were employed to merge the concepts of progress and spread. The approach of Berger and Luke (14, 85) was used to determine the isopathetic rates. These rates were calculated by plotting the gompit lines for the same disease assessment at 0.3 and 8.5 m from the source, against time. The interlinear distance (8.2 m) was divided by the number of days needed for disease at 0.3 m to reach the same level at 8.5 m. The averages and standard deviations of isopathetic rates from the two seasons and two directions were calculated.

Jeger (60) proposed some equations to describe the spread and progress of epidemics: $y = 1/(1+a*\exp(bd-ct))$, $y = 1-a*d^b*\exp(-ct)$, and $y = 1/(1+a*d^b*\exp(-ct))$. These equations plus $y = \exp(-b*\exp(-kt)-cd)$ were fitted to the data by nonlinear regression and the fits were compared, as discussed before. Besides the fitting, response surfaces of the data versus distance and time were also constructed (22).

RESULTS AND DISCUSSION

Disease Progress

Nonlinear Curve Fitting

The monomolecular, logistic, and Gompertz models were fitted to the disease progress data. Initially, the R^2 and the sum of squares of residuals (SSR) were used to compare the fit of the three models. However, as the number of parameters in a model increases, the R^2 increases (95) and the SSR decreases. A better way to compare the fit of nonlinear models would be to study the individual residuals. A computer program was prepared in BASIC, to calculate the residual for each point, for each model. The residuals for six points in time for the three models were compared and ranked (Table 1). Thus, suppose that at a point, the observed value was 0.3, the predicted value for the logistic model was 0.4, for the Gompertz was 0.5, and for the monomolecular was 0.6. At that point in time, the logistic was ranked 1, the Gompertz ranked 2, and the monomolecular ranked 3. The ranks of each model throughout the curve were summed. The best model was the one which gave the smallest sum of ranks. The best overall fit (considering the disease progress from the three inoculum sources, in the three

Table 1. Comparison of the Fit of the Monomolecular (M), Logistic (L), and Gompertz (G) Models for Progress of Bean Rust, Based on the Ranking of the Individual Residuals^x

Assessment Type ^y	Source Type	Spring			Fall-W			Fall-E		
		Model								
		L	G	M	L	G	M	L	G	M
INCPL	Area	63	53	65	37	45	50	45	42	45
	Line	48	44	74	42	37	56	43	41	64
	Point	62	49	103	44	44	60	40	46	69
INCLF	Area	70	46	84	53	65	85	69	53	87
	Line	63	50	82	60	66	80	57	61	81
	Point	60	50	97	56	49	70	43	46	76
SEV	Area	52	52	80	63	52	91	54	52	96
	Line	43	40	85	67	63	76	50	55	84
	Point	56	44	96	46	43	63	58	42	65

^xThe minimum $\Sigma_{\text{rank}} = 36$, i.e., all first-place ranks.

^y

INCPL = incidence of diseased plants; INCLF = incidence of diseased leaves; SEV = disease severity.

assessments, and in the two seasons) was found with the Gompertz model, using the three comparisons (R^2 , SSR, and rank of residuals).

Vanderplank (132) theorized that the simple interest (monomolecular) model is to describe epidemics of monocyclic diseases. In bean rust, typically compound interest disease, sometimes the incidence of diseased plants (INCPL) was fitted well by the monomolecular model. The goodness of fit of the monomolecular model for progress of INCPL decreased from area to point source and from 0.3 m distance to the source to 8.5 m distance. Thus, the fit for the monomolecular model to INCPL data was better where there was more initial inoculum than where there was less. In the higher initial inoculum (area source and closer to the source), all plants soon had at least one pustule. Under these conditions, the progress of INCPL was not dependent on secondary inoculum, which resulted in the monocyclic trend. In epidemics of many monocyclic diseases, INCPL was assessed (132) and the monomolecular pattern observed. Is the monocyclic pattern a general trend when INCPL is assessed in polycyclic diseases and when the source is strong? To describe epidemics, the model choice may be more dependent on the assessment type. The increase in incidence of diseased plants occurs at a very fast rate which happens in polycyclic diseases (the fungal spores are widely disseminated and most plants have at least one lesion). The process appears monocyclic not from the limitation of inoculum or environment, but from the rapid

rate at which plants have at least one lesion. The process is limited only by the number of healthy plants. The multiple infections which occur play no role in determining incidence of diseased plants. In this study, the plants at each assessment point were relatively few, and the monomolecular trend may have been an artifact because of the low number of plants. Monomolecular growth also implies very fast rates in the beginning of the epidemic, and slower rates as the disease level approaches a maximum (89).

Vanderplank (132) suggests the logistic model be used to describe compound interest (polycyclic) diseases; however, the Gompertz model gave a better fit for many epidemics (13, 15), including bean rust (102). In this study, the Gompertz also had a better fit for the progress of INCPL, INCLF, and SEV, that developed from area, line, and point sources, and during both seasons. The Gompertz model may be appropriate when the maximal rate occurs earlier than the logistic (89), which happened in the bean rust epidemics.

The use of a model to describe epidemics is more a statistical problem than just a matter of personal choice. The choice of a model should be considered very carefully in epidemiological research. With the correct choice of a model, parameters will be portrayed more reliably, the plant disease epidemics will be better understood, and the selection of control measures will be more rational.

Linear Fitting

The choice of the transformation of disease progress data is dependent upon the type of growth of the epidemic, as well as the adequate linearization (101). In nonlinear regression, the disease progress data were fitted well by both the Gompertz and logistic models. Therefore, simple linear regression was also used to test the linear fit for logits and gompits against time. In the logistic transformation, the estimated 'y' was transformed to $Y = \log_e(y/(y_{\max}-y))$ (logits), and in the Gompertz, 'y' was transformed to $Y = -\log_e(-\log_e(y/y_{\max}))$ (gompits). The 'y_{max}', the disease asymptote (61) was the maximum disease observed in each assessment: 1., 0.95, and 0.6, for INCPL, INCLF, and SEV, respectively. The gompits regressed over time gave the better fit, as assessed by the R^2 and CV (Table 2). The gompits were observed to give a better fit than logits for other pathosystems (13, 15, 85, 102). For bean rust, the supremacy of the gompit transformation was evident for assessments based on SEV and INCLF. For INCPL, neither transformation gave a very good fit. Probably for this assessment type, a monomolecular linearization would be better. However, to standardize and to facilitate comparisons of the disease data, disease proportions were transformed to gompits, and regressed on time to implement the use of Zellner's procedure (149) for comparison of parameters.

Table 2. Comparison of Statistics from Linear Regression of Logit (y) and Gompit (y) versus Time for Bean Rust Epidemics^x

Source Type		Distance from the Source (m)	Trans-formation ²	Spring										
				Assessment Type ^y										
				INCPL				SEV		INCLF				
				R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a
Area	0.3	Logit	.7497	28.68	3.5274	.3281	.9335	478.6	-2.8638	.2005	.9007	32.01	-21.46	.1234
		Gompit	.7497	28.62	3.5383	.3277	.9781	33.19	-1.230	.1331	.9299	20.66	.2102	.1062
	0.7	Logit	.7778	34.35	1.9399	.3967	.9393	52.90	-4.1815	.1978	.9455	35.42	-.767	.1211
		Gompit	.7769	34.13	1.9956	.3943	.9952	71.60	-1.5992	.0985	.9680	17.76	-1.885	.0987
	1.5	Logit	.7962	42.04	.1801	.4740	.8484	47.52	-7.3894	.2802	.8906	418.3	-2.1188	.1521
		Gompit	.7928	40.37	.5415	.4578	.9812	16.34	-2.1089	.0816	.9477	45.65	-.8609	.1022
	2.7	Logit	.9080	44.75	-3.1943	.5407	.9151	29.95	-9.2288	.3296	.8582	117.5	-3.1806	.1638
		Gompit	.8834	44.2	-2.2385	.4982	.9831	11.32	-2.3808	.0799	.9531	242.4	-1.213	.0877
	4.3	Logit	.8876	68.5	-3.7791	.452	.8539	34.78	-9.3624	.3100	.8667	83.72	-3.4507	.1644
		Gompit	.8335	67.26	-2.6060	.4006	.9797	8.8	-2.3196	.0647	.9617	370.2	-1.3018	.083
	6.3	Logit	.8629	82.26	-3.9555	.4490	.9592	13.83	-9.8918	.2904	.8029	87.64	-4.3872	.1974
		Gompit	.8005	78.8	-2.7334	.3959	.9876	5.11	-2.4112	.056	.9417	119.6	-1.5068	.0853
	8.5	Logit	.8316	103.67	-4.2202	.4465	.8138	29.28	-8.836	.2435	.9013	43.53	-3.5794	.1448
		Gompit	.7609	95.92	-2.9523	.3927	.9305	10.36	-2.1911	.0450	.979	36.94	-1.3470	.0671

Table 2. continued

Line	Source type	Distance (m) from the Source	Trans-formation ²	Spring											
				Assessment Type ^y						INCLF					
				INCPL			SEV			R ²			CV		
				R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a	b
0.3	Logit			.7879	35.89	1.4542	.4186	.9768	19.23	-3.612	.143	.9105	49.25	-.9697	.1413
	Gompit			.7865	35.5	1.5558	.4141	.9973	12.27	-1.4135	.0691	.9387	26.28	-.2932	.1138
0.7	Logit			.7963	42.96	.0218	.4807	.8495	44.72	-7.6693	.2838	.8919	254.02	-2.1526	.1437
	Gompit			.2925	41.06	.4277	.4625	.9817	14.06	-2.1357	.078	.9537	48.89	-.8637	.0935
1.5	Logit			.9063	64.51	-4.0317	.4672	.8467	35.36	-9.315	.3067	.794	97.38	-4.0761	.188
	Gompit			.8514	63.21	-2.6675	.407	.9737	9.98	-2.3169	.0643	.9179	214.7	-1.4114	.0841
2.7	Logit			.8775	85.47	-4.3874	.4661	.9307	18.75	-9.1447	.2722	.6893	109.2	-6.9982	.3062
	Gompit			.8075	80.99	-2.9442	.4035	.9969	2.60	-2.3022	.0545	.8667	106.6	-1.8615	.0945
4.3	Logit			.9698	38.77	-1.5814	.1722	.9565	12.78	-10.3366	.2846	.8343	53.53	-4.6178	.1801
	Gompit			.9405	35.17	-.8391	.1433	.9988	1.28	-2.4296	.0484	.9548	37.03	-1.5744	.070
6.3	Logit			.7549	201.9	-8.3956	.4512	.7978	40.52	-14.8741	.4817	.6999	91.01	-10.31	.4261
	Gompit			.9886	89.79	-2.2336	.1796	.9503	9.9	-2.7554	.0619	.911	45.65	-2.3176	.0976
8.5	Logit			.7461	202.9	-8.3208	.4456	.9528	34.36	-15.445	.5072	.6791	94.42	-10.171	.4185
	Gompit			.9734	88.68	-2.2427	.1777	.9774	6.89	-2.8202	.0653	.8924	48.61	-2.2696	.0941

Table 2. continued

Fall-West Position from the Source														
Source Type		Distance (m) from the Source	Trans-formation ^z	Assessment Type ^y										
				INCPL			SEV			INCLF				
				R ²	CV	a b	R ²	CV	a b	R ²	CV	a b		
Area	0.3	Logit	1	0	11.513	0	.7488	67.04	-.8271	.1414	.7028	30.76	1.3174	.1058
		Gompit	1	0	11.513	0	.7816	42.61	-.0379	.1132	.7025	28.77	1.4700	.1005
	0.7	Logit	1	0	11.513	0	.6937	344.5	-1.9986	.0937	.7722	26.06	.6508	.0542
		Gompit	1	0	11.513	0	.7685	135.4	-.6731	.0536	.7798	21.13	.8779	.0480
	1.5	Logit	.4286	30.09	6.7417	.1789	.6634	157.06	-2.3670	.0942	.6950	35.06	.4931	.0538
		Gompit	.4286	29.97	6.787	.1783	.7431	491.87	-.8092	.0483	.6998	27.43	.7630	.0464
	2.7	Logit	.4286	34.95	6.0825	.2036	.8013	88.49	-3.3406	.1249	.8448	34.68	-.033	.818
		Gompit	.4286	34.54	6.1365	.2016	.8996	281.4	-1.2197	.0603	.8681	24.24	.3514	.0697
	4.3	Logit	.4286	37.17	5.7905	.2146	.8250	51.44	-4.0379	.1277	.8205	59.46	-.5964	.0854
		Gompit	.4286	36.50	5.8782	.2130	.9012	56.24	-1.4222	.0524	.8534	33.25	-.0168	.0672
	8.5	Logit	.7138	35.86	2.3713	.3224	.8247	41.28	-4.7991	.1372	.8188	127.4	-1.1636	.0889
		Gompit	.7133	35.19	2.5013	.3176	.8815	37.15	-1.5941	.0484	.8545	46.44	-.3507	.0639

