Dynamic Pathogen Distribution and Logistic Increase of Plant Disease

X. B. Yang and D. O. TeBeest

Department of Plant Pathology, University of Arkansas, Fayetteville 72701.
We thank C. L. Campbell and L. V. Madden for comments and suggestions.
Approved for publication by the director of the Arkansas Agricultural Experiment Station.
Accepted for publication 8 January 1992 (submitted for electronic processing).

Random distribution of propagules is one of the basic assumptions of logistic growth models of plant disease (12). Numerous investigations on the spatial patterns of plant diseases commonly have found clustered distributions for soilborne fungal diseases (1), virus diseases vectored by insects (7,8), bacterial disease spread by rain (9), and fungal disease spread by air (10) or by mycelial growth (3,15). In a theoretical study, Waggoner and Rich (13) found that aggregation should slow the rate of disease development if the degree of aggregation remains constant over the course of the epidemic.

Although many experiments show that the logistic assumption of randomness is not true, we still lack a complete fundamental understanding of how aggregation affects logistic disease dynamics (5,6) and, in return, how these dynamics determine the spatial pattern. This letter attempts to examine these relationships.

Detection of aggregation effects. Two methods have been used to examine the effect of aggregation on disease development. The method used by Waggoner and Rich (13) plots disease logistic values (logits) against the time. A departure from a straight line will be observed if the logistic model is not appropriate. The second method is to plot the rate of disease increase against an aggregation index for a group of epidemics with different degrees of spatial clustering but with similar environmental conditions (7). Using the second method, Madden et al (7) demonstrated a negative correlation between the estimated logistic infection rate parameter and final Lloyd's index of patchiness (LIP) in dwarf mosaic of maize. The degree of aggregation is positively associated with LIP. Yang et al (15) also found that the logistic rate parameter was negatively correlated with initial LIP in soybean foliar blight caused by *Rhizoctonia solani*. In examination of additional data on rice sheath blight caused by *R. solani* (3), we found that the rate of increase of this disease was significantly reduced with increasing degrees of spatial aggregation (Fig. 1). A modified logistic differential equation (15) that incorporates LIP fitted the data better than a logistic differential equation without this correction (Table 1). In the above examples, as well as in the calculation of Waggoner and Rich (13), aggregation was assumed a constant during the epidemics.

Rate of increase, constant aggregation, and disease mean. Differential equations for the rate of disease increase can be expressed as:

$$dY/dt = rY(1 - Y) \quad (1)$$

$$dY'/dt = rY(1 - Y)^{(1 + 1/k)} \quad (2)$$

in which dY/dt is logistic rate of increase for randomly distributed disease (12), and dY'/dt is rate of increase proposed by Waggoner and Rich for disease following a negative binomial distribution (13). In both equations, r is the intrinsic growth rate (unit per time), and Y is disease intensity mean. The difference between Equations 1 and 2 is the correction factor $(1 - Y)^{(1 + 1/k)}$, in which k is the dispersion parameter of negative binomial distribution. Values of k are negatively correlated with the degree of aggregation; very small values of k result in tremendous negative feedback. When k approaches infinity, disease distribution converges to random distribution, and Equations 1 and 2 are the same.

To examine how rate of increase is affected by aggregation and by disease mean, a simple relation can be derived by dividing Equation 2 by Equation 1: $P = (dY'/dt)/(dY/dt)$, or $P = [rY(1 - Y)^{(1 + 1/k)}]/[rY(1 - Y)]$. With simplification, P can be written as:

$$P = (1 - Y)^{1/k} \quad (3)$$

From the above derivation, we know that P represents the ratio between rates of increase for disease distributed by aggregation and for disease randomly distributed. The properties of Equation 3 are shown in Figure 2; the effect of aggregation on the rate of increase is determined by both k and Y. P increases geometrically as k increases. When $k > 10$, the effect of aggregation on the rate of increase is unremarkable ($P > 0.9$ for $Y < 0.7$). At a given k, P decreases geometrically as Y increases. When Y is low, the reduction of the rate of increase is small. For the intermediate or low level of aggregation ($k > 4$), reduction is not noticeable in the low to intermediate range of disease means. At this value of k, to cause 20% reduction in the rate of disease increase ($P < 0.8$), the disease mean must be greater than 0.6.

