Primary disease gradients of wheat stripe rust, caused by the aerially dispersed fungal pathogen *Puccinia striiformis*, were measured in Madras and Hermiston, OR in the springs of 2002 and 2003. Disease foci were created by inoculating a 1.52m x 1.52m area in each of three replicate field plots (6.1m wide x 131 to 171m long) in each of the four experiments. Primary disease gradients were measured both upwind and downwind of the focus. Four gradient models—the power law, the modified power law, the exponential model, and the general model proposed by Lambert *et al.*—were fit to the data. Five of the eight gradients were better fit by the power law, modified power law, and Lambert model than by the exponential, revealing the non-exponentially bound nature of the gradient tails. The other three datasets, which were comprised of the fewest data points, were equally well fit by all the models. By truncating the largest datasets it was shown how the relative suitability of dispersal models can be obscured when data is available only at a short distance from the focus. The truncated datasets were also used to examine the risks of extrapolating gradients to distances beyond available data. The
power law and modified power law predicted dispersal at large distances well, even given limited data, while the Lambert and exponential consistently and sometimes severely underestimated dispersal at large distances. Field data on disease gradients are useful for helping to confirm or refute the validity of gradient models derived from the physical mechanisms of dispersal, as well as to provide accurate information for models designed to predict the behavior of expanding epidemics.

The velocity of expansion of focal epidemics was studied using an updated version of the simulation model EPIMUL. Dispersal data were derived from the Hermiston 2002 field experiment described above. Lesion growth rate, latent period, infectious period, multiplication rate, and dispersal gradient steepness were varied within ranges reasonable for P. striiformis. All but the infectious period had a strong influence on velocity. Three different equations were employed in turn to describe dispersal: the modified power law, the exponential, and Lambert’s general model. The exponential, which fit the gradient data from the field epidemic poorly, yielded an epidemic that expanded at a constant velocity, after an initial buildup period. Both the modified power law and the Lambert model fit the field data well, and produced gradient curves of similar shape. Simulations run with the modified power law and the Lambert model resulted in velocities that increased over time for the entire course of the epidemic, supporting the existence of dispersive epidemic waves.
Modeling Disease Gradients and Understanding the Spread of Wheat Stripe Rust Using Simulated Epidemics

by
Kathryn E. Sackett

A THESIS
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APPROVED:

Redacted for privacy

Major Professor, representing Botany and Plant Pathology

Redacted for privacy

Chair of the Department of Botany and Plant Pathology

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Dean of the Graduate School

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Kathryn E. Sackett, Author
ACKNOWLEDGEMENTS

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INTRODUCTION

Focal plant epidemics are those in which disease spreads outward over time from a source of pathogen inoculum. A number of diseases, such as potato late blight (caused by *Phytophthora infestans*) and wheat stripe rust (caused by *Puccinia striiformis*) exhibit this type of spread in the field (13). There is some controversy over whether such epidemics expand at a constant velocity (a traveling wave) or whether the velocity of a focal epidemic continues to increase over time (a dispersive wave) (11). Analytic and simulation studies have come to different conclusions regarding this question (2,3,6,9,10,12,14).

The basic nature of the velocity of focal epidemics is of interest, but it would also be useful to know what factors can influence the speed with which a plant disease occupies and causes damage in new territory. Environmental conditions (e.g. temperature, humidity, dew period), host vigor and genetic resistance, pathogen virulence, and pesticide application can all affect the ability of a pathogen to reproduce and spread by influencing various components of the pathogen’s life cycle. These components include sporulation rate, infection efficiency, length of latent period, length of infectious period, and rate of lesion growth (1,8).

The epidemic simulator EPIMUL (4,5) was used to study the effect of pathogen life cycle components on epidemic velocity in the context of a particular plant-pathogen system, wheat stripe rust. The model incorporates lesion growth, which is often neglected in epidemic models, but which is an important component in some diseases,
including wheat stripe rust (1). I also altered the simulator code in order to (a) allow a larger area to be modeled, (b) provide greater flexibility in representation of pathogen dispersal, (c) allow asymmetrical upwind/downwind dispersal, and (d) produce output data files relevant to the study of epidemic velocity.

Key to constructing realistic models of focus growth is an understanding of the spatial distribution of disease propagules as they are dispersed from a source. To this end, primary disease gradient data were gathered from artificially inoculated field epidemics of wheat stripe rust in Hermiston and Madras, OR in 2002 and 2003. The field data were analyzed in terms of simple empirical mathematical models that are commonly employed for this purpose in the phytopathological literature. Using these data, the issues surrounding model fitting to gradient data were explored, as well as the importance of obtaining information on dispersal relatively far from the source, in the tails of the gradient.

The most extensive gradient datasets were from the Hermiston 2002 epidemic, and this epidemic was used as a baseline: using EPIMUL, a simulated epidemic was constructed that closely resembled this field epidemic. Input parameters for the model were derived, when possible, from the field epidemic, and otherwise were estimated from the literature. The latent period, infectious period, lesion growth, and multiplication rate (a combination of sporulation rate and infection efficiency), and dispersal gradient steepness were then varied individually within ranges reasonable for wheat stripe rust, and the resulting epidemic velocities were evaluated.

EPIMUL was also used to study the effect of using different dispersal gradient models on velocity. Although the various equations used in the literature to describe
dispersal gradients describe superficially similar curves, it has been hypothesized that the seemingly small difference in the tails of dispersal gradients can have a qualitative impact on epidemic velocity (3,7), and may be the key to the controversy over traveling vs. dispersive epidemic waves.
LITERATURE CITED


Title:

Fitting Models to Primary Disease Gradients of Wheat Stripe Rust

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ABSTRACT

Field data on disease gradients are useful for helping to confirm or refute the validity of gradient models that are derived from the physical mechanisms of dispersal, as well as to provide accurate input information for models designed to predict the behavior of expanding epidemics. In this study, primary disease gradients of wheat stripe rust, caused by the aerially dispersed fungal pathogen *Puccinia striiformis*, were measured in Madras and Hermiston, OR in the springs of 2002 and 2003. Disease foci were created by inoculating a 1.52m x 1.52m area in each of three replicate field plots (6.1m wide x 131 to 171m long) in each of the four experiments. Primary disease gradients were measured both upwind and downwind of the focus. Four gradient models—the power law, the modified power law, the exponential model, and the general model proposed by Lambert *et al.*—were fit to the data. Five of the eight gradients were better fit by the power law, modified power law, and Lambert model than by the exponential, revealing the non-exponentially bound nature of the gradient tails. The other three datasets, which were comprised of the fewest data points, were equally well fit by all the models. By truncating the largest datasets it was shown how the relative suitability of dispersal models can be obscured when data is available only at a short distance from the focus. The truncated datasets were also used to examine the risks of extrapolating gradients to distances beyond available data. The power law and modified power law predicted dispersal at large distances well, even given limited data, while the Lambert and exponential consistently and sometimes severely underestimated dispersal at large distances.
INTRODUCTION

Dispersal of plant pathogen propagules plays an essential role in the spatial spread of plant diseases. Field studies to quantify dispersal usually involve measuring the number of propagules or amount of disease at several distances from a source of inoculum. Fitting simple empirical models to the resulting dispersal or disease gradients can facilitate the interpretation and comparison of gradients (10). Accurate models of spore dispersal are also necessary to create realistic models of the combined temporal and spatial dynamics of plant disease epidemics (7).

Historically, the models used most frequently to represent dispersal and disease gradients have been the exponential model (11),

\[ y = a \exp(-bx) \]  \hspace{1cm} (1)

and the (inverse) power law (10),

\[ y = ax^{-b} \]  \hspace{1cm} (2)

where \( y \) is the number of propagules or amount of disease at a distance \( x \) from the source, \( a \) is proportional to the strength of the source, and \( b \) is a measure of the steepness of the gradient. The power law has the serious disadvantage that it has an infinite value at the source \( (x=0) \), and therefore cannot quantify autoinfection. Mundt and Leonard (16) recommended the following modification of the power law to alleviate this problem:

\[ y = a(x+c)^{-b} \]  \hspace{1cm} (3)

where \( c \) is an offset parameter greater than zero that allows \( y \) to be finite at \( x=0 \). The shape of the curve described by this modified power law is the same as the original power law, but the curve is shifted \( c \) length units to the left along the \( x \)-axis. It has been
suggested that an appropriate value for $c$ is the radius of the source (2,16). Another more general model was proposed by Lambert et al. (12):

$$y = a \exp(-bx^n).$$

(4)

The Lambert model contains a shape parameter, $n$, which enables it to take on a variety of shapes, from a power law (as $n$ approaches 0), to an exponential ($n=1$), to a Gaussian (normal) curve ($n=2$).

