

Focus Expansion in Plant Disease. III: Two Experimental Examples

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ABSTRACT

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Experiments on focus expansion and its underlying processes were performed with stripe rust (*Puccinia striiformis*) on wheat and downy mildew (*Peronospora farinosa*) on spinach. Models for the spore production by an individual lesion as a function of time after victimization (the time kernel) and the spatial distribution of daughter lesions around one mother lesion (the contact distribution) were fitted to experimental data. Two methods to estimate the gross reproduction of the pathogen are described. Velocities of focus expansion were estimated using field

observations from artificially established foci. "Expected" velocities of focus expansion were calculated from the time kernels, contact distributions, and gross reproductions. For both diseases, the observed and expected rates were in good agreement. A sensitivity analysis shows that the accuracy of the estimated variance of the contact distribution has a large influence on the accuracy of the predicted velocity. When the gross reproduction is small, the accuracy of its measurement also strongly contributes to the accuracy of the expected velocity of focus expansion.

Additional keywords: epidemiological models.

van den Bosch et al (9) introduced a model for the spatial development of plant disease foci in two dimensions. The model predicts that after an initial buildup phase the velocity of focus expansion will be constant. Moreover it permits calculating this velocity from the gross reproduction, γS_0 , which is the number of daughter lesions per mother lesion in an otherwise uninfected field, the time kernel, which is the spore production as a function of the time since infection, and the contact distribution, which represents the spatial distribution of daughter lesions. Realistic submodels for the time kernel and the contact distribution were introduced in a second paper (10), which also discussed qualitative relationships between the model parameters and the velocity of the focus expansion and the steepness of the disease gradient at the focal front.

In the present paper, we study the model's performance in real-life situations. We shall pay attention to the ease with which the model parameters can be obtained, the fit of the various submodels, and the precision with which the velocity of focus

expansion is predicted. Several unpublished data sets, originally collected for other purposes, were used. These concern wheat (*Triticum aestivum* L.) with stripe rust (*Puccinia striiformis* West.) and spinach (*Spinacia oleracea* L.) with downy mildew (*Peronospora farinosa* (Fr.) Fr. f. sp. *spinaciae* Byford). It will be shown that the submodels for time kernel and contact distribution reasonably fit the data and that the velocity of focus expansion is fairly well predicted. Possibilities to improve experimental work on focus expansion will be discussed, using the sensitivity of the predicted velocity to the various measurement errors as a guideline.

Symbols are explained in Table 1.

TIME KERNEL

P. striiformis. In 1976, wheat seedlings of cultivar Rubis were grown in pots in a growth chamber, inoculated at growth stage decimal code (DC) = 1.1 (13) with *P. striiformis* race 232E137 (Clement race) (4), and incubated for 1 day at 4 C in a water-saturated atmosphere. During the following week, the incubated plants were placed in a growth chamber at 15 C and about 80%

relative humidity, with a day length of 12 hr and a light intensity of about 4 klx. Subsequently, the plants were placed at the target temperature of 10 C (other conditions as above) to study the course of sporulation. The spores produced were collected daily by means of a cyclone collector (5) and counted with a hemacytometer.

***P. farinosa*.** Spinach plants of cultivar Huro were inoculated on the first pair of true leaves with *P. farinosa* f. sp. *spinaciae* race 3 (2). The plants were incubated 6 days in a growth chamber with saturated atmosphere at 15 C and a day/night regime of 16/8 hr and a light intensity of about 7.5 klx. These conditions match the latency period, p , to that observed in the experiments on focus expansion. The relative rate of sporulation was assessed by observing the size of the sporulating area.

Parameter estimation. The gamma-density function (10) was fitted to the spore production during the infectious period by means of least squares regression after logarithmic transformation (see Appendix I). The latency period of *P. striiformis* for the mean temperature of the experiments on focus expansion was obtained from Zadoks (12). For *P. farinosa*, no descriptive model is available relating latency period to environmental conditions; the latency period was directly observed during the initial phase of the experiment on focus expansion.

CONTACT DISTRIBUTION

***P. striiformis*.** In the spring of 1977, five *P. striiformis* "point" infections were established at the centers of adjoining 15- \times 13-m plots in a winter wheat crop (cultivar Tadorna, DC = 33, and leaf area index [LAI] = 2 m² of foliage per square meter of soil) by planting seedlings infected with race 37E132 (Tadorna race, earlier named race 60) (4,7). The sources planted measured approximately 0.1 \times 0.2 m. Between 2 and 2.5 latency periods after infection (calculated from the latency period-temperature relationship [12] and measured temperatures), the disease severity expressed as the fraction of the foliage visibly infected by rust, at various distances, x , from the centers of infection was determined in four compass directions. Data were averaged over compass directions and replicates. At low disease severities, as used here, disease severity is (almost) proportional to the number of spores landed at distance x .

