Focus Expansion in Plant Disease. IV: Expansion Rates in Mixtures of Resistant and Susceptible Hosts

F. van den Bosch, M. A. Verhaar, A. A. M. Buiel, W. Hoogkamer, and J. C. Zadoks

First author: Institute of Theoretical Biology, State University of Leiden, Kaiserstraat 63, 2311 GP Leiden, The Netherlands. Remaining authors: Department of Phytopathology, Agricultural University, P.O. Box 8025, 6700 EE Wageningen, The Netherlands.
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ABSTRACT

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The radial expansion of plant disease foci in ideal (i.e., infinitesimally fine grained) mixtures of a susceptible and a resistant cultivar is described. If the only difference between the cultivars is in their susceptibility to disease, theory predicts that the radial velocity of focus expansion increases linearly with the logarithm of the fraction of susceptible plants. This hypothesis was tested in the field by using the wheat-stripe rust pathosystem. The results of the experiment were generally consistent with the hypothesis, although minor deviations were present. There was an indication that in plots with a small fraction of susceptible plants, the velocity of focus expansion did not attain its final value.

Additional keywords: cultivar mixtures, epidemiology, model, *Puccinia striiformis, Triticum aestivum.*

van den Bosch et al (23,24) developed a theory of focus expansion in two-dimensional space, based on a model by Diekmann (4,5) and Thieme (21,22). According to the theory, the radial velocity of focus expansion approaches a constant value, c, after an initial phase of focus buildup. This value, called the asymptotic velocity of focus expansion, can be calculated from three entities: the gross reproduction, the time kernel, and the contact distribution. The gross reproduction is defined as the total number of progeny lesions descending from one parent lesion in an otherwise uninfected field. The time kernel is the relative rate of spore production as a function of the time since infection. The contact distribution represents the relative spatial distribution of progeny lesions around a parent lesion. Formal definitions can be found in van den Bosch (23). van den Bosch et al (24) described submodels for the time kernel and the contact distribution. With these submodels, several qualitative relations were found between model parameters and the velocity of focus expansion. In a third paper (25), the model performance in real-life situations was investigated. When input parameters were carefully estimated, using archive data of stripe rust (*Puccinia striiformis* Westend.) on wheat (*Triticum aestivum* L.) and downy mildew (*Peronospora farinosa* Fr.) on spinach (*Spinacia oleracea* L.), the model predicted observed velocities of focus expansion reasonably well. The model, thus validated, can be used as a tool generating hypotheses that can be tested experimentally.

In this paper we investigate, theoretically and experimentally, the velocity of focus expansion in ideal mixtures of susceptible and resistant plants. In theory, an ideal mixture consists of an infinitesimally fine-grained mixture. In practice, ideal mixing is approximated when the spore cloud around a lesion on a plant encloses a large number of other plants. This situation is obtained by random seed mixing. For related work on nonideal mixtures we refer to Mundt et al (16–18). Throughout the paper we assume the total plant density (susceptibles plus resistant) to be constant.

THEORETICAL CONSIDERATIONS

The gross reproduction of a disease in a crop is defined as the total number of progeny lesions produced by one parent lesion, during its whole lifetime, in an otherwise uninfected field. We are interested in the gross reproduction in an ideal mixture of a resistant and a susceptible cultivar. Denote by $\gamma\tilde{S}_0$ the gross reproduction in a field consisting of plants of the susceptible cultivar only. Assume that the plants of the susceptible and resistant cultivars have the same morphology, the same development rate, the same spore trapping characteristics, etc. Then, the movement of spores through the canopy is independent of the fraction of susceptible plants, f, and spores are deposited alike on leaves of susceptible and resistant plants. The only effect of the cultivar mixture then is that a fraction of the spores, $1 - f$, is removed from the epidemic process. This leads to

$$\gamma S_0 = f \cdot \gamma\tilde{S}_0 \tag{1}$$

Note that here $\gamma\tilde{S}_0$ is a parameter, f is the independent variable.