Interestingly, although the exponential and the power law were originally used simply as descriptive, empirical models, their utility has also been confirmed by researchers attempting to derive gradient models from the physical principles involved in dispersal. Van den Bosch et al. (19) derived an exponential dispersal gradient for their model of focal expansion of plant disease based on turbulent diffusion within the plant canopy. Minogue and Fry's (14) assumptions that a constant proportion of spores are deposited as the spores move away from the source also yielded an exponential gradient. Aylor and Ferrandino (2) used a modified power law with exponent $b=2$ to describe gradients of bean rust close to a source by assuming that the spores were dispersed in a radially diverging flow field. Ferrandino's (4) inclusion of a vertical movement of spores out of the plant canopy also led to a power law gradient.

Aylor (1) suggested that the processes of diffusion and deposition were responsible for the power law and exponential components of dispersal gradients, respectively. The two processes may be at work in different proportions, depending on the particular circumstances of dispersal, producing gradients of different shapes. Aylor cautions against inferring the mechanism of dispersal based on gradient shape, though,
pointing out that diffusion may dominate over deposition, even when a gradient is fit well by the exponential.

The shapes of the power law and the exponential are superficially similar, especially near the source, and they often fit field data equally well (5,6). They have important differences, however, and some gradient data are fit better by one than the other. Log-linear plots of the models reveal the differences most clearly. Log transformation of the exponential yields the equation \( \ln y = \ln a - bx \), which is a straight line on a log-linear plot. On the same plot, however, the transformed power law, \( \ln y = \ln a - blnx \), is curvilinear. In the tails of the gradient, the power law exceeds the exponential (e.g., see Fig. 1.1). That is, compared to the exponential, the power law represents a dispersal pattern where more propagules travel far from the source. The importance of this difference plays out in the development of epidemics over many generations of the pathogen. According to analytic and simulation studies, epidemics with exponentially bound dispersal gradients (e.g., exponential or Gaussian) expand at a constant rate, as a so-called traveling wave. On the other hand, epidemics with non-exponentially bound dispersal gradients (e.g., power law) expand at a rate that increases with time, i.e., a dispersive wave (1,8,14,15,19,21). The latter type of epidemic has the potential to spread faster and cause more damage than the former.

The Lambert model (equation (4) above) provides a compromise between the exponential and the power law. It can take on shapes intermediate between the power law and the exponential when \( n \) is between zero and one, and it is defined at \( x=0 \). Its tails may be exponentially bound or not, depending on the value of \( n \).
In the present study, primary disease gradients of stripe rust on wheat (caused by _Puccinia striiformis_) from inoculated foci were measured in field experiments in two years and two locations both upwind and downwind of the foci. _P. striiformis_ is aerially dispersed via urediospores produced in foliar lesions. The gradient data were used to investigate issues surrounding the fit of gradient models to field data, particularly in the case when the range of distances is limited. The fits of the exponential model, power law, modified power law, and Lambert model to the field data were evaluated. In some of the experiments, it was possible to gather gradient data quite far from the inoculated focus, and those datasets were used to evaluate the appropriateness of using the various gradient models to estimate dispersal to distances beyond available data.

**MATERIALS AND METHODS**

**Field plots.** Field experiments were conducted at Hermiston and Madras, OR during the springs of 2002 and 2003 as part of a larger study on the effect of cultivar mixtures on epidemic velocity (3). Both sites experience prevailing winds from the west, Hermiston having both stronger winds and stronger directionality than Madras. The growing season is earlier, warmer, and drier at the Hermiston site, in the Columbia Basin of eastern Oregon, than at the Madras site, which is located just east of the Cascade Mountains in central Oregon. No wheat cultivars susceptible to _P. striiformis_ were commonly grown in either area at the time this study was conducted, and risk of contamination of field plots by indigenous populations of the pathogen was low.

The winter wheat cultivar Jacmar was planted in the fall of each year in three plots measuring 6.1m wide by 131 to 171m long, oriented with the long axis parallel to
the dominant wind direction. The plots of Jacmar, which is susceptible to *P. striiformis*, were separated from one another and from mixed-cultivar plots (not involved in this part of the study) by 16.8-m buffer zones planted to the resistant winter wheat cultivar Stephens. Standard commercial cultural practices for each location were applied, including regular irrigation starting in early spring.

A 1.52m x 1.52m area of each plot was inoculated with *P. striiformis* race CDL 5 (CDL = USDA-ARS Cereal Disease Laboratory, Pullman, WA) in the spring of each year. This focus was placed in the center of the plot widthwise, and was shifted upwind of the center lengthwise. The inoculation was accomplished by dusting the wheat plants with a mixture of urediospores and talc powder, under a tent of plastic sheeting the same size as the inoculated square. The inoculated plants were then covered overnight with a plastic sheet, preventing spread of spores beyond the intended inoculation area, as well as ensuring an environment moist enough for spore germination and infection.

**Disease assessments.** For the larger study (3), weekly assessments of disease severity were made beginning at the appearance of sporulating lesions, and continuing until the plants naturally began to senesce at the end of the growing season. In order to characterize the primary disease gradient, an intensive disease assessment was conducted seven to nine weeks after inoculation. For each of the four experiments, this date was chosen to be shortly after the appearance of the second generation of the pathogen, and well before the appearance of the third generation. This second generation was the first to appear outside the focus, and originated from spores produced from lesions that resulted from the artificial inoculation. Assessments were made at markers in the focus, at 0.9, 1.5, 2.1, 3.0, 4.6, 6.1, 9.1, and 12.2m from the center of the focus, and at every
6.1m thereafter, in both the upwind and downwind directions. The outermost marker in each direction was 6.1m or more from the edge of the plot, to avoid the eddying effects that sometimes occur there.

At each marker, two 0.3-m sections of plants were assessed, one in the second row north of the center aisle (where the planter tires ran during planting), and one in the second row south of the aisle. The centers of these rows were 0.9m apart. In each section, the total number of lesions on a designated leaf of each tiller was counted, as well as the number of tillers. The designated leaf was the F-3 leaf in Hermiston both years, the flag leaf (F) in Madras 2002, and the F-2 leaf in Madras 2003. In each case, the leaf was chosen to maximize the amount and accuracy of information gathered. Leaves higher on the plant had fewer lesions, and the counts decreased to zero not far from the focus. Lower leaves had more lesions, which at high severities coalesced, making individual lesions difficult to discern and count. In 2002 at both locations, the level of disease near the focus was too high to count individual lesions on the designated leaf. At these markers only, the severity (percent leaf area covered by lesions) was assessed for that leaf, and later converted to an approximate lesion count by estimating the maximum possible number of lesions per leaf. This procedure was followed at five markers (0 to 3.0m downwind) in Hermiston, and three markers (0 to 1.5m downwind) in Madras. All three replicate plots were assessed in each experiment, except at Hermiston in 2002, where one of the three plots had planting irregularities that prevented accurate assessment. The entire assessment for each of the four experiments took place in either a single day or two consecutive days. Each plot (replicate) was completed in a single day.
Data analysis and model fitting. Data were expressed as the mean number of lesions per leaf for each assessment point. These raw lesion count data represent the primary disease gradient (except in the focus, where two generations of the pathogen were superimposed). To characterize the dispersal gradient, an adjustment was made for multiple infections. Assuming a finite number of infection sites on a leaf's surface, and assuming that, on the scale of a leaf, established infections reduce the likelihood of new infection in proportion to the number of sites they occupy, the number of "effective spores" required to achieve \( y \) lesions of \( N \) total possible lesions on that leaf is \( N \ln(N/(N-y)) \) (9). The term "effective spores" is defined as those spores that, in the absence of prior infection, would succeed in causing a lesion. Following Minogue's advice (13), the transformation was applied to severity values above 10%. None of the data points originally collected as lesion counts corresponded to severities high enough to necessitate the transformation.

In the focus, quantifying the second generation of the pathogen was complicated by the presence of the first generation. (Here, the convention is that the "zeroth" generation consists of the spores used in the inoculation; the first generation consists of the initial lesions in the focus; and the second generation consists of lesions caused by the spores of the first generation.) The principles of the multiple infection transformation were applied in order to estimate dispersal of spores to the focus from infected plants within the focus. As an illustration of the calculation, say the severity of the first generation of lesions on a leaf is 34%. If the number of possible infection sites per leaf is 100, then 34 sites are filled. The multiple infection transformation predicts that 42 effective spores would be necessary to achieve that severity. Say also that after the
second generation appears, the severity is 95%. According to the transformation, a total of 300 effective spores would be required. The difference (300 – 42 = 258) is the number of effective spores needed to increase the severity on that leaf from 34% to 95%, and this figure may be used to quantify second generation dispersal in the focus. Similar calculations were made for the focus in each of the four experiments, using the severity at the time that the primary disease gradient data were gathered (first plus second generation) and the severity two weeks prior (first generation).

