

EFFECT OF FIELD GEOMETRY ON THE SPREAD OF CROP DISEASE

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ABSTRACT

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The spread of plant diseases which have passive airborne dispersal stages has been studied using mathematical models which include time-dependent features of pathogen population growth and dispersal. These reaction-diffusion models, which are based on cereal rust biology, demonstrated that field geometry can have a major influence on epidemic development. Assuming pathogen dispersal approximates a random diffusion process, it was shown that the greater the perimeter to area ratio of the fields in an agricultural region, the slower the increase of disease within those fields. Hence, when a constant proportion of a region's acreage was allocated to a crop, the models indicated that decreasing field size and elongating field shape can retard disease progress and thus reduce yield losses. Moreover, knowledge of the prevailing direction of pathogen dispersal by wind can be used to further augment the advantages of designing fields with large perimeter to area ratios.

The results are discussed in terms of previous work in plant disease epidemiology in particular and population dynamics in general. It is suggested that multilines may represent the limiting case for decreasing field sizes when a fixed fraction of the region is sown to each constituent variety. Methods for testing the results in the field are considered.

INTRODUCTION

Aerial dispersal has long been recognized as a significant factor in the spread of many of the most destructive parasitic diseases of man's crops. The importance of determining how the design of agroecosystems affect epidemic development stems directly from man's struggle to feed his exploding populations in the face of increasing losses in agricultural production to plant pathogens (Pimentel, 1977).

When a given total acreage within a region is planted with a certain crop or variety, the chances of disease spreading from one field to another may be a direct consequence of the size and shape of the fields. Van der Plank (1948, 1949, 1960) and Waggoner (1962) studied this problem for pathogens which

are passively dispersed by wind. They assumed that all fields are the same size and that shape and orientation are constant.

Noting that much inoculum falls back into the field from which it originates, Van der Plank reasoned that changes in the amount of escaping inoculum would be inconsequential to disease progress within the field but crucial to the spread of disease between fields. According to his mathematical model, the probability of infection per unit area in one field by propagules released from another decreases as the distance between fields increases. Since the distance between fields is proportional to the size of individual fields when a constant acreage is grown, he recommended large fields.

Subsequently, Waggoner (1962) suggested that since disease spreads relatively easily once established within a field, the probability of infection per field may be a more appropriate indicator of potential epidemic development than Van der Plank's measure, the probability per unit area within a field. Waggoner based his analysis of the aerial spread of disease between fields on Gregory's (1945) equation. Gregory adapted Sutton's (1932) theory of atmospheric eddy diffusion to describe the decrease in density of inoculum deposited with distance from the point of release.

Waggoner's (1962) model indicated that the probability of infection in one field by inoculum from another decreases as the field area decreases. In contrast to Van der Plank (1948, 1949, 1960, 1963), he concluded that decreasing the size, and therefore the isolation, of fields into which a region's cropland is subdivided could inhibit epidemic development. Van der Plank and Waggoner have apparently maintained their opposing viewpoints ever since (Zadoks and Kampmeijer, 1977).

However, both Van der Plank and Waggoner implicitly assumed steady state conditions for their models. Hence, their conclusions are suspect if either the spread of disease over space, or the rate of inoculum production at the source, or both, change with time. Since both dispersal and production are time dependent during epidemic development (Stakman and Harrar, 1957; Cammack, 1958; Aylor, 1978; Berger and Luke, 1979), this problem deserves further analysis.

In this paper diffusion-reaction models are used to study the spread of plant disease when both dispersal and inoculum production are time-dependent. Although our models are designed specifically for cereal rusts, we believe that their applicability extends to many other plant pests with passively airborne dispersal stages. In Sections 1-6 we develop mathematical models to study the relationship between inoculum dispersal and production in single isolated fields for a variety of circumstances. Analytic and numerical techniques are used in Section 7 to study the effect of changing the size or shape of associated fields on disease spread; this is followed by an exposition of the general principles behind our results. Examples of the application of these principles in general, and of reaction-diffusion models in particular, to the population dynamics of other systems, including multilines, are discussed. Procedures for testing our conclusions in the field are outlined.

PROPOSITIONS

(1) *The model*

The model is developed around a structure similar to that used by Fleming (1980b). Since this concerns situations where the rates of inoculum dispersal and production change markedly with time, our attention is restricted to occasions of rapid population growth when these circumstances occur most acutely. Assuming that low pathogen densities prevalent during these occasions exert a negligible influence on the density and genotypic content of the crop, we exclude host dynamics from the model.

Let $N(x, y, t)$ be the pathogen density, the total pathogen biomass (mycelium, pustules, and spores) per unit area, at position (x, y) at time t . Then the rate of change of pathogen density with time can be written

$$\frac{\partial N}{\partial t} = F_G(N) + F_D(N) \quad (1.1)$$

where $F_G(N)$ and $F_D(N)$ describe the rates of change of $N(x, y, t)$ due to population growth and dispersal, respectively.

Population growth, $F_G(N)$, can be separated into two components:

$$F_G(N) = G(N) - L(N) \quad (1.2)$$

where $G(N)$ and $L(N)$, respectively, represent the rates at which increases and decreases in parasite density at (x, y) occur as a result of local population dynamics.

Let B be the per unit rate of pathogen biomass production (through sporulation and lesion expansion), and let H be the fraction of this production lost when spores landing in the field fail to infect vulnerable host tissue (through e.g., deposition on the ground). Then

$$G(N) = Bf(N)(1 - H) \quad (1.3)$$

where B depends on the age structure of the pathogen population, B and H depend on the microclimatic conditions, and $f(N)$ is the effective parasite density. The function $f(N)$ describes the effects of density dependent competition among pathogens for the finite resources such as space, energy, or nutrients of the host plants.

Gregory (1973) has shown that early in the growing season, when the proportion of healthy vulnerable host tissue is high, the probability of multiple infection is low. Under these conditions competition is negligible and

$$f(N) = N \quad (1.4)$$

In (1.2) $L(N)$ represents the rate of loss of pathogen biomass per unit area due to: parasite senescence on healthy host tissue, disease-caused host deaths, plus natural host death. Since competitive or synergistic interactions among individual parasites are insignificant during the initial stages of epidemic

development (Van der Plank, 1963)

$$L(N) = MN \quad (1.5)$$

where M is the intrinsic rate of "natural" pathogen mortality. "Natural" mortality includes all forms of mortality except losses due to natural enemies which are assumed to be negligible.

The term $F_D(N)$ in (1.1) accounts for the fundamentally passive and air-borne movement of disease inoculum (e.g. spores). We assume that the effect of inoculum dispersal on $N(x, y, t)$ is a diffusion process. After a short time δt we assume that the offspring of a single reproducing parasite will be normally distributed about their parent with variance $\sigma^2 \delta t$ when dispersal occurs over a uniform surface of unlimited extent in all directions. Skellam (1951) gives the maximum likelihood estimate of σ^2 , the mean square dispersion rate during the time interval $(0, \delta t)$, as

$$\hat{\sigma}^2 = \frac{1}{n \delta t} \sum_{i=1}^n r_i^2$$

Here r_i is the distance of the i -th unit of pathogen biomass (e.g., spore) from its parent (e.g., pustule) and n is the number of observed values of r_i .

The random diffusion hypothesis implicitly assumes that a constant proportion of the pathogen population is dispersing at all times; that dispersal results in transport which is uncorrelated and random with respect to direction and distance; and (indirectly) that the probability of new infection decreases monotonically with distance from the source. The effects of wind on propagule dissemination, which can invalidate all of these assumptions, are considered in the discussion section. Briefly, we suggest that our conclusions are robust (Fleming, 1980a) when wind effects are included in the analysis.

Under the assumptions of random diffusive dispersal, Skellam (1951) shows that

$$F_D(N) = D^2 \left(\frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} + \frac{\partial^2}{\partial z^2} \right) N \quad (1.6)$$

where $D^2 = \sigma^2/2q$ for diffusion in q dimensions, $q = 1, 2, \text{ or } 3$ (see Appendix I).