***P. farinosa*.** One data set was derived from field experiments. Spinach (cultivar Noorman) was sown in the spring of 1983 with 20-50 plants per meter of row length, at a row distance of 0.15 m. Point sources were established at the centers of adjoining 1.5 \times 1.5 m plots using race 3. An observation grid with grid cells of 0.15 \times 0.15 m was placed over the plots with the inoculated plants in the center cell. Twice a week the number of infected leaves per grid cell was counted over an area of about 1 \times 1 m. The counts made at a

time equivalent to about 2.5 latency periods after inoculation were used to determine the contact distribution. A second data set was derived from a similar experiment, performed in the greenhouse, in the spring of 1984, using Huro (approximately 20 plants per meter row length at a row distance of 0.16 m) and race 3. The experiment was made in four replications at about 15 C. Wind simulated by a ventilator promoted turbulent spore dispersal. Because the data sets of the two experiments did not show quantitative differences, the data were combined.

Parameter estimation. Bessel densities were fitted to the data using nonlinear least squares (see Appendix I).

GROSS REPRODUCTION

When the infectious period of a fungus is shorter than the latency period, the first few generations of an epidemic have little overlap. Therefore, the gross reproduction can be estimated directly as the number of sporulating leaves of the first generation divided by the number of inoculated and sporulating leaves (the 0th generation). When the number of first-generation infections is small relative to the number of susceptibles at risk, correspondence between leaves and lesions is approximately one to one. When the number of first-generation infections is larger, it is better to count the number of lesions. Alternatively, one may apply a multiple infection transformation (MIT) (3). If the number of first-generation infections is really large, one should even apply a MIT to the number of lesions. A practical problem is that any MIT is necessarily based on specific assumptions about the sampling distribution of spores landing in an area element, be it Poisson or negative binomial, which should be validated separately (11). Fortunately, a MIT did not appear to be necessary in any of the experiments reported in this paper.

When the latency period is much shorter than the sporulation period, the first two generations already grossly overlap. Then, γS_0 must be determined indirectly. When an arbitrary but, relative to the density of susceptibles, small initial infection is established, the number of victims increases exponentially during a certain period. During this exponential phase, the number of victims Z increases as:

$$Z(t) = Z_0 e^{rt} \quad (1.1)$$

where $Z(0) = Z_0$ and r satisfies (6):

$$1 = \gamma S_0 \int_0^{\infty} e^{-r\tau} i(\tau) d\tau. \quad (1.2)$$

Using the time kernel, $i(\tau)$, described by equation 3.1 in reference 10, the equation 1.2 can be written as follows:

$$\gamma S_0 = e^{rp} \left(1 + \frac{r}{\beta}\right)^n \quad (1.3)$$

which gives γS_0 in terms of the exponential growth rate r and the parameters of the time kernel. The size of the initial infection is an empirical matter. It should be such a small infection that we can observe a period of exponential growth of a duration sufficient to estimate r . Theoretically, the initial infection does not need to be homogeneous, provided that the total number of victims in the study area is counted.

***P. striiformis*.** The infectious period of stripe rust exceeds the latency period. Therefore the indirect method was used. In three small plots in fields of cultivar Clement, point infections were established with rust race 232E137 (Clement race) in 1982, 1983, and 1984. Every week the number of victims in a group of 100 leaves, selected in advance, was counted. The exponential phase of the resulting epidemics occurred in spring, in the period when DC rose from 31 to about 61 and LAI increased from about 4 to 6.

***P. farinosa*.** The infectious period of *P. farinosa* was about 9 days, 2 days more than the latency period of 7 days. The spore production after the first few days of the infectious period is small. Therefore, the conditions for the use of the direct method are

TABLE 1. List of symbols

Symbol	Explanation	Dimension
$A(t)$	Area of the focus at time t	L ²
β	Constant of the gamma density	T ⁻²
c	Velocity of focus expansion	L.T ⁻¹
c_{expected}	Velocity of focus expansion predicted by the model	L.T ⁻¹
c_{observed}	Velocity of focus expansion observed in the field	L.T ⁻¹
DC	Decimal code of growth stage (12)	-
ϵ	Constant correcting for initial period of focus buildup	L
γS_0	Gross reproduction	1
$i(\tau)$	Time kernel	T ⁻¹
LAI	Leaf area index	1
n	Constant of the gamma density	1
p	Latency period	T
r	Exponential growth rate	T ⁻¹
$R(t)$	Radius of the focus at time t	L
σ_2	Parameter of the contact distribution	L ²
x	Distance from parent infection	L
$Z(t)$	Number of victims at time t	N _{vi}
Z_0	Number of victims at time 0	N _{vi}

approximately fulfilled. In the autumns of 1981, 1982, and 1983, point infections were established in spinach crops (Noorman) using race 3. Grids were established and counts made as described above. Because data taken at a time equivalent to about 2.5 latency period, used to calculate γS_0 , were relatively low, no MIT was applied.

Parameter estimation. Parallel lines were fitted to the stripe rust data using least squares.

WAVE VELOCITY

Assuming the foci to be rotationally symmetric, we write for the area, A , of the focus

$$A(t) = \pi R^2(t) = \pi(\epsilon + ct)^2 \quad (1.4)$$

where R is the radius, t is time, c is the velocity of focus expansion, and ϵ is a constant correcting for an initial period of focus buildup. From equation 1.4 we can conclude that a straight line should be found plotting the square root of A against t , when the initial period of focus buildup has passed by. If so, c can be calculated from this straight line.