The number of lesions per leaf at each distance upwind and downwind of the focus, transformed as described above, was then averaged over the replicates (two in Hermiston, 2002; three in the other experiments). Before fitting the gradient models to the data, mean lesion counts were transformed using the natural logarithm. This transformation improved the residual patterns of the data, correcting the tendency of variances to increase with increasing lesion counts. Data points with mean zero were excluded from the analyses, since the logarithm is not defined at zero. Points beyond these zeroes where lesions were found were also excluded, so as not to introduce bias in those regions. By calculating the means before applying the log transformation, the amount of information available for model fitting was maximized. Applying the logarithm first would have required excluding datapoints where only one of the replicates had zero lesions. Those points were in fact included, since the means at those locations were non-zero.

The exponential model, power law, modified power law, and Lambert’s general model were fit to each of the eight datasets (two directions x two locations x two years) using least squares regression on the logged lesion counts. The focus was excluded from
analysis for the power law, since the negative exponent causes an infinite value to be predicted there. In the case of the modified power law, two different regressions were carried out for each dataset. In the first, all three parameters were estimated concurrently. In the second, the offset parameter, c, was fixed and set at the half-width of the inoculated focus (0.762m). The other two parameters were then estimated using nonlinear regression. To evaluate the relative abilities of the models to fit each dataset, the coefficients of determination ($R^2$) were calculated, and residual plots were examined.

The three largest datasets (downwind gradients for Hermiston 2002, Madras 2002, and Madras 2003) were used to investigate the ability of the gradient models to predict dispersal of wheat stripe rust, given data with a limited distance range. The datasets were truncated to within 30.5m, 18.3m, and 6.1m of the focus, and regressions were performed using the gradients models as described above. Fitted curves derived from the truncated datasets were compared to the full datasets to see how well extrapolations based on those curves described dispersal farther away from the source.

Ferrandino (5) proposed a formal statistical test to determine whether a dataset is significantly better fit by the power law or the exponential. This F-test was performed on all eight datasets, plus the truncated ones derived from the three largest datasets. Since the power law is not defined at $x=0$, observations in the focus were not included for this test.

RESULTS

Although the field plots were similar sizes in both years and both locations, the distance over which non-zero lesion counts were observed did vary. When conditions
were more conducive to disease, the number of data points available for analysis was greater. For each of the eight datasets, there were one or two non-zero observations beyond the first zero observation. These were excluded, along with the zeroes, from the regression analyses.

Based on R²-values and residual patterns, the power law, modified power law, and Lambert model fit the dispersal data considerably better than the exponential did for the five largest datasets (Hermiston 2002 upwind and downwind, Madras 2002 upwind and downwind, and Madras 2003 downwind). The other three datasets, each of which consisted of seven or fewer observations and extended no farther than 6.1m from the focus, were equally well described by all the models (Table 1.1 and Fig. 1.1). In the cases where there was a difference in goodness of fit among the models, the exponential always underestimated values near the source and in the tail of the gradient (Figs. 1.1 and 1.2). For each dataset, the shapes of the fitted curves were similar for the power law, modified power law, and modified power law with c fixed (not shown). The R²-values for these three models also tend to be about the same for a given dataset, although it should be noted that the non-modified power law was fit to a reduced dataset (the focus was excluded).

The Lambert model performed as well as the three versions of the power law in most cases, but showed slightly more bias in the residual plots in the two largest datasets (Hermiston and Madras 2002 downwind). The Hermiston 2002 downwind dataset was the largest and shows the clearest differences among the models (Fig. 1.2). In all cases, the estimated value of the shape parameter of the Lambert model, n, was between zero
TABLE 1.1. Parameter estimates and coefficients of determination ($R^2$) derived from fitting four models to primary disease gradient data of wheat stripe rust from two locations (Hermiston and Madras, OR) in two years

<table>
<thead>
<tr>
<th></th>
<th>Downwind</th>
<th>Upwind</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$a^*$</td>
<td>$b^*$</td>
</tr>
<tr>
<td>Hermiston 2002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exponential</td>
<td>18.59</td>
<td>0.106</td>
</tr>
<tr>
<td>Power Law (focus excluded)</td>
<td>184.9</td>
<td>2.07</td>
</tr>
<tr>
<td>Modified Power Law</td>
<td>431.8</td>
<td>2.29</td>
</tr>
<tr>
<td>Modified Power Law (c fixed)</td>
<td>380.4</td>
<td>2.25</td>
</tr>
<tr>
<td>Lambert</td>
<td>895.9</td>
<td>2.85</td>
</tr>
<tr>
<td>Hermiston 2003</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exponential</td>
<td>4.979</td>
<td>1.005</td>
</tr>
<tr>
<td>Power Law (focus excluded)</td>
<td>1.653</td>
<td>1.94</td>
</tr>
<tr>
<td>Modified Power Law</td>
<td>255.0</td>
<td>4.26</td>
</tr>
<tr>
<td>Modified Power Law (c fixed)</td>
<td>5.052</td>
<td>2.40</td>
</tr>
<tr>
<td>Lambert</td>
<td>7.848</td>
<td>1.66</td>
</tr>
<tr>
<td>Madras 2002</td>
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<td></td>
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<tr>
<td>Exponential</td>
<td>2.372</td>
<td>0.149</td>
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<tr>
<td>Power Law (focus excluded)</td>
<td>14.20</td>
<td>2.09</td>
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<tr>
<td>Modified Power Law</td>
<td>17.25</td>
<td>2.12</td>
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<tr>
<td>Modified Power Law (c fixed)</td>
<td>40.50</td>
<td>2.39</td>
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<tr>
<td>Lambert</td>
<td>227.9</td>
<td>3.92</td>
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<tr>
<td>Madras 2003</td>
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<td></td>
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<tr>
<td>Exponential</td>
<td>1.805</td>
<td>0.245</td>
</tr>
<tr>
<td>Power Law (focus excluded)</td>
<td>5.804</td>
<td>2.11</td>
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<tr>
<td>Modified Power Law</td>
<td>112.6</td>
<td>3.02</td>
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<tr>
<td>Modified Power Law (c fixed)</td>
<td>10.16</td>
<td>2.24</td>
</tr>
<tr>
<td>Lambert</td>
<td>10.30</td>
<td>1.59</td>
</tr>
</tbody>
</table>

* $a$, $b$, $c$, and $n$ are parameters of the models fit to the gradient data. The models are as follows:
- exponential $y = a^\cdot \exp(-bx)$;
- power law $y = a^\cdot x^b$;
- modified power law $y = a^\cdot (x+c)^b$; and
- Lambert $y = a^\cdot \exp(-bx^c)$; where $y$ is the number of lesions per leaf at a distance $x$ in meters from the center of the inoculated focus. Where $c$ of the modified power law was fixed, its value was the half-width of the inoculated focus.
Fig. 1.1. Primary disease gradients of wheat stripe rust from four field experiments (two locations x two years) downwind and upwind of an inoculated focus, fit to three models. Open circles represent the means of two or three replicate plots.
and one, indicating a non-exponentially bound shape intermediate between the power law and the exponential.

The models responded differently to the effect of truncation (Fig. 1.3). For the exponential, the fitted line became steeper as the amount of data used to fit the model was reduced. As a consequence, the underestimation of dispersal far from the source was more severe when the data was truncated more. The Lambert model also underestimated dispersal far from the source when the data was truncated, although somewhat less than the exponential. The three power laws did the best job of predicting dispersal accurately for distances beyond those used in the regressions. The modified power law with \(c\) fixed at the half-width of the focus was less satisfactory than the other two power law models: although it did as well as the others in predicting dispersal far from the source, values near the source were underestimated. The Madras 2002 downwind gradient, which did not decline smoothly with distance from the source, was difficult for all the models to fit, with or without complete data.
Fig. 1.3. Validity of extrapolation. Downwind disease gradients of wheat stripe rust from three field experiments, fit to five models. Open circles represent means of two or three replicate plots. Lines show results of regressions using either complete or truncated data. Legends show the distance from the focus to the farthest data point used for model fitting. The focus (distance=0) was excluded from power law calculations.
Ironically, although the tendency of the exponential to underestimate dispersal far from the source worsened when the data was truncated, the model appeared to fit the data included in the regression better when there were fewer data points (Fig. 1.4). While the exponential provided a clearly inferior fit to the full datasets, the $R^2$ was nearly the same as for the other models when the data were truncated to 6.1 m. That is, when data are available only relatively close to the focus, it becomes more difficult to distinguish the relative suitability of the models. This same effect was probably also at work in the three smallest untruncated datasets (Hermiston 2003 upwind and downwind, and Madras 2003 upwind), where no model fit better than any other.