Substituting (1.4) into (1.3)

$$G(N) = BN(1 - H)$$

and then combining this expression and (1.5) in (1.2), the local population dynamics are described by

$$F_G(N) = (B - BH - M)N = RN \quad (1.7)$$

This represents exponential population growth.

Using (1.6) and (1.7) in (1.1) to describe the growth of the disease population in two dimensions (see Appendix I)

$$\frac{\partial N}{\partial t} = RN + D^2 \left(\frac{\partial^2}{\partial x^2} + \frac{\partial^2}{\partial y^2} \right) N \quad (1.8)$$

Here we implicitly assume that the pathogen's age distribution is stable; that any decline in the diffusion rate, D , with crop growth is negligible; and that net changes in the growth rate of the parasite population, R , due to shifting environmental conditions or genetic adaptation to the host are small.

We have developed equation (1.8) in terms of pathogen biomass. However, following Okubo (1980, pp. 217–220), it can be shown that the same equation applies when N represents the number of pathogens in a disease population undergoing simple birth and death processes and age-dependent dispersal. In this case the “macroscopic” parameters R and D represent corresponding quantities averaged over the (microscopic) age structure. Thus, equation (1.8) can be thought of in terms of pathogen numbers as well as pathogen biomass density.

It is instructive to compare the representation of space in (1.8) with that of simulation models which have also been used to describe the growth and spread of plant disease in time and space. Both Shrum (1975) and Kampmeijer and Zadoks (1977) divided space into a number of compartments or cells, each of which could include a large number of host plants. They adopted functions which decreased monotonically with distance to describe the “long range” movement of spores between cells. But by spreading spores evenly within cells, they implicitly assumed that for “short range” dispersal the probability of infection is not a function of distance from the source.

Inconsistencies arise on the boundaries between cells. For example, consider two plants colinear with a point source of inoculum in such a simulation model. The probability of infection may be quite different at the two plants if they are separated by a cell boundary, while the probabilities would be exactly the same at the two plants even if they were separated by a much greater distance, provided they lay within the same cell.

Kiyosawa (1976) avoids these inconsistencies by modeling dispersal from plant to plant but his resulting computer program was so cumbersome and time consuming that he was forced to restrict his simulations to plots of only 97×97 plants. Our approach, equation (1.8), allows us to consider realistic field sizes without having to “compartmentalize” space and tolerate the attendant inconsistencies.

(2) Critical dimensions for rectangular fields: no survival outside

We begin by studying the effect of field size on pathogen population growth where death is instantaneous outside the field. Consider a rectangular field with sides of length a and b , respectively, in the x and y directions. It has one corner at the origin, $(x, y) = (0, 0)$ and its diagonally opposite corner at (a, b) . Outside the field we assume that the environment is so physiologically unsuit-

able for the parasite that it cannot survive there. This requires that the density $N(x, y, t)$ vanish on the field boundaries. Hence we look for solutions of equation (1.8) which satisfy the boundary conditions

$$N(x, y, t) = 0 \quad \text{at} \quad x = 0, x = a, y = 0, y = b; t > 0 \quad (2.1)$$

and initial conditions

$$N(x, y, 0) = N_1(x, y), \quad 0 < x < a, 0 < y < b \quad (2.2)$$

The function $N_1(x, y)$ describes the pathogen density at time zero and hence is positive somewhere within the field and negative nowhere.

It can be verified directly that

$$\sin \frac{m\pi x}{a} \sin \frac{n\pi y}{b} \exp [F(m, n)t] \quad (2.3)$$

where

$$F(m, n) = R - \pi^2 D^2 \left(\frac{m^2}{a^2} + \frac{n^2}{b^2} \right) t$$

is a solution of (1.8) and (2.1) for any positive integers m, n . Since any smooth initial density $N_1(x, y)$ can be written as a superposition of the sine functions occurring in (2.3), we can write the solution of (1.8) and (2.1) which satisfies (2.2) as

$$N(x, y, t) = \sum_{m=1}^{\infty} \sum_{n=1}^{\infty} A_{mn} \sin \frac{m\pi x}{a} \sin \frac{n\pi y}{b} \exp [F(m, n)t] \quad (2.4)$$

To find the constants A_{mn} we set $t = 0$ in equation (2.4) and multiply both sides by

$$\sin \frac{p\pi x}{a} \sin \frac{q\pi y}{b}$$

where p and q are integers, and integrate the result from $x = 0$ to a and from $y = 0$ to b . On rearranging the integral

$$A_{mn} = \frac{4}{ab} \int_0^a \int_0^b N_1(x, y) \sin \frac{m\pi x}{a} \sin \frac{n\pi y}{b} dy dx$$

As t increases, the behavior of the expansion (2.4) becomes dominated by its first term (the coefficient A_{11} is necessarily > 0):

$$N(x, y, t) \approx A_{11} \sin \frac{\pi x}{a} \sin \frac{\pi y}{b} \exp [F(1, 1)t], \quad \text{for large } t$$

Thus, at any point (x, y) inside the field, the pathogen density N eventually increases exponentially with time, remains the same, or decreases exponential-

ly with time, depending on whether

$$F(1,1) = R - \pi^2 D^2 / d^2 \geq 0 \quad (2.5)$$

where

$$d^2 = (a^{-2} + b^{-2})^{-1}$$

is a measure of the size of the field in terms of diffusion. It follows that any initial infection eventually either dies out or becomes an epidemic as d is respectively either less or greater than d_c where

$$d_c = \pi D / \sqrt{R} \quad (2.6)$$

The quantity d_c , with dimensions of length, is the critical value of d (above) for the occurrence or otherwise of infection spread within the field.

Since the loss of inoculum occurs through the field boundaries, the rate of loss is roughly proportional to the perimeter. In contrast, the production rate is proportional to the field's area because production occurs at every point within the field. Since a smaller field has a greater perimeter relative to its area than a larger field, dispersal has a relatively stronger effect in smaller fields. Equation (2.6) specifies the size (d_c) of fields for which production exactly

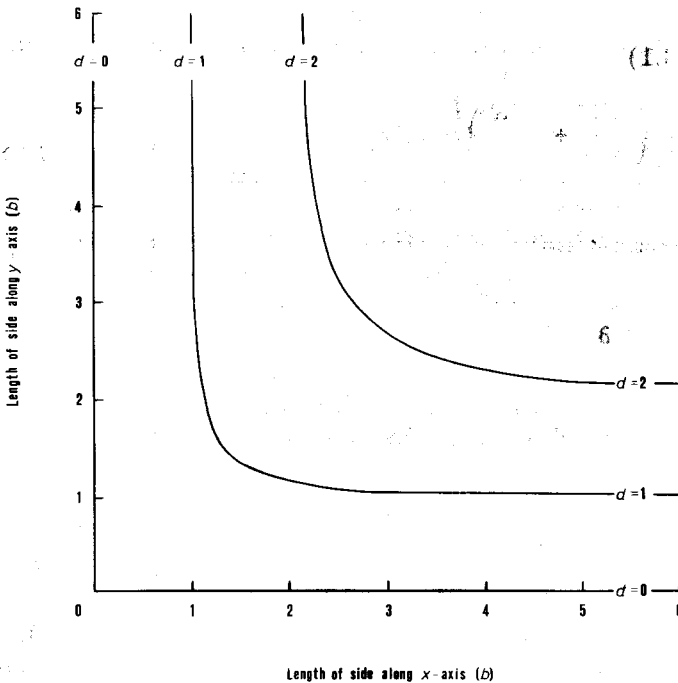


Fig. 1. Loci of d -isopleths in terms of the dimensions of a rectangular field isolated in an otherwise completely uninhabitable region (equation 2.5). The parameter d measures the size of the field in terms of the diffusing pathogen population; the larger d , the greater the likelihood that disease will increase in the field.

compensates for dispersal losses. In smaller fields ($d < d_c$) the pathogen population decreases with time because its growth rate in the field cannot offset the losses due to dispersal out of the field. On the other hand, in larger fields where $d > d_c$, population growth exceeds dispersal losses so pathogen biomass increases with time (Fig. 1). Note, however, that transient effects can occur before the lowest term in the series ($m = n = 1$) has achieved dominance in (2.4) and established a subsequently consistent trend (see Appendix II).