***P. striiformis*.** In 1976, five foci were established artificially in a winter wheat crop (Clement) using the race 232E137 (Clement race [8]). The growth of the foci was carefully monitored in each of eight compass directions (see Fig. 6.26 in Zadoks and Schein [14]). The foci developed in the period when DC rose from 31 to 75 and LAI increased from 2 to 3. The resulting data were averaged to obtain the area A within which the disease severity exceeded 0.01, 0.05, 0.1, and 0.5, respectively.

***P. farinosa*.** In the fall of 1982, artificial foci were established in spinach plots (Noorman), 20 plants per meter row length at a row distance of 0.15 m, using race 3. Counting grids were established as described above. Observations were made at about weekly intervals.

Parameter estimation. Parallel lines were fitted to the data using least squares.

RESULTS

Table 2 summarizes the estimated parameter values of both plant-pathogen systems. Numerical values are given in the representation: estimate \pm standard deviation.

***P. striiformis*.** Figures 1 and 2 show that the submodels for the time kernel and the contact distribution reasonably fit the data. In the experiments used to determine r , the number of victims in the infected plots increased exponentially during a few weeks (Fig. 3). Figure 4 shows that, after a phase of focus buildup, the square root of the focal area increases linearly with time, as predicted. The velocity of focus expansion estimated from these data shall be called c_{observed} . Using the estimated parameters of the various submodels, we also calculated, following van den Bosch et al (9,10), a predicted velocity c_{expected} . The calculation procedure is described in some detail in Appendix II; Appendix III treats ways

TABLE 2. Summary of the estimated parameter values of *Puccinia striiformis* on wheat and *Peronospora farinosa* on spinach^a

Factor		Stripe rust, wheat	Downy mildew, spinach	Units
Time kernel				
Latency period	p	10.0	7.0	day
	\hat{n}	3.0 ± 0.18	3.1 ± 0.9	l
Infectious period	β	0.2 ± 0.02	0.9 ± 0.6	day
	cov. ($\hat{\beta}, \hat{n}$)	0.0025	0.11	day
Contact distribution	$\hat{\sigma}_2$	34.7 ± 5.8	16.3 ± 4.7	cm
Gross reproduction	γS_0	(55.4 ± 16.4)	3.2 ± 1.8	l
Exponential growth rate	\hat{r}	0.20 ± 0.02	...	day ⁻¹

^aThe estimated values are given in the usual representation, estimate \pm standard deviation. The gross reproduction of stripe rust on wheat was determined implicitly (see text).

to calculate approximate values of the standard deviation of c_{expected} . The resulting values are as follows:

$$c_{\text{expected}} = 8.0 \pm 1.5 \text{ cm day}^{-1}$$

$$c_{\text{observed}} = 9.4 \pm 0.8 \text{ cm day}^{-1}.$$

The effect of the precision of various measurements of the input variables (the gross reproduction, γS_0 , the contact distribution, D , and the time kernel, i) on the output variable (the velocity of focus expansion) was studied by means of a sensitivity analysis. Assuming that all but one parameter of the input variables have zero variance, the variance of the output variable was calculated. Note that the standard deviation of c_{expected} as given above is equal to the square root of the sum of the variances calculated for all separate input variables. From Table 3 we can conclude that the variance in the parameter of the contact distribution contributed most to the variance of the expected expansion velocity.

***P. farinosa*.** The delayed gamma density appears not to fit the data so well (Fig. 5). There are various ways of improving the apparent fit, e.g., by assuming a slightly longer latency period or by doing a nonlinear least squares on the untransformed data. However, because at a low γS_0 the wave velocity is hardly influenced by the parameters of the gamma density (see Fig. 3 of

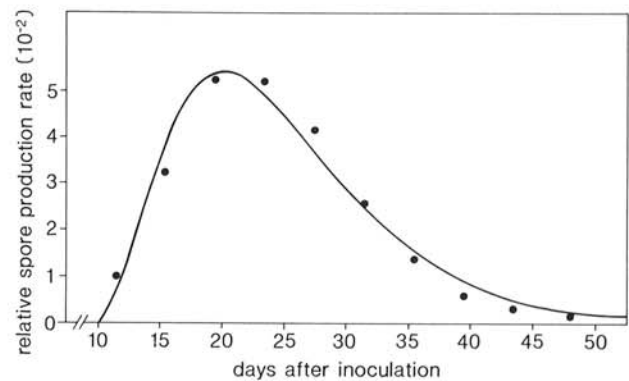


Fig. 1. Time kernel of *Puccinia striiformis* race 232E137 on primary leaves of winter wheat cultivar Rubis, determined in the greenhouse, in 1976, at about 10 C. Continuous curve: best fitting gamma density. Dots represent successive observations. Area under the curve is equal to 1.