Ferrandino’s F-test (5) indicated no significant difference in the fit of the power law versus the exponential for any dataset with a range of less than 18 m ($p>0.05$), including the artificially truncated ones. The power law fit significantly better for the three largest datasets (Hermiston 2002 downwind, and Madras 2002 and 2003).
downwind) \((p<0.05)\). The Hermiston and Madras 2002 downwind gradients were fit significantly better by the power law, even when truncated to 30 or 18 m \((p<0.05)\).

**DISCUSSION**

The importance of characterizing the shape of the dispersal gradients of plant pathogens has been demonstrated in previous analytic and simulation studies on the spread of plant diseases. Power law dispersal has been shown theoretically to produce focal epidemics with continually increasing rates of expansion, in contrast with the constant rate of expansion associated with exponential dispersal \((4,8,14,15,19,21)\). In addition, Shaw \((18)\) showed that, when the dispersal gradient was non-exponentially bound (he used a Cauchy distribution), an irregular pattern of daughter foci surrounding the original focus of infection was possible. When the exponential was used for dispersal in his simulations, no daughter foci appeared.

The current study illustrates both the difficulty and the necessity of gathering gradient data relatively far from the source in order to distinguish among competing gradient models. Of the three gradients that Ferrandino’s F-test \((5)\) indicated were significantly better fit by the power law, none revealed this characteristic within 6.1 m of the focus. This can be seen graphically in log-linear plots \((\text{Fig. 1.3})\), where an exponential gradient is represented by a straight line and a power law gradient has curvature. The steep portions of the gradients at close to the focus are equally well described by either model.

As this study also demonstrates, it may not always be possible or practical to obtain sufficient data to adequately characterize the gradient. Upwind gradients were
always steeper than downwind gradients in the same year and location, which is a typical pattern (6,7). This was indicated by the larger upwind $b$-values of both the power law and the exponential (Table 1.1). (Interpretation of $b$ for the modified power law and Lambert model is trickier, since the three parameters are highly correlated when estimated simultaneously using nonlinear regression.) This means that lesion density decreased more rapidly upwind than downwind, and fewer non-zero observations could be made (Fig. 1.1).

The largest pair of datasets was taken in Hermiston in 2002, when both the initial inoculation and the second generation of the pathogen were extremely successful. The resulting high lesion counts allowed an accurate and unambiguous interpretation of the gradients as non-exponentially bound. The conditions in Madras in 2002 were also quite conducive to disease, and gradients clearly show the curvature in the log-linear plots that indicates a better fit by a power law than the exponential (Fig. 1.1). In this experiment, and in Madras in 2003, it was also possible to detect lower lesion densities than in Hermiston, which extended the number of non-zero observations. Higher tiller densities at the Madras site meant that smaller lesion densities (lesions per tiller) could be detected. Counting lesions on more tillers at each assessment site in Hermiston might have yielded more information about the gradients, but this was deemed impractical, as the procedure used was already quite labor intensive.

Unambiguous characterization of gradients may not be possible in all situations. In the current study, plot size was not a hindrance—zero observations were made before the end of the plot was reached in all eight cases—but in some studies this may be a limitation. High disease levels were also necessary to obtain gradient data far from the
source. This required both a large source of inoculum (from a successful initial inoculation) and favorable conditions for infection outside the focus. The ability to detect very low levels of disease is also an advantage, and should be considered when designing a sampling method. To further complicate matters, the length scale required to characterize gradient tail shapes likely differs depending on the pathosystem and environmental conditions such as wind speed.

Ferrandino’s F-test (5) formally confirmed the conclusion that the downwind gradients in Hermiston 2002 and Madras 2002 and 2003 were better fit by the power law model than the exponential. Examination of log-linear plots, residual plots, and $R^2$-values suggested two more datasets (Hermiston and Madras 2002 upwind) where the fit of the exponential was inferior to the other models. For all five of these datasets, there was obvious curvature in the log-linear plots (Fig. 1.1), indicating poor fit by the exponential.

The Lambert model described the gradients well in all cases, but even with the added flexibility provided by the shape parameter, it did no better than the power law or modified power law (Table 1.1, Fig. 1.1). In all cases, the Lambert model achieved a shape between the power laws and the exponential, which is evident both in the plots of the fitted curves (Fig. 1.1) and in the fact that the estimated $n$-values are between zero and one.

Truncating the larger datasets illustrates what might happen when gradient data are available only relatively near the source. The three datasets used for this exercise were the same ones identified by Ferrandino’s F-test (5) as significantly better fit by the power law than the exponential. Log-linear plots also showed this relationship plainly
(Fig. 1.1). However, when the distance range of the data was reduced to 6.1m, the preference for the non-exponentially bound models disappeared. $R^2$-values for the exponential became quite high and were comparable to those of the other models. This phenomenon alone may be responsible for the fact that no difference in model fit could be detected in the three smallest datasets, which extended to only 3.0m, 4.6m, and 6.1m from the source.

Since gradient data are often available only near the source of disease propagules, those constructing models of disease spread may be forced to make assumptions—explicit or implicit—regarding the behavior of gradients at distances beyond the available data. With the three largest datasets presented here, there exists the opportunity to address the question of whether, if less extensive data had been available, empirical gradient models would be capable of giving reasonable estimates of dispersal in the tails of the gradient.

As the amount of data was reduced, the steepness of the exponential increased (Fig. 1.3), a phenomenon also noted by Aylor (1). This means that, given a gradient of power law form, the exponential underestimates dispersal at large distances more severely when the distance range of the data is smaller. Evaluated on the basis of the smaller dataset only, though, the exponential may appear to perform just as well as other models (Fig. 1.4).

Of the four remaining models, the two with only two free parameters—the power law and the modified power law with $c$ fixed—predicted the shape of the tails of the gradients the best. The other two models—the modified power law and the Lambert—contain an additional parameter. The flexibility provided by the third parameter
apparently allows the models to be more easily "mislead" by limited data. This effect was less serious for the modified power law: only the Hermiston 2002 gradient was severely underpredicted in the tail, and only when the dataset was truncated to 6.1m. In every case, though, the flexibility of the Lambert model caused the tails of the gradient to be underestimated, given incomplete data (Fig. 1.4).

Another useful observation from the truncation exercise depicted in Fig. 1.3 is that, when the models were inaccurate in extrapolating beyond the available data, the error was almost always an underprediction. This could seriously affect predictions of the rate of epidemic expansion made on the basis of these models.

In this study, an effort was made to use disease gradient data of wheat stripe rust to provide information about the dispersal gradient. The dispersal gradient is the spatial distribution of disease propagules around the source of those propagules. Since not all propagules produce disease, the disease gradient that results from dispersal even in a spatially homogeneous crop may not have exactly the same shape as the dispersal gradient. Gregory's multiple infection transformation (9) was used to estimate the number of propagules necessary to achieve the observed levels of disease, and therefore the gradients presented here were taken to be proportional to dispersal gradients.

Consequently, the gradients presented here may be comparable to gradients predicted by physical models of dispersal (e.g., 1,2,4,14,19). In that context, it is notable that the estimated value of the steepness parameter for the power law ($b$) for the three best-defined gradients was around two (Table 1.1). This was the value predicted by Aylor (1) and close to the value (1.89) that Ferrandino used in his study on dispersive epidemic waves (4). Also, when constructing models of the combined spatial and
temporal dynamics of plant disease epidemics, it is the dispersal gradient—or some approximation of it—and not necessarily the disease gradient that should be used to represent dispersal.

In conclusion, under some circumstances, it is possible to measure disease gradients well enough to characterize the nature of their tails. Data may need to be gathered at relatively large distances from the source of disease propagules, though, to make this determination. A variety of factors, including small plot size, low disease levels, and insufficiently sensitive sampling techniques, can hinder the collection of sufficient data. More information is needed about the shape of dispersal gradients to help resolve the question of whether, and under what circumstances, traveling or dispersive epidemic waves exist (20). They are hypothesized to be associated with exponentially bound and non-exponentially bound dispersal gradients, respectively (4, 8, 15,17). In this study, gradients of wheat stripe rust were fit better by non-exponentially bound functions (power law, modified power law, or Lambert) whenever there was a difference in fit among models. And indeed, all of the epidemics that developed following the gradient measurements presented in this paper spread as dispersive waves (3).
LITERATURE CITED


Title:

The Effects of Dispersal Gradient and Pathogen Life Cycle Components on Isopathic Velocity in Computer-Simulated Plant Epidemics

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ABSTRACT

The velocity of expansion of focal epidemics was studied using an updated version of the simulation model EPIMUL. Input parameters for the model were based on wheat stripe rust, caused by *Puccinia striiformis*, an aerially dispersed foliar pathogen. Dispersal data were derived from an artificially inoculated field epidemic of stripe rust. Lesion growth rate, latent period, infectious period, multiplication rate, and dispersal gradient steepness were varied within ranges reasonable for this disease. All but the infectious period had a strong influence on velocity. Three different equations were employed in turn to describe dispersal: the modified power law, the exponential model, and Lambert's general model. The exponential, which fit the gradient data from the field epidemic poorly, yielded an epidemic that expanded at a constant velocity, after an initial buildup period. Both the modified power law and the Lambert model fit the field data well, and produced gradient curves of similar shape. Simulations run with the modified power law and the Lambert model resulted in velocities that increased over time for the entire course of the epidemic, supporting the existence of dispersive epidemic waves.