Table 2. continued

Fall-West Position from the Source													
Source Type	Distance (m) from the Source	Trans-forma-tion ²	Assessment Type ^y										
			INCPL			SEV			INCLF				
			R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a
Line 0.3	Logit Gompit	.4286	32.27	6.4432	.1901	.8268	406.8	-2.1215	.1053	.7686	32.04	.4254	.0647
		.4286	32.06	6.4721	.1890	.8734	92.64	-.8101	.0649	.7724	25.83	.6972	.0568
0.7	Logit Gompit	.7142	33.59	2.794	.3078	.793	73.73	-2.9555	.1032	.889	33.16	-.2505	.0866
		.7146	33.13	2.8839	.3045	.8716	168.3	-1.0925	.0500	.9154	21.19	.1827	.0729
1.5	Logit Gompit	.7131	36.83	2.2029	.328	.8432	56.08	-3.6907	.1240	.8442	53.65	-.6048	.0892
		.7123	36.11	2.3454	.3227	.9114	80.14	-1.3446	.0556	.8703	31.42	-.0364	.0713
2.7	Logit Gompit	.7140	38.85	1.829	.3417	.8753	36.39	-4.5978	.1365	.7658	120.09	-1.0661	.0907
		.7133	37.65	2.0527	.3334	.9384	30.96	-1.5843	.0517	.8071	49.43	-.2625	.0653
4.3	Logit Gompit	.7141	45.44	.7214	.3818	.8242	48.84	-5.8913	.1821	.6734	3475.8	-2.1113	.1123
		.7132	41.78	1.3262	.3592	.7631	.7398	-1.8623	.0631	.7365	106.09	-.6701	.0654
8.5	Logit Gompit	.8793	40.25	-2.2517	.4482	.8339	38.68	-8.1612	.2306	.8184	162.7	-3.2144	.1414
		.8683	37.4	-1.3516	.415	.949	16.34	-2.1642	.0551	.9253	153.54	-1.1983	.0759

Table 2. continued

Fall-West Position from the Source														
Source Type	Distance (m) from the Source	Assessment Type ^y												
		INCPL				SEV				INCLF				
		R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a	b	
Point	0.3	Logit	.7114	45.2	.8027	.3768	.8123	54.22	-4.8027	.1525	.8327	179.5	-1.9327	.1302
		Gompit	.7081	42.9	1.2316	.3609	.8982	51.81	-1.5953	.057	.8609	57.12	-.7416	.0905
0.7	Logit	.714	43.14	1.0964	.3675	.8303	48.15	-5.4983	.1705	.8606	228.9	-2.0703	.12	
		Gompit	.7124	40.82	1.5018	.3525	.9133	39.5	-1.7499	.0585	.9174	47.32	-.8128	.0882
1.5	Logit	.7133	44.89	.821	.3768	.8212	50.97	-6.9496	.2178	.8492	1367.8	-2.2883	.1268	
		Gompit	.7107	42.31	1.2862	.3596	.9361	30.93	-2.01	.0651	.9187	58.18	-.8929	.0811
2.7	Logit	.8757	46.02	-3.0933	.4748	.7716	54.08	-8.2504	.2483	.8295	218.4	-3.4497	.1602	
		Gompit	.8588	41.72	-1.8269	.4281	.9327	23.45	-2.1562	.0611	.9355	100.8	-1.2916	.0879
4.3	Logit	.8298	99.31	-7.5783	.6409	.6657	.7787	-12.5525	.3944	.596	.1739	-8.5412	.3358	
		Gompit	.8628	45.34	-2.5471	.4531	.9111	24.96	-2.5225	.0685	.8453	721.4	-1.9209	.0992
8.5	Logit	.913	124.9	-9.4042	.6323	.7184	60.52	-13.2029	.3924	.6511	129.3	-9.1092	.34	
		Gompit	.8869	57.7	-4.1351	.4381	.9461	15.28	-2.6397	.0638	.9105	118.8	-2.1642	.0968

Table 2. continued

Fall-East Position from the Source														
Source Type	Distance (in) from the Source	Transformation ^a	Assessment Type ^y											
			INCLP				SEV				INCLF			
			R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a	b
Area	0.3	Logit	.4286	20.71	8.0938	.1282	.6964	382.4	-1.9632	.0934	.9015	18.85	.6174	.086
		Compit	.4286	20.7	8.0953	.1282	.7647	130.1	-.6601	.0539	.908	161.2	-.8271	.0794
0.7		Logit	.4286	33.83	6.2323	.1980	.7396	101.3	-2.8664	.1059	.8891	26.31	-.0891	.0768
		Compit	.4286	33.52	6.2741	.1965	.8362	460.9	-1.0325	.0519	.9031	19.33	.4206	.0667
1.5		Logit	.4286	30.74	6.6524	.1823	.7247	61.78	-3.4244	.1034	.7945	40.72	-.0414	.0670
		Compit	.4286	30.6	6.6724	.1815	.8075	73.82	-1.2021	.0427	.8147	27.7	.3735	.0557
2.7		Logit	.4286	40.67	5.3405	.2315	.8415	32.59	-4.6225	.1216	.8206	120.28	-1.1208	.0875
		Compit	.4286	39.3	5.5153	.2249	.9086	24.64	-1.5568	.0417	.88	40.35	-.3206	.0629
4.3		Logit	.7140	45.17	.7642	.3793	.8268	35.24	-6.4999	.1733	.7349	482.5	-2.2573	.1101
		Compit	.7119	42.25	1.2777	.3602	.9296	17.96	-1.9066	.0466	.8281	106.58	-.7813	.0638
8.5		Logit	.9164	46.14	-3.9413	.4422	.7903	40.47	-9.4459	.2543	.7413	110.14	-5.1626	.1959
		Compit	.8825	45.73	-2.7073	.3970	.935	14.09	-2.2809	.0497	.9211	182.9	-1.6349	.0784

Table 2. continued

Fall-East Position from the Source													
Line	Source Type	Distance (m) from the Source	Assessment Type ^y										
			INCPL			SEV			INCLF			R ²	CV
			R ²	CV	b	a	b	a	R ²	CV	b		
0.3	Logit		.4286	34.96	6.0813	.2037	.8156	77.63	-2.9244	.1066	.8975	28.15	-.0812
	Gompit		.4286	34.56	6.1354	.2017	.879	270.4	-1.0967	.0535	.9147	20.02	-.2937
0.7	Logit		.4286	37.99	5.6837	.2186	.8595	51.80	-4.179	.1396	.8534	56.99	-.7615
	Gompit		.4286	37.19	5.7876	.2147	.9171	68.54	-1.4889	.0597	.8868	31.19	-.1327
1.5	Logit		.7083	43.40	1.1463	.3639	.8932	37.53	-5.1525	.1611	.8674	100.7	-1.386
	Gompit		.7055	41.87	1.4444	.353	.9498	33.09	-1.7361	.0606	.9076	38.09	-.4952
2.7	Logit		.7097	43.48	1.1097	.3655	.9446	19.52	-5.1519	.1408	.893	177.66	-1.5618
	Gompit		.7069	41.83	1.4292	.3538	.9804	12.53	-1.7539	.050	.9403	37.04	-.5972
4.3	Logit		.8763	42.66	-2.559	.4565	.841	37.59	-8.2689	.2346	.826	182.17	-3.2579
	Gompit		.8617	40.06	-1.6307	.4225	.9784	10.76	-2.2042	.0569	.9226	130.5	-1.2192
8.5	Logit		.9458	55.21	-6.3782	.5414	.8661	32.85	-11.056	.3069	.8030	93.56	-5.2297
	Gompit		.9178	42.23	-3.4638	.4265	.9814	7.95	-2.5089	.0572	.9593	133.9	-1.726

Table 2. continued

Fall-East Position from the Source														
Source Type	Distance from the Source (m)	Trans-formation ²	Assessment Type ^y											
			INCLP			SEV			INCLF					
			R ²	CV	a	b	R ²	CV	a	b	R ²	CV	a	b
Point	0.3	Logit	.7017	43.76	1.1979	.3611	.8922	37.08	-4.9044	.1522	.8033	215.45	-1.9123	.1263
		Gompit	.6994	42.44	1.4496	.3520	.9318	38.99	-1.6808	.0587	.8165	68.12	-.7263	.0874
0.7	0.7	Logit	.6622	39.48	2.4396	.3240	.8779	50.43	-5.6423	.1758	.9075	156.2	-2.3984	.1377
		Gompit	.6622	37.35	2.8316	.3100	.9231	52.55	-1.7785	.061	.9086	50.24	-1.00	.0928
1.5	1.5	Logit	.7086	48.99	.2638	.395	.9021	30.97	-6.5736	.1929	.8706	386.7	-2.7897	.1398
		Gompit	.7035	45.81	.8389	.3739	.9677	17.67	-1.995	.0593	.9142	83.30	-1.117	.0852
2.7	2.7	Logit	.8611	48.47	-2.963	.4651	.9027	26.81	-7.4312	.0204	.9089	92.96	-3.8204	.1627
		Gompit	.8426	45.72	-2.007	.4309	.9896	7.24	-2.1132	.0539	.9771	162.53	-1.4909	.0871
4.3	4.3	Logit	.8572	109.2	-8.8452	.6712	.7422	56.28	-13.4466	.3974	.6492	127.69	-9.0981	.3379
		Gompit	.8380	56.97	-3.4203	.4751	.9583	13.16	-2.6784	.0642	.9223	102.44	-2.1818	.0963
8.5	8.5	Logit	.9554	141.95	-10.9794	.6748	.8444	38.44	-14.786	.4243	.8309	69.19	-11.0768	.395
		Gompit	.8715	70.78	-4.768	.4517	.9878	6.27	-2.8244	.0635	.9852	30.02	-2.662	.1088

χ^2 = coefficient of determination, CV = coefficient of variation, a = intercept of transformed line: logit (a) or gompit (a), b = apparent infection rate (r) of Vanderplank for logistic model and epidemic rate (k) for Gompertz model.

y INCLP = incidence of diseased plants, INCLF = incidence of diseased leaves, SEV = disease severity.

z Logits calculated as $\log_e(y/y_{\max} - y)$;

gompits calculated as $-\log_e(-\log_e(y/y_{\max}))$.

Estimates and Comparison of Initial Disease and Disease Progress Rates

Initial disease (y_0) and disease progress rate (k) were estimated from the bean rust data transformed to gompits (Table 3) by Zellner's procedure, through the SYSREG technique of SAS. The estimated y_0 's and k 's were also compared by Zellner's procedure.

Disease progress parameters estimated for the three assessment types were compared among all three inoculum sources, all distances from the sources, both seasons, and both directions from the source in fall. Overall, the y_0 's of the INCPL were higher than the y_0 's from INCLF, which were higher than the y_0 's of SEV. When disease was first detected in the fields, INCPL had reached a level much higher than in SEV (just because of the kind of assessment). Although not statistically compared, the observed ' $y_{\max}$ ' from INCPL (1.0) was higher than ' $y_{\max}$ ' from SEV (0.6). Imhoff et al (54) observed severity values of 0.95-0.97 in bean rust, but these values were for the total damage to bean plants, which was more than the damage due to rust alone. Overall, faster rates (k) were estimated for INCPL than for SEV and INCLF. This tendency was most typical at the point source. At the other two sources, INCPL reached 1.0 earlier, and since no more increase could occur, the average progress rate was reduced. Overall, the rates of progress of INCLF and SEV were similar. The correlation between disease severity and incidence of diseased leaves of bean rust is low (54), as in other pathosystems (118). However the k 's of both assessment

Table 3. Estimated Initial Disease (y_0) and Disease Progress Rate (k) of Bean Rust Epidemics^x