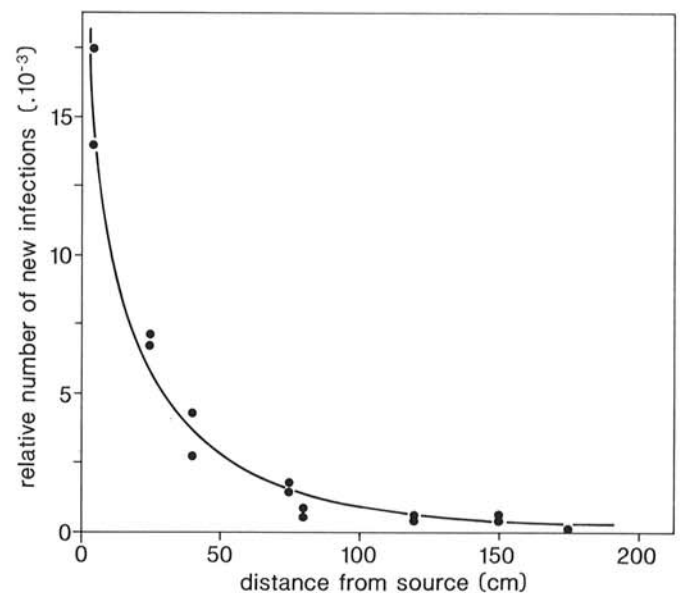


Fig. 2. Contact distribution of *Puccinia striiformis* race 37E132 on winter wheat cultivar Tadorna, observed in the field in 1977. Continuous curve: best fitting Bessel density. Each dot is the average value of four compass directions. Area under the curve is equal to 1.

reference 10), we chose the easy way and worked with the estimates as indicated. The Bessel density again gave a good fit to the data (Fig. 6). Figure 7 shows that the square root of the area of the focus does increase linearly, with a negligible period of focus buildup.

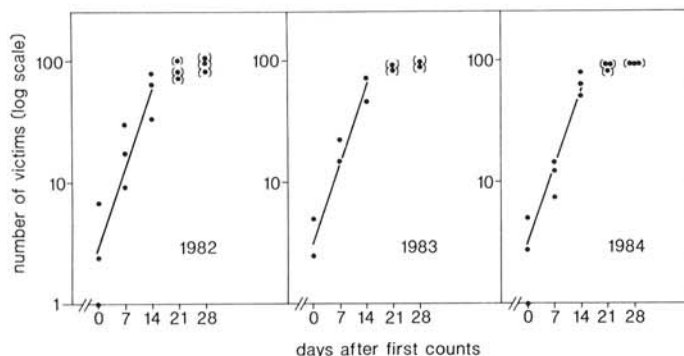


Fig. 3. Exponential increase of an epidemic of *Puccinia striiformis* race 232E137 in small plots of winter wheat cultivar Clement, 1982–1984. After 14 days, saturation becomes apparent. Data within brackets were not used. Continuous curve: best fitting straight lines with a common slope. Dots represent successive observations.

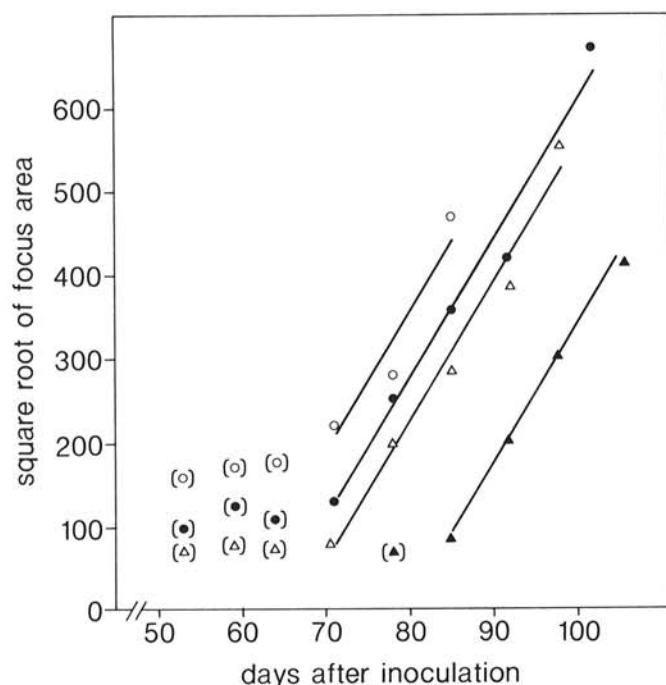


Fig. 4. Focus development of *Puccinia striiformis* race 232E137 on winter wheat cultivar Clement in 1976. In a commercial field, foci were initiated in five replications. Severity levels: ○ = 0.01; ● = 0.05; △ = 0.1; and ▲ = 0.5. Data from the period of focus buildup (within brackets) were not used. Continuous lines: best fitting straight lines with a common slope. Symbols represent successive observations.

TABLE 3. Sensitivity analysis of the constant rate of focus expansion, c , for *Puccinia striiformis* on wheat and *Peronospora farinosa* on spinach^a

Factor with variance	Contribution to the variance of c	
	Stripe rust, wheat	Downy mildew, spinach
Time kernel	0.05	1.15
Contact distribution	2.22	2.05
Exponential growth rate, r	0.11	...
Gross reproduction	...	2.53

^a The contribution to the variance of c was calculated for each one of the three factors, assuming that the other two factors had zero variance.

Following the same route as in *P. striiformis* on wheat, we find:

$$c_{\text{expected}} = 3.0 \pm 2.4 \text{ cm day}^{-1}$$

$$c_{\text{observed}} = 2.3 \pm 0.2 \text{ cm day}^{-1}.$$

The results of the sensitivity analysis are given in Table 3.