INTRODUCTION

One of the fundamental tasks of plant pathology is to understand the spread of plant diseases in time and space. Focal epidemics, in particular, have drawn considerable interest (64,67). These are epidemics in which disease spreads outward over time from the initial site of infection. Diseases such as potato late blight (caused by *Phytophthora infestans*) and wheat stripe rust (caused by *Puccinia striiformis*), among others, exhibit focal spread in the field (69). To quantify this type of disease spread, Berger & Luke (8)
introduced the concept of *isopathic velocity*. An *isopath* is a contour in space of constant disease level. As a focal epidemic progresses, isopaths expand outward from the center of the focus. The rate of movement of an isopath in a particular direction is its *velocity*, and is expressed in units of distance per time (e.g., m/day).

Understanding how quickly epidemics spread in space has important implications for controlling disease, as fast-moving epidemics may be far more challenging to combat than slow-moving ones. In addition, since dispersal is a universal phenomenon in ecology, work on the spread of plant pathogens may inform study of a variety of other systems of practical interest, including spread of arthropod pests and weeds (23), spread of invasive animal species (2), restoration and conservation of natural vegetation (23), geographic movement of genetic traits in a population (51), deployment of biocontrol organisms (19,23,67,69), and the potential dispersal of genetically modified organisms (GMOs) (69).

Research on the expansion of plant disease foci in the field is expensive both in terms of labor and the amount of land required (37), and field data on epidemic velocity are limited (but see 1,2,6,8,10,20,25,35,40,46,59). Typically, the spatial extent of experimental plots restricts the ability of researchers to characterize velocity over an extended period of time, as isopaths quickly move beyond the borders of small plots. Analytic and simulation-based approaches have yielded some insight, but have also given rise to a controversy that field experiments have yet to resolve (67). The disagreement lies in whether plant epidemics expand at a constant velocity, after a brief buildup period, or whether velocity is unbounded and increases over time. Epidemics with constant velocity are said to expand as *traveling waves*, with disease gradients (plots of disease
level vs. distance from the source) that retain a constant shape after the buildup period is past. *Dispersive waves*, on the other hand, are exhibited by epidemics with increasing velocity; disease gradients become shallower as the epidemic progresses (15).

Analytic studies have resulted in conflicting conclusions regarding this controversy. While the models developed by Minogue and Fry (39) and van den Bosch *et al.* (60,61) predicted a constant velocity, Ferrandino (15) found that velocity should increase with time. All three models represent fungal diseases that are spread via airborne spores. The important difference appears to be in the assumptions regarding the dispersal of those spores. In order to incorporate a spatial aspect into disease models, it is necessary to make assumptions about the distribution of propagules around a source plant. This dispersal gradient (or, equivalently, contact distribution) may be derived from physical principles such as turbulent diffusion (15,18,60) or may be empirical, based on experimental observations of spore dispersal (14,17).

Mollison (41) showed that traveling wave solutions are expected only if the tail of the dispersal gradient is exponentially bound, while dispersal gradients with non-exponentially bound tails produce dispersive waves. Indeed, the physical assumptions of Minogue and Fry (39) and van den Bosch *et al.* (61), though different, resulted in exponentially bound dispersal gradients—the double geometric and Bessel distributions, respectively—and their models predicted traveling waves. In contrast, Ferrandino’s dispersal gradient was not exponentially bound and resulted in a dispersive wave (15). Simulation studies of epidemic velocity show the same connection between dispersal gradient and velocity (14,52,68,70).
The issue, it seems, would be simple enough to resolve if the “true” nature of the tails of dispersal gradients (i.e., exponentially bound or not) could be determined. The nature of the tails of the dispersal gradient may be different for different plant-pathogen systems, depending on such factors as the mechanism of dispersal, crop structure, and local environmental conditions (18). Fitt et al. (17) suggested that dispersal gradients of splash-dispersed droplets were fitted better by the exponential model, while those for fungi with small (<10μm) air-borne spores were fitted better by the power law, which is not exponentially bound in the tails. There is, however, no definitive rule for predicting the character of the dispersal gradient, and indeed it is often impossible to determine from a given dataset whether the gradient tail is exponentially bound or not (16,17,20). One difficulty is that exponentially bound functions (such as the exponential, equation (1) below) and non-exponentially bound functions (such as the modified power law, equation (2) below) can take on very similar values, particularly near the source, where data are easiest to come by. Dispersal data collected at relatively large distances from the source, therefore, are critical to characterizing a dispersal gradient and have the potential to shed light on the behavior of epidemic velocity as well.

Factors influencing the shape of the dispersal gradient, then, can have an effect on epidemic velocity. Generally speaking, however, they cannot easily be manipulated to control disease spread. Other factors that may influence velocity are characteristics of the pathogen’s life cycle, which may in turn be influenced by environmental conditions (e.g., temperature, humidity, dew period), host vigor and genetic resistance, pathogen virulence, and pesticide application. Berger and Luke found that resistance in oats to crown rust was associated with reduced epidemic velocity (8). Jeger et al. observed
reduced spread of *Septoria nodorum* on wheat in mixtures of susceptible and resistant plants, relative to pure stands of the susceptible variety (25). On the other hand, Minogue and Fry's field study detected no difference in the rate of spread of potato late blight among plots of susceptible plants, moderately resistant plants, and susceptible plants with a fungicide applied (40).

Components of a pathogen's life cycle that may affect the progress of plant epidemics include sporulation rate, infection efficiency, length of latent period, length of infectious period, and rate of lesion growth (7,44). There is a good deal of recent interest in quantifying these components for a variety of pathosystems in order to assess host resistance (e.g. 11,12,55,58), select pathogen strains for biocontrol of weeds (32), and evaluate the effect of cultural practices on disease (48). The interaction of these components over multiple generations is complicated, however, and analytic methods and simulations have been used to integrate their effects into succinct measures of disease, such as rate of disease increase ($r$), area under the disease progress curve (AUDPC), severity at a given date (30,34,45,49,54,56,63), and velocity (15,39,61). Of those studies where velocity has been examined, none has incorporated lesion growth as a disease component. For diseases with indeterminate lesion size, such as wheat stripe rust and potato late blight, the rate of lesion growth can significantly affect disease progress (7,29).

The current study examined the velocity of focal epidemic expansion in the context of a particular pathosystem, wheat stripe rust. The computer simulator EPIMUL was used to demonstrate the sensitivity of velocity to the mathematical representation of
the dispersal gradient, and to address the question of how components of the pathogen’s life cycle, including lesion growth, affect epidemic velocity.

MATERIALS AND METHODS

A baseline simulation was created using archival and field data to mirror an actual wheat stripe rust epidemic that took place as part of a field experiment. Each of six factors (dispersal gradient equation, steepness of dispersal gradient, latent period, infectious period, lesion growth rate, and reproduction rate) was then varied one at a time to determine its effect on the velocity of expansion of the focal epidemic.

Simulation model. The computer simulator EPIMUL was first introduced by Kampmeijer & Zadoks (26) to address the effectiveness of host mixtures and mosaics in controlling disease. The version of EPIMUL used in this study is essentially the same as the original, with a few enhancements (power law dispersal (43) and lesion growth (29), plus asymmetrical dispersal and exponential or Lambert model dispersal, as described below).

EPIMUL divides the host plant population into a two-dimensional spatial grid, in which each cell of the grid may represent an individual plant, a group of plants, or a field, depending on the application. The user may specify the arrangement in the grid of hosts of varying degrees of susceptibility to a pathogen, as well as the location and quantity of pathogen inoculum that initiates the epidemic. Each cell of the grid is divided into a number of potential infection sites, representing unit lesions, each of which exists in one of four states: healthy, latent, sporulating, or removed.
Disease propagules (spores) are dispersed daily among the cells according to one of three dispersal functions: the modified power law model (42),

\[ y = a(x+c)^b \]  

(1)

the exponential (27),

\[ y = a\exp(-bx) \]  

(2)

or a more general model proposed by Lambert et al. (28),

\[ y = a\exp(-bx^n) \]  

(3)

which is capable of describing curve shapes between those of the first two models. In each dispersal function above, \( y \) is the number of effective spores dispersed to a distance of \( x \) from the source lesion. The parameter \( a \) is proportional to the amount of inoculum produced at a source location; \( b \) influences the steepness of the gradient; \( c \) is an offset parameter that allows the power law to have a finite value at \( x = 0 \); and \( n \) is the shape parameter of the Lambert model. (Note, however, that when performing regressions to fit the modified power and Lambert models to data, the parameters are strongly correlated, so interpretation of their values should be approached cautiously.)