(3) Effects of field size on production and exit rates

Since the total pathogen biomass in the rectangular field of Section 2 at time t is

$$P(t) = \int_0^a \int_0^b N(x, y, t) dy dx \quad (3.1)$$

where $N(x, y, t)$ is given by (2.4), its rate of change is

$$\frac{dP}{dt} = \int_0^a \int_0^b \frac{\partial N}{\partial t} dy dx$$

Hence, by (1.8) and (3.1)

$$\begin{aligned} \frac{dP}{dt} &= \int_0^a \int_0^b \left\{ RN + D^2 \left(\frac{\partial^2 N}{\partial x^2} + \frac{\partial^2 N}{\partial y^2} \right) \right\} dy dx \\ &= RP - P_E \end{aligned}$$

where

$$P_E = D^2 \left\{ \frac{\partial}{\partial x} \int_0^b N dy + \frac{\partial}{\partial y} \int_0^a N dx \right\} \quad (3.2)$$

Here RP is the net rate at which pathogen biomass is created within the field, while P_E is the rate at which biomass is lost as inoculum leaves the field across the boundaries. We can write

$$\frac{dP}{dt} = (R - E)P, \quad \text{where } E = P_E/P \quad (3.3)$$

Here E is the per unit rate at which pathogen biomass leaves the field. It is the two-dimensional analogue of Kierstead and Slobodkin's (1953) "leakage".

Generally, the exit rate E is time-dependent, but as t becomes large, P and P_E become dominated by the lowest terms in their series. Hence, for steady state values, $m = n = 1$ in (2.4), (3.1), and (3.2), and substituting for P_E and

P in (3.3):

$$E \approx \pi^2 D^2 / d^2 \quad \text{when } t \text{ is large} \quad (3.4)$$

This relationship shows clearly that the per unit exit rate, E , depends on the field dimensions, d . Using this approximation in (3.3), at the steady state

$$(1/P)(dP/dt) = R - \pi^2 D^2 / d^2 \quad (3.5)$$

Hence, the smaller the size of the field in terms of diffusion, d^2 , the slower the per capita rate of pathogen biomass increase within an isolated field.

These relationships lead us to question the conclusions of Van der Plank (1960). He claimed that parasite dispersal between fields would be reduced by planting a few large fields which were far apart when a certain proportion of a region is allocated to a crop. However, (3.5) suggests that if fields are small enough, disease may be unable to establish itself at all.

(4) General critical dimensions: no survival outside

McMurtrie (1978) has generalized equation (2.6) for the critical value of d , the size of the field in terms of diffusion, to

$$d_c = kD/\sqrt{R} \quad (4.1)$$

where k is a constant of order unity. The value of k , like the expression for d , depends on the shape of the field. For instance, Skellam (1951) and Kierstead and Slobodkin (1953) have shown that if (1.8) describes the growth of a population in a strip of infinite length in one dimension and of width W in the other, then

$$d = W \quad \text{and} \quad k = \pi \quad (4.2)$$

This result follows directly from (2.5) by letting $b \rightarrow \infty$ and putting $a = W$.

A number of workers (e.g. Skellam, 1951, Landahl, 1959) have shown that (4.1), (4.2) also apply to populations undergoing logistic growth in an infinite strip of width W in an otherwise completely unfavorable environment. For logistic growth (1.7) takes the form

$$F_G(N) = RN(1 - N/N_{\max}) \quad (4.3)$$

where N_{\max} is the maximum population density the habitat can sustain. As before, the population collapses when $W \leq W_c$, the critical width, but when $W > W_c$ population growth is now limited to an equilibrium density N^* where $0 < N^* < N_{\max}$. This equilibrium density, N^* , increases with W , the width of the strip.

Bradford and Philip (1970) further extend these results by showing that criteria similar to (4.1), (4.2) arise for any form of local population growth, $F_G(N)$, which is a positive function for $0 < N < N_{\max}$ with $F_G(0) = F_G(N_{\max}) = 0$.

Skellam (1951) and Kierstead and Slobodkin (1953) have also shown that if (1.8) describes the growth of a population in a circular area of radius a in an otherwise totally inhospitable environment, then

$$k = 2.4048 \quad \text{and} \quad d = a \quad (4.4)$$

The number 2.4048 is the smallest positive root of the Bessel function of the first kind of order zero. It appears here because the spatial part of (1.8) in polar coordinates with radial symmetry is Bessel's equation of order zero (equation 5.2).

(5) *Critical dimensions for isolated circular areas: survival outside*

Having generalized (4.1) in terms of field geometry and population growth, we now extend our conclusions to situations where the environment outside the field is more hospitable. Equation (2.1) implies that inoculum suffers instantaneous death as soon as it reaches a field boundary. More realistically, survival is difficult, not impossible, outside the field.

To investigate how the hostility of the exterior environment affects the critical dimensions, consider dispersal in two dimensions with radial symmetry and assume that a patch of susceptible hosts is isolated in an otherwise inhospitable environment. Since R , as defined in (1.7), represents the survival and reproductive abilities of the parasite, it seems reasonable (Yarwood and Sylvester, 1959; Dimond and Horsfall, 1965) to set

$$R = R_i \quad \text{inside the patch: } r < a$$

and

$$R = -R_0 \quad \text{outside the patch: } r > a \quad (5.1)$$

Both R_i and R_0 are positive.

We begin by transforming (1.8) into polar coordinates. Setting $x = r \cos \theta$ and $y = r \sin \theta$ and recalling the radial symmetry about the centre of the patch, the rate of change in disease severity at time t at a distance r from the centre of the patch is

$$\frac{\partial N}{\partial t} = RN + D^2 \left(\frac{\partial^2 N}{\partial r^2} + \frac{1}{r} \frac{\partial N}{\partial r} \right)$$

We seek steady state solutions of this equation to determine how the severity of the external environment, which increases with R_0 , affects the critical radius of the patch. Hence, we put $\partial N / \partial t = 0$ and rearrange the resulting steady state equations into the forms of Bessel's equation of order zero ($R > 0$) and Bessel's modified equation of order zero ($R < 0$), respectively

$$\frac{d^2 N}{dQ^2} + \frac{1}{Q} \frac{dN}{dQ} + \frac{R}{|R|} N = 0 \quad (5.2)$$

where

$$Q = r\sqrt{|R|}/D$$

If pathogen densities inside and outside the patch are $N_i(r)$ and $N_o(r)$, respectively, then bounded solutions at the centre of the patch require that

$$\frac{dN_i}{dr} = 0 \quad \text{at } r = 0 \quad (5.3)$$

At the edge of the patch, where $r = a$, we make the biologically reasonable assumptions (Skellam, 1951) of continuity in the density and flux, respectively, of pathogen biomass:

$$N_i(a) = N_o(a)$$

and (5.4)

$$\frac{dN_i}{dr} = \frac{dN_o}{dr} \quad \text{at } r = a$$

Without continuity in density, an infinitely large flux must exist at the patch perimeter; and without continuity in flux, an infinitely large change in density must exist in an infinitesimally small neighborhood of the perimeter.

For $r > a$, the solution $N_o(r)$ of (5.2) which is bounded and nonnegative for large r is

$$N_o(r) = BK_0(r\sqrt{R_o}/D)$$

where $B > 0$ is constant and K_0 is the modified Bessel function of the second kind of order zero.

For $r < a$, the solution of $N_i(r)$ satisfying (5.3) is

$$N_i(r) = AJ_0(r\sqrt{R_i}/D)$$

where $A > 0$ is constant and J_0 is the Bessel function of the first kind of order zero.

Matching the solutions $N_o(r)$ and $N_i(r)$ given above at the field boundary $r = a$ with conditions (5.4), we find that non-trivial solutions for $N(r)$ exist only if

$$J_1(a\sqrt{R_i}/D)K_0(a\sqrt{R_o}/D) - \sqrt{R_o/R_i}J_0(a\sqrt{R_i}/D)K_1(a\sqrt{R_o}/D) = 0 \quad (5.5)$$

Here J_1 and K_1 are the first order counterparts to J_0 and K_0 , respectively.