Change of aggregation over time. It is well established from experimental data that aggregation varies with plant disease mean and, therefore, usually with time (5). The decrease of aggregation or a transition from aggregation to randomness during the epidemic has been reported in studies in which disease spatial patterns were monitored over time. This phenomenon was observed for diseases caused by bacteria (9), viruses (7,8), soilborne fungi (2), airborne fungi (10), and leafborne fungi (fungi that spread among leaves by mycelial growth) (3,15). Changes in aggregation in these studies were calculated with different aggregation indices, such as k and LIP. If the same sampling units are repeatedly measured during the epidemic, the change of k over time should reflect the change of aggregation.

As suggested by Taylor (11), potential dependence of k on disease mean can be expressed in terms of dk/dY. For example, the decrease of aggregation of wheat powdery mildew (caused by *Erysiphe graminis*) is much faster than that of rice sheath blight and soybean foliar blight, two Rhizoctonia diseases (Fig. 3). The k-Y relation appears exponential for powdery mildew. Powdery mildew approached a random distribution when $Y > 0.13$ (10), but the Rhizoctonia diseases were still highly aggregated at $Y = 0.16$. Table 2 shows that Rhizoctonia foliar blight has a much slower change in aggregation than that of powdery mildew (Fig. 3).

A modified logistic equation can be written by incorporating an aggregation factor that changes with disease mean:

$$dY/dt = rY(1 - Y)^{(1 + 1/f[Y])} \quad (4)$$

In Equation 4, $k = f(Y)$. Equations of $f(Y)$ were estimated as $f(Y) = 30Y$ for quick dispersal disease using powdery mildew data (10) and as $f(Y) = 6Y$ for a slow dispersal disease using aerial blight data (1988 Ben Hur in Table 2). For diseases with different $f(Y)$, the mean at any point of epidemic can be integrated

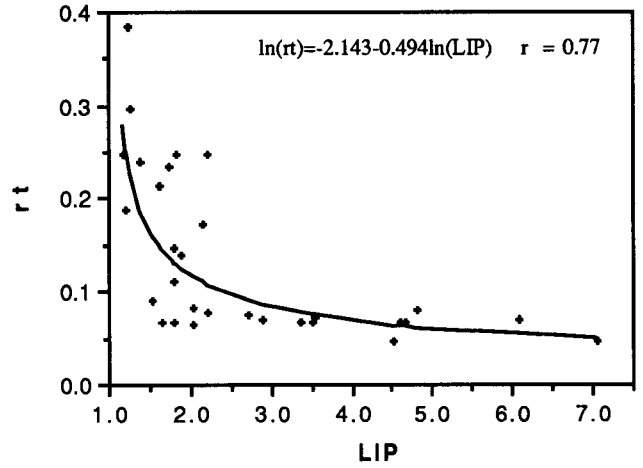

Fig. 1. Relationship between aggregation (expressed as Lloyd index of patchiness [LIP]) and disease increase [$rt = \ln(Y_2/[1 - Y_2]) - \ln(Y_1/[1 - Y_1])$] of rice sheath blight from growth stages heading to late milk. LIP and rt were calculated from data of Gou et al (3, Table 4), in which disease patterns of 30 fields (200 contiguous hills per field) were investigated.

TABLE 1. Regression to determine effect of aggregation on development of rice sheath blight[a]

Model	B_0	B_1[b]	B_2 (LIP[c])	r^2	$P > F$
Logistic	0.0679	1.478	...	0.76	0.0001
Modified					
LIP (time 1)[d]	0.019	0.984	0.168	0.83	0.0001
LIP (time 2)	−0.181	1.006	0.344	0.87	0.0001

[a]Effect can be detected by comparing logistic disease model [$\ln(Y_2/[1 - Y_2]) = B_0 + B_1\ln(Y_1/1 - Y_1])$] with modified logistic model [$\ln(Y_2/1 - Y_2)) = B_0 + B_1\ln(Y_1/[1 - Y_1])) + B_2$LIP], in which aggregation effect on disease increase is corrected. In logistic model, $rt = B_0$. In modified model, $rt = B_0 + B_2$LIP, in which B_2LIP is to correct the effect of aggregation on increase (for derivation see Yang et al [15]). Improvement of modified model is shown by values of the coefficient of determination (r^2), reduction of B_1 toward 1, and the significance of regression coefficient for LIP (B_2). Calculated from data of Gou et al (3, Table 4).