After dispersal, new infections are initiated in proportion to the number of uninfected sites in the destination cell. New lesions begin producing spores after a fixed latent period, and continue until the infectious period is complete, after which the site is considered removed—neither infectable nor infectious. While infectious, lesions grow at a rate proportional to the number of healthy sites in the cell. Plant tissue that becomes infected via lesion growth does not pass through a latent period before beginning to produce spores. The rate of pathogen reproduction is the daily multiplication factor (DMFR): the number of daughter lesions per parent lesion per day that would be
produced in an unlimited, completely healthy and susceptible population. This parameter incorporates spore production, infection efficiency, and loss of spores to the ground and atmosphere. It can therefore be thought of as the number of “effective spores” to originate from a lesion each day.

EPIMUL was modified for this study from previous versions in order to (a) allow a grid as large as 105 x 105 cells; (b) include the exponential and Lambert models as options for dispersal gradients, in addition to the modified power law model already available (43); (c) allow the input of both upwind and downwind gradients, to simulate the asymmetrical dispersal typical of directional wind patterns; and (d) produce an additional output data file to speed calculations of isopath velocities.

Field experiment. The baseline simulated epidemic was constructed to mimic a field experiment conducted in Hermiston, OR during the spring of 2002. This experiment was chosen from four possible experiments (two locations, two years) for the precision and extent of the dispersal data available (47). A full description of the experiment is given by Cowger et al. (10).

The winter wheat cultivar Jacmar, which is highly susceptible to P. striiformis, was planted in three 6.1m-wide field plots. The lengths of the plots were 131m, 146m, and 171m. Plots were oriented approximately parallel to the prevailing winds and were separated from one another and other inoculated plots by 16.8m swaths of the resistant winter wheat cultivar Stephens. The wheat was planted in October 2001. In March 2002, a square area in each plot measuring 1.52m x 1.52m, centered in the crosswind direction, and shifted upwind of center in the other direction, was inoculated using urediospores of
*P. striiformis* race CDL 5 (CDL = USDA-ARS Cereal Disease Laboratory, Pullman, WA).

The severity (percentage of leaf area visibly affected by stripe rust) was then assessed weekly in the three plots at 6m or closer intervals upwind and downwind from the focus (the location of the inoculation), until the plants began to senesce naturally at the beginning of June (Fig. 2.1A). An additional, more intensive round of data collection took place after sporulating lesions began to appear outside the focus, but before a third generation of the pathogen was evident (47; also Fig. 2.2). To get a precise estimate of the shape of this primary disease gradient, disease assessments were made at 39 locations in each of two plots, including the focus (assessments in the third plot were impossible due to planting irregularities). At each location, the assessment was made on the F-3 leaf (the third leaf beneath the flag leaf) of each tiller in a 0.3-m section of each of two rows 0.9m apart, one on each side of the long axis of the plot. The assessment consisted of either a count of the total lesions or a visual assessment of percent severity (leaf area covered), and was later converted to an estimate of the number of lesions per leaf (47).

**Baseline simulation. Plot layout.** The same plot layout was used for all simulations, and was constructed to correspond as closely as possible to the conditions of the field experiment. One cell in the EPIMUL grid represented an area the same size as the inoculated area in the field experiment (1.52m x 1.52m). Since the shape of the dispersal gradient may change with the size and shape of the inoculum source (22), the dispersal data collected in the field is directly applicable only to a source the same size and shape. The simulated plot was three rows wide and 89 columns long, with the inoculated focus placed in the second row of the 24th column. This width is slightly
Fig. 2.1. Disease gradients for field and simulated epidemics. A: Six weekly assessments of wheat stripe rust severity in an artificially inoculated field plot. Data shown are the means of three replicate plots. B: Corresponding data output from a computer-simulated epidemic modelled on the field experiment.

Fig. 2.2. Wheat stripe rust disease gradients from Hermiston, OR 2002 field epidemic, fitted to three gradient models. Open circles are mean lesion counts from two replicate plots, transformed with the natural logarithm.
narrower than the field plot, but preserves the position of the focus in the center of the plot.

A number of different calculations were made to determine an appropriate number of potential infection sites per cell. Estimates derived both from field observations and from the literature came to approximately 500,000 infection sites per cell. An initial disease level of 115,000 lesions in the focus cell was calculated using the first generation severity observed in the focus in the field (23% severity * 500,000 infection sites). Since data were gathered weekly for the field experiment, severity data were also extracted from EPIMUL at seven-day intervals.

**Dispersal gradients.** The three gradients models were fit to the lesion count data (means of two replicates) after one generation of dispersal using either linear regression (for the exponential model) or nonlinear regression (for the modified power law and Lambert models) on the logged response. Taking the natural logarithm of lesion counts before analysis improved the residual patterns, correcting the tendency for variances to increase with increasing number of lesions. Locations in the field where no lesions were found in either replicate were excluded from analysis, since the logarithm is not defined at zero. Data points beyond these zeroes where lesions were found were also excluded, in order not to introduce bias in those regions. (This was the remedy for zero readings preferred by Minogue (37)). The modified power law gave a superior fit for both the upwind and downwind gradients, when evaluated using residual plots and $R^2$ values (47; see also Table 2.1 and Fig. 2.2), and therefore was used for the baseline simulation. The modified power law also provided a good fit for three other datasets (six gradients) from similar experiments (47).
TABLE 2.1. Dispersal function parameters estimated from Hermiston, OR 2002 primary disease gradient data

<table>
<thead>
<tr>
<th>Gradient Equation</th>
<th>Downwind</th>
<th>Upwind</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$a^*$</td>
<td>$b^*$</td>
</tr>
<tr>
<td>Modified Power Law</td>
<td>431.8</td>
<td>2.29</td>
</tr>
<tr>
<td>Exponential</td>
<td>18.59</td>
<td>0.106</td>
</tr>
<tr>
<td>Lambert</td>
<td>895.9</td>
<td>2.85</td>
</tr>
</tbody>
</table>

* $a$, $b$, $c$, and $n$ are parameters of the models fit to the gradient data. The models are as follows: exponential $y = a\exp(-bx)$, modified power law $y = a(x+c)^b$, and Lambert $y = a\exp(-bx^q)$, where $y$ is the number of lesions per leaf at a distance $x$ (in meters) from the center of the inoculated focus.

**Latent period.** Temperature is the main factor influencing latent period (the time between infection and first production of the next generation of spores) for stripe rust on wheat. Based on the mean daily temperature at the Hermiston field station during the spring of 2002, 11.7°C, the average latent period was estimated to be about 17 days (50,57,65). Also, in a growth chamber experiment using a diurnal temperature regime similar to that observed in the field at Hermiston during the spring of 2002 and using the same wheat cultivar and pathogen isolate as the field experiment, the average latent period was 17 days (data not shown).

**Infectious period.** Luo and Zeng (33) reported wheat stripe rust infectious periods ranging from 6.05 to 7.39 days in four slow-rusting varieties, and 11.61 days in a susceptible variety. Data given by van den Bosch et al. (59) showed that the bulk of stripe rust spores were produced in the 14-day period starting 6 days after inoculation. The most conservative choice of infectious period for a susceptible cultivar would be the largest of these values, and so 14 days was used for the baseline simulation.

**Daily multiplication factor.** This parameter, also called the DMFR, is the most difficult to estimate. Since it is a measure of effective spores, determining its value directly would mean counting all the daughter lesions produced by a single lesion in a
healthy, susceptible population of infinite extent. In the present case, it would also require an accurate count of the total number of parent lesions in the inoculated focus before dispersal. Instead, an indirect approach was used. The isolated effect of the DMFR is most evident when the first dispersed generation of the pathogen appears outside the inoculated focus. At this time, there is no overlap of generations outside the focus, and thus no reduction of infection efficiency due to already infected tissue. Also, growth of lesions beyond their initial size has not yet begun outside the focus. Trial simulations with various DMFRs were run, and the disease gradient at this time was compared to the field epidemic. The gradients were most similar when the DMFR for the simulation was set at five daughter lesions per parent lesion per day.

**Lesion growth.** Field estimates of the lesion growth rate of *P. striiformis* range from 15% to 76% per day (13,29,34). In preliminary trial simulations, a rate of 25% per day produced a simulated epidemic more similar to the field epidemic than larger or smaller values. This value was thus used for the baseline simulation.