Fig. 2 is a typical plot of relationship (5.5). The critical radius increases at a decreasing rate with R_o . As $R_o \rightarrow \infty$, equation (5.5) reduces to (4.1), (4.4). Substituting (4.4) into (4.1) we find, as indicated in Fig. 2, that the critical radius for $R_o \rightarrow \infty$ is

$$d_c = a_c = 2.4048 D/\sqrt{R}$$

Gurney and Nisbet (1975) also studied a radially symmetric version of

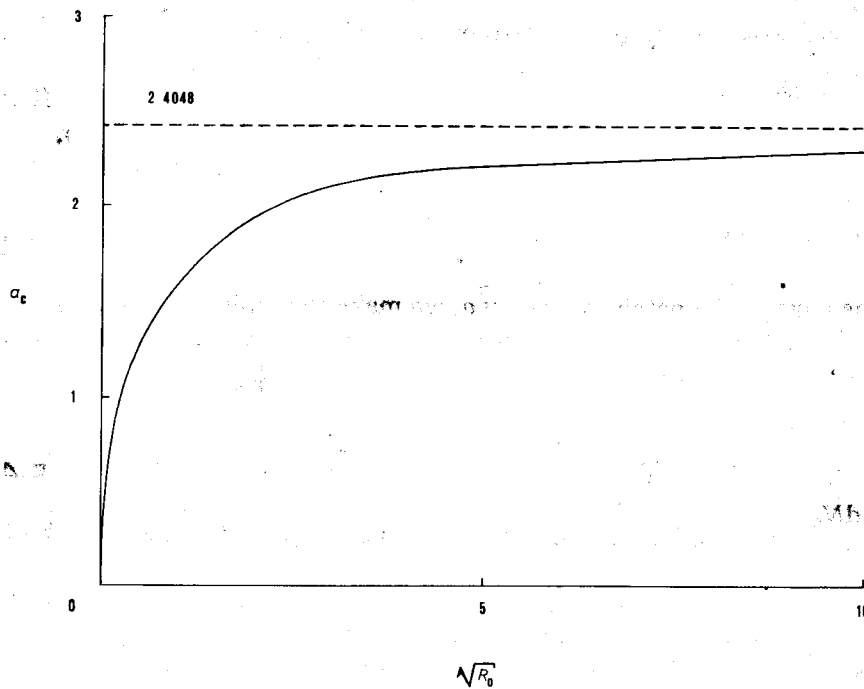


Fig. 2. Locus of critical radii, a_c , of an isolated circular patch of susceptible hosts against the square root of the exponential decay rate in the exterior environment, $\sqrt{R_0}$. This illustrates equation (5.5) for $D = R_i = 1$.

(1.8) for a habitable patch of radius a in an otherwise severe environment. Instead of the abrupt change of conditions at the edge of the patch implied by (1.7) and (5.1), they assume a gradual environmental deterioration with increasing distance from the origin. Their population growth function is of the form

$$F_G(N) = R[1 - (r/a)^2]N$$

Although (1.7), (5.1) may be suitable for spatially discontinuous agricultural systems, natural environments often exhibit the gradual cline assumed by Gurney and Nisbet (1975). Their analytical results are also consistent with (4.1) (cf. McMurtrie, 1978).

(6) Critical dimensions for exponential and logistic growth in isolated infinite strips: survival outside

The one-dimensional analogue of the previous problem concerns a strip of infinite length in the y -direction and of width W in the x -direction

$$\begin{aligned} R &= R_i & \text{inside the strip: } |x| < W/2 \\ R &= -R_0 & \text{outside the strip: } |x| > W/2 \end{aligned} \quad (6.1)$$

where R_i and R_0 are positive.

At steady state conditions in one-dimension (1.8) becomes

$$\frac{d^2N}{dx^2} + \frac{R}{D^2} N = 0 \quad (6.2)$$

Given the boundary conditions

$$\frac{dN}{dx} = 0 \quad \text{at } x = 0$$

$$N_i\left(\frac{W}{2}\right) = N_o\left(\frac{W}{2}\right) \quad (6.3)$$

and

$$\frac{dN_i}{dx} = \frac{dN_o}{dx} \quad \text{at } x = W/2$$

where N_i and N_o are, respectively, the solutions for $|x| < W/2$ and $|x| > W/2$; and given the requirement that $N_o(x)$ be non-negative and bounded for $x > W/2$, Ludwig et al. (1979) show that the critical width of the strip is

$$W_c = (2D/\sqrt{R_i}) \arctan \sqrt{R_o/R_i} \quad (6.4)$$

Fig. 3 is a typical graph of the critical width, W_c , against R_o , the hostility of the exterior environment. As $R_o \rightarrow \infty$, W_c approaches the value expected when survival is impossible beyond the boundary; substituting (4.2) into (4.1)

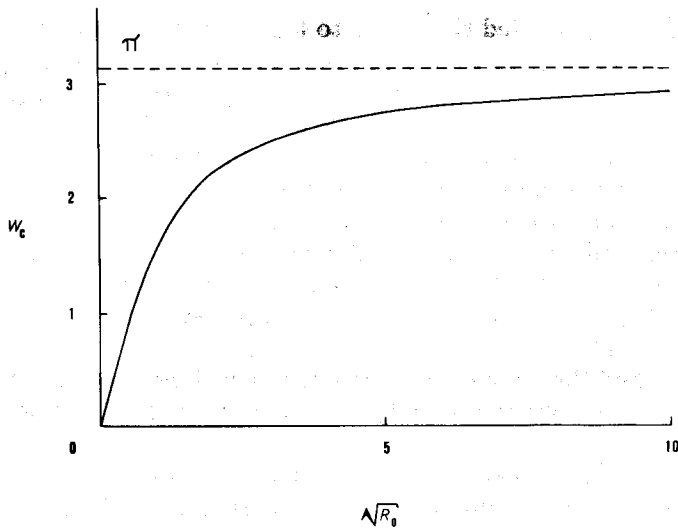


Fig. 3. Critical width, W_c , of a strip of infinite length is plotted against the hostility of the exterior environment, $\sqrt{R_o}$, according to equation (6.4) for $D = R_i = 1$.

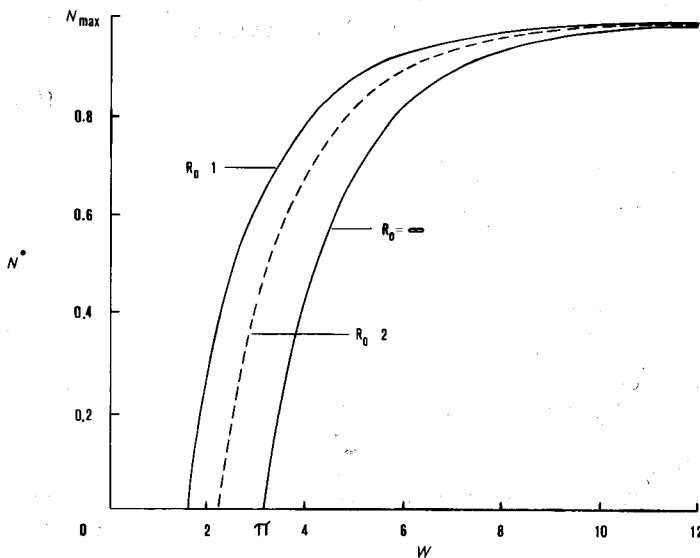


Fig. 4. Plot of the steady state density, N^* , against the width of the strip, W , for various decay rates outside the strip, R_0 , when logistic growth occurs inside. The parameters are: diffusion rate, $D = 1$; carrying capacity, $N_{\max} = 1$; and intrinsic rate of increase in the strip, $R_i = 1$ (after Ludwig et al., 1979).

demonstrates that this value is

$$W_c = \pi D / \sqrt{R_i}$$

Ludwig et al. (1979) have extended this result to logistic growth (4.3) inside the strip. However, as in the situation with instantaneous death at the boundary, parasite growth is limited to a maximum density, $0 < N^* < N_{\max}$, when the strip width exceeds the critical width. This density is plotted in Fig. 4 against the strip width for various values of R_0 when $D = N_{\max} = R_i = 1$. The points of intersection with the W axis are the critical widths defined in (6.4). Fig. 4 suggests that even when the field dimensions exceed the critical dimensions, smaller fields will have lower disease densities.