Dis- tance (m) from the Source	Assess- ment Type ^y	Spring					
		Source Type					
		Area		Line		Point	
		y_0^z	k	y_0	k	y_0	k
0.3	INCPL	3.435	.292	2.585	.371	1.463	.437
	SEV	-1.23	.133	-1.414	.069	-1.669	.064
	INCLF	.210	.106	-.293	.114	-1.011	.092
0.7	INCPL	1.917	.745	.45	.424	-2.841	.495
	SEV	-1.599	.099	-2.136	.078	-2.495	.092
	INCLF	-.189	.099	-.864	.093	-1.580	.126
1.5	INCPL	.586	.403	-2.477	.383	-4.164	.448
	SEV	-2.109	.082	-2.317	.064	-2.752	.096
	INCLF	-.861	.102	-1.411	.084	-2.461	.142
2.7	INCPL	-1.941	.445	-2.764	.381	-4.025	.435
	SEV	-2.381	.080	-2.302	.055	-2.934	.097
	INCLF	-1.213	.088	-1.862	.094	-2.645	.157
4.3	INCPL	-2.333	.366	-.839	.143	-4.293	.442
	SEV	-2.320	.065	-2.42	.048	-2.967	.095
	INCLF	-1.302	.083	-1.574	.070	-2.464	.146
6.3	INCPL	-2.468	.363	-2.234	.180	-4.314	.440
	SEV	-2.411	.056	-2.755	.062	-2.992	.098
	INCLF	-1.507	.085	-2.318	.096	-2.705	.156
8.7	INCPL	-2.70	.360	-2.243	.178	-4.573	.469
	SEV	-2.191	.045	-2.82	.065	-2.973	.099
	INCLF	-1.347	.067	-2.27	.094	-2.738	.146

Table 3. continued

		Fall-West Direction from the Source					
Dis- tance (m) from the Source	Assess- ment Type ^y	Source Type					
		Area		Line		Point	
		y_0^z	k	y_0	k	y_0	k
0.3	INCPL	11.556	.023	5.966	.167	1.172	.331
	SEV	-.038	.113	-.81	.065	-1.595	.057
	INCLF	1.47	.101	.697	.057	-.742	.09
0.7	INCPL	11.519	-.0012	2.814	.27	1.443	.324
	SEV	-.673	.054	-1.093	.05	-1.75	.058
	INCLF	.878	.048	.183	.073	-.813	.088
1.5	INCPL	6.198	.154	2.278	.289	1.229	.333
	SEV	-.809	.048	-1.345	.056	-2.01	.065
	INCLF	.763	.046	-.036	.071	-.893	.081
2.7	INCPL	5.592	.178	1.987	.3	-1.685	.399
	SEV	-1.22	.060	-1.584	.052	-2.156	.061
	INCLF	.351	.070	-.262	.065	-1.292	.088
4.3	INCPL	5.349	.188	1.262	.328	-2.411	.425
	SEV	-1.422	.052	-1.862	.063	-2.522	.069
	INCLF	-.017	.067	-.67	.065	-1.921	.099
8.7	INCPL	2.429	.281	-1.186	.381	-3.937	.414
	SEV	-1.594	.048	-2.164	.055	-2.64	.064
	INCLF	-.351	.064	-1.198	.076	-2.164	.097

Table 3. continued

Dis- tance (m) from the Source	Assess- ment Type ^y	Fall-East Direction from the Source					
		Source Type					
		Area		Line		Point	
		y_0^z	k	y_0	k	y_0	k
0.3	INCPL	9.168	.116	5.639	.180	1.391	.323
	SEV	-.66	.054	-1.097	.054	-1.681	.059
	INCLF	.827	.079	.294	.077	-.726	.082
0.7	INCPL	6.195	.156	5.307	.194	1.718	.315
	SEV	-1.033	.052	-1.489	.06	-1.811	.062
	INCLF	.421	.067	-.133	.077	-1.169	.098
1.5	INCPL	6.12	.158	1.378	.320	.785	.348
	SEV	-1.202	.043	-1.736	.061	-1.995	.059
	INCLF	.373	.057	-.495	.079	-1.117	.085
2.7	INCPL	4.977	.201	1.365	.322	-1.868	.402
	SEV	-1.557	.042	-1.754	.05	-2.113	.054
	INCLF	-.321	.063	-.597	.07	-1.491	.087
4.3	INCPL	1.204	.323	-1.462	.388	-3.287	.448
	SEV	-1.907	.047	-2.204	.057	-2.678	.064
	INCLF	-.781	.064	-1.219	.08	-2.182	.096
8.7	INCPL	-2.406	.361	-3.214	.396	-4.573	.428
	SEV	-2.281	.05	-2.509	.057	-2.824	.063
	INCLF	-1.635	.078	-1.726	.083	-2.662	.109

^xEstimated by Zellner's Procedure based on the transformation with the Gompertz model: $-\log_e(-\log_e(y)) = -\log_e(-\log_e(y_0)) + k*t$ (t = time and ranged from 1 to 36 days).

^yINCPL = incidence of diseased plants, INCLF = incidence of diseased leaves, and SEV = disease severity.

^z y_0 expressed as gompits.

types might be correlated. INCPL is primarily an alloinfection process, while INCLF and SEV are predominantly autoinfection processes (107). Therefore, the epidemic rates calculated for INCLF and SEV might be expected to be similar. More studies to compare these three assessment types are needed to define the relationship among them.

The y_0 's and k 's estimated from the progress observed in the three assessment types, in the three inoculum sources, in the two seasons, and in both directions in fall were also compared along the several distances from the source of inoculum. As distance from the inoculum source increased, the occurrence of initial infections was progressively delayed and the estimated y_0 's became lower. This trend was also observed by Emge and Shrum with wheat rust (29). The delay, and the smaller y_0 farther from the source is probably due to the inoculum gradient. From ecological theory, local increase in population density decreases the growth rate of individuals which remain close to the parental site (83). Therefore in soybean downy mildew, higher epidemic rates were observed farther from the source (84). However, as an overall trend, the progress rates of SEV and INCLF of bean rust estimated closer and farther from the source did not differ. Similar results were reported by Emge and Shrum (29) with wheat rust and by Luke and Berger (85) with oat rust. Seemingly, the rusts are limited in their epidemic rate as distance from the source increases. As observed, because of the inoculum gradient, there is a time delay to start the epidemic farther from the source. Although, far from the

source there is more tissue to be colonized, the tissue may no longer be as susceptible as when the epidemic began at the source. This is because bean leaves (136, 150), as well as cereal leaves, become more resistant to rust with age. A good test to prove this theory would be to study disease progress at increasing distances from an inoculum source, in a pathosystem in which the host susceptibility is not altered with age. In most of the cases that INCPL was assessed, 'k' increased with distance from the inoculum source. For this assessment, just one pustule on one leaflet would be enough for a plant to be considered diseased. Considering the size of the plot, the inoculum soon reached all points. Therefore, the change of resistance of the host was not a limiting factor for the rate of progress of INCPL.

The epidemics originated from the three source types were compared, for the three assessment types, the two seasons, two directions, and at all distances from the source. The y_0 's from epidemics originating from point sources were always smaller than from the area sources. This is logical, since the strength of the area sources was higher. The y_0 's that developed from the line sources were intermediate. The comparison of epidemics that result from inoculum sources of different strengths has an important practical application in sanitation theory. Vanderplank (132) proposed that if initial inoculum is reduced, the final disease is reduced in the same proportion. However, as experimentally observed (102, 110), as ' y_0 ' decreases, 'k' increases. This trend was also observed in the bean-rust pathosystem, typically when

the k 's estimated from epidemics that resulted from point and area sources were compared. The faster rates were observed for epidemics from the point source. The k 's from epidemics from the line source were somewhat intermediate.

The y_0 's and the k 's estimated in all assessment types, from the three source types, and at all distances from the sources were compared between epidemics from the spring and fall. The y_0 's were statistically higher in fall than spring. This difference probably occurred because the epidemics were begun differently in the two seasons. The inoculation of bean plants in situ seems to be more efficient than transplanting rust-diseased plants. However, the disease progress rates of spring and fall were similar, overall. It seems contradictory that ' y_0 ' was higher in fall, but the rates were similar between the two seasons. Besides the difference in initial disease, other variables, weather for instance, also affected the epidemic progress.

In the fall, epidemics were generated in two directions from the source. The y_0 's and k 's estimated at both directions were compared in the three assessments, three source types, and six distances from the source. In half of the comparisons, differences in y_0 's were detected; the estimated y_0 's in the west-direction (predominantly downwind direction) epidemics were higher. However, in the other half of comparisons, no differences were detected between the y_0 's. The level of infection was expected to be delayed in the predominantly upwind direction (29), but the results of bean rust were not conclusive. Overall, no statistical

difference between the k's estimated in the epidemics which developed in the two directions from the source was detected. This similarity in epidemic rates of upwind and downwind directions also has been found for wheat rust (29).

Disease Spread

Nonlinear Fitting

The seven models tested to describe the disease gradients can be separated into three families: i). power law , in which $Y = a * d^{f(b)}$ (the models of Gregory (44) , and Prunty (103)); ii). exponential law, in which $Y = a * \exp[f(d)*b]$ (the models of Kiyosawa and Shiyomi (74) , Bateman (7), and Daniel and Wood (23)); iii). mixed power and exponential (the models of Lambert et al. (80), and Hoerl (23). These models were compared to describe spread of bean rust in three assessment types, from three inoculum sources, two seasons, two directions in fall, and at six times.

Of the seven models, four produced a good fit in terms of R^2 and SSR: Gregory, Kiyosawa and Shiyomi, Lambert et al., and Hoerl. When the R^2 and SSR of these four models were compared, the models of Lambert et al. and Hoerl gave the best fit, whereas the model of Gregory gave the poorest fit. The differences between the fit of the models were small. Since the R^2 is not a good parameter to compare nonlinear models (95), other techniques were employed. The ranking of residuals of the four models were compared using a program written by me in BASIC, as done for the models of disease

progress (Table 4). Overall, the models of Lambert et al. and Hoerl provided the best fit.

The methods to choose the best model for gradient analysis are not well defined and there are examples in the literature in which the researcher used a model just as a matter of personal preference (69, 75). In the cases where the Gregory and Kiyosawa and Shiyomi models were compared, sometimes the Gregory model fit the data better (69, 87) and in others the model of Kiyosawa and Shiyomi was better (74, 114). When both models were compared to describe disease gradients of bean rust, a very definite tendency was found: the model of Gregory gave the best fit for severity data, while the model of Kiyosawa and Shiyomi was the best for incidence of both diseased plants and diseased leaves. This tendency was not observed when other authors compared these two models. A good approach would be to compare both models for epidemics of other pathosystems, when assessed by incidence and severity.

The models of Gregory and of Kiyosawa and Shiyomi have two parameters, whereas the models of Hoerl and of Lambert et al. have a third parameter. It was not surprising that the three-parameter models gave a better fit to the data (52); i.e., the third parameter increased the precision of the model. Hoerl's model has been used in statistics (23) to describe nonlinear trends, and its use in this work is the first time it has been applied to plant disease gradients. Hoerl's model fit the gradients as well as the model of

Table 4. Comparison of the Fit of the Models of Gregory (G), Kiyosawa and Shiyomi (K), Hoerl (H), and Lambert et al. (L) to Gradients of Bean Rust, Based on the Ranking of the Individual Residuals^x

Assess- ment Type ^y	Source Type	Spring				Fall-W				Fall-E			
		Model											
		G	K	L	H	G	K	L	H	G	K	L	H
INCPL	Area	88	64	81	65	53	48	125	49	75	56	109	64
	Line	93	82	81	74	73	51	122	59	79	52	118	61
	Point	75	78	65	63	76	64	95	86	62	54	102	65
INCLF	Area	85	98	69	92	93	84	78	91	115	79	57	87
	Line	75	105	77	68	97	86	69	83	111	90	64	71
	Point	84	76	71	93	114	74	59	86	100	78	62	83
SEV	Area	78	83	63	60	71	119	81	68	94	95	56	66
	Line	72	80	61	69	74	117	68	66	85	84	56	52
	Point	69	86	48	91	92	78	60	83	89	61	62	76

^xThe minimum $\Sigma_{\text{rank}} = 42$, i.e., all first-place ranks.

^y INCLP = incidence of diseased plants; INCLF = incidence of diseased leaves; SEV = disease severity.

Lambert et al., but it was much better than the model of Lambert et al. to describe INCPL data, because the model of Lambert et al. seldom estimated $INCPL = 1.0$. An intensity of 1.0 was observed progressively farther from the source as the disease progressed in time. Therefore in the situations in which the maximal disease intensity of 1.0 is reached, Hoerl's model would likely be a better choice.

Another advantage of the model of Hoerl is that the 'c' parameter (which is usually negative), becomes positive when gradients are U-shaped (disease increases more than expected farther from the source, because of background contamination or gradients from two distinct inoculum sources). More research is needed to test the suitability of this model to describe disease gradients.

The models of Hoerl and of Lambert et al. gave the best overall fit for gradients observed for the three source types, two seasons, and two directions in the fall. The model of Kiyosawa and Shiyomi was better than the model of Gregory under the same conditions. Therefore with the bean rust data, the type of assessment was the most important determining factor in the choice of the model.

Disease spread has received less attention by researchers than disease progress. Therefore, the most suitable models for different types of diseases and for different types of assessments need to be examined. With a more complete knowledge of gradients, we will be able to determine the distance to isolate fields in space against diseases and

execute other sanitation measures, and even to conduct field experiments with plant diseases to avoid the common interplot interference.

