Probability Models For Host Infection By Soilborne Fungi

Christopher A. Gilligan

Visiting professor, Department of Botany and Plant Pathology, Colorado State University, Fort Collins 80523.
Permanent address of author: Department of Applied Biology, University of Cambridge, Pembroke Street, Cambridge CB2 3DX, U.K.
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ABSTRACT

Gilligan, C. A. 1985. Probability models for host infection by soilborne fungi. Phytopathology 75:61-67.

Some theoretical models for the infection of hosts by soilborne fungi are derived. Emphasis is given to the effects of the following phenomena on the probability of infection: spatial pattern of inoculum, displacement of soil by the growth of host organs, and distance of a propagule of inoculum from the surface of the host. A binomial model, of the form $P=(1-\phi)^N$, is presented for the probability (P) that a host unit, such as a root, seed, hypocotyl, or (in the case of hyperparasitism) a sclerotium, should remain uninfected when exposed to soil randomly infested by N propagules of a pathogen. The probability that the host encounters a fungal propagule, and is infected by it, is given by ϕ. The zone within which encounter and infection can occur is designated the "pathozone." The relationship between the binomial model and the Poisson model is discussed. A negative binomial model, of the form $P=(1+N\phi/k)^{-k}$, is presented for the probability that a host unit should escape infection when exposed to soil in which propagules of the pathogen or parasite are clumped; k is an index of the degree of clumping. Refinements of the models to allow for thresholds of numbers of infections required to cause disease are given and some effects of clumped and randomly dispersed inoculum upon the probability of disease escape are shown. Models in which allowance is made for the displacement of soil by the host unit in estimating ϕ are compared with those that assume no displacement. Differences in the predictions of the models are illustrated for an example of hyperparasitism of sclerotia. More complex binomial models, of the form $P=(1-\theta\psi)$, for the probability of a host unit escaping infection are presented, in which θ is the probability that a propagule occurs in the pathozone, and ψ is the probability that the propagule can infect the host, conditional upon its occurrence in the pathozone.

Additional key words: rhizosphere, spermosphere.

There is a certain volume of soil associated with a subterranean plant organ within which a propagule of a fungal pathogen must occur if it is to have any chance of infecting that organ. Propagules outside this volume either do not respond to the presence of the host or, if they do, have insufficient reserves of nutrients to reach and infect the host. The dimensions, particularly the width, of this exclusive volume are of interest to epidemiologists because they prescribe the proportion of the total soil population of a pathogen that encounters host organs. This relationship has been used (10,14) to obtain an expression for the expected number of infections per host organ. Rearrangement of the expression allows estimation of the width of the volume when the number of infections per host unit is either known or estimated. In the case of roots, this facilitates estimation of the width of the rhizosphere. Baker et al (5) had earlier proposed a different model to describe the initial infection of host units by propagules of soilborne fungi. That model was based upon the theory of solid packing and surface density, as developed, for example, in physical chemistry (21). It allowed a qualitative distinction between rhizosphere and rhizoplane infection so long as pathogen propagules were regarded as points (ie, without volume). The two approaches to modeling of root infection have been subjects for lively debate (2,4,5,10,11,14, 17,20,26). Baker and Drury (4) concede that if the propagules can be regarded as having volume, then the surface density model is inappropriate. They also assert that the alternative models, designated by Gilligan (17) as probability models, are useless without some estimate of the *infection efficiency* of a pathogen population. By infection efficiency is meant the proportion of the total soil inoculum capable of causing infection of hosts, if favorably placed to do so.

In this paper the simple probability model is developed further. The models presented below are envisaged as applying primarily to host-parasite systems involving large propagules, such as sclerotia. Infection efficiencies may be estimated by direct placement of the propagules adjacent to susceptible host units (15,23). Four problems in particular are addressed. First, allowance is made for multiple infection so that the investigator only has to score for presence or absence of infection and not for the total number of infections. Second, allowance is made for clumping of the inoculum in soil. Third, the shape of the target volume is considered. Whether or not the target is taken to be solid or hollow depends upon whether allowance is made for displacement of soil by the host unit. Fourth, the effects of distance of propagules from the surface of the host is considered in relation to the probability of infection.