Exponential growth rate. The total number of lesions in a field, Z, increases exponentially during a certain period, if we start with a small initial number of lesions (25). So,

$$Z(t) = Z_0 \cdot \exp(rt), \tag{2}$$

where Z_0 is the initial number of lesions and r is the exponential growth rate. The value of r can be calculated from the so-called Euler-equation (12,19, eq. 1.2 in [25]). For the delayed gamma-density (our submodel for the time kernel), the Euler-equation is given by eq. 1.3 in (23). Using eq. 1 we arrive at

$$f = \frac{1}{\gamma\tilde{S}_0} \cdot \exp(r\,p) \cdot [1 + r/\beta]^n \tag{3}$$

where p is the latency period, and n and β are parameters of the gamma-density (24). Note that here $\gamma\tilde{S}_0$, p, β, and n are parameters, f is the independent variable, and r is the dependent variable. We will use eq. 3 to estimate $\gamma\tilde{S}_0$.

Velocity of focus expansion. Under the assumptions mentioned above, the spatial distribution of progeny lesions around a parent lesion does not depend on f. In (24) it is shown that, except for γS_0 near unity, the velocity of focus expansion, c, increases logarithmically with γS_0. This is explained (24) by considering the effect of a line source parallel to the front of the epidemic wave. We observed (24) that the effective distance, x_{eff}, beyond

which the number of progeny lesions per unit of distance decreases below a value κ is given by

$$x_{\text{eff}} = (\sigma_2/\sqrt{2}) \ln (\gamma S_0/\kappa \sqrt{2}\, \sigma_2), \quad (4)$$

where σ_2 is the variance of the marginal contact distribution. Note the typographical error in eq. 4.3 in (24).

The effective distance measures the distance covered by one generation. Therefore, it is reasonable to assume that the velocity of focus expansion is linearly proportional to the effective distance, X_{eff},

$$c = A_1 X_{\text{eff}} + B_1, \quad (5)$$

where A_1, B_1 are unknown constants. Substitution of eq. 4 into eq. 5 yields after rewriting

$$c = A \ln (\gamma S_0) + B \quad (6)$$

where A and B are unknown constants (depending on A_1, B_1, κ, and σ_2). Finally, substitution of eq. 1 in eq. 6 leads to the conclusion that

$$c = A \ln(f) + \tilde{c} \quad (7)$$

where A is an unknown constant and $\tilde{c}$ is the velocity of focus expansion in a field consisting of susceptible plants only. Thus the radial velocity of focus expansion should increase logarithmically with the fraction of susceptible plants.

MATERIALS AND METHODS

Experimental design. In a large field of winter rapeseed 12 wheat plots were established at least 70 m apart (Fig. 1). Each plot consisted of 11 × 11 hills of wheat plants, planted at a center-to-center distance of 0.3 m. Each hill consisted of a mixture of a susceptible (Okapi) and a resistant cultivar (Sarno). Four treatments, with mixing ratios of 1:0, 1:1, 1:2, and 1:4, respectively, were planted in three replicates.

For plot establishment, seeds of the winter wheat cultivars Okapi and Sarno were mixed as described, sown in small peat-pots, and placed in a greenhouse (January 1987). After emergence (February 1987), the pots were placed in frames ('Dutch lights') for vernalization. In April, the pots with wheat plants (DC = 11, [28]) were transplanted to the field in South Flevopolder.

Twenty-four pots with Okapi plants (DC = 11) were inoculated with *P. striiformis* race 105 E 137. The plants were incubated in a growth chamber at 15 C in a saturated atmosphere for 2 days. Pots were then transferred to the field. Foci were initiated by planting two pots with infected wheat plants at the central position of each plot.