[b]B_1 is expected to be 1 if there is no aggregation effect on disease increase or if such an effect has been corrected.

[c]Lloyd's index of patchiness.

[d]LIP at first or second rating. Transformation of LIP in regression = 1/LIP was made based on Figure 1.

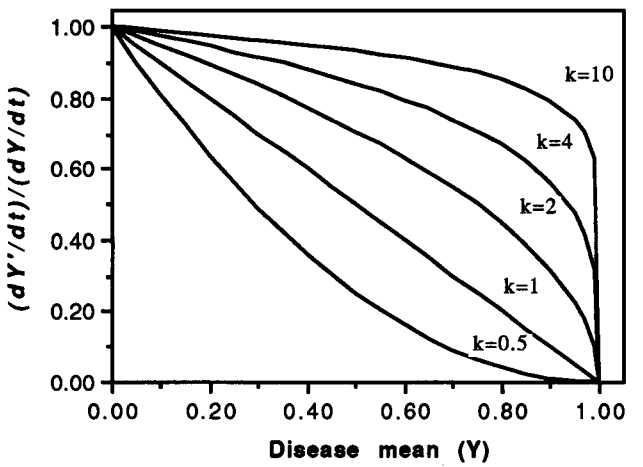

Fig. 2. Relationships among rate of disease increase, disease mean, and degree of aggregation (expressed as k of negative binomial distribution). P is the ratio between rate of increase for aggregated population [$dY'/dt = rY(1 - Y)^{(1 + 1/k)}$] and rate for randomly distributed population [$dY/dt = rY(1 - Y)$], $P = (1 - Y)^{1/k}$.

numerically by using Equation 4 (assuming $r = 0.2$ and $Y_0 = 0.01$) with 1-day intervals. Aggregation effects can be inspected by the method of Waggoner and Rich (13) shown in Figure 4. For a disease similar to powdery mildew (i.e., aggregated at an early stage but spreading quickly), the modified logistic rate of increase is very close to the response for expected random distribution. For an aggregated disease with slow dispersal rate (e.g., the Rhizoctonia diseases), the modified logistic line departs remarkably from the line for expected random distribution. For constant aggregation ($k = 0.5$), the modified logistic increase is the curve departing greatly from the logistic expectation, which is a finding of Waggoner and Rich (13). Another significant point of Figure 4 is that the modified logistic increase of disease is linear for a situation in which aggregation decreases during an epidemic. Such a linearization may explain the fact that the logistic model fits many polycyclic disease curves well, although distributions of the diseases are known to violate the assumption of randomness. Therefore, the apparent infection rates calculated under aggregated conditions are underestimates when compared with the definition of this parameter proposed by Vanderplank (12).

Spread and aggregation. Because dispersal of inoculum results in the spatial dispersion of a disease, there is a relationship between spread and spatial pattern. One theoretical relationship between aggregation and spread of plant disease may be derived from