In this paper, the term *pathozone* means the region of soil surrounding a host unit within which the center of a propagule must lie for infection of the host unit to be possible. The term pathozone has several advantages over previously used terms such as rhizosphere or spermosphere. It is a more general term that may be applied to a root, seed, hypocotyl, epicotyl, or mesocotyl and, in the case of hyperparasitism, for example, to a sclerotium or an oospore. Pathozone does not carry the connotation of extent of exudation of nutrients (sugars, amino acids, and organic acids) that may be attributed to the term *rhizosphere*. Hiltner (24) originally proposed the term rhizosphere to describe the zone of soil surrounding the roots of legumes in which the growth of bacteria might be influenced by nitrogenous compounds. Although it has come to be more broadly defined as the zone of influence surrounding roots (35), most work on the extent of the rhizosphere has concentrated on measurement of changes in density of

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saprophytic microorganisms in relation to nutrient exudation (see, eg, the reviews of Rovira [34] and Bowen and Rovira [6]). The concentration of exuded nutrients drops off sharply within the first 1–2 μm from the root surface (see the calculations of Newman and Watson [29]). Nevertheless, stimulation of the growth of mycelium from sclerotia has been demonstrated over much larger distances (7,32), presumably in response to volatiles that can diffuse through soil for greater distances (8). In other instances, host exudates may not be important in stimulating germination of fungal sclerotia (8). Spontaneous germination of sclerotia in the absence of hosts has been reported for *Phymatotrichum omnivorum* (25), *Rhizoctonia solani* (31), *Sclerotium rolfsii* (1), and *Helicobasidium purpureum* (41).

Other terms have been proposed to describe the same concept as the pathozone. Earlier I used the term "zone of potential infection" (15), but reference to an infection zone may confuse the concept with that of the infection court (3). Grogan et al (20) favored the term *competence volume*, and defined competence as the ability of propagules to germinate and infect, if located near enough to the surface of the host. Competence, however, is defined in the dictionary as a sufficiency without superfluity (R. Drury, R. Baker, and G. Griffin, *personal communication*: see, eg, [30]). The concept of sufficiency might arguably be applied to the ability of propagules to germinate and infect. The qualification superfluity, or rather nonsuperfluity, is an unnecessary restriction, however, when considering a quantal response. The term pathozone, as defined above, avoids this difficulty.

A glossary of symbols, used to represent variables and parameters in the text, is given in Table 1.

TABLE 1. Glossary of symbols used to represent variables and parameters in the text

Variable or parameter	Symbol	Definition
Host	M	Number of hosts within volume, V, of soil.
	L	Length of host root exposed to infection.
	r_r	Radius of root.
	r_{sd}	Radius of seed.
	H	Expected number of infections per host.
	U	Expected number of uninfected hosts.
	D	Expected number of diseased hosts.
Pathogen	N	Number of propagules within volume, V, of soil.
	I	Inoculum density (propagules per unit volume).
	r_i	Radius of propagule of inoculum.
	m	Mean number of propagules within pathozone ($\equiv N\phi$).
	k	Index of aggregation of propagules.
Environment	V	Total volume of soil within which inoculum and hosts occur.
Host/pathogen	ϕ	Probability that a propagule occurs in the pathozone and infects the host.
	ϕ_{mit}	Estimate of ϕ, based upon the multiple infection transformation.
	θ	Probability that propagule occurs in the pathozone.
	ψ	Probability that propagule infects hosts, given that propagule occurs in the pathozone.
	v	Volume of the pathozone.
	R	Outer radius of pathozone.
	w	Component of radius of pathozone, where $R = r_r + w + r_i$.
	r	Radial distance from center of pathozone.
	p_r	Observed proportion of propagules that can infect a host from a distance r.
	n	Number of infections per host less than a threshold number above which disease is observed.
	α, β,	Parameters of function, $\beta\exp(-\alpha r^2)$, for decrease in probability of infection with increasing distance of propagules from the host.
	γ, λ	Parameters of function $\alpha = \gamma\exp(-\lambda t)$ for decrease in probability of infection with increasing age, t, of propagules.
	B	Dummy variable ($\equiv H/MI\pi L$).