(7) Dispersal between fields

So far we have established the existence of critical dimensions for a variety of different geometries; for logistic, exponential, and other forms of population growth; and for a wide range of survival rates outside an isolated field. The general existence of critical dimensions suggests that parasite populations may be incapable of increasing on small fields, and if there is little disease in the fields there can be little dispersal between them.

We now address this hypothesis explicitly. We consider an infinite number of identical strips of width W running parallel to each other and each extending for an infinite distance in the y -direction. Due to the inherent symmetry

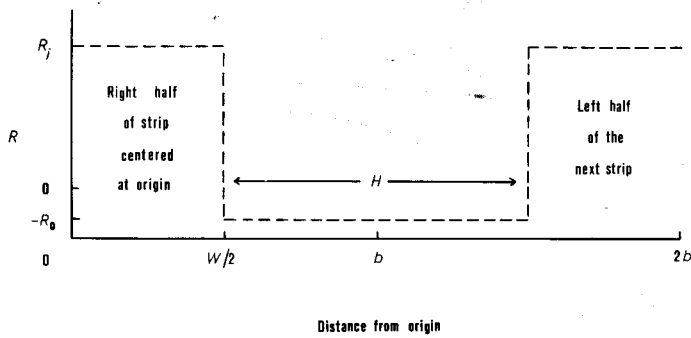


Fig. 5. Geometry used to study dispersal between fields. The rate of exponential pathogen biomass growth, R , is plotted against distance from the origin. A distance H separates infinite strips of width W . One strip is centered at the origin and others are centered every $2b$ distance units on either side of it.

of the problem we need only consider the region $0 \leq x \leq b$ where $H = 2b - W$ is the distance between strips and a strip is centered at the origin (Fig. 5).

Our objective now is to solve the system

$$\frac{d^2N}{dx^2} + \frac{R}{D^2} N = 0 \quad (7.1)$$

where

$$R = R_i \quad \text{when } 0 \leq x < W/2$$

and

$$R = -R_0 \quad \text{when } W/2 < x \leq b$$

subject to the boundary conditions (6.3) and the extra condition, arising from continuity and symmetry about $x = b$

$$\frac{dN_0}{dx} = 0 \quad \text{at } x = b \quad (7.2)$$

When $0 \leq x \leq W/2$, the solution of (7.1) satisfying the symmetry condition (6.3) at $x = 0$ is

$$N_i(x) = A \cos(x \sqrt{R_i}/D)$$

where $A > 0$ is constant. When $W/2 < x \leq b$, the general solution is

$$N_0(x) = B \cosh(x \sqrt{R_0}/D) + C \sinh(x \sqrt{R_0}/D)$$

where B and C are constants. Using (7.2) and the boundary conditions (6.3) at $x = W/2$, we find, after some calculation, that the critical strip width is

$$W_c = (2D/\sqrt{R_i}) \arctan [\sqrt{R_0/R_i} \tanh(H\sqrt{R_0}/2D)] \quad (7.3)$$

where $H = 2b - W$. The right hand side of (7.3) approaches that of (6.4) as $H \rightarrow \infty$.

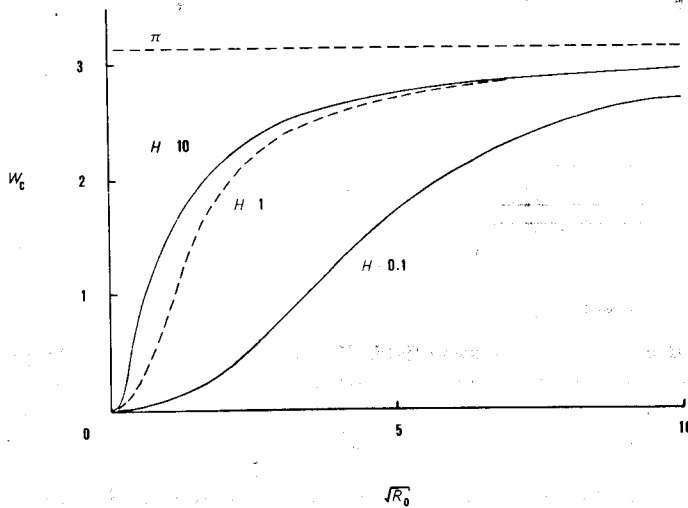


Fig. 6. Plot of the critical width of a strip, W_c , as a function of the decay rate in the exterior environment, R_0 , for different distances between strips, H . The parasite grows exponentially at rate $R_i = 1$ in the strip and diffuses at rate $D = 1$. The graph illustrates equation (7.3).

In Fig. 6 the critical strip width, W_c , is plotted against the square root of the decay rate outside, R_0 , for different values of H , the distance between parallel strips. For a particular separation distance (H), those combinations of strip width (W) and hostility of the exterior environment ($\sqrt{R_0}$) represented by points above the critical curve allow disease development. In contrast, initial infections eventually die out for combinations below the curve. Hence, regardless of the initial distribution of inoculum in a region with a certain crop acreage, if the fields are narrow enough, the pathogen population may not sustain itself.

Fig. 6 shows that increasing the strip width, W , or reducing the hostility of the exterior environment, R_0 , for a given strip separation, H , can turn very sparse populations into epidemics. Increases in W appear concomitantly with recent trends towards large scale mechanized agriculture (Apple, 1978). Moreover, modernization of cultural practices frequently decreases R_0 , the hostility of the environment outside the field. For instance, Rotem and Palti (1969) report that the introduction of irrigation for non-host crops nearby can enhance pathogen survival by its affect on the microclimate.

Fig. 7 clarifies the relationship between the critical strip width, W_c , and the fraction of the agricultural region sown to susceptible hosts, $W_c/(H + W_c)$, for various values of R_0 in (7.3). A population of disease organisms is unable to maintain itself against dispersal losses from the fields and therefore collapses in agricultural regions whose geometric parameters are contained under the curve defined by the appropriate R_0 . The same disease can spread in regions where the geometric parameters lie above the appropriate locus of critical values.

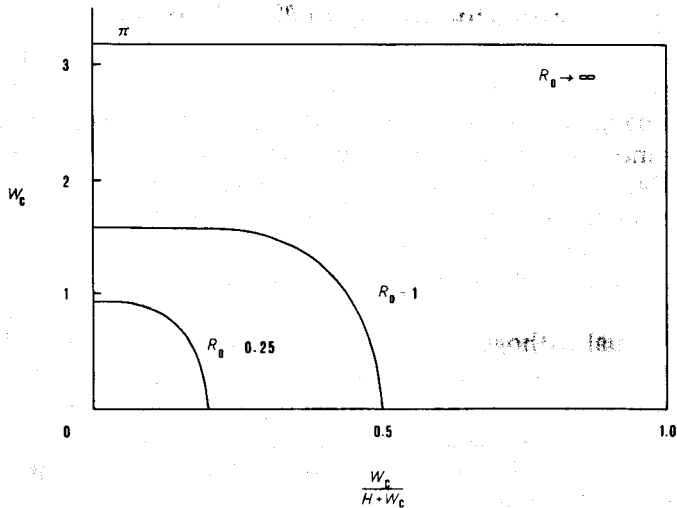


Fig. 7. Locus of critical values in (7.3) for $D = R_i = 1$. An infinite number of parallel strips of width, W_c , a distance H apart, are planted to susceptible hosts for various values of R_0 , the hostility of the exterior environment; $W_c/(H + W_c)$ is the proportion of the area planted to susceptible hosts.

Fig. 7 supports the common belief (e.g. Johnson, 1953) that increasing the relative area planted to a particular crop, $W/(H + W)$, can increase disease severity. However, more important to our purpose here, Fig. 7 also shows that increasing field sizes from $W < W_c$ to $W > W_c$, while maintaining the same fraction of total area allocated to the crop, changes a situation of population decline and hence negligible disease severity at the steady state into one of population growth and potential epidemics. Therefore, in contrast to Van der Plank (1948, 1949, 1960), we conclude that increasing the area, and hence, isolation of the fields into which a region's crop acreage is subdivided, can increase the danger of epidemic development.