The plants of one hill, resulting from one pot with seedlings, were considered to form one experimental unit covering 0.3 × 0.3 m. The number of sporulating lesions per hill was counted weekly. When this number exceeded 20, the number was estimated in increments of five. Lesion coalescence was not considered because its effect was negligible at the low lesion densities considered here. The course of the epidemic was followed until the end of June, when the high day temperatures (over 25 C) became unfavorable for pathogen reproduction, disease development, etc.

Data analysis. The plants used to initiate the foci were severely damaged by heavy infection at an early growth stage. As the development of these central hills and of the rust on them was atypical, they were not considered in any calculation.

After log-transformation of the total number of lesions in a plot, $Z(t)$ in eq. 2, the exponential growth rate r was estimated by using linear least squares.

For the parameters n and β of eq. 3 we used the estimates $\hat{n} = 3.0$ and $\hat{\beta} = 0.2$ from van den Bosch et al (25). The latency period, p, of *P. striiformis* for the mean temperature of the experimental period was estimated at 15.4 days (27). Eq. 3 was fitted to the estimated exponential growth rates from the 12 plots, using nonlinear least squares. This procedure provided the estimate of $\widetilde{\gamma S_0}$, the gross reproduction in a field with the susceptible cultivar only. With this estimate of $\widetilde{\gamma S_0}$ and eq. 1 the γS_0 in fields with given fractions of susceptible plants can be calculated. In order to test the fit of eq. 3 to the data, we performed an F test for the lack of fit as described by Draper and Smith (6) for the linear regression situation with repeated observations at a number of values of the independent variable.

The radial velocity of focus expansion was estimated for each plot. A comparative study (3) on methods to calculate the velocity

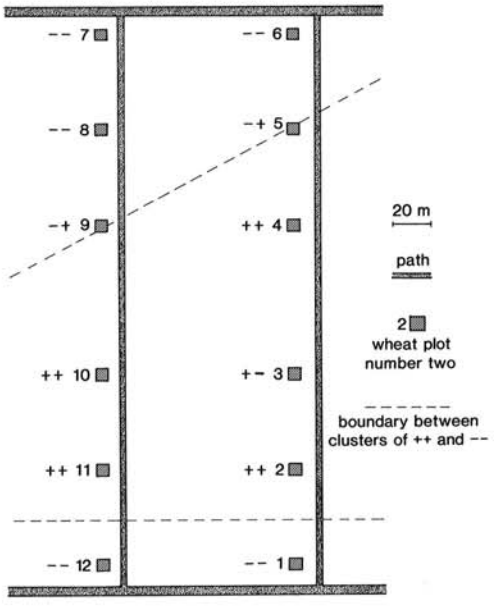

Fig. 1. Positions of experimental wheat plots in a winter swede rape field near Almere in the South-Flevopolder (The Netherlands). The + and − signs for each plot indicate whether the numerical values of the observed velocity of focus expansion (c, left hand sign) and the observed rate of exponential increase in the number of lesions (r, right hand sign) are larger (+) or smaller (−) than the values calculated by equations 3 and 7.

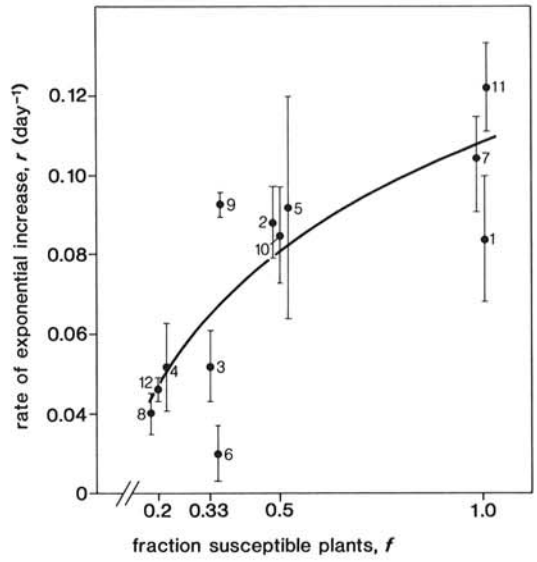

Fig. 2. The rate of exponential increase, r, in the number of lesions as a function of the fraction susceptible plants, f, in the experimental plots. Points represent observed r values, vertical bars their standard deviations. Data points are scattered slightly in the horizontal direction to avoid obscuring data points in similar positions. Entries are plot numbers (Fig. 1). Bars indicate variation (±1 standard deviation). The curve is the fitted equation 3.