BINOMIAL AND NEGATIVE BINOMIAL MODELS FOR INFECTION

Binomial model. Gilligan (14) and Ferriss (10) proposed simple models to predict the number of infections, H, that should occur on M hosts growing in a total volume, V, of soil with uniform inoculum density of I propagules per unit volume. The expected number of infections is given by:

$$H = MN\phi \quad (1)$$

in which, $N = IV$ is the total number of propagules within the soil and ϕ is the probability that one host unit is infected by one propagule. The probability, ϕ, is given by v/V in which v is the volume of the pathozone for each of the M hosts. Rearranging equation 1 allows ϕ, and hence v, to be estimated when H, M, N, and V are known, thus:

$$\phi = H/MN. \quad (2)$$

The number of infections usually cannot be counted directly and recourse must be made to a multiple infection transformation (10,14,18,43) to estimate the mean number of infections per host unit (H/M) from the observed proportion of infected hosts. Allowance for multiple infection, however, may be conveniently incorporated into a simple binomial model for infection.

Suppose, as before, that a propagule of a pathogen occurs within a pathozone, with probability ϕ, and that a host unit (eg, seed, root, hypocotyl, or [in the case of hyperparasitism] a fungal sclerotium) becomes infected if one or more propagules occur within the pathozone. For a system involving only one host and one pathogen propagule, the probability that a host remains uninfected is $1-\phi$. For N propagules and M hosts, the expected number of uninfected hosts (U) is given by:

$$U = M(1-\phi)^N. \quad (3)$$

If N is known and U/M is observed, the corresponding value of ϕ may be calculated thus:

$$\phi = 1-(U/M)^{1/N}. \quad (4)$$

In using this binomial model the experimenter has only to score for presence or absence of infection on each host unit. The assumptions of the model are twofold: infection ensues if the center of a propagule occurs within the pathozone; the probability of occurrence within a pathozone is constant, and independent for all propagules. Confidence limits for U/M may be obtained as for a binomial proportion for a given level of significance. Tables of confidence limits are given in Snedecor and Cochran (37). Substitution of the upper and lower limits for U/M into equation 4, in turn gives estimates of the variability of ϕ for a single determination. I am grateful to one reviewer who noted that since the values calculated by equation 3 vary with the value of V, the estimate of U/M increases with increasing volume of the container, V, even when inoculum density, $I = N/V$, remains constant. This effect is small: for $I = 0.1$, $v = 1.0$, and $V = 10$, 10^2, 10^3, or 10^4, the corresponding estimates of U/M are 0.900, 0.90438, 0.90479, and 0.90483, respectively.

If $v \ll V$, as would be expected in most experimental situations, equations 3 and 4 may be simplified. Since $N = IV$, $(1-v/V)^N = (1-v/V)^{IV}$ but $(1-v/V)^V = (1-v)$, if $v \ll V$, so equation 3 becomes

$$U = M(1-v)^I.$$

Similarly, equation 4 may be simplified to

$$v = 1-(U/M)^{1/I}.$$

Relationship between binomial model, the Poisson model, and original model of Gilligan (14). The Poisson distribution can be considered as the limiting case of the binomial when the probability of an event occurring is very small and the number of trials is very large. Hence, for small values of ϕ and large values of N, ie, high inoculum densities, the Poisson distribution applies and the probability of a host unit remaining uninfected is given by $\exp(-N\phi)$ in which $N\phi$ is the mean number of infections per host unit. Hence:

