# Focus Expansion in Plant Disease. I: The Constant Rate of Focus Expansion

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## ABSTRACT

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According to theory, a focus of disease in a crop expands radially at a rate that asymptotically approaches a constant value. This value can be calculated from the time kernel, contact distribution, and gross reproduction. Time kernel describes the inoculum production through time and contact distribution describes inoculum dispersal. Gross reproduction is the total number of victimized individuals produced by a single infectant placed in a population consisting of suscepts only. Definitions and mathematical evidence are given.

*Additional key words*: epidemiology, focus, model.

From a geometric point of view, epidemics are classified as general, focal, or intermediate (18,20). Focal epidemics begin with foci that can be initiated by a single infectious unit. Such epidemics were described for stripe rust (*Puccinia striiformis* West.) on wheat by Zadoks (18) and for potato late blight (*Phytophthora infestans* (Mont.) de Bary) by Zaag (17). At the other end of the scale are the general epidemics, where initial inoculum is so abundant that foci are inconspicuous. General epidemics occur when stripe rust inoculum overwinters profusely or when massive amounts of windborne stem rust (*Puccinia graminis* Pers.) inoculum arrive from afar.

When a stone is thrown into a still pond, the disturbance of the mirror-flat water surface appears as a series of circular waves that expand gradually. The outer wave seems to travel at a constant radial velocity. A disease focus, originating from a single diseased individual, expands in a circular wavelike pattern not dissimilar from the outer traveling wave on the water surface. Kampmeijer and Zadoks (4,19), studying focal behavior by means of the dynamic simulator EPIMUL, found that the front of the focus expanded radially at a constant velocity once an initial phase of focus buildup had passed by. In other words, the velocity of the epidemic wave approaches a limit value (Fig. 1).

In recent papers, Minogue and Fry (9,10) studied the spread of an epidemic in one-dimensional space. They used a constant inoculum production and fixed latency and infectious periods. The dispersal function was a double geometric distribution. Application of stochastic and deterministic versions of the model provided much insight in the spatial spread of epidemics. They indicated the need for extension of the model to cover asymmetric inoculum dispersal, more realistic patterns of spore production, and spread through two-dimensional space.

Except for asymmetric dispersal, this extension can be found in the more general and abstract epidemiologic model developed independently by Thieme (13,14) and Diekmann (1,2). A detailed account of the model formulation in the nonspatial case is given (7,8). The present paper elaborates on some phytopathologically interesting aspects of their models.

The term *front* is defined as that part of the disease profile that is in the presaturation phase—i.e., the foremost tail of the profile (4,19). The disease profile is quantified in terms of densities of diseased individuals per unit of area. In experimental situations, all quantities should be measured as numbers of individuals (sites, leaves, or plants) and not as percentage of leaf area. At low disease severities, there is an approximately linear correspondence between diseased leaf area and the number of diseased individuals, so that—with caution—percentage values can be used.