Linear Fitting

As mentioned earlier, researchers have used nonlinear models to describe disease gradients without testing their suitability first. Similarly, the suitability of linearization models to regress disease on distance has been examined infrequently. As in disease progress, the choice of the transformation should be based on the fit of the nonlinear model. Therefore, the linear transformations of the four gradient models used in my study were compared. In addition, a fifth gradient model was used based on a gompit transformation of the disease proportions against linear distance, rather than $\log_{10}(d)$, as Danos et al. (25) used.

As the fit of the nonlinear models did not vary between different seasons and directions, the linear regressions were implemented only with the data for the fall experiment that were west from the inoculum sources (Table 5). The linear equations compared were

- i). Gregory: $\log_{10}(Y) = \log_{10}(a) - b * \log_{10}(d)$;
- ii). Kiyosawa and Shiyomi: $\log_e(y) = \log_e(a) - b * d$;
- iii). Lambert et al.: $\log_e(-\log_e(y/a)) = \log_e(b) + c * \log_e(d)$;
- iv). Hoerl: $\log_e(y) = \log_e(a) - b * \log_e(d) - c * d$;
- v). Gompit: $-\log_e(-\log_e(y)) = -\log_e(-\log_e(a)) - b * d$.

Table 5. Comparison of Five Linear Models for Disease Gradients of Bean Rust Based on Regression Statistics, West from Source, Fall 1984^x

Assessment Type	Source Type	1					8				
		Time (Days)									
		R ²	CV	a	b	c	R ²	CV	a	b	c
INCL AREA	GR	.8905	39.6	-.0376	-.1362	--	.378	215.7	-.0007	-.0068	--
	KS	.9514	26.4	.0044	-.0551	--	.7304	141.9	.0059	-.0037	--
	LA	.8219	49.6	-7.1482	3.7505	--	.3775	28.8	-11.136	1.6340	--
	H0	.9715	23.4	-.0203	-.0483	-.0379	.873	112.5	.0110	.0099	-.0072
	G0	.5172	81.7	8.5488	-1.1633	--	.7304	18.9	12.943	-.8887	--
LINE	GR	.8277	44.9	-.2524	-.7146	--	.6188	92.8	-.019	-.0753	--
	KS	.9706	18.6	-.0517	-.3079	--	.9030	46.8	.0238	-.0362	--
	LA	.9824	35.9	-1.0465	.9408	--	.7258	52.2	-5.6787	2.5472	--
	H0	.9720	20.9	-.0877	-.0687	-.2831	.9358	43.9	.0455	-.0414	-.0511
	G0	.8155	116.2	1.5444	-.3410	--	.4124	76.4	6.5326	-.7640	--
POINT	GR	.7283	58.1	-1.3069	-3.4713	--	.5951	69.7	-.1122	-.2463	--
	KS	.8460	43.7	-.4587	-1.4885	--	.8788	38.1	-.0357	-.1191	--
	LA	.8151	43.6	.7073	.7675	--	.6571	47.1	-1.6057	.5888	--
	H0	.8480	50.	-.6817	-.4247	-1.3351	.9148	36.9	.0403	.1446	-1.713
	G0	.8504	39.2	-.1968	-.3119	--	.8234	33.8	2.0686	-.2622	--

Table 5. continued

Assessment Type\ Source		Time (Days)									
		15					22				
Type	Model	R ²	CV	a	b	c	R ²	CV	a	b	c
INCL AREA	GR	1	0	0	0	--	1	0	0	0	--
	KS	1	0	0	0	--	1	0	0	0	--
	LA	1	0	-11.513	0	--	1	0	-11.513	0	--
	H0	1	0	0	0	0	1	0	0	0	0
LINE	G0	1	0	11.513	0	--	1	0	11.513	0	--
	GR	.3717	216.8	-.00083	-.00804	--	1	0	0	0	--
	KS	.7182	145.2	.00709	-.00445	--	1	0	0	0	--
	LA	.3717	29.6	-11.1187	1.6627	--	1	0	-11.513	0	--
POINT	H0	.8634	116.7	.0134	.0120	-.0088	1	0	0	0	0
	G0	.7182	19.8	-12.9009	-.9196	--	1	0	11.513	0	--
	GR	.5571	129.7	-.0242	-.1549	--	.3717	216.8	-.0011	-.0103	--
	KS	.8846	66.2	.0928	-.0777	--	.7182	145.2	.0091	-.0057	--
	LA	.7948	42.2	-8.8933	3.9242	--	.3717	30.6	-11.107	1.7119	--
	H0	.9503	50.1	.1595	.1269	-.1235	.8634	116.7	.0171	.0153	-.0112
	G0	.7417	47.3	11.2358	-1.5083	--	.7182	20.5	13.0243	-.9468	--

Table 5. continued

		Time (Days)										
		29					36					
Assessment Type	Source Type	Model	R ²	CV	a	b	c	R ²	CV	a	b	c
INCL AREA	GR		1	0	0	0	--	1	0	0	0	--
	KS		1	0	0	0	--	1	0	0	0	--
	LA		1	0	-11.513	0	--	1	0	-11.513	0	--
	H0		1	0	0	0	0	1	0	0	0	0
LINE	G0		1	0	11.513	0	--	1	0	11.513	0	--
	GR		1	0	0	0	--	1	0	0	0	--
	KS		1	0	0	0	--	1	0	0	0	--
	LA		1	0	-11.513	0	--	1	0	-11.513	0	--
POINT	H0		1	0	0	0	0	1	0	0	0	0
	G0		1	0	11.513	0	--	1	0	11.513	0	--
	GR		1	0	0	0	--	1	0	0	0	--
	KS		1	0	0	0	--	1	0	0	0	--
	LA		1	0	-11.513	0	--	1	0	-11.513	0	--
	H0		1	0	0	0	--	1	0	0	0	--
	G0		1	0	11.513	0	--	1	0	11.513	0	--
			1	0	0	0	--	1	0	0	0	--

Table 5. continued

Assessment Type		1					8				
		CV	a	b	c	R ²	CV	a	b	c	
SEV	AREA										
	GR	.9933	2.48	-1.7487	-1.002	--	.9278	14.4	-.8546	-.9333	--
	KS	.8407	12.1	-3.4967	-.3604	--	.9595	10.8	-1.3635	-.3711	--
	LA	.9833	2.8	1.3603	.2305	--	.9819	9.1	.5916	.3941	--
	H0	.9944	2.6	-3.9729	-.9309	-.0306	.9916	5.7	-1.5732	-.4098	-.2259
	G0	.7479	10.8	-1.2520	-.0786	--	.852	25.8	-.3776	-.1435	--
LINE											
	GR	.8561	17.2	-2.1839	-1.8527	--	.9610	8.9	-1.380	-1.2522	--
	KS	.9900	4.5	-3.6729	-.7927	--	.9411	10.9	-2.3937	-.4930	--
	LA	.9495	5.5	1.5704	.3017	--	.9977	1.8	1.0935	.3387	--
	H0	.9928	4.5	-3.8042	-.2499	-.7024	.9975	2.6	-2.7737	-.7236	-.2317
	G0	.9468	5.7	-1.3782	-.1199	--	.8475	14.2	-.9099	-.1242	--
POINT											
	GR	.8642	18.6	-4.1149	-4.0025	--	.9605	4.4	-2.1100	-.8437	--
	KS	.8658	18.5	-6.9132	-1.5940	--	.9112	6.7	-4.3465	-.3270	--
	LA	.9261	5.71	2.1816	.3477	--	.9767	2.1	1.5667	.1605	--
	H0	.9068	17.8	-8.0045	-2.0780	-.8434	.9844	3.2	-4.6383	-.5555	-.1263
	G0	.8459	8.2	-1.9785	-.1322	--	.8537	5.2	-1.4771	-.0597	--

Table 5. continued

Assessment Type	Source Type	Time (Days)										
		15					22					
		Model ^z	R ²	CV	a	b	c	R ²	CV	a	b	c
SEV	AREA	GR	.9735	7.4	-.5707	-.5214	--	.9591	7.6	-.4893	-.3559	--
		KS	.8738	16.1	-1.0209	-.1932	--	.8994	12.	-.9173	-.1348	--
		LA	.9675	22.3	.1940	.3569	--	.9606	33.7	.0688	.2863	--
		H0	.9828	6.9	-1.2317	-.4122	-.0472	.9799	6.2	-1.0419	-.2436	-.0485
		G0	.7366	63.7	-.0260	-.1218	--	.7667	81.9	.0733	-.1100	--
LINE		GR	.9059	13.9	-.8504	-.7483	--	.9482	8.7	-.6562	-.4851	--
		KS	.9721	7.6	-1.4471	-.3084	--	.9187	10.9	-1.2105	-.1190	--
		LA	.9758	8.6	.6196	.3192	--	.9660	13.8	.3662	.2846	--
		H0	.9904	5.1	-1.8802	-.2536	-.2168	.9798	6.3	-1.3647	-.2936	-.0839
		G0	.9050	17.1	-.4303	-.1223	--	.8180	32.03	-.2124	-.1042	--
POINT		GR	.8900	12.3	-1.3051	-.8855	--	.7856	19.3	-.9026	-.6579	--
		KS	.9657	6.8	-2.3939	-.3670	--	.9954	2.8	-1.5560	-.2946	--
		LA	.9504	6.6	1.0688	.2514	--	.8534	17.1	.7013	.2540	--
		H0	.9806	5.9	-2.5375	-.2735	-.2682	.9976	2.4	-1.5128	.0822	-.3243
		G0	.9207	8.3	-.9131	-.0984	--	.9875	5.1	-.5154	-.1089	--

Table 5. continued

Assessment Type		29					36					
		Source Type	Model ²	R ²	CV	a	b	c	R ²	CV	a	b
SEV	AREA	GR	.9368	8.10	-.4651	-.2765	--	.92	9.8	-.4660	-.2963	--
		KS	.8259	13.5	-.9183	-.1015	--	.8756	12.2	-.8961	-.1130	--
		LA	.9164	58.7	.0288	.2418	--	.9216	56.9	.0294	.2527	--
		H0	.9425	8.9	-1.0362	-.2305	-.0199	.9449	9.3	-.9943	-.1920	-.0450
		G0	.7031	110.6	.086	-.0828	--	.752	101.3	.0991	-.0893	--
LINE		GR	.4660	27.5	-.5832	-.2791	--	.9576	6.4	-.5779	-.3353	--
		KS	.5705	24.7	-1.1279	-.1229	--	.9001	9.8	-1.1290	-.1293	--
		LA	.4609	70.07	.2662	.1719	--	.9738	13.2	.2569	.2270	--
		H0	.5705	28.8	-1.1242	.0071	-.1254	.9783	5.3	-1.2490	-.2286	-.0468
		G0	.4862	68.4	-.1507	-.0702	--	.8301	33.6	-.1333	-.0834	--
POINT		GR	.9597	5.4	-.8198	-.4056	--	.7849	8.1	-.7148	-.1954	--
		KS	.9794	6.7	-1.6338	-.1596	--	.9501	3.9	-1.4968	-.0855	--
		LA	.986	4.4	.6163	.1932	--	.7942	13.7	.4926	.1080	--
		H0	.9960	2.0	-1.7572	-.2349	-.0748	.9501	4.5	-1.4975	-.0012	-.0851
		G0	.8903	12.3	-.5047	-.0730	--	.9318	7.9	-.4125	-.0465	--

Table 5. continued

Assessment Type	Source Type	Time (Days)										
		1						8				
		Model	R ²	CV	a	b	c	R ²	CV	a	b	c
INCLF	AREA	GR	.8907	19.89	-.3455	-.4281	--	.8441	23.8	-.1667	-.2001	--
		KS	.9775	9.02	-.5022	-.1754	--	.9807	8.4	-.2393	-.0843	--
		LA	.9692	151.4	-.3275	.4232	--	.9483	16.8	-1.0506	.4141	--
		H0	.9904	6.8	-.5644	-.1216	-.1323	.9833	9	-.2527	-.0262	-.0751
		G0	.9042	267.1	.5703	-.1598	--	.885	25.1	-1.2930	-.1564	--
LINE		GR	.8870	24.8	-.6724	-1.1009	--	.7985	28.1	-.2952	-.3600	--
		KS	.9627	14.2	-.7883	-.4561	--	.9929	5.3	-.3987	-.1597	--
		LA	.9807	18.1	.2665	.5549	--	.9306	66.3	-.4644	.3982	--
		H0	.9775	12.8	-.9666	-.3395	-.3370	.9936	5.8	-.3854	.0253	-.1689
		G0	.8629	48.3	.0456	-.2071	--	.9697	43.8	.7290	-.1617	--
POINT		GR	.7254	46.4	-2.0162	-3.7272	--	.7185	20.6	-.6621	-.4194	--
		KS	.8440	34.9	-1.9001	-1.5995	--	.9690	6.7	-1.1730	-.1939	--
		LA	.8085	22.4	1.3421	.5532	--	.7530	33.3	.3971	.2215	--
		H0	.8458	40.2	-2.1313	-.4403	-1.4405	.9821	5.9	-1.1044	.1306	-.2410
		G0	.9635	18.9	-.9658	-.2275	--	.9545	14.3	-.2212	-.0992	--