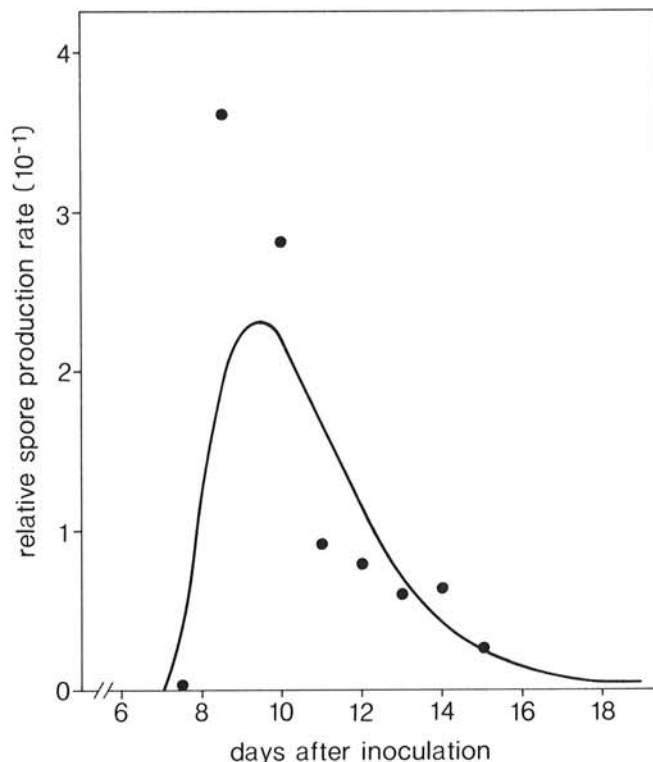


Fig. 5. Time kernel of *Peronospora farinosa* race 3 on spinach cultivar Huro, determined in a growth chamber at 15 C. Continuous curve: best fitting gamma density. Dots represent successive observations.

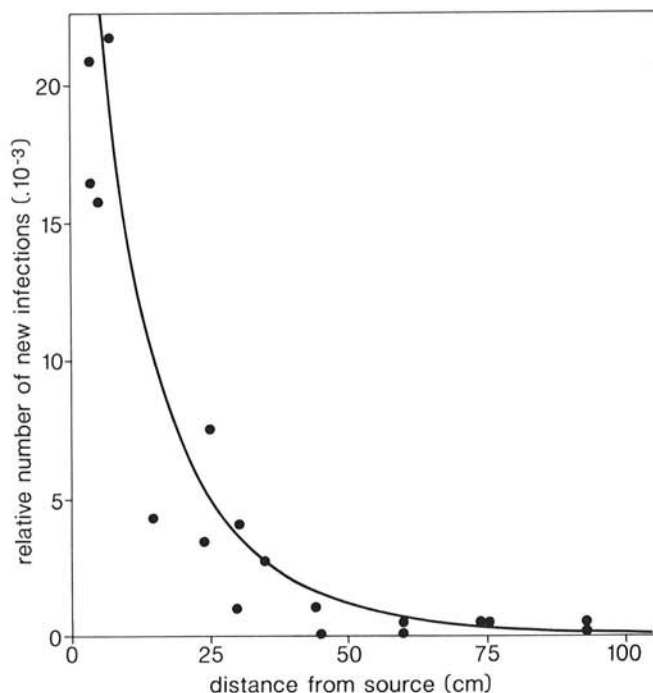


Fig. 6. Contact distribution of *Peronospora farinosa* race 3 on spinach cultivar Noorman (field data) or cultivar Huro (greenhouse data). Each dot is the average value of four wind directions.

Both the variances of the gross reproduction and of the parameter of the contact distribution greatly contribute to the variance of the expected expansion velocity.

DISCUSSION

Validity aspects. The validity of any experimental check of a model of a more general nature depends on three considerations. First, the output variable to be predicted and the input parameters, on which that prediction is based, must be determined independently. In our case the output variable is the velocity of focus expansion, and the input variables are the time kernel, the contact distribution, and the gross reproduction of the pathogen. Second, the experimental objects must be representative for the class of objects presumed to be covered by the model. Third, the extent to which circumstances had to be selected to obtain a good agreement between model and experiment must be examined.

P. striiformis belongs to the Uredinales, a major group of pathogens, many of which have windborne spores, polycyclic epidemics, and distinct focus formation. This pathogen lives on wheat, a host plant with a dense canopy. Most data for this pathosystem came from field experiments, where winter wheat was inoculated in early spring and rust development occurred during

shooting and heading (DC = 31 to 61). The time kernel was determined (in 1976) in a growth chamber, using seedlings of a highly susceptible cultivar. The contact distribution was determined (in 1977) using Tadorna and its compatible race. The gross reproduction (1982–1984) and the velocity of focus expansion (1976) were determined using Clement and its compatible rust race. Most data were collected under more or less similar conditions of crop growth and environment, although they come from a variety of experiments performed in different years.

P. farinosa belongs to the Peronosporales. Many species in this group have windborne spores and show distinct focus formation. Spinach, used as the host of this pathogen, is much lower and has a more open canopy structure than wheat. As in *P. striiformis*, most parameters for this species were derived from field experiments. The time kernel was determined (in 1978) indoors using Huro and *P. farinosa* race 3. The contact distribution was derived from field experiments with Noorman (in 1983) and greenhouse experiments using Huro, both with race 3. Field data, chosen for determining the gross reproduction of the pathogen, were collected during field experiments with Noorman and race 3 (in 1981, 1982, and 1983) under conditions only moderately favorable to downy mildew; under favorable conditions, much higher values were found. The velocity of focus expansion was determined (in 1982) in fields of Noorman using race 3, again under moderately favorable conditions.