of focus expansion showed that the area method described by van den Bosch et al (25) is most suitable. For every observation date, the area within which the disease severity exceeded 5 and 10 lesions per hill, respectively, was determined for each plot. Using linear least squares, eq. 7 was fitted to the estimates of the radial velocity of focus expansion from the 12 plots. The F test described above was used to evaluate the adequacy of eq. 7.

The rule of thumb (24) that the velocity of focus expansion increases linearly with $\ln\gamma S_0$, is an approximation only. This approximation rule is derived from the results of the epidemic model in van den Bosch et al (23,24). We will refer to this model as the 'full model' (as opposed to eq. 7), described in detail in (23–25). Instead of using our rule of thumb we could also calculate the velocity of focus expansion, c, as a function of the fraction of susceptible plants, f, from the full model. Deviations between results of the full model and from the rule of thumb give insight in the adequacy of the latter in the present experimental situation. Using the parameter estimates as discussed above, we are left with one parameter to be estimated, the variance of the marginal distribution of the contact distribution, $\sigma_2{}^2$ (24). The standard deviation σ_2 is a scaling factor of the velocity of focus expansion (23,24). With all parameters known except σ_2/μ, the scaled velocity of focus expansion, c^*, from the full model (24) is related to the actual velocity c as

$$c = \frac{\sigma_2}{\mu}\, c^*$$

where $\mu = n/\beta$. Using the parameter estimates as defined above, we can calculate c^* for each value of f. Least squares were used to estimate σ_2/μ and its variance.

Deviations of the estimates for r and c of a plot from the fitted eqs. 3 and 7 can have two causes. First, stochasticity at the level of the population of lesions and, presumably more importantly, errors made during data collection caused deviations. This type of deviation is the usual estimation error. Second, deviations can be induced by differences between the wheat plots. When the spatial scale of the factors inducing the differences between the plots is sufficiently large, we also expect plots with deviations of the same sign in both r and c to be clustered in the rapeseed field. Consider the sign of the residual. (A residual is the numerical value of the difference between the estimate and the fitted curve.) Standard probability theory shows that, if the combination of the signs of the residuals of r and c for a plot are determined purely by chance, the number of ++ combinations follows a hypergeometrical distribution (14).

RESULTS

The estimated r values showed considerable variation within and between plots (Fig. 2). Nevertheless, the curve representing eq. 3 fitted the data well. The F test, summarized in Table 1, shows that eq. 3 had no significant lack-of-fit to the data. From eq. (3) we found $\widetilde{\gamma S_0} = 19.4$.

The value c showed considerable variation within and between plots (Fig. 3). Eq. 7 fitted the data well, without significant lack-of-fit (Table 1). From the fit of the full model we found $\widehat{(\sigma_2/\mu)} = 1.27 \pm 0.36$. Using the parameter, μ, of the time kernel given in (24), and the method to calculate approximate standard deviations described in (24), we found $\hat{\sigma}_2 = 19.0 \pm 5.6$ [cm]. The fitted curve of the full model was (almost) indistinguishable from a straight line (Fig. 3). Although the fit of the full model was slightly less than that of eq. 7, there was no significant lack-of-fit (Table 1). Several data points for small f values are below the model predictions.

Nine out of 12 plots had an equal sign for the deviations in r and c, and three had opposite signs. The probability of finding at least this amount of regularity if deviations in r and c were purely random was approximately 0.12.

The spatial arrangement of the plots and the signs is given in Figure 1. Clustering of plots with ++ and plots with −− was obvious. Two of the three plots with opposite signs were located on the boundary of a cluster (plots 5 and 9).