Table 5. continued

		15					22					
Assessment		Source					Time (Days)					
Type	Type	Model ²	R ²	CV	a	b	c	R ²	CV	a	b	c
INCLF AREA	GR		.9082	16.8	-.0709	-.080	--	.8381	20.9	-.0655	-.0645	--
	KS		.9021	17.4	-.1135	-.0312	--	.9471	20.3	-.1099	-.0254	--
	LA		.8914	12.9	-1.9475	.4462	--	.8249	14.7	-2.0165	.4041	--
	H0		.9498	14.3	-.1357	-.0433	-.0158	.8843	20.4	-.1263	-.0321	-.0140
	G0		.6825	22.11	2.1589	-.1527	--	.6234	21.5	2.2051	-.1374	--
LINE	GR		.7147	34.9	-.1188	-.1428	--	.8428	23.3	-.0976	-.1138	--
	KS		.9412	15.9	-.1563	-.0652	--	.9873	6.6	-.1406	-.0490	--
	LA		.8839	16.9	-1.373	.3860	--	.9226	11.8	-1.5719	.3917	--
	H0		.9483	17.2	-.1384	.0340	-.0775	.9888	7.2	-.1465	-.0112	-.0449
	G0		.9412	12.1	1.6349	-.1585	--	.9332	10.9	1.8249	-.1567	--
POINT	GR		.7859	30.7	-.2720	-.3543	--	.8825	18.0	-.1370	-.1421	--
	KS		.9816	8.9	-.3486	-.1576	--	.9913	4.9	-.2142	-.060	--
	LA		.9221	55.2	-.5575	.4206	--	.9716	8.6	-1.2209	.3637	--
	H0		.9825	10.1	-.3331	.0295	-.1682	.9985	2.4	-.2303	-.0306	-.0489
	G0		.9552	41.8	.8355	-.1703	--	.9254	13.9	1.4424	-.1412	--

Table 5. continued

		Time (Days)											
		29						36					
Assess- ment Type	Source Type	Model	z	R ²	CV	a	b	c	R ²	CV	a	b	c
INCLF AREA	GR	.7691			21.12	-.0598	-.0456	--	.7459	21.9	-.0593	-.0438	--
	KS	.7295			22.9	-.1107	-.0174	--	.7089	23.4	-.1105	-.0167	--
	LA	.7426			14.2	-2.0746	.3239	--	.7243	14.3	-2.0816	.3152	--
	H0	.7889			23.3	-.1261	-.030	-.0068	.7657	24.3	-.1251	-.0285	-.0066
	G0	.5553			18.6	2.2239	-.1095	--	.5418	18.5	2.2270	-.1066	--
LINE	GR	.8358			21.7	-.0899	-.0925	--	.8330	18.5	-.0800	-.0665	--
	KS	.9728			8.9	-.1388	-.0397	--	.8023	20.1	-.1433	-.0260	--
	LA	.8845			12.3	-1.6391	.3500	--	.8617	10.8	-1.7465	.3040	--
	H0	.9748			9.8	-.1445	-.0108	-.0358	.8586	19.6	-.1650	-.0413	-.0111
	G0	.9298			9.6	1.8736	-.1428	--	.7888	13.3	1.9245	-.1157	--
POINT	GR	.9208			14.7	-.1283	-.1354	--	.8130	16.4	-.102	-.0668	--
	KS	.9591			10.6	-.2055	-.0550	--	.9743	6.1	-.1825	-.0291	--
	LA	.9798			7.1	-1.2941	.3768	--	.8535	10.2	-1.4844	.2380	--
	H0	.9873			6.8	-.2352	-.0566	-.0345	.9744	7.0	-.1838	-.0023	-.0282
	G0	.8848			16.8	1.5118	-.1429	--	.9433	6.4	1.6515	-.0995	--

x²r² = coefficient of determination, CV = coefficient of variation, a = intercept (disease at the source), b and c = gradient slopes.

yINCLP = incidence of diseased plants, INCLF = incidence of diseased leaves, SEV = disease severity.

zGR = Gregory; KS = Kiyosawa and Shiyomi; LA = Lambert et al.; H0 = Hoerl; G0 = Gompertz

The comparison among models was based upon the regression which yielded the largest R^2 and smallest CV. Using this principle, the best overall fit was given by the model of Hoerl, which yielded one intercept and two slope components. The linear fit of the model of Lambert et al. (one of the best in nonlinear fitting) was not as good. This is probably because the 'a' parameter estimated in the nonlinear regression was supposed to be included in the linear equation (equation iii) to estimate the 'b' and 'c' parameters. Therefore, 162 'a' values would have been needed to implement the linear regression of the model of Lambert et al. for the data set collected. However, this procedure was not followed here. Nevertheless, the linearization equation of the model of Lambert et al. indicates that a type of gompit transformation sometimes could be adequate to describe disease gradients, as done by Danos (24). The gompit transformation (equation v) of the data (regressed against linear distance) did not give a good fit for the gradients of bean rust. The linearization of the model of Gregory gave the worst fit. A good fit was observed when the linearized Kiyosawa and Shiyomi model (equation ii) was used. This linearization only transforms the disease intensity data (less manipulations), and it has just two parameters (simpler interpretation). Therefore, equation (ii) was chosen to generate and compare parameters by Zellner's procedure.

As in disease progress, the fit of gradients is of capital importance. The main objective of transforming gradient data and regressing them on distance is to estimate the gradient parameters and to facilitate the comparison of these parameters. Therefore the choice of the transformation, which is the scale to represent the data, is essential in the study of gradients. In the linear equations of Gregory, Kiyosawa and Shiyomi, and Hoerl, the slopes increased over time (Table 5). In the equations involving the gompits, the slopes remained unaltered. However, when Danos (24), regressed gompit (y) on $\log(d)$, an increase of slopes over time was observed. The author did not compare the fit of this transformation with others. It seems that whether the slopes change in time is a function of the linearization model chosen. Therefore, more studies are needed to determine the suitability of the transformations and to choose a more reliable one. For bean rust data, the linear equation of Kiyosawa and Shiyomi was considered adequate because its statistical fit was good. The nonlinear fit of the same model to the bean rust data was also good. Moreover, in the nonlinear model, fitted with untransformed data, the slopes also increased in time.

Estimates and Comparison of Disease at the Source and Gradient Slopes

Disease at the source (a) and gradient slope (b) for the bean rust data transformed to $\log_e(y)$ were estimated and compared by Zellner's procedure (149), using the SYSREG

technique of SAS (Table 6). The a's and b's were also compared by Zellner's procedure.

The parameters estimated from the gradients of three types of assessments, observed for three source types, in two seasons, and in two directions in fall were compared over six assessment times. In all comparisons, 'a' increased over time. The increase of 'a' was expected since it is a measure of disease intensity at the source the epidemics progressed. The b's for all sources and all assessments also increased over time. The increase in slope is the flattening, which is a debatable point in gradient theory (24, 44, 76, 85, 86, 147). Gregory (44) was one of the first to observe that secondary spread flattens the primary gradient. According to Zadoks and Schein (147), it is difficult to determine which part of the flattening of gradients is due to the enlargement of the primary focus, which to secondary spread, and which to the unavoidable background contamination. Other researchers have discarded the possibility of background contamination under their experimental conditions (90). Background contamination likely was not important in this research. Rust incidence was observed to be low in the bean area outside the experimental plots, which probably resulted in a reduced invasion of inoculum. To reduce interplot interference, all plots were surrounded by millet borders. As a result, rust only appeared in the noninoculated plot late in the epidemic. However, the flattening began with the second assessment. In bean rust, the slope often rose quickly from the first to the

Table 6. Estimated Disease at the Source (a) and Gradient Slope (b) of Bean Rust^X

Time (Days)	Assess- ment Type ^Y	Spring					
		Source Type					
		Area		Line		Point	
		a	b	a	b	a	b
1	INCPL	-.476	-.353	-.260	-1.406	-5.025	-1.054
	SEV	-6.439	-.729	-6.966	-1.470	-12.395	-.987
	INCLF	-1.811	-.435	-2.396	-1.470	-7.310	-1.058
8	INCPL	-.019	-.098	-.112	-.189	-4.620	-1.031
	SEV	-3.301	-.488	-4.115	-.729	-1.877	-.782
	INCLF	-.543	-.204	-.974	-.319	-4.62	-1.03
15	INCPL	-.026	-.034	-.020	-.075	-4.426	-1.797
	SEV	-1.841	-.484	-3.134	-.502	-.673	-.047
	INCLF	-.287	-.133	-.460	-.194	-4.426	-1.797
22	INCPL	-.0108	-.0088	-.015	-.028	-10.97	-5.609
	SEV	-1.051	-.420	-2.263	-.331	-.039	-.171
	INCLF	-.121	-.098	-.341	-.116	-10.97	-.561
29	INCPL	-.00003	0	-.0099	-.0092	-22.65	-15.07
	SEV	-.885	-.291	-1.659	-.275	-.069	-.135
	INCLF	-.124	-.065	-.268	-.091	-22.65	-15.06

Table 6. continued

		Fall-West Position from the Source					
		Source Type					
Time (Days)	Assess- ment Type ^y	Area		Line		Point	
		a	b	a	b	a	b
1	INCPL	-.0068	-.056	-.052	-.308	-.459	-1.489
	SEV	-3.479	-.37	-3.673	-.793	-6.913	-1.594
	INCLF	-.495	-.180	-.788	-.456	-1.900	-1.60
8	INCPL	-.006	-.004	-.024	-.036	-.036	-.119
	SEV	-1.348	-.38	-2.394	-.493	-4.347	-.327
	INCLF	-.236	-.086	-.399	-.16	-1.173	-.194
15	INCPL	-.00002	-.000002	-.0071	-.0044	-.093	-.078
	SEV	-1.011	-.198	-1.447	-.308	-2.394	-.367
	INCLF	-.112	-.032	-.156	-.065	-.349	-.158
22	INCPL	-.00002	-.000002	-.00003	0	-.009	-.0057
	SEV	-.912	-.138	-1.21	-.19	-1.556	-.295
	INCLF	-.109	-.026	-.141	-.049	-.214	-.060
29	INCPL	-.00002	-.000002	-.00003	0	-.00002	0
	SEV	-.914	-.104	-1.128	-.123	-1.634	-.160
	INCLF	-.110	-.018	-.139	-.040	-.206	-.055
36	INCPL	-.00002	-.000002	-.00003	0	-.00002	0
	SEV	-.891	-.116	-1.129	-.129	-1.497	-.086
	INCLF	-.11	-.017	-.143	-.026	-.183	-.029

Table 6. continued

		Fall-East Position from the Source					
		Source Type					
Time (Days)	Assess- ment Type ^y	Area		Line		Point	
		a	b	a	b	a	b
1	INCPL	-.315	-.410	-.55	-.833	-.0148	-1.542
	SEV	-3.576	-1.017	-4.260	-1.115	-5.839	-1.704
	INCLF	-.028	-.813	-.829	-.717	-1.833	-1.611
8	INCPL	-.062	-.047	-.048	-.114	-.330	-.513
	SEV	-1.92	-.615	-3.159	-.635	-4.265	-.714
	INCLF	-.162	-.241	-.507	-.256	-1.055	-.615
15	INCPL	-.040	-.025	-.044	-.031	-.0708	-.103
	SEV	-1.415	-.38	-1.906	-.418	-2.924	-.445
	INCLF	-.109	-.119	-.164	-.139	-.582	-.217
22	INCPL	-.0051	-.0032	-.0029	-.002	-.0208	-.013
	SEV	-1.179	-.289	-1.37	-.295	-1.676	-.389
	INCLF	-.084	-.084	-.138	-.072	-.171	-.130
29	INCPL	-.00003	0	-.00003	0	-.00002	0
	SEV	-3.185	-.224	-1.316	-.217	-1.61	-.227
	INCLF	-.097	-.058	-.142	-.051	-.217	-.053
36	INCPL	-.00003	0	-.00003	0	-.00002	0
	SEV	-1.236	-.177	-1.231	-.137	-1.477	-.131
	INCLF	-.109	-.045	-.129	-.034	-.211	-.028

^xEstimated by Zellner's Procedure based on the transformation with the model of Kiyosawa and Shiyomi: $\log_e(y) = \log_e(a) - b \cdot d$.