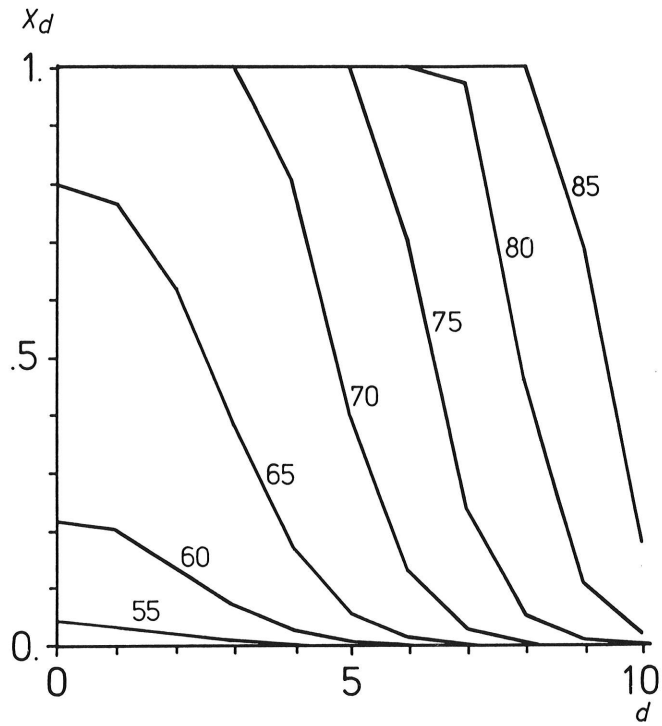


**Fig. 1.** Disease gradients of growing focus plotted at various days. Focus was started by single effective spore. Abscissa: distance $d$ from center of focus measured in compartments. Ordinate: severity $x_d$. Entries: number of days from start. From Zadoks and Kampmeijer (19). After 70 days, distance between two successive disease profiles is about constant and rate of focus expansion has reached limit value. (Reprinted with permission of Ann. NY Acad. Sci. 287:171 (1977))

## DESCRIPTION OF THE MODEL

The model will be derived to calculate the asymptotic wave velocity of the front of a focal epidemic. The model is based on knowledge of the underlying phenomena such as spore production, spore dispersal, and effectiveness of infectious units. The model is based on a set of assumptions that will be discussed first. Symbols are explained in Table 1. Physical dimensions of variables are specified within square brackets.

**Assumptions about the host population.** 1a. The host population consists of individuals, called suscepts, that are all equally susceptible to the disease. For *individual*, one may read plant, leaf, or site (a limited area of the leaf that can become a lesion [4]). The population of suscepts is distributed homogeneously over an infinite two-dimensional habitat and initially has density $S_0$ $[N_{su}L^{-2}]$.

1b. After infection, a suscept becomes a victim. A victim passes through three phases. Phase 1 is noninfectious because the disease is in its latency period. During phase 2, the victim is infectious; it is then called an infectant. During phase 3, the victim is again noninfectious because of death or immunity. A victim thus cannot become a suscept again.

1c. A suscept occupies an area $\zeta$. Therefore, $\zeta S_0$ is the fraction of the two-dimensional habitat covered by suscepts plus victims.

1d. The population of suscepts and the total number of victims are so large that a deterministic model can be used. Single individuals within the population can, however, be subject to stochastic processes.

1e. Changes in the number of suscepts by growth, birth, death, or migration are disregarded except when death is caused by the disease under consideration.

TABLE 1. List of symbols

| Symbol | Explanation | Dimension[a] |
|---|---|---|
| $\alpha$ | $= p/i$ | [1] |
| $c$ | Asymptotic velocity of frontal wave | $[L \cdot T^{-1}]$ |
| $c^*$ | $= ic/\sigma$, scaled wave velocity | [1] |
| $\gamma S_0$ | Gross reproduction ratio | [1] |
| $D(\vec{x}, \vec{\xi})$ | Contact distribution, two-dimensional distribution of inoculum produced by individual positioned at $\vec{\xi}$ | $[L^{-2}]$ |
| $\tilde{D}(x_1)$ | Marginal distribution of $D(\vec{x}, \vec{\xi})$ | $[L^{-1}]$ |
| $\zeta$ | Area occupied by suscept | $[L^2 \cdot N_{su}^{-1}]$ |
| $I(\tau)$ | Time kernel, number of infectious units produced per infectant per unit of time | $[N_{iu} \cdot T^{-1} \cdot N_{vi}^{-1}]$ |
| $\bar{I}$ | Total number of infectious units produced per infectant | $[N_{iu} \cdot N_{vi}^{-1}]$ |
| $i(\tau)$ | Normalized time kernel | $[T^{-1}]$ |
| i | Infectious period | [T] |
| $\lambda$ | Shape parameter of epidemic wave front | $[L^{-1}]$ |
| $L$ | Auxiliary function (Diekmann [1]) | [1] |
| $l(c, \lambda)$ | $= \ln L(c, \lambda)$ | [1] |
| $\vec{\xi}$ | Source position | [−] |
| p | Latency period | [T] |
| $S(t, \vec{x})$ | Density of suscepts at time $t$ and position $\vec{x}$, in number per unit area | $[N_{su} \cdot L^{-2}]$ |
| $\sigma^2$ | Variance of contact distribution | $[L^2]$ |
| $t$ | Time | [T] |
| $\tau$ | Period of time evolved since infection of individual: age of infection | [T] |
| $\vec{x}$ | Target position or position where epidemic is evaluated | [−] |
| $y(t, \vec{x})$ | Relative rate of victimization at target position $\vec{x}$, or relative infection rate, in number of victims per number of suscepts per unit of time | $[T^{-1}]$ |
| $S_0$ | Initial density of suscepts | $[N_{su} \cdot L^{-2}]$ |
| $\psi$ | Effectiveness, in number of victims per infectious unit | $[N_{vi} \cdot N_{iu}^{-1}]$ |

[a] For use of dimensions, see Zadoks and Schein (20). Dimension number [N] is subdivided by indexes iu = infectious units, su = suscepts, and vi = victims.