$$U = M\exp(-N\phi) \quad (5)$$

and

$$\phi = -(1/N)\ln(U/M).$$

The model given in equation 2 implies a Poisson model. It is identical to that of equation 5, when the multiple infection transformation is used to estimate H/M. The assumptions for the Poisson model are the same as for the binomial model, but with the additional conditions of small ϕ and large N. Consequently the binomial and Poisson models are identical when ϕ is small and N is large. The mathematical relationship between equations 2 and 4 for ϕ (and hence between the binomial model and the original (10,14) implied-Poisson model) is given below. From the first term of the Poisson series:

$$U/M = \exp(-H/M)$$

so

$$H/M = \ln(M/U).$$

Substituting $H/M = \ln(M/U)$ into equation 2 gives an estimate for ϕ, say ϕ_{mit}, based upon the multiple infection transformation

$$\phi_{mit} = \ln(M/U)/N = -\ln(U/M)/N$$

and

$$\exp(-\phi_{mit}) = (U/M)^{1/N}.$$

But

$$\exp(-\phi_{mit}) = 1 - \phi_{mit} + \phi_{mit}^2/2-\ldots$$

and if ϕ_{mit} is very small, terms of the order ϕ_{mit}^2 can be ignored so:

$$\phi_{mit} = 1 - (U/M)^{1/N}$$

and hence, for small values of ϕ, ϕ_{mit} estimated from equation 2, is identical to ϕ estimated in equation 4.

Negative binomial model. A random distribution of propagules of inoculum in soil is assumed in both the binomial and the Poisson models. It is probable, however, that the pattern of inoculum in naturally infested soils is clumped (39). The negative binomial distribution is one member of the binomial family of distributions that may be used to approximate the distribution of clumped inoculum. It has two parameters, a mean, $m = N\phi$, and an index of aggregation, k. The more aggregated the inoculum, the smaller is the value of k (see, eg, 38). For randomly distributed inoculum, k tends to infinity and if ϕ is small, the negative binomial distribution tends to the Poisson distribution. Use of the negative binomial model to predict the number of uninfected hosts implies no more than that the inoculum was aggregated. No inference ought to be made about the mechanism causing the aggregation without additional independent evidence (43).

For a negative binomial distribution of inoculum in soil, and correspondingly of infection, the probability that a host unit remains uninfected, ie, unexposed to infection, is given by the first term of the negative binomial distribution, $(1 + N\phi/k)^{-k}$. Thus :

$$U = M(1+N\phi/k)^{-k}$$

and

$$\phi = k[(M/U)^{-1/k}-1]/N.$$

The form of the equation, using v and I is trivially different:

$$v = k[(M/U)^{-1/k}-1]/I$$

Values for the probability of a host remaining uninfected are given in Table 2 for the binomial and negative binomial models for representative values of k, the index of aggregation (22,39). Large differences between the models are apparent when the product $N\phi$ is relatively large. The discrepancy is greatest when the index of aggregation is smallest. Taylor et al (39) recorded a value of 0.96 for k and Hau et al (22) obtained values in the range 2.09 to 2.77 for k for the pattern of inoculum of *Cylindrocladium crotalariae* in naturally infested soil.

Multiple infection to cause disease. Death of a host organ is relatively easy to score for and is much less equivocal than scoring for infection. It is probable, though, with many host-pathogen and host-hyperparasite systems, that infection from more than one propagule would be necessary to cause death. It should be noted that the concept of a threshold of infection necessary to cause death is not at variance with Vanderplank's 'law of the origin' (42). Vanderplank (42) compiled evidence to refute the hypothesis of Gaümann (13) and others, that a minimum number of spores is necessary to establish certain diseases. The evidence, however, is sufficient only to show that infection can be established by single propagules; it does not discredit the hypothesis that numerical thresholds of infection may exist for producing symptoms of disease or causing death of the host.