So far we have considered only steady state solutions of (1.8) for boundary conditions which represent dispersal between fields. However, transient solutions are often more relevant to plant disease management. For instance, success in cereal rust control is frequently measured in terms of how long epidemic development can be delayed (e.g. Fleming and Person, 1978; Kranz, 1978; Fleming, 1980b). Time-dependent solutions to the system (6.3), (7.1), and (7.2) were obtained by numerical integration. Moore Lee (1978) describes the numerical integration program in detail.

In Figs. 8–11 we use parameter values which appear reasonable for cereal rusts. We set the width of the strip at 200 m, the length of the side of a square 4-ha field, a representative size for European conditions (Potts and Vickerman, 1974). Yarwood and Sylvester (1959) estimated the half-life of *Puccinia coronata* at 70 days from Forbes' (1939) data. This corresponds to $R_0 \approx 0.01 \text{ day}^{-1}$. We estimated the same value from Chester (1943) for the decay in population size of *P. recondita* over a mild winter. $R_i = 0.05 \text{ day}^{-1}$ does

not seem unreasonable for a variety with some resistance and Cammack's (1958) classic experiment with *P. polysora* provides data for an estimate of the diffusion coefficient: $D \approx 1 \text{ m day}^{-1/2}$ (see Appendix III).

Fig. 8 illustrates the change in relative pathogen density over space after five consecutive 20-day intervals. Initially, pathogen densities are uniform in the field and zero outside. Confidence in models is often established by noting qualitative similarity between the overall patterns of model predictions and the appropriate field data (Holling, 1978). In this context we note that in field tests of cereal rust dispersal, Roelfs (1972) obtained flat-topped gradients similar to those of Fig. 8.

Initially uniform and equal pathogen densities inside and outside the fields were used to describe the effect of simultaneous spore showers originating from outside the agricultural region at the beginning of the disease season. The 100-day profiles in Fig. 8 were essentially unchanged when initial pathogen densities were zero outside the field. These last initial conditions represent the overwintering of parasites in the fields.

Fig. 7 showed that increasing strip widths through the critical size changes declining pathogen populations into growing ones. Fig. 9 extends these conclusions. It shows that enlarging fields further beyond the critical size leads to even greater growth rates for populations of disease organisms.

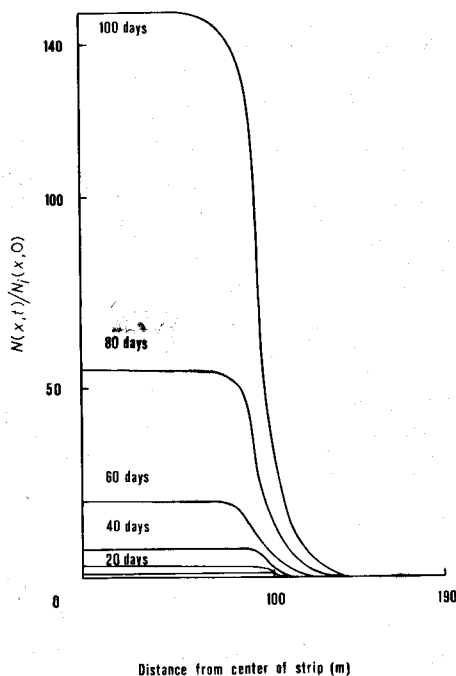


Fig. 8. Relative pathogen density across an infinite strip of width $W = 200 \text{ m}$ for five consecutive 20-day intervals after an initially uniform infection within the strip, $N_i(x, 0)$. The $N(x, t)$ represent numerical solutions of the system (1.8), (6.3), and (7.2) with $D = 1$, $R_i = 0.05$, and $R_0 = 0.01$. The difference between $H = W$ and $H = 5W$ is negligible.

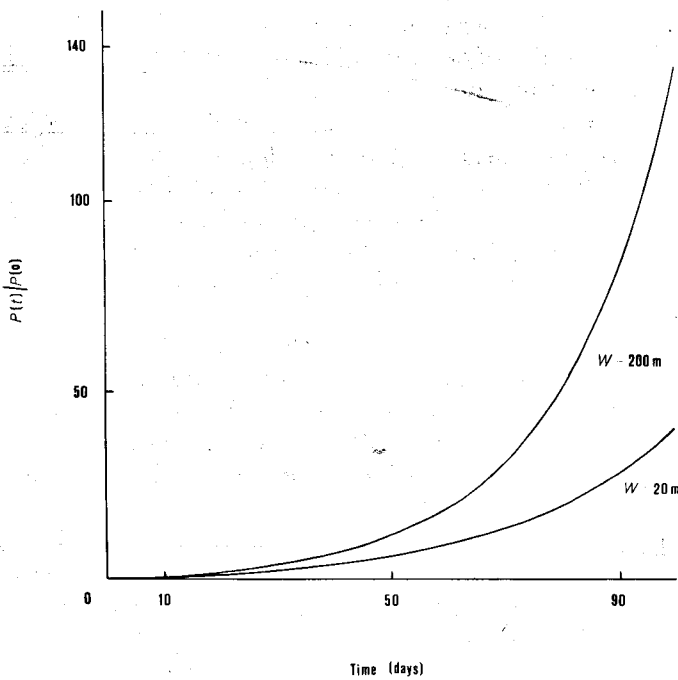


Fig. 9. Effect of field size on regional disease progress. The total parasite biomass in the strip at time t , $P(t)$, relative to the uniformly distributed initial number, $P(0)$, is plotted against time for strips of infinite length and width W . Parameter values and initial conditions are identical to those used in Fig. 8.

In Fig. 10 we plot the relative pathogen biomass at harvest as a function of field size when a tenth of the region has been planted to the crop. We assume that the cereal has a growing season of 100 days as is common for oats (Frey et al., 1977) and wheat (Chester, 1943; Peterson, 1965) and note that the cereal is susceptible to attack throughout this period (Peterson, 1965).

Together, Figs. 7, 9, and 10 suggest that when all fields are the same size, with shape and orientation constant, yield losses to disease decrease as a region's total crop acreage is progressively subdivided into an increasing number of smaller fields. Waggoner (1962) and Kampmeijer and Zadoks (1977) also indicated overall advantages to having many small fields in terms of epidemic control. In contrast, Van der Plank (1948, 1949, 1960, 1963) and during the 1971 epidemiology conference in Wageningen (Zadoks and Kampmeijer, 1977) has steadfastly recommended few large fields.

Fig. 11 shows that reducing relative strip size is generally more effective at inhibiting epidemic development for smaller strips than for larger strips. For example, decreasing the strip width from 200 m to 100 m decreases pathogen biomass at harvest by only 8% whereas a reduction from 20 m to 10 m leads to an 82% decrease.

Therefore, it is important to determine the width of an infinite strip, W ,

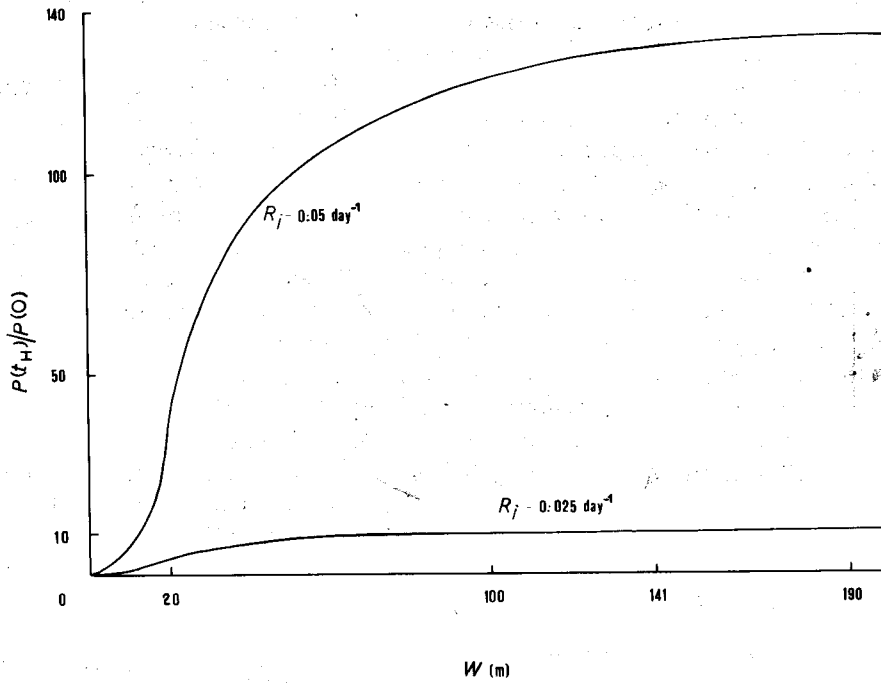


Fig. 10. Effect of strip size, W , on relative pathogen biomass at harvest, $P(t_H)/P(0)$. The graph illustrates the results of numerical solutions to equations (6.3), (1.8) and (7.2) for $R_i = 0.05$ and 0.025 when $H = 5W$ and $t_H = 100$. Initial conditions and the values of D and R_0 are identical to those of Figs 8 and 9.