**Simulations performed.** *Dispersal gradient equation.* Keeping all other model parameters at their baseline values, simulated epidemics were run in EPIMUL using the modified power law, the exponential, and the Lambert models, in turn, for dispersal of spores. Dispersal model parameters were derived from regressions (linear or nonlinear) on the logged lesion count data from the Hermiston 2002 field experiment. In each case, upwind and downwind data were fitted separately. Parameter estimates for all three models are shown in Table 2.1. The *a* parameter in each equation has no effect on the shape of the gradient, but is used to determine the proportionality between upwind and downwind gradients. EPIMUL normalizes the combined upwind and downwind
gradients before using them for dispersal, so the form of the gradient equation does not affect the quantity of spores dispersed from each lesion, which is governed by the DMFR.

**Numerical parameters.** To test the effect of the steepness of the dispersal gradient on epidemic velocity, the exponent, \( b \), of the modified Gregory model was varied, with \( a \) and \( c \) held constant. For simplicity, the same gradient was used to model dispersal in both the upwind and downwind directions in this round of simulations. The offset parameter, \( c \), was kept at the baseline downwind value of 0.848m. Using downwind gradients for both upwind and downwind dispersal slows disease spread slightly, but does not qualitatively affect disease gradients or velocity. (In Fig. 2.4, compare the baseline curve in the dispersal gradient slope plot to the baseline curves in the other plots.) Lesion count data from four different field experiments (in Hermiston and Madras, OR in 2002 and 2003) were used to provide a range of reasonable values for \( b \). When regressions were done on upwind and downwind data to fit the modified Gregory model with \( c=0.848\text{m} \), estimates of \( b \) were 2.29, 2.30, 2.43, 2.52, 3.14, 3.52, 4.18, 5.89. The simulations were run using values of 1, 2, 3, 4, 5, and 6; larger values indicate steeper gradients and in these experiments the largest values were associated with upwind dispersal.

Each of the pathogen life cycle parameters (latent period, infectious period, lesion growth rate, and multiplication rate) was then varied, in turn, to explore its effect on epidemic velocity. Model parameters were chosen to encompass a reasonable range of values for stripe rust on wheat. In some cases, more extreme values were also included.

Latent and infectious periods ranging between 5 and 30 days were used. Latent periods of 11.3 to 23.8 days have been reported for stripe rust on ten cultivars of spring
wheat raised in growth chambers (9). Infectious periods between 6 and 14 days have been observed (33,59).

Lesion growth rates were varied from zero to 100% per day.

As noted above, DMFR combines the effects of two disease parameters: spore production and infection efficiency. DMFR is directly proportional to each and is therefore also proportional to the product of the two. The product (spore production) x (infection efficiency) may vary by a factor of up to 100 in cultivars differing in their susceptibility to a pathogen (31,34). In addition to the baseline value of five lesions/lesion/day, simulations were run with DMFRs of 0.05, 0.5, and 50 lesions/lesion/day.

**Velocity calculations.** In order to calculate the velocity of an isopath at a particular date, the location of the isopath was first estimated. In most cases, the isopath was between two data assessment locations in the field (or in the simulation output). The estimated location of the isopath was calculated by linear interpolation between these two points. The velocity in meters per week was the distance between this location and the previous week’s location. When severity exceeds an isopath level at all points in the plot in the direction of interest, the location of the isopath, and therefore the velocity, cannot be estimated.

**RESULTS**

**Baseline simulation compared to field epidemic.** Disease gradients for both the simulated (power law dispersal) and field epidemics flattened with time (Fig. 2.1). Both epidemics also exhibited isopathic velocities that increased over time (Fig. 2.3, thin
Fig. 2.3. Effect of dispersal gradient form on the velocity of a simulated wheat stripe rust epidemic. Velocity of several isopaths are shown for the Hermiston 2002 field epidemic and three computer-simulated epidemics with different dispersal functions.
thick solid lines, respectively). This was true at all levels of severity, in both the upwind and downwind directions. Velocities in the field epidemic tended to be smaller than corresponding velocities in the simulation.

In the field, disease approached 100% severity at a slower pace than in the simulation. Comparing the severity at the focus (Fig. 2.1) illustrates this difference.

The latent period of stripe rust changes with temperature, shortening as the spring growing season progresses. EPIMUL, however, employs a constant latent period throughout the simulation. The latent periods match well during the bulk of the epidemic (approximately weeks three through nine of the eleven week period). Cold weather in the weeks immediately following inoculation in the field delayed the field epidemic relative to the simulation. To correct for this effect, comparisons between field and simulated epidemics were made starting at the end of the second latent period, rather than at inoculation (Figs. 2.1 & 2.2).

**Effect of dispersal gradient equation on epidemic velocity.** Although the parameters for each of the gradient equations (modified power law, exponential, and Lambert) were estimated using the same field data, the behavior of velocity as a function of time depended strongly on which equation was used in the simulation (Fig. 2.3). The modified power law model, which best fit the dispersal data from the field, resulted in isopath velocities that increased over time. The Lambert model also produced epidemics with increasing velocities, although these were smaller in magnitude than the modified power law velocities.

Simulations performed using the exponential model had velocities that increased for a time, then leveled off. Disease gradient plots of simulations using exponential
dispersal (not shown) reveal gradients that become parallel after the initial buildup period. As expected with parallel disease gradients, the asymptotic velocity of these epidemics does not appear to depend on isopath level. The steeper upwind dispersal gradient, however, resulted in a smaller asymptotic velocity: approximately 2m/week upwind versus 15m/week downwind.

**Effects of life cycle parameters and gradient steepness on epidemic velocity.**

Since the qualitative behavior of the isopathic velocity does not appear to depend on severity level (Fig. 2.3), a single isopath, the 20% severity level, was chosen to represent the results of this round of simulations.

The latent period, rate of lesion growth, multiplication rate, and steepness of the dispersal gradient each had a large impact on epidemic velocity (Fig. 2.4). Increasing the latent period delayed the epidemic, while having little effect on the slope of the velocity curve. Increased lesion growth rate was associated with increased velocity. Decreasing the multiplication rate (DMFR) reduced velocity by decreasing the slope of the velocity curve. Steeper disease gradients, characterized by larger exponents \( b \) in the modified power law model, were associated with shallower velocity curves and smaller velocities. The length of the infectious period did not strongly influence the velocity of the epidemic when the infectious period was between 10 and 30 days. Only a very short infectious period, five days, was capable of delaying the epidemic.

It is also interesting to note that changes in these parameters had no effect on the overall shape of the velocity curve. In every case, velocity increased as the epidemic proceeded.
Fig. 2.4. Effect of disease cycle components on the velocity of the 20% severity isopath in simulated wheat stripe rust epidemics. Model parameters are baseline values (see text), except where specified. Figures show downwind velocities calculated at seven-day intervals.
DISCUSSION

EPIMUL was able to re-create the overall characteristics of the Hermiston 2002 field epidemic: greater spread downwind than upwind of the inoculated focus; long tailed disease gradients that flatten over time; and accelerating movement of isopaths. Early in the epidemics, there is a slight hump in the curves of velocity versus time for both the field and simulated epidemics, indicating a temporary increase in velocity due to the fact that the pathogen generations are relatively discrete in this stage. Disease increases in a stepwise fashion initially, until the generations begin to overlap.

Conceptually, EPIMUL is a fairly simple model of plant epidemics. It is based on Vanderplankian increase of disease in time (62); it is deterministic; and it takes as constant a number of parameters that in reality fluctuate with factors such as weather and plant maturity. Even so, the model does a remarkably good job of simulating the behavior of an epidemic as it progresses in time and space. EPIMUL also has the advantage of requiring relatively few input parameters to produce this realistic response, making it possible to apply it to a variety of pathosystems without undue expense. This version also allows the incorporation of different gradients in different directions, which sets it apart from other models. And, although we did not take full advantage of it in the current study, where the plots were long and narrow, EPIMUL offers the possibility of simulating asymmetrical dispersal in two dimensions.

There were also some differences between the field and simulated epidemics. Upwind of the focus, velocity generally increased over time, although the increase in the field seemed to trail off in the last week of observation, while the simulation continued its acceleration. In Oregon, wheat cultivars generally become less susceptible to stripe rust
with age, and environmental conditions become less conducive for disease as temperatures rise during late spring and early summer. It is likely that these factors simply countered the normal tendency for velocity to increase for the slower movement that occurred upwind. In fact, we have noticed a similar phenomenon for a combination of suboptimal environmental conditions and use of cultivar mixtures, which further reduces epidemic velocity (10).