TABLE 1. F tests for the lack-of-fit of models describing the rate of exponential increase and the velocity of focus expansion of wheat stripe rust in field plots

	Source	df[a]	SS[b]	F ratio	P
Exponential growth rate, r	Lack of fit	3	4.0	0.4	
(Equation 3) Fig. 2	Pure error	8	28.3		>0.5
Velocity of focus expansion, c					
(Equation 7)	Lack of fit	2	0.2	0.3	
Fig. 3, continuous line	Pure error	8	1.9		>0.5
Velocity of focus expansion, c					
(Full model, see [23][24])	Lack of fit	3	1.3	1.8	
Fig. 3, broken line	Pure error	8	1.9		>0.5

[a]df = degrees of freedom.
[b]SS = sum of squares.

DISCUSSION

The estimated value of the gross reproduction in a plot consisting of the susceptible cultivar only, $\widetilde{\gamma S_0}$, was roughly one third of the value given by van den Bosch et al (25). The parameters p, n, and β used to fit eq. 3 to the data on the exponential rate of increase, r, were calculated from a different cultivar and race than the ones used in the present experiments. Cultivars with similar infection types can differ in various components of resistance. Thus, the use of a different cultivar race combination may have had an effect on the estimate of $\widetilde{\gamma S_0}$. We believe, however, that the difference in $\widetilde{\gamma S_0}$ is not due to the use of a different wheat race combination but rather to less favorable weather conditions for stripe rust in 1987. Persistent rainfall during the experimental period washed away the spores that would otherwise have caused infection and it damaged lesions that otherwise would have produced more spores. Such effects of rain have been shown for *Puccinia arachidis* (20).

The value of the standard deviation, σ_2, of the marginal contact distribution given by van den Bosch et al (25) was between 1.5 and 2 times larger than the one estimated here. The difference is probably due to the following factors: 1) in 1987 the focus

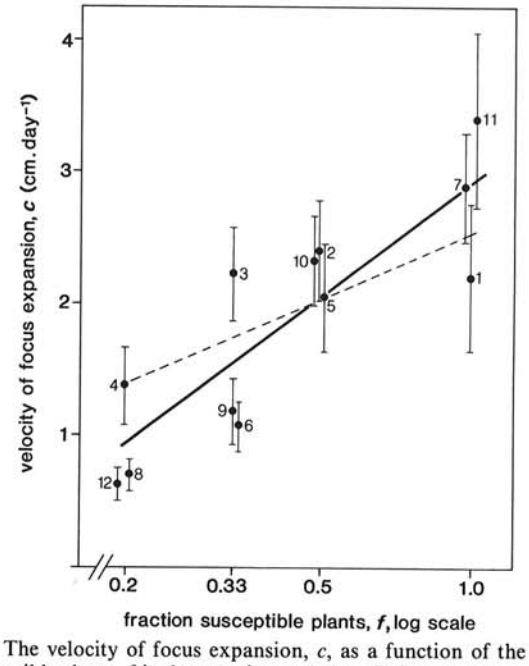

Fig. 3. The velocity of focus expansion, c, as a function of the fraction of susceptible plants, f, in the experimental plots. Points represent observed c values, vertical bars their standard deviations. Data points are scattered slightly in the horizontal direction to avoid obscuring data points in similar positions. Entries are plot numbers (Fig. 1). The drawn line is fitted equation 7, the broken line is the fitted 'full model.'

development began later and the wheat was accordingly taller; 2) the 1987 plots had a slightly higher plant density; and 3) the 1987 plots were surrounded by a relatively high rapeseed crop acting as a windbrake.

We conclude that the smaller values of the parameters $\widetilde{\gamma S_0}$ and σ_2 in the 1987 experiment as compared to those given in (25) can be explained by differences in experimental conditions. These smaller parameter values resulted in a velocity of focus expansion which is about one third of the value reported earlier (25).