The models given above can be adopted to take account of numerical thresholds of infection. Suppose for example that death of a host unit occurred only after infection by four or more propagules and that the occurrence of a propagule within the pathozone resulted in infection. Equations for the expected numbers of dead hosts (D) are:

TABLE 2. Effects of using binomial and negative binomial probability density functions to approximate the frequency of occurrence of infective propagules in the pathozone of, for instance, a seed on estimates of the probability of the host remaining uninfected.

Mean no. of propagules within a pathozone ($N\phi$)	ϕ[a]	Total no. of propagules in soil (N)	Probability of host remaining uninfected			
			Binomial model[b]	Negative binomial model[c]		
				k[d] = 0.5	k = 2.0	k = 10.0
0.1	0.0001	10^3	0.905	0.913	0.907	0.905
1.0	0.001	10^3	0.368	0.577	0.444	0.368
10.0	0.01	10^3	0.001	0.218	0.028	<0.001
100.0	0.1	10^3	0.001	0.071	<0.001	<0.001

[a] Probability of infective propagule occurring in unit volume of soil and infecting the host located therein.
[b] Probability of host remaining uninfected = $(1-\phi)^N$.
[c] Probability of host remaining uninfected = $(1 + N\phi/k)^{-k}$.
[d] Index of aggregation: k = 0.5, 2.0, and 10.0 represent intense aggregation, moderate aggregation, and near-randomness, respectively.

binomial model,

$$D = M[1 - \sum_{n=0}^{n=3} \binom{N}{n} \phi^n (1-\phi)^{N-n}]; \quad (6)$$

Poisson model,

$$D = M[1 - \sum_{n=0}^{n=3} (N\phi)^n \exp(-N\phi)/n!]; \quad (7)$$

negative binomial model,

$$D = M[1 - \sum_{n=0}^{n=3} \binom{n+k-1}{k-1} (N\phi/k)^n (1 + N\phi/k)^{-(k+n)}]. \quad (8)$$

The second term within the square brackets in equations 6 to 8 gives the probability that a host will be alive. The equations cannot be solved directly for ϕ, but iterative solution for ϕ is possible. The effects of different combinations of values for N and ϕ on the probability of host units remaining alive are compared for the binomial and negative binomial models in Table 3. The index of aggregation, k, was set at 0.5 for the negative binomial model, to represent marked clumping of propagules. The threshold value for death was put at four infections.

As with the presence or absence of infection, large discrepancies in the probabilities predicted by the models are apparent when $N\phi$ is relatively large. The probability of escape given by the negative binomial model, for aggregated propagules, was less than or approximately equal to that given by the binomial model, for randomly distributed propagules, when the mean number of propagules per pathozone, $N\phi$, was ≤ 2.24 (with $N = 2.24 \times 10^2$ and $\phi = 0.01$, for the binomial model). Above that value the chance of hosts escaping death was much greater when propagules were clumped than when they were randomly distributed. Note, however, that the cross-over value of 2.24 propagules per pathozone is not unique for this system. The variables N and ϕ always occur as the product, $N\phi$, in the negative binomial model, but they are treated separately in the binomial model. Hence, there are numerous possible values given by the binomial model for each value of the negative binomial model that corresponds to a given mean, $N\phi$. Nevertheless, the discrepancy between the estimates from the binomial and negative binomial models highlights the importance of first checking the distribution of inoculum within soil before selecting the model for the infection zone.