**Assumptions about the disease.** 2a. Disease is physically transferred from an infectant to a suscept in the form of inoculum consisting of infectious units (20), as (for example) fungal spores.

2b. The function describing the mean production of infectious units by a victim at time $\tau$ after infection ($I(\tau)$) is called *time kernel* $[N_{iu} \cdot T^{-1} \cdot N_{vi}^{-1}]$. (For symbol explanations, see Table 1.) The $I$ function includes latency period and infectious period: during latency $I(\tau) = 0$.

2c. The progress of the disease within an individual is fully autonomous, e.g., not influenced by other victims.

**Assumptions about the inoculum.** 3a. Inoculum (in the form of infectious units) moves from an infectant (the source) to a suscept (the target) with positions $\vec{\xi} = (\xi_1, \xi_2)$ and $\vec{x} = (x_1, x_2)$, respectively.

3b. The infectivity of the infectious units is constant.

3c. The probability per unit area that an infectious unit released at $\vec{\xi}$ will be deposited at $\vec{x}$ is described by a function $D(\vec{x}, \vec{\xi})$ called *contact distribution*. This probability is a function of the distance between $\vec{\xi}$ and $\vec{x}$ only. Consequently, $D(\vec{x}, \vec{\xi})$ is rotationally symmetric.

$$D(\vec{x}, \vec{\xi}) = D(\sqrt{|x_1 - \xi_1|^2 + |x_2 - \xi_2|^2}).$$

The dimension of $D$ is $[L^{-2}]$. Conceptually the contact distribution and Gregory's (3) primary gradient are equal apart from being defined in two and one dimension, respectively.

3d. The decrease in the density of suscepts at time $t$ and position $\vec{x}$, which is also the increase in the density of victims, equals the product of the density of suscepts and the rate of victimization $y$ at $\vec{x}$. The rate of victimization, which is the probability per unit of time that a suscept becomes a victim, is only dependent on the total number of infectious units arriving at $\vec{x}$ from all sources $\vec{\xi}$. This is the usual "law of mass action" assumption.

3e. An infectious unit is not necessarily effective. It may be deposited on the ground or it may not infect even though deposited on a suscept. The former effect is accounted for by $\zeta S_0$, the fraction of the habitat covered by individuals, which is also the chance that an infectious unit will be deposited on an individual. The latter effect is accounted for by the effectiveness $\psi$, the chance that an infectious unit will be effective. The dimension of $\psi = [N_{vi} \cdot N_{iu}^{-1}]$. Note that $\psi$ does not refer to Vanderplank's (15) correction factor $(1 - x)$. The correction for decreasing numbers of susceptibles is accounted for by the occurrence of $S(t, x)$, the current density of suscepts (instead of $S_0$), in equation (1.1).

## DEVELOPMENT OF THE MODEL

Here we shall give a phytopathologically oriented derivation, accompanied by a discussion of the dimensions. A more abstract derivation can be found in Thieme (13) and Diekmann (1).

In accordance with assumption 3d, we can write:

$$\frac{\partial S}{\partial t}(t, \vec{x}) = -S(t, \vec{x}) \times y(t, \vec{x}) \qquad (1.1)$$

$$[N_{su} \cdot L^{-2} \cdot T^{-1}] = [N_{su} \cdot L^{-2}] \times [T^{-1}].$$

The minus sign indicates loss of suscepts, victimization. For symbol explanations, see Table 1.

To calculate $y(t, \vec{x})$ we observe that victimization is the result of two different groups of infectants. The first group is the infectants that were introduced into the susceptible population at $t = 0$. These individuals started the epidemic. The relative rate of victimization resulting from these individuals will be denoted by $y_0(t, \vec{x})$. The second group is the individuals that were victimized after $t = 0$. The relative rate of victimization resulting from these individuals will be denoted by $y_1(t, \vec{x})$.