^y

INCPL = incidence of diseased plants, INCLF = incidence of diseased leaves, SEV = disease severity.

second assessment, and did not change much in later assessments. Minogue and Fry (93, 94) also observed, that for potato late blight, the gradients were initially quite steep and rapidly became flatter over time. Within 7-10 days of the first observation, the flattening ceased, and the gradients were stable thereafter (94). MacKenzie (86) found no statistical indication of the flattening. However, he made just two assessments, at the very end of the epidemic, when there was probably no difference between the two slopes.

Berger and Luke (14) suggest the inappropriateness of the Gregory transformation was responsible for the flattening of gradients. As already mentioned, the increase in slope values for bean rust could be observed even in the nonlinear models, using nontransformed data. Therefore in bean rust, the flattening was not from an aberration of the transformation. Nevertheless the transformation is a shift in the scale of plotting the data, and must be considered when models are applied to disease gradients.

Some authors mention the reduction of tissue available for infection as the cause of flattening (74, 111). For bean rust, as the disease progressed in time, the 'a' values became closer, i.e., as the epidemics approached finality, the maximum asymptote of disease was reached at the source and close to it. At these points there was limitation of healthy tissue. Therefore, the main mechanism behind the flattening was the disease-carrying capacity: no more susceptible plants or sites to be diseased. This phenomenon

was very noticeable in the INCPL: 1.0 was reached very quickly, and the fastest flattening of the gradient was observed in INCPL.

Although noticed for SEV, the flattening of gradients was less pronounced than for INCLF and INCPL. Therefore, the flattening can be observed in some conditions, but not in others. As mentioned by Gaumann (37), each epidemic has its own "genius epidemicus". Thus, the flattening might be a phenomenon typical of bean rust epidemics, as found in this research and by Imhoff et al (54), but does not occur in wheat rust, even with high final severity (86).

The effect of the type of the inoculum source (point, line, and area) on the gradient parameters was tested for three assessment types, two seasons, two directions in fall, and six assessment times. According to Zadoks and Schein (147), 'a' should increase for epidemics from point, to line, to area sources. For the bean rust data, 'a' was always higher in the area source than in the point source. However, the 'a' from line source was not always strictly intermediate; sometimes nearly equal to the point source, and sometimes it approached the 'a' of the area source. Gregory (43, 44) reports that the gradients from a point source are steeper than those from a line source, which are steeper than the gradients from an area source. Other authors (81, 142), cited this principle, but without testing its validity. When gradients slopes of bean rust were compared, the slopes of the area source were flatter than the slopes of the point source, until the second- to third-assessment time. However,

as the epidemic progressed to the end, no difference was detected among the slopes of the three inoculum sources. Therefore, Gregory's principle should be tested carefully with other pathosystems, because it was not entirely applicable with bean rust. As with 'a', the 'b' parameter estimated in the line source was intermediate. The differences in source sizes used in this research probably were not great enough to delineate clearly area, line, and point sources.

The slopes and intercepts estimated in the three assessment types were also compared in the three inoculum sources, two seasons, two directions in fall, and six assessment times. The three 'a's were different in all the sources and throughout all the assessment times. The difference in the 'a's was probably a result of the difference among the maximum disease intensities of each assessment type. Berger (personal communication) suggested that the gradient slope is a function of the assessment type. This was observed for bean rust: the gradient slope was statistically flatter successively in SEV, INCLF, and INCLP. Therefore, when gradients for different pathosystems are to be compared, the assessment type should be specified. Moreover, the same assessment type should be used to compare disease gradients of different pathosystems or different treatments.

Paysour and Fry (99) state that disease gradients are often asymmetrical around the source, due to factors as strong prevailing winds. According to Vanderplank (135), one

way in which wind affects the gradient is that more inoculum leaves the source in one direction than in another. However, Emge and Shrum (29) did not find significant differences between downwind and upwind gradients. This is probably because wind direction fluctuates during dispersal (43). The effect of direction from the source on the gradients of bean rust was also studied in the fall experiment. Therefore, the parameters estimated from the three sources of inoculum, in the three assessment types, and in the six assessment times, were compared between the west and east direction from the inoculum source. No difference was detected between the 'a's estimated west and east from the source. This observation seems logical, as the inoculum source was common to both directions. A statistical difference was detected between the 'b's of the two directions. Overall, the slopes observed in the west (the predominantly downwind direction) were flatter than the slopes estimated in the east direction. Of the three source types, the largest differences in the gradient slopes were detected in the area source. Of the three assessment types, the largest difference in gradient slopes were found in INCPL. Both cases represent the largest source strength. The significance of this observation is to be elucidated. The fact that different slopes are estimated for different directions from the source is important in planning experiments. Ideally, to estimate and compare gradients, disease should be assessed at all directions around the source to eliminate the variation due to wind direction. However, when this type of design is not

possible, and disease gradients are to be used to compare treatments, this comparison should be done when disease was assessed at the same direction from the source in all of the treatments. This procedure will reduce the effect of wind direction as a source of variation.

The gradients were also compared between epidemics of the two seasons. Therefore, the parameters estimated for the gradients from the three inoculum sources, for the three assessment types, and at six assessment times were compared between spring and fall. Overall, higher intercepts were estimated in fall rather than in spring. As in disease progress, this difference probably resulted from a more efficient way of starting the epidemics in fall. Therefore, the inoculation of bean plants in the field was more effective than transplanting inoculated plants. Overall, the slopes were also different. The slopes of the spring gradients were steeper than in the fall. This difference in slopes probably was from the influence of source strengths on the gradient slopes, as already discussed.

The progress of epidemics in time has received much more attention than the spread of disease in space. Thus, many factors in experimentation with disease progress are already understood, which is not the case for gradients. More research is needed to understand disease spread and the factors that govern it. This will generate more reliable information that will lead to the rational control of epidemics.

Trends of Parameters of Disease Progress and DiseaseGradients

Some authors tried to compare the gradient slope with the disease progress rate. MacKenzie (86) tried to merge vanderplankian (132) and gregorian (44) models to compare the resistance of wheat varieties to rust. He found no correlation between the two slopes. Progress and spread are necessarily two related phenomena. However, the quantification of this relationship seems complicated. The two slopes (gradient and progress rate) estimated in epidemics of bean rust were also compared (Table 7). As observed, sometimes both slopes had the same trends. For example, the progress rates in INCPL were faster than in SEV and the gradient slopes estimated in INCPL were flatter than SEV. However, in other occasions the slopes had completely opposite trends. For example, the progress rates that originated from point sources were faster than from area sources, and the gradient slopes from point sources were steeper than from area sources. Therefore, it was difficult to compare conclusively slopes of both spread and progress.

MacKenzie (86) noted that the intercept of the disease gradient was associated with the rate of slow rusting of wheat. However, for these studies with bean rust higher intercepts of disease gradients were not always associated with faster disease progress. As an example, the progress rates estimated in epidemics that originated from the point sources were faster than epidemics from the area sources.

Table 7. General Trends of the Parameters Estimated by the Models of Progress and Gradient^x

	Intercept		Slope	
	Progress (y_0)	Spread (a)	Progress (k)	Spread (b)
Assessment Type	INCPL > INCLF > SEV ^y	INCPL > INCLF > SEV	INCPL > INCLF > SEV	INCPL > INCLF > SEV
Season	Fall > Spring	Fall > Spring	Fall = Spring	Fall > Spring
Position	Downwind = Upwind	Downwind = Upwind	Downwind = Upwind	Downwind > Upwind
Source Type	Area > Line > Point	Area > Line > Point	Point > Line > Area	Day 1: Area > Point Day 36: Area = Point
Distance	8.5 > 0.3 m	--	No differences	--
Time	--	Day 36 > Day 1	--	Day 36 > Day 1

^x" y_0 " and "k" are initial disease and epidemic rate, respectively, estimated by the model of Gompertz; "a" and "b" are disease at the source and gradient slope, respectively, estimated by the model of Kiyosawa and Shiyomi.

^yINCPL = incidence of diseased plants; INCLF = incidence of diseased leaves; SEV = disease severity.

However, the strengths of point sources were lower than the area sources.

A direct relationship was found between the intercepts of the two models to describe epidemics of bean rust. That is, when the estimate of initial disease (intercept of the progress model) was low, disease at the source (intercept of the gradient model) was also estimated to be low. Both intercepts are directly dependent upon the source strength.

Both intercepts were related to the gradient slope; i.e., the higher the intercept value, the flatter the gradient slope. Therefore, the gradient slope was related to the source strength. Thus, sanitation measures should reduce the spread of epidemics. This hypothesis, if valid, can be of great value in the control of epidemics, and should be quantified in future research.

These exercises of comparisons may prove very useful to understand and to control epidemics. However, models that merge the concepts of both progress and spread are needed.

Tridimensional Approach to Describe Epidemics

Isopathetic Rates

A good start in merging the concepts of progress and spread was taken by Berger and Luke (14) when they developed the concept of isopathetic rates. According to them, "the outward spread of disease from infection foci can be considered moving annuli of disease of equal intensity. "

These authors used isopathetic movement to rank the resistance of oat cultivars to rust and found values of 0.4 and 0.9 m/day for resistant and susceptible cultivars, respectively.

Isopathetic rates of bean rust were calculated for the spring and fall experiments, the two directions from the source in fall, the three assessment types, and the three source types. The isopathetic rates did not vary much between seasons and directions. Therefore the rates observed in the two seasons and two positions were averaged (Table 8).

As proposed by Berger and Luke (14), isopathetic rates are a good parameter for comparative epidemiology. When the isopathetic rates, estimated in the three inoculum sources of bean rust were compared, the isopathetic rates (0.56-0.73 m/day) from the point sources were consistently faster than the rates (0.35-0.48 m/day) from the area sources. The isopathetic rates (0.33-0.46 m/day) from line sources were similar to the rates from area sources. The disease progress rates also increased from point to area sources.

Although the gradient slopes of bean rust differed between seasons and between directions, the disease progress rates did not differ. The isopathetic rates of bean rust also did not vary between seasons or between directions in the fall.

Berger and Luke (14) found that the gradient slopes of rust did not differ among oat cultivars, but the disease progress rates and the isopathetic rates did. Hence, the disease progress rates and the isopathetic rates were faster in

Table 8. Isopathetic Rates of Bean Rust Observed for Three Assessment Types and Three Source Types Calculated for Day 1 to Day 36

Assessment Type ^x	Source Type ^y	Isopathetic Rate (m/day)	Standard Deviation	Standard Error of Mean
INCPL	Area	.48	.12	.07
	Line	.46	.15	.09
	Point	.69	.07	.04
INCLF	Area	.35	.09	.05
	Line	.35	.05	.03
	Point	.56	.03	.02
SEV	Area	.35	.13	.08
	Line	.33	.01	.01
	Point	.73	.14	.08

^xINCPL = incidence of diseased plants; INCLF = incidence of diseased leaves, SEV = disease severity.

^yAverages of the isopathetic rates of spring and fall and the west and east direction in fall.

the susceptible than in the resistant cultivars, even though there was no difference in gradient slopes.

For both oat rust (14, 85) and bean rust pathosystems, conditions that caused faster disease progress rates also caused faster isopathic rates. Although the concept of isopathic rate embodies both progress and spread, disease progress probably is more important than disease spread to determine the isopathic rate. Further field work and simulation should be conducted to verify this hypothesis.

Isopathic rates may have considerable utility in epidemiological research. The concept of isopathic rates may provide the foundation for models to merge progress and spread.

Tridimensional Representation

Instead of solely viewed as "change with time" (89), epidemics should be viewed as "changes with time and space". Considering this concept, the progress and spread of bean rust were plotted as response surfaces (Figures 1-3). These plots are very useful to view simultaneously the trends in progress and spread. Thus, epidemics originated from point sources progressed faster than epidemics from area sources. Similarly, epidemics based on INCPL progressed faster than when based in INCLF or SEV. The maximum disease asymptote for the three assessment types is easily noticed: in INCPL the maximum of 1.0 was soon reached; in INCLF the maximum was around 0.9; and in SEV the asymptote was about 0.5. Disease spread is also visualized; the gradients of epidemics

generated from point sources were steeper than the gradients from area sources from the early assessment times. The flattening of gradients in time is more clearly seen when INCLF and INCPL were assessed.

Although the response surfaces are valuable to aid in the interpretation of disease progress and spread, a more analytical approach is needed to quantify these factors. Therefore, models which merge both progress and spread would be ideal.