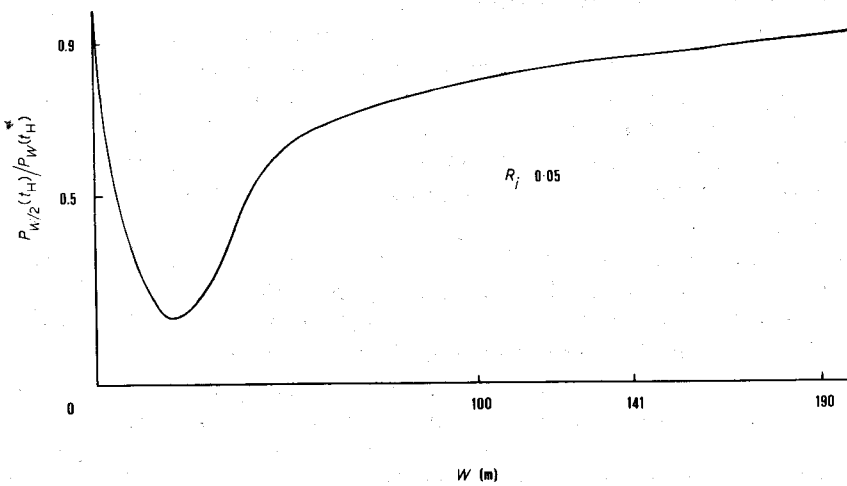


Fig. 11. Effect of halving field size on regional disease development. $P_W(t_H)$ and $P_{W/2}(t_H)$ are the pathogen biomasses at harvest in infinite strips of width W and $W/2$, respectively. Parameter values and initial conditions are identical to those of Fig. 10 except that $R_i = 0.05$, only.

corresponding to a field. In Fig. 8 the width is 200 m, the side of a square 4 ha field. When an infinite death rate exists outside, (2.5) gives a measure of the size of such a 200 m \times 200 m field in terms of diffusion. The value of W which provides the same value of d in (2.5) for an infinite strip is 141 m. Nonetheless, Fig. 11 shows that even at this lower value of W , halving the strip width reduces cereal rust biomass at harvest by just 14%. Under these circumstances, the economics of the situation will dictate the value of changing field sizes.

However, Fig. 1 suggests that field shape may also be an important factor in disease progress. The effect of field shape can be measured in (2.5) by d^2 , the size of a rectangular field in terms of diffusion when pathogens cannot survive outside. For example, if a 4 ha field is square then $d^2 = 20000 \text{ m}^2$, but if its dimensions are 20 m \times 2000 m then d^2 is only 400 m^2 . Similarly, $d^2 = W^2$ for an infinite strip of width W . Thus, infinite strips of widths $W = \sqrt{20000 \text{ m}^2} = 141 \text{ m}$ and $W = \sqrt{400 \text{ m}^2} = 20 \text{ m}$, respectively, are of the same size in terms of diffusion as 200 m \times 200 m and 20 m \times 2000 m fields with no survival outside. Therefore, assuming (2.5) holds approximately when $R_0 = 0.01$ per day, Fig. 10 indicates that the 20 m \times 2000 m field ($W = 20 \text{ m}$) will have 70% less cereal rust biomass at harvest than a square field of the same 4 ha area ($W = 141 \text{ m}$) when $R_i = 0.05$ per day.

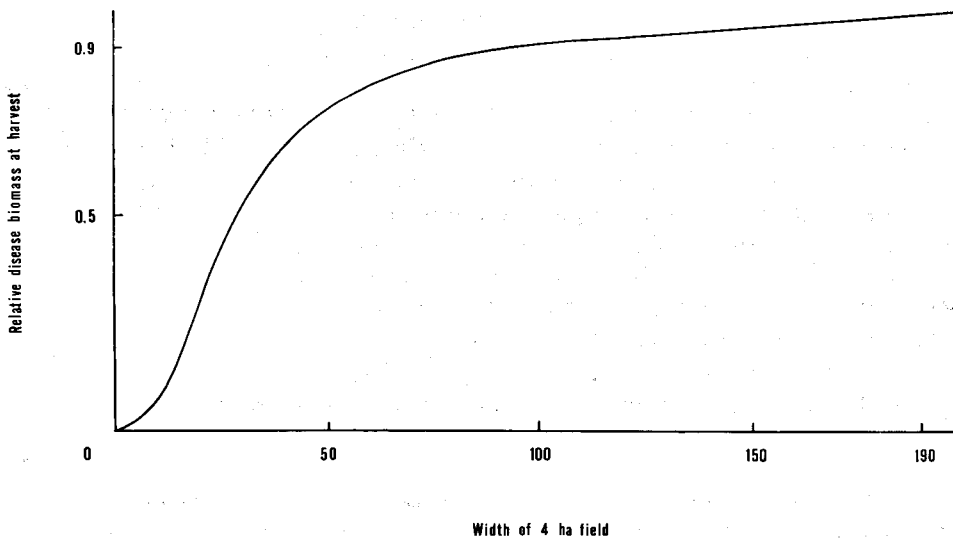


Fig. 12. Effect of field shape on the relative pathogen biomass in a region's fields at harvest. The vertical axis, $P_W(t_H)/P_{141}(t_H)$, gives the pathogen biomass at harvest in an infinite strip of width $0 < W \leq 141 \text{ m}$ relative to that in an infinite strip of width $W = 141 \text{ m}$. The horizontal axis gives the width of the 4 ha rectangular field which has the same value of d^2 in equation (2.5), the field size in terms of diffusion when survival outside is impossible, as the infinite strip of width $0 < W \leq 141 \text{ m}$. The maximum value of d^2 attainable by a 4 ha field occurs when the field is square (i.e. width = 200 m); the corresponding value of W is 141 m.

Generally, if (2.5) holds reasonably well for realistic decay rates outside the field, then elongating fields of a given area can substantially reduce disease (Fig. 12).

DISCUSSION

Van der Plank (1948, 1949, 1960) and Waggoner (1962) studied the significance of field geometry on the spread of plant disease with models which ignore the effects of time dependence in population growth and dispersal. Generally, however, dispersal and population growth are time-dependent processes which interact simultaneously to produce changes in population densities over space and time. This has been emphasized by much recent work in population dynamics on a range of insect (e.g. Wellington, 1964; Myers and Campbell, 1976; Jones, 1977; Clark, 1979), large mammal (e.g. Sinclair and Norton-Griffiths, 1979), marine (e.g. Steele, 1978), microtine (e.g. Krebs and Myers, 1974), plant pathogen (e.g. Shrum, 1975; Kiyosawa, 1976) and theoretical (e.g. Levin, 1978) systems.

Diffusion-reaction equations provide a relatively simple means of modeling the simultaneous dynamics of population growth and dispersal. A number of very readable recent reviews (e.g. Levin, 1976, 1978; Okubo, 1980) discuss the use of diffusion-reaction equations in describing the effects of environmental heterogeneity on population dynamics.

This approach has proved to be particularly successful in describing the formation of patches of phytoplankton.