Multiplication of the time kernel by the contact distribution gives the inoculum supply at target $\vec{x}$ resulting from victims at position $\vec{\xi}$ that were infected $\tau$ ago:

$$I(\tau) \times D(\vec{x}, \vec{\xi}) \qquad [N_{iu} \cdot T^{-1} \cdot N_{vi}^{-1}] \times [L^{-2}]. \qquad (1.2)$$

Furthermore, at position $\vec{\xi}$ the rate of victimization $\tau$ ago is:

$$-\frac{\partial S}{\partial t}(t-\tau, \vec{\xi}) \qquad [N_{su} \cdot L^{-2} \cdot T^{-1}]. \tag{1.3}$$

Multiplication of equations 1.3 and 1.2, the area occupied by a single suscept ($\zeta$), and the efficiency factor ($\psi$) gives the rate of victimization at target $\vec{x}$ resulting from all infectants at source $\vec{\xi}$ infected a period $\tau$ ago. Integration of the product over $\tau$ and over all sources $\vec{\xi}$ produces the rate of victimization at position $\vec{x}$. Equation 1.1 becomes:

$$\frac{\partial S}{\partial t}(t, \vec{x}) = S(t, \vec{x}) \times \left\{ \int_0^t \int_{\mathbf{R}^2} \frac{\partial S}{\partial t}(t-\tau, \vec{\xi}) \times I(\tau) \times D(\vec{x}, \vec{\xi}) \times \zeta \times \psi \times d\vec{\xi} \times d\tau - y_0(t, \vec{x}) \right\} \tag{1.4}$$

$$[N_{su} \cdot L^{-2} \cdot T^{-1}] = [N_{su} \cdot L^{-2}] \times \left\{ [N_{su} \cdot L^{-2} \cdot T^{-1}] \times [N_{iu} \cdot N_{vi}^{-1} \cdot T^{-1}] \times [L^{-2}] \times [L^2 \cdot N_{su}^{-1}] \times [N_{vi} \cdot N_{iu}^{-1}] \times [L^2][T] \right\}.$$

Note that the integration over the habitat is a double integration over the two variables $\xi_1$ and $\xi_2$. Therefore, the dimension of $d\vec{\xi}$ is given as $[L^2]$.

The initial rate of victimization $y_0(t, \vec{x})$ can be described in terms of $I(\tau)$, $D(\vec{x}, \vec{\xi})$, $\zeta$, and $\psi$ as done above for $y_1(t, \vec{x})$. However, the influence of the initial inoculum that started the epidemic will approach zero when time $t$ becomes sufficiently large; consequently, $y_0(t, \vec{x})$ may then be neglected.

## GROSS REPRODUCTION

The foregoing model is based on a set of underlying phenomena defined in the assumptions. These phenomena can be measured, at least in principle. However, some of them, especially the efficiency factor $\psi$, are difficult to measure. Several factors can be lumped into a gross reproduction $\gamma S_0$, which is relatively easy to assess. This gross reproduction is defined theoretically as the number of daughter victims that would be produced by a single mother victim, during the whole course of its disease, if the surrounding population were to consist permanently of suscepts only (e.g., by every daughter victim being replaced immediately by a new suscept).

The total number of infectious units produced by a single individual during the whole course of its disease is given by:

$$\bar{I} = \int_0^\infty I(\tau) d\tau \tag{2.1}$$

$$[N_{iu} \cdot N_{vi}^{-1}] = [N_{iu} \cdot N_{vi}^{-1} \cdot T^{-1}] \times [T].$$

Define

$$i(\tau) = I(\tau)/\bar{I} \tag{2.2}$$

$$[T^{-1}] = [N_{iu} \cdot N_{vi}^{-1} \cdot T^{-1}] \times [N_{iu} \cdot N_{vi}^{-1}]^{-1}.$$

The resulting function $i(\tau)$ has unit area. Only the shape of $I(\tau)$ is retained in $i(\tau)$. $i(\tau)$ is a relative rate. The function $i$ is called *normalized time kernel.*