Tridimensional Models

As noted by Vanderplank (133), the integration of spatial concepts into temporal disease progress models has been difficult. There are sophisticated mathematical models considering just time, but, until recently, there have been no analytical models to describe disease in time and space (147). There are simulators which consider this approach (70, 119), but they have limited value (60). Based on the concept of isopathic rates, Jeger (60) developed several models to merge progress and spread. From eight models, three were considered the best:

- i). $y = 1 / (1 + a \cdot \exp(b \cdot d - c \cdot t))$ which corresponds to the classical polycyclic diseases;
- ii). $y = 1 - a \cdot d^b \cdot \exp(-c \cdot t)$ which is to be used for classical monocyclic disease;
- iii). $y = 1 / (1 + a \cdot d^b \cdot \exp(-c \cdot t))$ which can describe the early stages of a polycyclic epidemic, with high inoculum at the focal center (60).

Figure 1. Progress and Spread of Bean Rust from Three Source Types and for Three Assessment Types, in the Spring of 1984.

Figure 2. Progress and Spread of Bean Rust from Three Source Types and for Three Assessment Types, in the Fall of 1984, West Direction from the Source.

INCIDENCE (PLANTS)

INCIDENCE (LEAVES)

SEVERITY

POINT

LINE

AREA

Figure 3. Progress and Spread of Bean Rust from Three Source Types and for Three Assessment Types, in the Fall of 1984, East Direction from the Source.

Jeger (60) reports that model (iii) may be appropriate when epidemics of a polycyclic nature are initiated by artificial inoculation, and especially where spread is local (as splash- rather than air-dispersed).

A logistic submodel is implied by Jeger (60) in the models for epidemics of polycyclic diseases (i and iii). Since the Gompertz model was the best to describe bean rust epidemics, a tridimensional model based on Gompertz was needed. Consequently a model in which the submodel for progress is the Gompertz was developed; i. e., iv). $y = a * \exp(-b * \exp(-kt) - c * d)$. These four models were tested to describe bean rust epidemics of fall and spring, that originated from the three source types, and for in the three assessment types (Table 9).

The overall best model was the one Jeger (60) recommended for polycyclic diseases, (i), which agrees with the general concept of bean rust epidemics. It is important to observe that the model (iii) gave the best fit in terms of R^2 and SSR. However, when the actual fit was compared, model (iii) provided the higher rank of residuals (poorer fit). Therefore, a word of caution should be addressed to modelers who rely too much in the R^2 or SSR when choosing a nonlinear model.

The fit of the tridimensional models was very repeatable and not dependent on the time of the year nor on the amount of initial inoculum. However, the behavior of the models changed according to the type of disease assessment; i. e., for SEV and INCPL, model (i) always gave the best fit. For

Table 9. Fit of Tridimensional Models to Merge Spread and Progress of Bean Rust^x

Assessment Type ^y	Source Type	Model ^z	Spring					Rank of Residuals
			a	b	c	SSR	R ²	
INCPL	AREA	i	.5377	.5756	.3688	.3688	.8887	1
		ii	.4428	.5008	.1315	.2969	.9104	2
		iii	.8919	1.7220	.3644	.0950	.9713	4
		iv	1.1743	.0226	.2404	1.154	.6517	3
	LINE	i	1.5961	.4057	.2430	.4082	.9019	1
		ii	.6445	.3266	.0893	.4947	.8810	2
		iii	2.8161	1.2369	.2558	.1658	.9601	4
		iv	1.8549	.0357	.1936	.9789	.7623	3
	POINT	i	21.998	.2382	.3643	.5384	.9071	1
		ii	.9376	.1679	.0882	.9625	.8338	2
		iii	29.938	.9935	.3929	.3162	.9454	4
		iv	13.353	.0070	.2818	.7564	.8695	3
SEV	AREA	i	9.046	.9127	.0880	.0624	.9365	1
		ii	.9425	.0858	.0077	.1943	.7019	2
		iii	3.531	.8170	.1165	.1283	.9541	4
		iv	3.3106	.7009	.0713	.0442	.9550	3
	LINE	i	31.6672	1.1377	.1027	.0074	.9672	1
		ii	.9845	.0374	.0038	.0607	.6352	2
		iii	6.7970	.8440	.1120	.1420	.9432	4
		iv	4.8916	.9540	.0581	.0063	.9621	3
	POINT	i	232.10	.0707	.1435	.0105	.9636	2
		ii	1.0245	.0144	.0064	.0382	.7813	3
		iii	17.495	.1873	.1600	.2521	.9229	4
		iv	8.5056	.0595	.0591	.0095	.9456	1
INCLF	AREA	i	2.2819	.2990	.1140	.2887	.8968	2
		ii	.7300	.2415	.0396	.2723	.9027	1
		iii	32.764	.9483	.0913	.0415	.9363	4
		iv	2.0133	.1138	.1367	.2874	.8973	3
	LINE	i	3.8086	.3181	.1043	.3985	.8405	1
		ii	.8261	.1896	.0282	.3907	.8435	1
		iii	126.01	.9170	.1039	.0029	.9826	3
		iv	2.6796	.1786	.1219	.2919	.8832	2
	POINT	i	16.2193	.0641	.1602	.2618	.9199	1
		ii	1.0794	.0457	.0457	.3573	.8907	2
		iii	241.56	.2067	.1416	.0065	.9755	4
		iv	6.1206	.0264	.1312	.1514	.9537	3

Table 9. continued

Assessment Type ^y	Source Type	Model ^z	Fall-West Position from the Source					Rank of Residuals
			a	b	c	SSR	R ²	
INCLP	AREA	i	.0992	.2651	.4482	.0217	.8978	2
		ii	.0923	.9429	.4138	.0068	.9847	1
		iii	.0913	1.0866	.460	.0046	.9784	4
		iv	.856	.0039	1.803	.102	--	3
	LINE	i	.3816	.6580	.6917	.0219	.9878	1
		ii	.3881	.5534	.2423	.0383	.9787	2
		iii	.6195	1.3736	.4464	.0244	.9864	4
		iv	.9623	.0092	.3738	.5453	.6970	3
	POINT	i	2.4481	.4070	.3460	.1451	.9632	1
		ii	.8595	.1871	.1418	.3226	.9181	2
		iii	4.9079	.7702	.3442	.2602	.9340	4
		iv	2.4873	.0164	.2673	.4420	.8879	3
	AREA	i	4.8227	.2341	.0443	.2169	.9106	2
		ii	.8878	.0999	.0105	.1415	.9221	1
		iii	.9729	.4949	.0883	.1782	.8732	4
		iv	1.9981	.1854	.0331	.1851	.7673	3
	LINE	i	10.3654	.2177	.0534	.1221	.7577	2
		ii	.9600	.0624	.0084	.0935	.8143	1
		iii	1.9118	.5468	.0924	.2893	.8718	4
		iv	2.808	.1815	.0324	.1039	.7937	3
	POINT	i	31.8375	.1769	.0681	.0272	.9375	1
		ii	1.0044	.0294	.0058	.0310	.8549	2
		iii	6.5515	.4510	.1136	.2981	.9037	4
		iv	4.1123	.1558	.0319	.0213	.9001	3
INCLF	AREA	i	.7818	.1740	.0895	.1979	.8591	2
		ii	.4896	.2876	.0566	.0998	.9290	1
		iii	7.2198	.5134	.0454	.1516	.8094	4
		iv	.8867	.0403	.1255	.2066	.8530	3
	LINE	i	1.4421	.2078	.0940	.3010	.8666	2
		ii	.6707	.2427	.0493	.1545	.9316	1
		iii	15.1097	.4551	.0539	.1001	.8012	4
		iv	1.5037	.0623	.1248	.2485	.8897	3
	POINT	i	4.8198	.1930	.1150	.2706	.9125	1
		ii	.9612	.1094	.0430	.2719	.9121	2
		iii	43.695	.3310	.0684	.0300	.8596	4
		iv	3.5194	.0701	.1220	.1336	.9567	3

Table 9. continued

Assessment Type ^y	Source Type	Model ^z	Fall-East Position from the Source					Rank of Residuals
			a	b	c	SSR	R ²	
INCPL	AREA	i	.1182	.9133	.8149	.0747	.9557	1
		ii	.1918	.8728	.1888	.1049	.9378	2
		iii	.0903	2.6401	.4906	.0561	.9667	4
		iv	.6927	.0166	.4449	.8173	.5150	3
	LINE	i	.5754	.4977	.3273	.0971	.9624	2
		ii	.4336	.5085	.1359	.1370	.9469	3
		iii	.9643	1.3578	.3055	.0459	.9822	4
		iv	1.0828	.0162	.2335	.7696	.7016	1
	POINT	i	2.1911	.4601	.2739	.1501	.9658	1
		ii	.7081	.3094	.0918	.5090	.8839	2
		iii	4.3989	1.3128	.2855	.1025	.9766	4
		iv	2.2108	.0173	.1688	.925	.7891	3
SEV	AREA	i	6.8831	.4707	.0444	.0715	.8390	1
		ii	.9319	.0766	.0061	.0766	.8273	1
		iii	1.4363	.7675	.0821	.2215	.9059	3
		iv	2.3339	.3936	.0299	.0564	.8228	2
	LINE	i	17.034	.2820	.0621	.0437	.9375	1
		ii	.9833	.0506	.0066	.0543	.8416	2
		iii	3.0431	.6560	.1033	.2061	.9257	4
		iv	3.4103	.2402	.0339	.0346	.8990	3
	POINT	i	34.4378	.2761	.0724	.0251	.9340	1
		ii	1.0015	.0335	.0051	.0440	.8842	2
		iii	10.6328	.6066	.1276	.2371	.9283	4
		iv	4.3884	.2443	.0351	.0196	.9484	3
INCLF	AREA	i	1.0189	.2851	.0855	.2281	.9031	2
		ii	.5484	.3520	.0399	.1484	.9371	1
		iii	14.442	.6286	.0455	.0606	.8633	4
		iv	1.1696	.0894	.1269	.2886	.8772	3
	LINE	i	2.1131	.2562	.1050	.2271	.9182	2
		ii	.7498	.2287	.0439	.1896	.9315	1
		iii	27.377	.4736	.0622	.0458	.8664	4
		iv	1.9554	.0773	.1204	.2467	.9110	3
	POINT	i	7.0649	.2380	.1262	.2665	.9194	1
		ii	.9626	.1303	.0374	.4342	.8686	2
		iii	56.664	.4339	.0736	.0291	.8624	4
		iv	5.028	.0829	.1266	.2494	.9245	3

Table 9. continued

^xa = parameter related to the intercept, b = gradient parameter, c = progress parameter, SSR = sum of squares of residuals, R² = coefficient of determination. The minimum rank of residuals is 1.

^yINCPL = incidence of diseased plants, INCLF = incidence of diseased leaves, SEV = disease severity.

^zModels were

$$\text{i} \quad y = 1/(1 + a*e^{(b*d-c*t)});$$

$$\text{ii} \quad y = 1 - a*d^b e^{(-c*t)};$$

$$\text{iii} \quad y = 1/(1 + a*d^b e^{-c*t});$$

$$\text{iv} \quad y = a*e^{(-ce^{-k*t}-b*d)};$$

$$d \text{ (distance)} = 0.3-8.5;$$

$$t \text{ (time)} = 1-36.$$

INCLF, model (ii), indicated for monocyclic diseases (60), was as good as model (i). The biological implication of this trend should be studied, with other pathosystems. Another aspect is to implement tridimensional models based on the Gompertz type of disease progress. When progress was considered apart from spread, the Gompertz model gave the best fit. However, when progress and spread were merged in the tridimensional model with the Gompertz submodel (model iv), it was worse than the model (i) (which had the logistic progress submodel). However, if a different gradient model had been merged with the Gompertz, the fit may have been better. Nevertheless, when disease progress solely was studied, the logistic model gave fit nearly equal to the Gompertz model for many curves.

Model (iii) is expected to give a good fit for experimental conditions in which there is a focal center of inoculum, as with *Septoria nodorum* in wheat (60). However, this fungus is spread by water splash (60), and *Uromyces phaseoli* is spread by wind (31). Probably in bean rust epidemics, the focal center is not an important inoculum source as in wheat blight epidemics. Therefore the general model for polycyclic diseases (model i) was more appropriate to describe epidemics of bean rust.

The three models of Jeger (60) and a subsequent variant of Jeger's model were very useful to explain progress and spread of bean rust. More studies, using other pathosystems and additional models, are required to implement and to

interpret the tridimensional modeling of epidemics. The tridimensional models should prove useful in the management of disease.