The results of Table 1 must be viewed with caution when considering the adequacy of equations 1 and 7. If the two sums of squares show some interdependence, the F ratio will only be approximately F distributed, and the critical region may be shifted somewhat in comparison with the one derived from the F distribution. Fortunately, the F ratios were far from the critical value determined from the F distribution.

The fit of eq. 3 to the data (Fig. 2) did not give reason to doubt the correctness of the assumptions leading to eq. 1. The curve of the full model (Fig. 3, broken line) was almost indistinguishable from a straight line. Moreover, considering Figure 3, eq. 7 and the full model seemed to fit the data equally well. We conclude that eq. 7 is, in the present case, a good description of the data.

Too small focus expansion velocities. The data points and the curve of the full model (Fig. 3, broken line) give the impression that the velocity of focus expansion in the plots with a low fraction of susceptible plants, f, was underestimated. The theory (24) only allows the calculation of the velocity with which the focus will eventually expand. Nothing is known about the duration of the initial phase of focus buildup. Literature on diffusion equations (1) suggests that, for these models at least, at small γS_0 the initial phase takes longer than at large γS_0. We hypothesize that in plots with low f the radial velocity of focus expansion had not yet attained its final value, whereas in plots with high f final values were realized. Extension of the theory is needed to clarify this point.

Plant variability. Several of the standard deviation bars do not overlap (Figs. 2 and 3). This suggests systematic differences between plots. The probability of finding at least six plots with equal signs of the deviations in r and c, given that the combinations of signs are due to pure chance, was 0.12. Although this is suggestively small, it is too large to reject the hypothesis that the combinations of the signs of the residuals of r and c for a plot are determined purely by chance. The spatial arrangement of the signs (Fig. 1) suggests a large-scale factor inducing systematic differences between plots, for estimates of r and c, respectively, at equal values of f. We conclude that our analysis strongly suggests differences between the wheat plots, but the results do not allow a firm statement.

The use of cultivar mixtures for disease control. The effectiveness of cultivar mixtures to control fungal diseases of the foliage in small grain crops is well documented (e.g. 2,7,15,26). Using both mathematical models and field experiments, several authors studied disease progress in cultivar mixtures, leaving the spatial component out of consideration. In a series of papers, Jeger et al (8–11) showed, both theoretically and experimentally, that disease levels (at a certain time after inoculation) in a cultivar mixture are close to the geometric mean of the disease levels found in the pure stands (i.e., with one cultivar only). Leonard (13) showed that in mixtures of a susceptible and a resistant oat cultivar the rate of stem rust increase is given by

$$r_m = r_s + E \ln (f) \qquad (8)$$

where r_s is the rate of stem rust increase in a plot with susceptible plants only, r_m is the rate of increase in a host mixture, f is the fraction of susceptible plants, and E is an unknown constant. Note the similarity with eq. 7 derived in this paper. Figure 2 indicates that eq. 8 holds approximately in our study. Since we only used these data to estimate γS_0 we used eq. 3 and did not consider this observation in more detail.

Recently, Mundt and Leonard (16–18) studied the effect of host genotype unit area (which is defined as the ground area occupied by an independent, genetically homogeneous unit of host plants) on epidemic development in mixtures of susceptible and resistant cultivars. The square units of susceptible plants were either homogeneously (16,17) or randomly (18) distributed in the field. In their simulation model as well as in their field experiments, the effectiveness of disease control of the mixture decreased with increasing genotype unit area. The most effective mixture thus is the ideal mixture. In the present study, the ideal mixture was approximated by random mixing, which is technically and economically more attractive than systematic seed placement.

For applications, it is helpful to have rules of thumb telling how certain epidemiological quantities, such as the velocity of focus expansion, vary with the fraction of susceptible plants in the field. Generalizing from the theoretical considerations and the present experimental evidence we can state the rule:

> In an ideal mixture of susceptible and resistant plants, the radial velocity of focus expansion increases linearly with the logarithm of the proportion of susceptible plants.

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