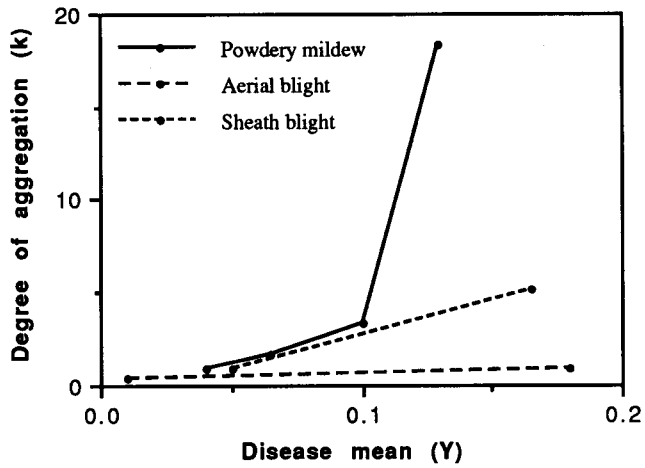

Fig. 3. Relationships between disease aggregation (k is an aggregation index of negative binomial distribution) and disease mean Y for wheat powdery mildew (10), rice sheath blight (calculated from data of 30 fields, [3]), and soybean foliar blight (see Table 2, 1987 Burden field A). Values of k are negatively correlated with the degree of aggregation. The rate of change of aggregation (dk/dY) is different for the three diseases. Sampling units were tillers for powdery mildew, plant for sheath blight, and 0.75- × 0.75-m quadrate for foliar blight, respectively. A smaller k would be expected for aerial blight if the sampling unit had been a plant.

TABLE 2. Change of aggregation (k of negative binomial) in relation to disease means for soybean foliar blight by *Rhizoctonia solani* in six fields in Louisiana with sampling quadrate, 0.75 × 0.75 m (calculated from Yang et al [15])[a]

		Disease mean		k of Negative binomial	
Year	Location	Time 1[b]	Time 2	Time 1	Time 2
1987	Lake Arthur	0.016	0.177	1.78	1.47
1987	Burden A	0.011	0.175	0.45	0.87
1987	Burden B	0.013	0.251	0.97	2.12
1988	Lake Arthur	0.008	0.265	0.28	3.03
1988	Ben Hur	0.014	0.386	0.51	3.13
1988	Burden	0.006	0.267	0.30	2.17

[a]In comparison with Figure 3, smaller values of k would be expected if a plant had been taken as sampling unit.

[b]Time 1 and time 2 were soybean growth stages V9–11 and R4, respectively.

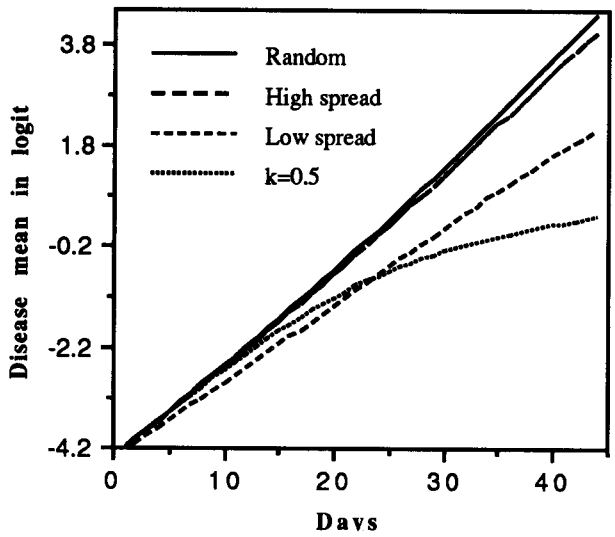

Fig. 4. Effects of aggregation on disease development. The effects are indicated by the departures of lines (logit scale) for aggregated diseases from the logistic expectation for a randomly distributed disease. A numerical simulation was done by using a modified rate equation $dY/dt = rY(1 - Y)^{(1 + 1/f(Y))}$, in which $f(Y)$ represents degree of spatial aggregation of disease expressed as a function of disease mean. For a disease with rapid rate of spread, degree of aggregation for $Y < 0.15$ was expressed as $f(Y) = 30Y$, and the random distribution was assumed for $Y > 0.15$ (estimated from data of Rouse et al [10]). For a disease with low rate of spread, degree of aggregation was as $f(Y) = 6Y$ for any point of epidemic (estimated from data of Yang et al [15]). If aggregation is constant during the epidemic, $f(Y) = 0.5$. For all cases, $r = 0.20$, $Y_0 = 0.01$.