Input and output variables were determined independently. The only exception is the latency period of *P. farinosa*, which was determined during the field experiment on the velocity of focus expansion. The latency period was estimated during the first part of the experiment from the time interval between successive sporulation waves, whereas the velocity of focus expansion was determined from the spatial development of the epidemic during the later part of the experiment. Thus, it can be concluded that the first requirement for the validity of the experimental check, that of independence, is almost ideally fulfilled.

It is obvious that the homogeneity of the data is far from ideal, even though we carefully selected the data sets to match experimental circumstances as closely as possible. The agreement between observed and predicted velocity can therefore be interpreted as an indication of the model's robustness.

Because *P. striiformis* and *P. farinosa* represent two major groups of pathogens and because the canopy structures of wheat and spinach show large differences, we infer that the experimental objects adequately represent the class of objects presumed to be covered by the model.

Some lessons from the present analysis. In this paper we showed that, although much experimental effort is needed, the parameters of the model for focus expansion are easily determined.

The sensitivity analysis indicates that the variance of the estimate of the parameter of the contact distribution, σ_2^2 , greatly contributes to the variance of the predicted velocity of focus expansion. Figures 2 and 6 show that this is not due to a great inaccuracy of the data but to the relatively limited sensitivity of the contact distribution submodel. For example, it would be difficult to distinguish, on the scale of Figures 2 and 6, two Bessel densities that differ as much in σ_2^2 as 5%. Therefore, it is unlikely that this estimate will be improved by better experimental methods. We conclude that the variance component of the predicted wave velocity due to the inaccuracy in the parameter of the contact distribution sets a lower boundary to any improvement in predicting the wave velocity.

The variance of the estimate of the gross reproduction of the downy mildew-spinach system contributed much to the variance of the expected wave velocity. In the stripe rust-wheat system, this variance, though relatively large, did not contribute much to the variance of the expected wave velocity. Due to the logarithmic dependence of c on γS_0 (10), the wave velocity is very sensitive to γS_0 when γS_0 is small, which explains the difference between the two pathosystems. The accuracy of the experimental determination of γS_0 , when this parameter is small, must be improved.

Given the validation of the model presented in this paper, we can now start incorporating the effect of environmental parameters on

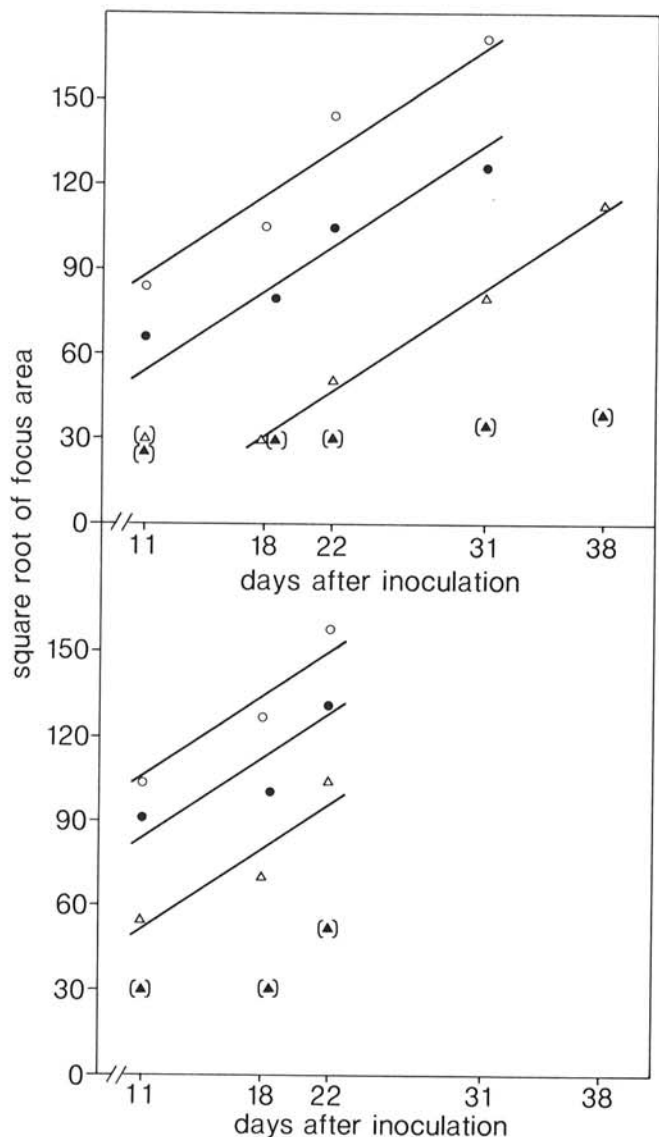


Fig. 7. Focus development of *Peronospora farinosa* race 3 on spinach cultivar Noorman in the field, autumn 1982. Severity levels: $\circ = 0.01$; $\bullet = 0.05$; $\Delta = 0.1$; and $\blacktriangle = 0.5$. Data within brackets were not used. Symbols represent successive observations.

model parameters. For instance, temperature is known to influence both the net reproduction and the parameters of the time kernel. Other examples are the effect of differences in resistance, cultivar mixtures, and plant density. Such studies, in connection with the study of the effect of model parameters on the velocity of focus expansion, c , can lead to predictions of the influence of environmental parameters on c .