Using empirically estimated parameter values Steele (1976) demonstrates that the result (4.1), (4.4) is supported by the general observation that plankton patches in the open sea appear at scales of the order of 10–100 km. Moreover, Platt and Denman (1975) and Wroblewski et al. (1975) show that modifications to this model to account for zooplankton grazing produce relatively small adjustments to the critical dimensions. Hence, the result (4.1), (4.4) is generally regarded as a theoretically and empirically robust description of plankton patchiness (Levin, 1978).

Our diffusion-reaction models indicate that the size and shape of fields can be important determinants of epidemic development. Because the net efflux of diffusing inoculum from a field occurs across the sides of the field, we argue that the rate is approximately proportional to the perimeter. However, since parasites are created at all points within a field, the production rate is proportional to the area. Hence the perimeter to area ratio of a field is roughly proportional to the ratio of diffusion losses to pathogen production in that field. Since the perimeter to area ratio increases as area decreases for fields of a given shape, small fields may sustain less disease than large fields.

In fact, Clark et al. (1978) have suggested that the same mechanism operates during outbreaks of spruce budworm (*Choristoneura fumiferana* Clem.) in the boreal forests of eastern North America. Here, small isolated

stands of even the most susceptible forest are unlikely to experience outbreaks of these defoliating insects (Van Raalte, 1972). Dispersal losses across the stand perimeters have been implicated ever since Morris and Mott (1963) first made the suggestion.

Extending this line of reasoning to an agricultural region as a whole, the rate of dispersal loss is roughly proportional to the sum of field perimeters while the production rate is proportional to the fraction of the region growing suitable hosts. Hence, for a given production rate, disease spread can be reduced by increasing the sum of field perimeters. There are two extreme approaches by which increases in the sum of field perimeters can be achieved: first, by changing the shape of fields such that the length to width ratio increases while their size remains the same, and second, by increasing the number of fields through a reduction in their size while their shape is fixed. This second method supports the recommendations of Waggoner (1962) and Zadoks and Kampmeijer (1977) for subdividing a region's cropland into many small fields. It argues against Van der Plank (1948, 1949, 1960, 1963) who claims that few large fields will provide superior disease control.

Borlaug (1959) and Browning and Frey (1969) were among the first to implement multilines, cultivars composed of several constituent lines each carrying different resistance genes, for disease control. In some ways each plant in a field sown to a multiline cultivar can be thought of as representing the limit to decreasing field sizes. Although our models are no longer strictly applicable when the typical distance travelled by a pathogen is large compared with "field size", the same general principles may apply. In fact, many workers (e.g. Frey et al., 1977; Trenbath, 1977; Burdon, 1978) have suggested that dispersal losses are significant for simple pathogenic races attacking multilines. The computer simulations of Kiyosawa (1976) and Kampmeijer and Zadoks (1977) provide support for this idea.

To estimate the relative effects of the two extreme approaches to increasing the sum of field perimeters we derived apparently reasonable parameter values for cereal rusts from the literature. With these values, our analysis suggests that elongating square fields is likely to be much more rewarding in terms of rust control than decreasing field size when a specific fraction of the region is devoted to susceptible cereals. Of course, economic considerations will be important determinants of the relative bias toward either approach in any mixed strategy to changing field geometry.

Meteorological factors can be important determinants of spore dissemination. Waggoner (1962) has emphasized the importance of orienting fields with respect to the prevailing wind direction. For example, suppose we are concerned with a field elongated perpendicularly to wind direction. Assuming dispersal is random, our model predicts that inoculum is symmetrically distributed across the width of the field with respect to the mid-width line (distance = 0 m in Fig. 8). In simple terms, the effect of wind can be imagined as a shift in the center of this distribution to a point downwind of the mid-width line, the magnitude of this shift being roughly proportional to the vec-

tor wind velocity. Although less inoculum would be lost at the upwind edge of the field, the loss would be greater at the downwind edge, and overall, the net loss from the field would be greater than if inoculum were randomly disseminated. Hence, our analysis underestimates the potential inhibition of epidemic development when dispersal is directed. Moreover, by the same reasoning, this underestimation is greatest for fields oriented perpendicularly to the prevailing wind direction and least for fields with an orientation parallel to wind direction.

Generally, statements above concerning the usefulness of elongated field shapes and small field sizes remain true when wind is considered. However, given a strong and steady wind, square fields may sometimes support less disease than fields elongated along the wind direction.

Our reaction-diffusion models also demonstrate that failure to consider dispersal can lead to misinterpretation of field data. For instance, neglect of the inequality in field sizes could lead to the erroneous conclusion that the difference between the curves of Fig. 9 is due to a difference in susceptibility of the varieties. Caution is always needed when parameter estimates are made from spatial averages (e.g. Jones, 1975). Hilborn (1979) recommends that estimates of the local site dynamics from the independent analysis of samples taken at single sites be used to provide insight about the dispersal processes.

It is convenient to split the field trials to test our results into two parts. The first part concerns the effect of field size and shape on disease development within single isolated fields. We suggest that comparisons of disease development between square and elongated "strip" fields be made for a number of different field areas. Then the effect of field size can be seen by comparing disease development in fields of the same shape, and the effect of field shape can be seen by comparing fields of the same size. If the prevailing direction of propagule dissemination can be predicted reasonably well, the length of elongated fields and two sides of each square field should be oriented perpendicularly to this direction. The distance separating one field from another must be great enough to inhibit dispersal between them and at the same time, be small enough to allow essentially equal environmental conditions. Inoculum densities as equal and as uniformly distributed as possible should be used to initiate disease in all fields. Then, following methods such as those described by Cammack (1958), disease severity could be measured at a number of locations in each field at various times during the disease season.

The second part to testing our results concerns the effect of field size and shape on disease development among groups of fields. If logistical problems prevent testing at a realistic scale, "scaled down" physical models of groups of fields may provide a reasonable alternative. For example, perhaps a few rows with a number of plants in each could be used to represent a group of elongated fields, etc. It may also be advantageous to consider just three groups of "fields": the first group having square "fields" of a convenient size, the second consisting of elongated "fields" of the same area, and the third group being square "fields" of a much different size. Each group of "fields" should

occupy a square "macroarea" of the same area and the fraction of it which is cultivated should be the same for all groups. A comparison of disease development in groups one and two would illustrate the effect of field shape while a similar comparison between groups one and three would illustrate the effect of field size. The details of field orientation, initial inoculum distribution, and the measurement of disease severity should be as outlined above. Care must be taken to separate the groups of fields by distances large enough that inoculum transfer between the groups is negligible, and yet small enough that the environmental conditions are not significantly different. Of course, in all the field trials proposed here, it is important to record the relevant meteorological variables and measure the intensity of incoming inoculum from distant sources.

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APPENDIX I

Implicit in (1.6) are two assumptions requiring the spatial scale of observation to be large. First, the assumption that D and R are constant in the field requires that the field be viewed as a uniform substrate, a 'carpet', rather than a collection of individually isolated plants. Second, (1.6) is derived from first principles (cf. Okubo, 1980, pp. 9-11) by visualizing propagule dissemination as a random walk process in which the length of each random step is very small compared to the spatial scale of observation.

APPENDIX II

Application and interpretation of the critical field size concept requires care. Pathogen biomass density, N , is distinct from disease severity, x , the visibly diseased fraction of susceptible host tissue. In contrast to N , x necessarily increases monotonically during the disease season as uredia become visible upon entering their infectious period, except perhaps during periods when the crop grows quickly. Hence, reports that x increased on small plots do not necessarily infer that N also increased, and therefore that the critical field size concept did not apply. On the other hand, because (1.6) holds only when the spatial scale of observation is large, the critical field size concept is not strictly applicable when realistic parameter values predict it to be very small.

We develop the critical field size concept to explain the behavior of the models at one extreme. Whether critical field sizes are realizable is another matter and is beside the thrust of our argument.

APPENDIX III

Such parameter values suggest exceedingly small critical field sizes in Figs. 2 and 3. Hence, since the models are strictly applicable only for large spatial scales of observation (as discussed in connection with 1.6), critical field sizes may be unrealizable. But the critical size depends on R_c , R_o , and D , so it will change during the growing season, it will depend on the host variety and rust race involved, and it will depend on the environment of the region. Critical area effects may be common but unrecognized features of cereal rust systems.