For each severity level, both upwind and downwind, the estimates of velocity according to the EPIMUL simulation were higher than observed in the field. Because of the multiple assumptions necessary to create a model with a reasonable number of parameters, it is difficult to tease out the sources of such discrepancies. Changes in parameters over time, which the model does not account for, may be involved. Inaccuracies in parameter estimation, particularly the multiplication rate (DMFR), which is difficult to measure, may contribute. Also, all real-world processes are stochastic at some level (52), while the EPIMUL simulations are deterministic. It has been observed (66,69) that an epidemic model with deterministic dispersal produced faster epidemics than the same model with stochastic dispersal.

In addition, the simulated epidemic approached 100% severity faster than the field epidemic. EPIMUL reduces infection efficiency in direct proportion to the amount of healthy host tissue available for new infections. Apparently, this adjustment is insufficiently restrictive for stripe rust on wheat. The model takes into account only the reduced likelihood that a spore will land on uninfected tissue as disease severity increases. In the field, additional factors may be at work. Plants may develop some physiological resistance to new infections as disease severity increases. Also, stripe rust
seems to infect more successfully on lush, green plants (65). The reduced vigor of plants as they become diseased may reduce infection efficiency and slow disease progress relative to the simulated epidemics. (In the Hermiston 2003 experiment, where the field was patchily infected with take-all of wheat, we observed that plants whose vigor was reduced as a result of take-all infection consistently showed less stripe rust than healthier neighboring plants.) This discrepancy may call into question direct comparison of isopaths at high severity levels (say, 60% and higher), but, again, the actual and simulated epidemics are qualitatively quite similar.

Any of a number of equations can be used to represent the hollow curve of a typical dispersal gradient (36). The modified power law, exponential, and Lambert’s equation were chosen for this study because of their common use in phytopathology literature, their ability to fit to our field data, and because they have a finite definition at the source (x=0). The last is a necessary condition for use in simulations, so that autoinfection can be quantified. Gregory’s power law, \( y = ax^{-b} \) (22), fit our dispersal data well when the focus (x=0) values were excluded from the analysis (47), but has the oft-noted property of predicting an infinite value of \( y \) at \( x=0 \). A variety of formulations similar to the modified power law shown above (equation 1) have also been used to represent dispersal gradients of plant pathogens. These include the Pareto probability distribution, \( y = \alpha \theta^{\alpha}(x + \theta)^{-(\alpha+1)} \) (37); and the Cauchy distribution, \( y = (2/\pi b)1/[1+(x/b)^2] \) (53); among others (e.g. 20). These are not necessarily equivalent, however, and neither the Pareto nor the Cauchy fit our field data well (47). The Cauchy, for example, followed the shape of the gradients fairly well at distances of 10m or more, but underestimated dispersal nearer the source.
Our simulations show that the velocity of an epidemic depends heavily on the pattern of spore dispersal. In particular, it seems that the tail of the dispersal gradient—the few spores that travel far from their source—drives the behavior of the epidemic. The three dispersal functions used in this study have different tail shapes. The tail of the exponential, of course, decreases exponentially with distance from the source. The modified power law decreases less than exponentially, producing a so-called fat-tailed distribution. For the dispersal data used here, the fitted Lambert model was intermediate in shape between the two.

Lambert’s general model has the interesting property that its tails can be either exponentially bound or not, depending on the value of the shape parameter, $n$. As $n$ approaches zero, the model converges to Gregory’s power law (22), which is not exponentially bound. Setting $n$ to 0.5 makes the model equivalent to the root function, $y = \sqrt{a_2} \exp(-ax^{1/2})$ (20), which is also not exponentially bound. The exponential is obtained with $n = 1$, and a normal curve results when $n = 2$ (28). The latter two have exponentially bound tails. In the present case, nonlinear regression to fit the Lambert model to dispersal data resulted in $n$ values near 0.3 (Table 2.1), and resulted in a gradient curve similar in shape to that of the modified power law (Fig. 2.2).

Simulations using the modified power law or Lambert’s model had velocities that increased over time, with no apparent tendency to level off. This was true over the wide range of $b$ values for the modified power law used in the simulations (Fig. 2.4), which is a further indication of the importance of dispersal gradient shape in determining the nature of epidemic velocity. The difference in the shape of the modified power law and Lambert gradients, though seemingly minor (Fig. 2.2), did cause consistent differences in
the velocity (Fig. 2.3). The modified power law velocity ran just under the Lambert velocity until the last few weeks of the epidemic, when it crossed over and overtook the Lambert.

The velocity of simulated epidemics with exponential dispersal behaved in a very different way from the modified power law and Lambert epidemics. With exponential dispersal, velocity increased at the beginning of the epidemic, then leveled off. This result is in agreement with the findings of other simulation and analytical studies: non-exponentially bound dispersal gradients result in accelerating epidemics, while exponentially bound gradients produce epidemics that reach some asymptotic velocity after a time (14,15,41,52). The present study had the advantage of combining dispersal gradient data derived directly from the field, subsequent estimates of epidemic velocity derived from those same field plots, and a spatially explicit simulation model that reliably reproduced the dynamics of the field epidemics.

These results suggest that characterizing the dispersal gradient is critical to understanding how a focal epidemic progresses in time and space. In the dispersal of *P. striiformis* spores in the windy conditions of Eastern Oregon, the modified power law provided a clearly superior fit to the data than did the exponential law. In determining this, we had the advantage of large plots and environmental conditions favorable to disease. The exponential and power law models tend to fit equally well close to the source (4,47). In fact, had dispersal data been collected to as far as 6m downwind of the focus, the two models would have been virtually indistinguishable ($R^2=0.939$ for the exponential and $R^2=0.992$ for the modified power law, with 7 data points). It became clearer as we moved outward from the focus that the exponential model was
inappropriate and underestimated dispersal at large distances as well as close to the source. Because conditions were favorable to successful infection, the plants in the focus provided an abundant source of inoculum, and during the first generation of dispersal, non-zero levels of disease were detected relatively far from the focus (79.2m downwind, 12.2m upwind). Other disease systems may require different scales of observation to characterize dispersal.

Having observed the acceleration of this epidemic in the field and determined that it can be explained via simulations using a power law dispersal model, the next logical question is whether this acceleration can continue over larger spans of time and space. At the present, this is unknown. The dispersal gradient may or may not continue to follow the power law on larger scales. Also, continued acceleration of epidemics may be hindered by geographic factors, climatological factors, and decreasing availability of suscepts farther from the source (5,23). Seasonal changes in weather and host availability could limit the ability of an epidemic to continue to accelerate maximally.

Our findings that latent period, multiplication rate, and dispersal gradient steepness had strong effects on epidemic velocity are consistent with the results of the analytic studies of van den Bosch et al. (61) and Minogue and Fry (39). Minogue and Fry (39) also concluded, as we did, that infectious period had little effect on velocity, except when the infectious period was very small. In addition, our model included lesion growth, which had a strong influence on velocity. These components of the pathogen’s life cycle have been shown in other studies to have similar effects on non-spatial measures of disease, such as the area under the disease progress curve (AUDPC) (34,45), disease severity over time (54,56,63), and rate of disease increase (r) (30). In summary,
our studies confirm that the shape of the dispersal function is key to the nature of epidemic velocity. In our simulations, a traveling wave of constant velocity was attained, after a period of stabilization, only for an exponential model. However, non-exponentially bound dispersal models provided better fits to primary disease gradient data of wheat stripe rust, and always resulted in dispersive epidemic waves when incorporated into simulations. Pathogen life cycle components had quantitative effects on epidemics velocity, though these were usually similar to previously reported effects on epidemic progression in time. More importantly, the life cycle components did not influence velocity qualitatively when power law dispersal was simulated: velocity always increased in time over the wide range of input values for life cycle components that we investigated. Thus, dispersive epidemic waves could result for a diversity of plant diseases, provided that their dispersal follows a non-exponentially bound function such as the power law.
LITERATURE CITED


CONCLUSION

Analysis of primary disease gradient data of wheat stripe rust led to the conclusion that dispersal of *Puccinia striiformis* urediospores was described better by non-exponentially bound dispersal models than by the exponential. When measurement of the primary disease gradient was restricted to within relatively short distances of the source of the spores, this relationship was obscured, since all gradient models fit equally well close to the source. It was also demonstrated that the power law and modified power law better predict dispersal to distances beyond the available data than the exponential and Lambert models, which tend to underpredict dispersal in that region.

EPIMUL simulations showed that latent period, pathogen reproduction rate, lesion growth rate, and gradient steepness can all have a strong impact on the velocity of expansion of wheat stripe rust epidemics, while the effect of infectious period was small. Simulations carried out using the exponential model for dispersal yielded epidemics that expanded at a constant rate, after a period of initial buildup. Simulated epidemics with power law or Lambert model dispersal had velocities that increased over time and resembled the field epidemic.