APPENDIX I

We have collected the recipes for calculating various parameter estimates discussed in the main text.

Gamma density. The m observations on the spore production, I , as a function of t are denoted by (I, t_i) , where t is the time since the onset of the infectious period. The equation to be fitted to the data is (10):

$$I(t_i) = \zeta \frac{\beta(\beta t_i)^{n-1} \exp(-\beta t_i)}{\Gamma(n)}$$

where ζ is a normalization factor. Taking logarithms gives the linear relation

$$y = \alpha + \delta_1 x_1 + \delta_2 x_2$$

where

$$y = \ln I, x_1 = \ln t_i, x_2 = t_i, \alpha = \ln[\zeta \beta^n / \Gamma(n)], \delta_1 = n - 1, \delta_2 = -\beta.$$

If the convenient and not unreasonable assumption is made that any measurement errors are proportional to I , so that on a log scale the error variance is constant, good estimates of α , δ_1 , and δ_2 can be obtained by ordinary least squares. The resulting estimates for n and β are:

$$\hat{\beta} = \frac{a_3 a_5 - a_1 a_4}{a_1 a_2 - a_3^2}; \hat{n} = \frac{a_1 + a_5 + \hat{\beta} a_3}{a_1}$$

where

$$a_1 = m \sum_i x_{1i}^2 - (\sum_i x_{1i})^2, a_2 = m \sum_i x_{2i}^2 - (\sum_i x_{2i})^2, \\ a_3 = m \sum_i x_{1i} x_{2i} - \sum_i x_{1i} \sum_i x_{2i}, a_4 = m \sum_i x_{2i} y_i - \sum_i x_{2i} \sum_i y_i, \\ a_5 = m \sum_i x_{1i} y_i - \sum_i x_{1i} \sum_i y_i,$$

$$\text{and } \alpha = m^{-1} \left\{ \sum_i y_i + \hat{\beta} \sum_i x_{2i} - (\hat{n} - 1) \sum_i x_{1i} \right\}.$$

The corresponding estimated variances and covariances of the estimators are:

$$\hat{\text{var}} \hat{\beta} = m \hat{\sigma}^2 \frac{a_1}{a_1 a_2 - a_3^2}, \quad \hat{\text{var}} \hat{n} = m \hat{\sigma}^2 \frac{a_2}{a_1 a_2 - a_3^2},$$

$$\hat{\text{cov}}(\hat{\beta}, \hat{n}) = -m \hat{\sigma}^2 \frac{a_3}{a_1 a_2 - a_3^2}$$

$$\text{where } \hat{\sigma}^2 = \frac{1}{m-3} \sum_i (y_i - (\hat{\alpha} + (\hat{n} - 1) x_{1i} - \hat{\beta} x_{2i}))^2.$$

Bessel density. Let y denote the observed spore deposition at a distance x from the source. Formula 2.4 from reference 10 can be rewritten as:

$$y_i = \rho K_0(\xi_i) \quad (\text{AI.1})$$

where $\xi_i = x_i / \sigma_1$, and K_0 is the so-called modified Bessel function of the second kind of the order zero (see, e.g., Abramowitz and

Stegun [1]), and ρ is an unknown constant. The value σ_1 can be calculated by numerically minimizing

$$Q = \sum_i \left\{ y_i - \frac{(\sum_i y_i K_0(\xi_i)) (\sum_i (K_0(\xi_i))^2)^{-1} K_0(\xi_i)}{\sum_i (K_0(\xi_i))^2} \right\}^2$$

using the approximation formulae from Abramowitz and Stegun (1) to calculate K_0 . An approximate estimate for the variance of the resulting estimator, derived by a local linearization of equation AI.1, is:

$$\text{var } \hat{\sigma}_1 \approx \hat{\rho}^2 \left\{ \sum_i K_0^2(\hat{\xi}_i) \right\} \left\{ \sum_i K_0^2(\hat{\xi}_i) \right\}^2 \left\{ \sum_i K_0(\hat{\xi}_i) y_i \right\}^{-1} \\ \times \left\{ \sum_i K_0^2(\hat{\xi}_i) \sum_i (\hat{\xi}_i K_1(\hat{\xi}_i))^2 - (\sum_i K_0(\hat{\xi}_i) \hat{\xi}_i K_1(\hat{\xi}_i))^2 \right\}^{-1}$$

where $\hat{\xi}_i = x_i / \hat{\sigma}_1$ and

$$\hat{\rho}^2 = \frac{1}{n-2} \sum_i \left\{ y_i - \frac{(\sum_i y_i K_0(\hat{\xi}_i)) (\sum_i (K_0(\hat{\xi}_i))^2)^{-1} K_0(\hat{\xi}_i)}{\sum_i (K_0(\hat{\xi}_i))^2} \right\}^2.$$