Multiplying $\bar{I}$ with the efficiency factor $\psi$ gives the total number of infectious units that will be effective when deposited on a suscept. The chance that an infectious unit actually is deposited on a suscept equals $\zeta S_0$. Therefore:

$$\gamma S_0 = \int_0^\infty I(\tau) d\tau \times \zeta \times S_0 \times \psi \tag{2.3}$$

$$[1] = [N_{iu} \cdot N_{vi}^{-1} \cdot T^{-1}] \times [T] \times [L^2 \cdot N_{su}^{-1}] \times [N_{su} \cdot L^{-2}] \times [N_{vi} \cdot N_{iu}^{-1}].$$

The procedure to measure $\gamma S_0$ implied by the theoretical definition is not feasible in practice. When the number of replacements per unit area is low in comparison with the density of suscepts, a fair approximation is obtained without replacements, though contamination by later generations of victims can be troublesome. In the third paper we shall discuss more practical ways to measure $\gamma S_0$.

## ASYMPTOTIC VELOCITY OF THE EPIDEMIC WAVE

If the contact distribution is rotationally symmetric, as we assumed, the focus should have circular symmetry as well. Therefore, contours of equal disease intensity behave like expanding circles. An ever-expanding circle will, in the long run, become locally indistinguishable from a straight line. For that reason, the focal front in a certain direction should eventually behave like a planar front. Therefore, it should be possible to deduce the behavior of the focal front by considering planar traveling wave solutions of equation 1.4; i.e., solutions having a constant disease profile in a certain (say the $x_2$) direction that moves unchanging with a constant speed (in the $x_1$ direction). If we consider such a planar wave, it necessarily satisfies in the $x_1$ direction a one-dimensional analog of equation 1.4 with the (marginal) contact distribution

$$\tilde{D}(x_1) = \int_{-\infty}^{\infty} D(x_1, x_2) dx_2. \tag{3.1}$$

(Calculating the number of infectious units deposited at a certain place that is derived from a straight line of infectants with a constant age distribution equals adding up all contributions from all victims on that line. Taking the marginal distribution just amounts to adding up these contributions.)

The results of Diekmann (1,2) and Thieme (13,14) imply that the speed, $c$, of the (only) traveling wave solution that matters can be calculated from

$$\begin{cases} l(c, \lambda) = 0 \\ \dfrac{\partial l}{\partial \lambda}(c, \lambda) = 0 \end{cases} \tag{3.2}$$

where

$$l(c, \lambda) = \ln L(c, \lambda) \tag{3.3}$$

$$L(c, \lambda) = \gamma S_0 \int_0^\infty e^{-\lambda c\tau} i(\tau) d\tau \int_{-\infty}^{\infty} e^{-\lambda x_1} \tilde{D}(x_1) dx_1. \tag{3.4}$$

The front of the wave has an exponential shape with parameter $\lambda$:

$$S_0 - S \propto \exp. \lambda(ct - x_1) \tag{3.5}$$

where $S_0 - S$ is the number of victims.

In theory, there also exist waves with speeds larger than $c$. However, Diekmann and Thieme have proved that if one moves from the center of the focus with a speed larger than $c$ one will eventually outrun the focus, whereas if one moves with a speed smaller than $c$ one will eventually encounter the terminal severity.

In conclusion, we can say that if the gross reproduction, time kernel, and marginal contact distribution are known, the asymptotic velocity of the epidemic wave can be calculated numerically from equations 3.2–3.4.

## AN EXAMPLE

The simulation model introduced in EPIMUL (4,19) simulates a spatial variant of the Vanderplank (15) equation. The time kernel in EPIMUL is a block function with a latency period p and an

infectious period i (Fig. 2). It is written as

$$i(\tau) = \begin{cases} 0 \text{ if } \tau < \text{p or } \tau > \text{p} + \text{i} \\ \\ \text{i}^{-1} \text{ if } \text{p} \leqslant \tau \leqslant \text{p} + \text{i} \end{cases} \quad [\text{T}^{-1}] \qquad (4.1)$$

and

$$\int_0^{\infty} e^{-\lambda c\tau}\, i(\tau)\mathrm{d}\tau = \int_{\text{p}}^{\text{p+i}} e^{-\lambda c\tau}\, \text{i}^{-1}\, \mathrm{d}\tau = e^{-\text{p}c\lambda}\,(1 - e^{-\text{i}c\lambda})/\,\text{i}c\lambda. \qquad (4.2)$$

The contact distribution in EPIMUL is a two-dimensional Gaussian distribution with variance $\sigma^2$. From statistical theory it follows that $\tilde{D}(x)$ is the one-dimensional Gaussian distribution, so that

$$\int_{-\infty}^{+\infty} e^{-\lambda x}\, \tilde{D}(x)\mathrm{d}x = e^{(1/2)(\lambda\sigma)^2} \qquad (4.3)$$

(see, e.g., Kendall [6]).