TABLE 3. Effects of using binomial and negative binomial density functions to approximate the frequency of occurrence of infective propagules in the pathozone of a susceptible host, on estimates of the probability of a host escaping disease[a]

Mean no. of propagules within a pathozone ($N\phi$)	ϕ[b]	Total no. of propagules in soil (N)	Probability of host escaping disease: Binomial[c] model	Probability of host escaping disease: Negative[d] binomial model
0.01	0.0001	10^2	>0.999	>0.999
0.10	0.001	10^2	>0.999	>0.999
1.00	0.01	10^2	0.982	0.919
2.50	0.01	2.5×10^2	0.758	0.759
5.00	0.01	5.0×10^2	0.264	0.602
10.00	0.01	10^3	0.010	0.455
100.00	0.1	10^3	≪0.001	0.154

[a] Hosts were deemed to have escaped disease if they encountered ≤3 propagules within the pathozone.

[b] Probability of infective propagule occurring in pathozone and infecting the host located therein.

[c] Probability of host remaining uninfected $= \sum_{n=0}^{n=3} \binom{N}{n} \phi^n (1-\phi)^{N-n}$.

[d] Probability of host remaining uninfected $= \sum_{n=0}^{n=3} \binom{n+k-1}{k-1} (N\phi/k)^n (1 + N\phi/k)^{-(k+n)}$

The index of aggregation was set at 0.5.

SHELL VERSUS DISPLACEMENT MODELS

The model originally proposed by Gilligan (14) included the host volume in the pathozone. Thus, for a radially symmetrical pathozone, the radius of the pathozone is the sum of the radii of the host, say a root (r_r), and inoculum (r_i) plus a distance w across which the pathogen can grow to cause infection. The volume of the pathozone for a root uniformly susceptible along its length (L) is, therefore, calculated by using the equation for the volume of a solid cylinder, of volume v that can be represented by:

$$v = \pi L(r_r + w + r_i)^2.$$

Such a model may be called a displacement model. It assumes that propagules within soil displaced by growth of host as well as those in the soil immediately surrounding the host should all take part in infection, if the pathozone extends far enough. Movement of roots through soil may be regarded as effecting localized changes in the bulk density of soil as soil is squeezed outward from the axis of the path of the growing root. A search of the literature has not revealed the radial extent of the change in bulk density for roots of given diameter. But if the furthest extent of the change in bulk density is less than $w + r_i$ from the root surface, then the displacement model holds.

Leonard (27) and Ferriss (10) challenged the displacement model. They proposed instead a model in which the volume of the host is excluded from the pathozone. The pathozone for a root uniformly susceptible along its length, L, is therefore a hollow cylinder of volume v that can be represented by:

$$v = \pi L[(r_r + w + r_i)^2 - r_r^2]$$

This may be regarded as a shell model. The primary use of the models proposed by Gilligan (14,15) and Ferriss (10) was to estimate the size of w. Working with the original form of the model given in equation 1, the equation for the calculation of w is:

$$w(\text{displacement model}) = B^{1/2} - (r_r + r_i)$$

in which $B = H/MI\pi L$. Ferriss (10) correctly noted that, for certain values of infections per root (H/M) and inoculum density (I), it was possible to obtain negative values for w from this equation. Indeed, this happened with my reanalysis (14) of the data of Rouse and Baker (33) and I adjusted these illogical values to zero. Ferriss (10) presented an alternative equation for the calculation of w:

$$w(\text{shell model}) = (B + r_r^2)^{1/2} - r_r \quad (9)$$

and suggested that since $B \geq 0$, negative values for w would not be obtained. Unfortunately, in doing so, Ferriss had altered the meaning of w so that 'w' in equation 9 was actually equal to the sum of w and r_i. The equation for w for the shell model should therefore be:

$$w(\text{shell model}) = (B + r_r^2)^{1/2} - (r_r + r_i) \quad (10)$$

Negative values for w would be obtained from equation 10, if $r_i > [B + r_r^2]^{1/2} - r_r$, although this inequality is unlikely to be satisfied, except in few cases. The models summarized in the form of equation 1 and indeed the models derived above, involve deterministic approximations for (eg) ϕ, and hence w, to what is a stochastic process. Negative values may therefore be attributed to w, when, due to chance, the number of successful infections is unusually low for a given inoculum density. Whether or not such negative values should be included in the estimation of w is still partly a subjective judgment. Given, however, that negative values may arise due to natural variation, I am inclined to include them in the estimation of w.