a spatial temporal model for polycyclic disease proposed by Jeger (see ref. 4, Eq. ii). His model is:

$$\partial Y/\partial t = rY(1 - Y) \quad (5a)$$

$$\partial Y/\partial s = -cY(1 - Y) \quad (5b)$$

in which $\partial Y/\partial t$ and $\partial Y/\partial s$ are the partial differential equations for the disease development in time and space, respectively. Constant c is the disease gradient parameter. By dividing Equation 5a by Equation 5b, Jeger expressed the rate of isopath movement as $\partial s/\partial t = -[(\partial Y/\partial t)/(\partial Y/\partial s)]$, in which $\partial s/\partial t$ reflects the rate of disease spread. When disease is aggregated, $\partial Y/\partial t$ in Equation 5a may be better described by the equation of Waggoner and Rich, and then we have:

$$\partial Y/\partial t = rY(1 - Y)^{1 + 1/k} \quad (6)$$

After dividing Equation 6 by Equation 5b, we have $\partial s/\partial t = (r/c)(1 - Y)^{1/k}$. Let $\partial s/\partial t = v$, and by algebraic manipulation, we have:

$$k = \ln(1 - Y)/\ln(vc/r) \quad 0 < Y < 1, \; vc/r < 1 \quad (7)$$

Because both denominator and numerator are negative, k is positively associated with v. Numerical results for Equation 7 (Fig. 5) were obtained by assigning c and r the same values as those used by Jeger (4). The relationship shows that as k declines, v should decline for any given value of Y. Stated another way, a higher rate of disease spread leads to a faster reduction in the degree of spatial aggregation. The above relationship, however, was derived through mathematical logic. To our knowledge, there are no data available for validation that directly relate spread to spatial pattern. Also, the assumption of a constant v and r during the epidemic may not occur in reality. Nevertheless, this idea may be a clue to tackle this challenging problem.

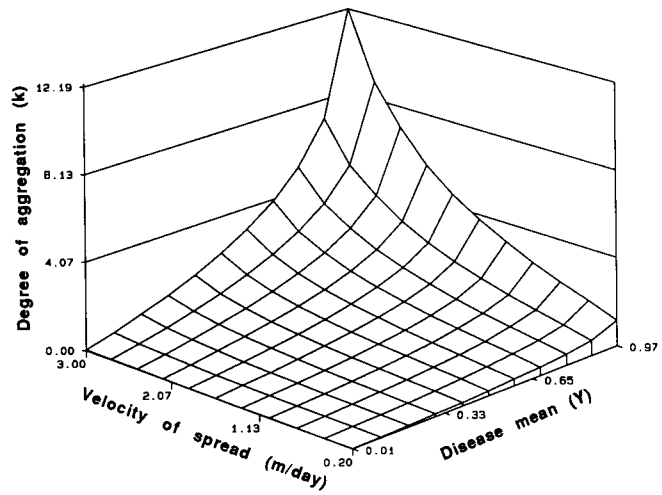

Fig. 5. Theoretical effects of velocity of disease spread and disease mean on expected degree of aggregation (expressed as k of negative binomial distribution). Values of k are negatively correlated with the degree of aggregation. Rate dk/dY increases as the rate of spread increases.

Based on the above discussion, one can argue that the effect of aggregation on the rate of disease development depends on the velocity of disease spread. For a quickly spreading disease such as wheat stripe rust (which may spread more than 900 m in a season [16]), high aggregation early in an epidemic season may have an unremarkable effect on rate of disease increase (Fig. 2). For slow spreading diseases such as Rhizoctonia foliar blight, which spreads about 2 m per season (14), relatively high aggregation at all levels of disease severity may reduce the rate of disease increase in a season remarkably (15). In the three cases in which correlations between increase of disease and aggregation were detected (7,15; Fig. 1), the diseases were highly aggregated because of low rates of either disease spread or insect vectors. For different diseases we may expect different relationships of k-Y.

Summary. Our results show that the effect of aggregation on the rate of disease increase is dependent on the disease mean. Changes in aggregation during a season may be associated with rate of disease spread. Violation of the assumption of random distribution depends on how quickly the distribution of a disease changes from aggregated to random (dk/dY). For a disease with a rapid rate of spread, aggregation early in the epidemic may not affect rate of increase greatly because of the low population of disease. Later in the epidemic, the effect of aggregation on the rate of increase still may be unnoticeable, because a rapid dispersion reduces aggregation. Further experimental data are needed to test these hypothesized relationships between the spatial pattern and spread of plant disease.

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