Combination of equations 3.4, 4.2, and 4.3 gives

$$L(c, \lambda) = \gamma S_0\, \frac{e^{-\text{p}c\lambda}\,(1 - e^{-\text{i}c\lambda})}{\text{i}c\lambda}\, e^{(1/2)(\lambda\sigma)^2} \qquad (4.4)$$

and with equation 3.3

$$l(c, \lambda) = \ln \gamma S_0 - \text{p}c\lambda + \ln (1 - e^{-\text{i}c\lambda}) + (1/2)(\lambda\sigma)^2 - \ln (\text{i}c\lambda).$$

After rearrangement, equation 3.2 becomes

$$\begin{cases} 1 - \alpha c^*\lambda^{-1} - \lambda^{-2} - [1 - (\alpha + 1)c^*\lambda^{-1} - \lambda^{-2}]e^{-c^*\lambda} = 0 \\ \\ \gamma S_0 = \dfrac{c^*\,\lambda\, e^{(\alpha \times c^* \times \lambda - (1/2)\lambda^2)}}{1 - e^{(-c^*\lambda)}} \end{cases} \qquad (4.5)$$

where $\alpha = \text{p/i}$, and $[\text{T/T}] = [1]$
$c^* = \text{i}c/\sigma$. $[\text{T} \cdot \text{L/T/L}] = [1]$

Unfortunately, equation 4.5 cannot be solved explicitly. For given values of $c^*$ and $\alpha$, $\lambda$ can be derived numerically from the first part of equation 4.5. Substitution of this $\lambda$ in the second part gives $\gamma S_0$. The numerical solution of such equations can be programmed on a programmable pocket calculator (12). Some results are given in Figure 3.

The parameters p/i and $\sigma$ also appear in EPIMUL, whereas $\gamma S_0$ equals the daily multiplication factor times the infectious period (i) (4). For Figure 1, the value of $c^*$ can be derived as $c^* = c\text{i}/\sigma$, where $c$ is the frontal displacement rate in compartments per day multiplied by the length of a compartment. Using the same values for the parameters in the Diekmann/Thieme model, we find:

| | |
|---|---|
| EPIMUL | $c^* = 2.63$ |
| Diekmann/Thieme | $c^* = 2.47$. |

The difference between the two values, possibly resulting from difference in habitat characterization, seems negligible. In EPIMUL the habitat is subdivided into discrete squares, whereas in the present paper the habitat is treated as a continuum.

A typical result of EPIMUL is that, contrary to Vanderplank's (16) opinion, the daily multiplication factor, equivalent to $\gamma S_0/\text{i}$, has little effect on the wave velocity (4).

Figure 3 confirms that, with fixed p and i, $\gamma S_0$ has little effect on the wave velocity when $\text{p/i} < 2$ and $\gamma S_0 > 10$. When, however, $\gamma S_0 < 10$ and/or $\text{p/i} > 2$, $\gamma S_0$ can have a marked retarding effect on the wave velocity. Biologically, this effect is understandable; both a high p/i ratio (due to a high p value) and a low multiplication rate (due to low $\gamma S_0$) will retard epidemic progress.

## DISCUSSION

This paper describes a generalized model for the spread of infectious disease in two-dimensional space. The advantage of the present model is that any pattern of inoculum production (time kernel) and of spatial distribution of inoculum (contact distribution) can be inserted. The model is a generalization of the models by Kendall (5), Mollison (11), and Minogue and Fry (9,10).