The practical importance of the difference between the shell and displacement models increases with the size of r_r relative to $w + r_i$. Consider, for example, the infection of sclerotia of *Sclerotinia sclerotiorum* by *Coniothyrium minitans* (40). Suppose the mean

radius of sclerotia is 0.5 mm, propagules of the hyperparasite comprise single pycnidia, of mean radius 0.6 mm (40), and w is assumed to be 0.2 mm. There is little difference between the estimates from the shell and displacement models for the probability of sclerotia escaping infection (Fig. 1A). However, if $w + r_i$ is reduced to 0.4 mm, as might happen if fragments of pycnidia were incorporated into soil, the discrepancy between the two models becomes large (Fig. 1B). Unfortunately, it is doubtful whether, in all but a few simple systems, techniques for investigating infection of below ground host units are sufficiently refined to allow practical distinction between the two models. The example given above is for illustrative purposes only. Judgment as to the applicability of the shell or displacement models needs to be based upon extraneous knowledge. Thus, one reviewer noted that if the host unit is large, such as a carrot, the displacement model is more likely to apply. Whereas, if the host unit is small and is formed in the soil pore spaces, then the shell model would apply.

ALLOWANCE FOR DISTANCE FROM HOST

It was assumed in the derivation of the models presented above that occurrence of a propagule in a pathozone is sufficient to ensure infection. Clearly, this is an oversimplification. Several investigators (19,32,36) have shown that the probability of germination of pathogen propagules declined with increasing distance from the host, while Punja and Grogan (33) and Henis and Ben-Yephet (23) demonstrated that the probability of infection declined with distance of the infective propagule from the host. A more realistic expression of the probability for infection of a host unit by a single propagule is given by:

$$P\{\text{host is infected}\} = P\{\text{propagule occurs in pathozone}\} \times P\{\text{propagule is capable of infecting host} \mid \text{propagule occurs in pathozone}\}$$

Let θ be the probability that the propagule occurs in the pathozone and ψ be the probability of the propagule infecting the host, conditional upon its occurrence in the pathozone. The value of θ is numerically equivalent to ϕ, as used above, but unlike ϕ it is used only to denote the probability of occurrence in the pathozone and not the probability of infection given occurrence.

Consider the infection of a root of radius r_r and length L, by a fungal sclerotium of radius r_i. If the root is uniformly susceptible to infection over its entire surface, and the shell model is assumed to apply, then the infection zone is described by a hollow cylinder of outer radius R (with $R = r_r + w + r_i$) and inner radius r_r. Then:

$$\psi = \int_{r_r}^{R} P\{\text{sclerotium occurs at distance } r \text{ from center of pathozone} \mid \text{sclerotium occurs within pathozone}\} \times P\{\text{sclerotium can infect root from a distance } r\}$$

The probability that the sclerotium can infect the root from a distance r could be expanded to allow for the conditional probabilities of germination, growth to the host, and the initiation of infection. It is left as a single expression, however, for simplicity in deriving the model. The probability that a sclerotium occurs at distance r from the center of the pathozone is derived first. Strictly, for a continuous random variable, the probability that a sclerotium occurs at a given distance r is zero. It is necessary to work, instead, with the probability that a sclerotium occurs between r and $r + \Delta r$. That is the probability that the center of a sclerotium occurs within the wall of a hollow cylinder of length L, and approximate area $2\pi\Delta r$, in which Δr is small enough to ignore the difference between the radii of the inner and outer surfaces of the cylinder.