Comparison of the Tridimensional Model with the Models of
Disease Spread and of Disease Progress

The tridimensional model that merged the concept of polycyclic diseases (logistic model) with exponential decrease in distance (Kiyosawa and Shiyomi's model) provided the best fit to the bean rust data. The model is $y=1/(1+a*\exp(bd-ct))$ (60). Although Jeger (60) provided no description of the parameters of the model, some conclusions were made: 'a' is related to initial disease. As the source strength increases, 'a' also increases. Therefore, for the bean rust data 'a' was successively higher in point, line, and area sources. For the same inoculum type, 'a' was successively higher in SEV, INCLF, and INCPL assessments. Similar conclusions were found when the intercepts of the models of progress and spread were studied individually (Table 7). The parameters 'b' and 'c' are the gradient and progress parameters, respectively. The general trends of these two parameters were studied (Table 10).

When these trends are compared with those from the models of disease progress and disease gradient (Table 7), some general conclusions can be drawn:

- 1). among the assessments, INCPL had the largest slope for both progress and gradient, in both tables;

- 2). between directions, downwind had a flatter gradient than upwind and the progress rate is the same for the two directions, in both tables;
- 3). between the seasons, the progress rates in spring and fall seemed not to differ in both tables; for the gradients, the slope estimated for INCPL was flatter in fall in both tables, in the other assessments the two tables differ;
- 4). among the sources, the point source had the largest progress rate in both tables; in Table 7, the gradient slope was steeper in the point source until the middle (time) of the epidemics; later, all slopes were equal. In Table 10 the difference in slopes was not well defined (probably, because the tridimensional model is a summation of the whole process of spread and progress).

This is the first time the trends of the parameters estimated in the tridimensional model have been compared to the trends of the parameters estimated from the individual models of progress and spread. The trends detected in the analysis with the tridimensional model and with the individual models of progress and spread were similar. Therefore, the inclusion of both time and distance in the same model is not only desirable but feasible to analyze epidemics. The tridimensional model can provide almost all the same conclusions as separate models for progress and for gradients. Besides being repeatable over different seasons and inoculum sources, the tridimensional model has the advantages of being simpler, and more points are given to

Table 10. General Trends of Gradient (b) and Rate (c) Parameters from Jeger's Tridimensional Model (i) for Bean Rust Data

	Parameters	
	Gradient	Progress
Assessment Type	INCLP > SEV ^x	INCLP > SEV
Season	INCLP: Fall > Spring	Fall = Spring
	INCLF, SEV: Fall = Spring	
Position	Downwind > Upwind	Downwind = Upwind
Source Type	Area > Point	Point > Area

^xINCPL = incidence of diseased plants; INCLF = incidence of diseased leaves; SEV = disease severity.

estimate the parameters. The parameters need additional interpretation, which can be done through further research and modeling.

The tridimensional approach has enormous potential to model and simulate epidemics. It is important to remember that the models are limited, and by definition, simplified (60). Nevertheless this approach should be considered more carefully in plant epidemiological research. Epidemics should be even viewed as "changes in disease with time and space". More accurate models are foreseen, and the use of "volume under disease progress surface" may become as familiar as the "area under disease progress curve", in comparative epidemiology.

SUMMARY AND CONCLUSIONS

Field experiments were conducted over two seasons (spring and fall), with two directions in the fall (east and west), to study the progress (change in time) and spread (change in distance) of bean rust. Disease intensity was assessed as incidence of diseased plants (INCPL; calculated as the proportion of diseased plants), incidence of diseased leaves (INCLF; calculated as the proportion of diseased leaves), and disease severity (SEV; calculated as the proportion of diseased tissue/plant), for epidemics that were initiated from three inoculum sources (area, line, and point).

The monomolecular, logistic, and Gompertz models (13, 89, 128) were fitted to the progress data. Overall, the Gompertz gave the best fit. In some cases, the monomolecular model gave a good fit: in the INCPL assessed in the area source, and in assessments closer to the source. Therefore, even polycyclic diseases can behave like monocyclic diseases, when there is limitation of host plants. In the bean rust pathosystem, relatively few plants were used to assess INCPL (a maximum of 20 in each assessment point). Thus the monocyclic pattern could be an artifact generated by this small number of plants. As observed before for several pathosystems (13) including the bean-rust pathosystem (102),

the Gompertz model fit the disease progress data better than the logistic. This fit was better in both nonlinear and linear regressions. The comparison of fits in the nonlinear regression was based on the R^2 sensu Kvalseth (79), sum of squares of residuals (SSR), and primarily in the ranking of the residuals. The study of residuals is suggested as the most valuable parameter to compare nonlinear models. The knowledge of the right model to describe an epidemic will increase the reliability of the estimated parameters and the comparison between them.

Traditionally, the comparison of epidemic rates (e.g., the progress rate yielded in the regression of gompits on time) has been done by Duncan's multiple range test. The Duncan's test may not be appropriate to compare rates estimated through regression, and other methods should be used. In the cases in which the regression lines are correlated (as for disease progress in a gradient), Zellner's procedure may be more appropriate (72, 149). Therefore Zellner's procedure was used to estimate and compare ' y_0 ' (initial disease) and ' k ' (disease progress rate) of the regression of gompits versus time.

Overall, the epidemic progress of INCPL was faster than INCLF and SEV, and INCLF and SEV were more similar. Although INCLF and SEV are not highly correlated (54, 117), they progress with a similar trend. INCLF and SEV are more autoinfection processes, while INCPL is more an alloinfection process, sensu Robinson (107). The ' y_0 's of the three assessments were all different. The biggest ' y_0 ' was found

in INCPL, and smaller successively in INCLF and SEV. Similarly, maximal disease was at the absolute maximum in INCPL, i.e. 1. In SEV, the maximum seldomly surpassed 0.5. Even though ' y_0 ' always was higher closer to the source than farther, in most of the cases, the ' k ' values were similar with increasing distance from the source. The ' k ' values were expected to become bigger with increased distance (83, 84), but the unchanging of ' k ' could probably be a common fact with the rusts (29, 85). The estimated ' y_0 ' decreased farther from the source and there was a time delay for the epidemic to start farther from the source. Although there is more available tissue farther from the source, this tissue could be resistant when epidemics start, because bean leaves become resistant with age (136, 150). An approach to prove this hypothesis would be a similar study with pathosystems in which aging does not increase resistance. Epidemics in spring and fall and in the west and east directions from the source in the fall progressed with similar ' k 's'. Both directions also shared the same ' y_0 '. However, the ' y_0 ' from fall was higher than the spring. A trend, observed before (110, 112), and of great significance to sanitation, was that the ' k 's' were higher for epidemics that originated from the point source (which had the smaller ' y_0 's') than from the area sources.

The knowledge of disease spread is as important as of disease progress in the control of epidemics. However, the models of disease spread are much less studied and compared.

Seven models were initially tested to describe gradient data. The models of Gregory (43), Kiyosawa and Shiyomi (74), Lambert et al. (80), and Hoerl (23) gave the best fit in terms of R^2 and SSR. When these four models were compared through the residuals, the best fit was observed for the models of Hoerl and Lambert et al. The model of Lambert et al. was inadequate to describe INCPL when the maximum of disease was 1.0. The two most commonly used gradient models, Gregory and Kiyosawa and Shiyomi, were compared. The model of Gregory best fitted SEV data, and the Kiyosawa and Shiyomi best fitted INCPL and INCLF data. Further research with other pathosystems is suggested, to determine whether this trend was typical of the bean-rust pathosystem or is a common one. In the transformed data, the best fit was observed with the linear regression of the model of Hoerl followed by the model of Kiyosawa and Shiyomi. This later transformation, because of its simplicity and less manipulation, was chosen to generate and compare parameters by Zellner's procedure (149). Thus, the parameters of disease at the source ('a') and gradient slope ('b') were generated and compared.

Both 'a' and 'b' increased over time. The increase in 'a' was expected, as the epidemic progressed. However, the increase in 'b' over time characterizes the flattening, which is a debatable point in gradient theory (24, 44, 74, 85, 86). Very little rust was detected outside the plots, and the movement of spores inside was reduced with borders of pearl millet. The flattening of gradients was observed even in the nonlinear models, with nontransformed data. Therefore,

background contamination and the inappropriateness of the transformation, factors commonly associated with the flattening of gradients (14, 70), were not considered as factors primarily responsible for the gradient flattening in the bean-rust pathosystem. Overall, the flattening of the slopes was probably due to the epidemics reaching the maximal asymptote of disease, which will result in no more increase of disease. This hypothesis has been suggested in other pathosystems (74, 111). The flattening was most obvious in the INCPL data, where the maximal asymptote of 1.0 was reached very early in time. An important concept in modeling disease spread is the effect of source strength in the gradient. As predicted (44, 147), 'a' was larger in the area than in the point. Gregory (43, 44) theorized and was supported by others (56, 81) that the gradient slope is successively steeper in the area, line, and point. However, according to the bean rust data, this principle only applied to the middle of the epidemics; later, there was no difference among 'b's', which is probably due to the flattening. The comparison of epidemics through gradients should be done using the same assessment type. As observed with bean rust, both slopes and intercepts of the gradient lines of the three assessment types were different. The ideal comparison of gradients would be when disease is assessed in all directions around the source. Although some authors (29, 43) suggest that wind direction does not affect the gradient slope, gradients of bean rust from two directions generated different slopes (even though no

difference in disease at the source was detected). The spring gradient was steeper than the fall, and the disease at the source was higher in the fall. Both ' y_0 ' and 'a' were higher in the fall probably as a reflection of a better inoculation technique in this season.

A consequence of studies with progress and spread would be to compare 'k' with 'b'. However, MacKenzie (86) found no correlation between gradient slopes and disease progress rates. Progress and spread are necessarily correlated phenomena, but they seem not to be statistically correlated, using the measurements we have. To exemplify, the disease progress rates over distances were compared with the gradient slopes over time. For INCPL, the flattening (increase in the gradient slope over time) was very accentuated and the disease progress rates typically increased with distance. However, in the other two assessment types, the progress rates tended not to change with distance, while the gradient slopes flattened over time. Correlation seemed to exist between ' y_0 ' and 'a', i.e., very often when ' y_0 ' was low, 'a' was also low. Thus, when the source strength was high, initial disease was also high.

Instead of comparing two models obtained independently, a better approach to understand epidemics would be to merge the two processes (progress and spread) in just one model. A good start in this direction was taken by Berger and Luke (14), when they presented the concept of isopathic rates. The isopathic rates were calculated for bean rust and proved to be very uniform between seasons and positions, and these

rates are considered a good parameter for comparative epidemiology. The isopathic rates differed among inoculum sources: they were faster for epidemics from the point source than from the area source. When bean rust and oat rust (14, 85) were compared, the isopathic rates were observed to be more affected by the disease progress rate than by the gradient slope. More research is needed to define whether this is a trend common to other pathosystems.

Simulation models that encompass both progress and spread already exist (70, 119), but they are limited in their use (60). More general models should be implemented. Based on the isopathic rate concept, Jeger (60) developed models to merge progress and spread. These models were tested for bean rust epidemics. The model $y = 1/(1+a*\exp(b*d-c*t))$, suggested for polycyclic diseases, gave a very good fit based on the residuals.

The tridimensional model was compared with the linearized models of progress (Gompertz) and spread (Kiyosawa and Shiyomi). Frequently, the spread parameter (b) and the progress parameter (c) of the tridimensional model had the same trends as the gradient slope (b) and the disease progress rate (k). Therefore, the simpler general model produced almost the same results as the two separate models to describe bean rust epidemics. The tridimensional approach should be studied more intensively to describe epidemics. More accurate models are foreseen and even the "volume under disease progress surface" should be considered in comparative

epidemiology. Even epidemics, instead of being seen as "changes in disease with time" (89), should be seen as "changes in disease with time and space".

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BIOGRAPHICAL SKETCH

Luiz Antonio Maffia was born on 21 June, 1953, in Cajuri, Minas Gerais, Brazil. He graduated as an Agronomic Engineer in December 1974 and completed the Master of Science in Plant Pathology degree in December 1977 at the Universidade Federal de Viçosa (UFV). He was on the staff of Empresa Brasileira de Pesquisa Agropecuária (EMBRAPA) from March 1975 until July 1977, when he accepted a faculty position in the Plant Pathology Department at the UFV. Today he is Assistant Professor in this department. In July 1981, he married Angela M. S. Carvalho. With a scholarship from Coordenação de Aperfeiçoamento do Pessoal de Nível Superior (CAPES), he came to the University of Florida (UF) in August 1981 to pursue the Ph.D. in plant pathology. In April 1985, he received a certificate of Presidential Recognition for Outstanding Services from Dr. M. Criser, President of the UF. Luiz A. Maffia belongs to the Sociedade Brasileira de Fitopatologia and to the American Phytopathological Society. He and his wife expect their first child in September 1985.

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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Professor of Plant Pathology

I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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August, 1985

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