Minogue and Fry (9,10) introduced the gradient parameter, $g$, as the slope of the straight line resulting from plotting the logit of disease severity against distance from the point of inoculation. It is a characteristic of the entire wave, and its use depends on the adequacy of the logistic equation as a description of the shape of the wave. In this paper we introduced the shape parameter, $\lambda$, to characterize the steepness of the front of the wave, which always has the shape of an exponential curve (1,13,14; van den Bosch et al, *in preparation*). The $\lambda$ is equivalent to $g$ for low disease levels. Note that the local apparent infection rate, $r$, of Minogue and Fry is the equivalent to $\lambda c$ in the Diekmann/Thieme model.

The assumptions underlying the model approximately fit the agricultural situation, where crops functioning as host populations are nearly homogeneous. Not infrequently, initial infections are sufficiently spread out for foci to develop.

Equations 3.1–3.4 allow calculation of the velocity with which the focus will eventually expand. The speed with which the asymptotic velocity is attained is a different matter. Zadoks and

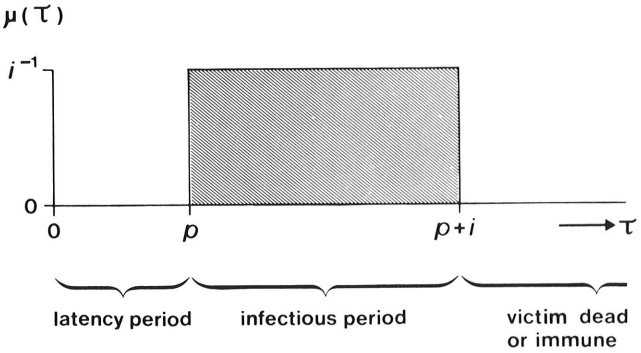


**Fig. 2.** Normalized time kernel showing relative infectiousness, $i(\tau)$, against time after infection, $\tau$, in shape of block function.

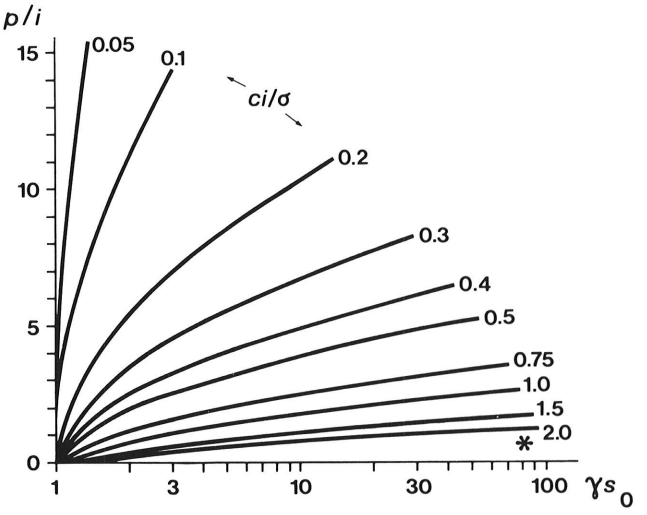


**Fig. 3.** Relation between $\alpha = \text{p/i}$ ratio (latency period divided by infectious period) and gross reproduction $\gamma S_0$ under various values of scaled wave velocity $c^* = c\text{i}/\sigma$.

Kampmeijer (19) numerically found a rapid convergence to a constant velocity. A similar result was obtained in a diffusion type model by M. Zawoľek (*personal communication*). The experimental examples to be discussed show rapid convergence to the asymptotic velocity within the time span of the experiments. The authors thus believe that the asymptotic velocity can be used in most situations.

The model assumes an infinite space available for focus expansion. In fact, because the area occupied by a focus is usually small in comparison with the area of the field, the assumption is reasonable.

Though inoculum plays a role in the model description, inoculum is not used per se. Inoculum is described in terms of its effect—that is, in terms of diseased individuals. The total number of infectious units produced by one diseased individual and the proportion of spores that actually cause new infections are brought together in $\gamma S_0$, a value comparable to $i \cdot R_c[T \cdot T^{-1}] = [1]$ in Vanderplank (15) and to the net reproduction $R_0$ in life table statistics (20). This parameter of effectiveness of disease has a simple interpretation and can be experimentally determined.

The next step would be to produce realistic submodels for the time kernel and the contact distribution and to calculate the associated wave velocity, $c$, and wave steepness, represented by $\lambda$. This will be the subject of the next paper. In a third paper we shall discuss parameter estimation and experimental examples.

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