APPENDIX II

Calculation of the wave speed. In the second paper of this series (10), we showed how to calculate γS_0 or p as functions of c when the other parameters are given. Here the interest is in the calculation of c , the wave speed, from given parameters. Equation A.1 from reference 10 reads as follows in unscaled form:

$$\left\{ \begin{aligned} \ln \gamma S_0 - \ln(1 - \frac{1}{2}(\lambda\sigma)^2) - \lambda c p - n \ln(1 + \frac{\lambda c}{\beta}) &= 0 \quad (\text{AII.1a}) \\ \frac{\lambda^2 \sigma^2}{1 - \frac{1}{2}(\lambda\sigma)^2} - \lambda c p - n \frac{\lambda c / \beta}{1 + \lambda c / \beta} &= 0 \quad (\text{AII.1b}) \end{aligned} \right.$$

Setting $\lambda c = \xi$, equation AII.1b can be written as:

$$(\xi^2 a_1 + (\xi) a_2 + a_3 = 0 \quad (\text{AII.2})$$

where

$$a_1 = -\frac{p}{\beta} (1 - \frac{1}{2}(\lambda\sigma)^2), \\ a_2 = (\lambda\sigma)^2 \frac{1}{\beta} - (p + \frac{n}{\beta}) (1 - \frac{1}{2}(\lambda\sigma)^2), a_3 = (\lambda\sigma)^2.$$

Solving equation AII.2 for ξ , taking into account that $\xi > 0$, gives:

$$\xi = \frac{-a_2 - \sqrt{a_2^2 - 4a_1 a_3}}{2a_1}.$$

Note that ξ still depends on λ . Substitution of ξ in equation AII.1a leads to an equation for λ that can be solved for given p, n, β, σ , and γS_0 using Newton-iteration. Substitution of this λ in the formulae for a_i gives ξ . Finally c can be calculated from

$$c = \frac{\xi}{\lambda}.$$

APPENDIX III

Sensitivity analysis. Assume for the time being that an explicit expression in terms of the model parameters is available for c :

$$c = f(\beta, n, \sigma, \gamma S_0).$$

When the variances of the estimators $\hat{\beta}$, \hat{n} , $\hat{\sigma}$, and $\gamma \hat{S}_0$ are small, and provided the pair β, n , and σ as well as γS_0 are estimated

directly in separate experiments, we may set to first order of approximation:

$$\begin{aligned} \hat{v} \hat{c} \approx & \left(\frac{\partial f}{\partial \beta} \right)^2 \hat{v} \hat{r}(\hat{\beta}) + 2 \frac{\partial f}{\partial \beta} \frac{\partial f}{\partial n} \text{cov}(\hat{n}, \hat{\beta}) + \left(\frac{\partial f}{\partial n} \right)^2 \hat{v} \hat{r}(\hat{n}) \\ & + \left(\frac{\partial f}{\partial \sigma} \right)^2 \hat{v} \hat{r}(\hat{\sigma}) + \left(\frac{\partial f}{\partial \gamma S_0} \right)^2 \hat{v} \hat{r}(\gamma \hat{S}_0) \end{aligned}$$

where the partial derivatives are evaluated at the estimated values of β , n , σ , and γS_0 .

Only c is not given explicitly but should be calculated from the system of equations (AII.1), which can be written as:

$$\begin{aligned} h_1(c, \lambda, \beta, n, \sigma, \gamma S_0) &= 0 \\ h_2(c, \lambda, \beta, n, \sigma, \gamma S_0) &= 0. \end{aligned}$$

In that case, $\partial f / \partial X$, where X stands for β , n , σ , or γS_0 , can be calculated from:

$$\frac{\partial f}{\partial X} = - \left(\frac{\partial h_2}{\partial \lambda} \frac{\partial h_1}{\partial X} - \frac{\partial h_1}{\partial \lambda} \frac{\partial h_2}{\partial X} \right) \left(\frac{\partial h_1}{\partial c} \frac{\partial h_2}{\partial \lambda} - \frac{\partial h_1}{\partial \lambda} \frac{\partial h_2}{\partial c} \right)^{-1}$$

A second complication is that, in the example of the stripe rust-wheat pathosystem, not γS_0 but r was estimated in a separate experiment. Proceeding as before, set (NB: f now has a different meaning)

$$c = f(\beta, n, \sigma, r).$$

This implies that in the expression for $\hat{v} \hat{r}(\hat{c})$ the last term has to be replaced by $(\partial f / \partial r)^2 \hat{v} \hat{r}(r)$ and $\partial f / \partial X$ must be calculated starting from:

$$h_i(c, \lambda, \beta, n, \sigma, r) = 0$$

with h_i given as

$$h_i(c, \lambda, \beta, n, \sigma, r) = p_i(c, \lambda, \beta, n, \sigma, \gamma S_0)$$

combined with

$$\gamma S_0 = g(\beta, n, r).$$

For $X = \beta, n$ this leads to

$$\frac{\partial h_i}{\partial X} = \frac{\partial p_i}{\partial X} + \frac{\partial p_i}{\partial \gamma S_0} \frac{\partial g}{\partial X},$$

and for $X = r$, this leads to

$$\frac{\partial h_i}{\partial X} = \frac{\partial p_i}{\partial \gamma S_0} \frac{\partial g}{\partial r}$$

where again all functions are evaluated at \hat{c} , $\hat{\lambda}$, $\hat{\beta}$, \hat{n} , $\hat{\sigma}$, $\gamma \hat{S}_0$, and \hat{r} .

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