$$P\{\text{center of sclerotium occurs between } r \text{ and } r + \Delta r \mid \text{sclerotium occurs within pathozone}\} = 2\pi r L \Delta r \Big/ \sum_{r_r}^{R} 2\pi r L \Delta r,$$

from which it follows that the probability density function is

$$2r/(R^2 - r_r^2) \qquad (11)$$

The probability that a propagule can infect a host decreases with distance from the host and approaches zero at the outer limit of the pathozone. A simple, empirically selected expression for the reduction in probability of infection with distance, r, is given by:

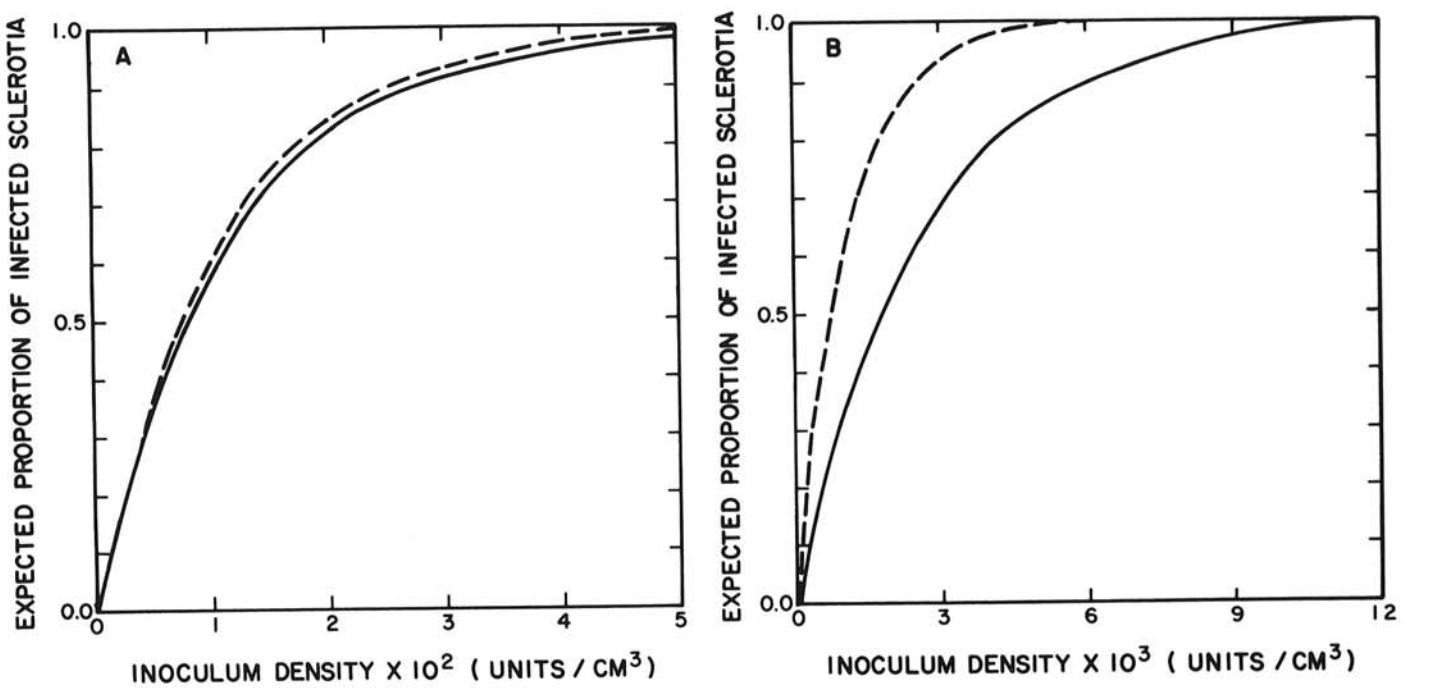

Fig. 1. Comparison of shell (——) and displacement (— —) models for infection of sclerotia, of radius 0.05 cm, of *Sclerotinia sclerotiorum* by *Coniothyrium minitans* (40). **A.** Radius of outer limit of pathozone, beyond which probability of infection is zero, was 0.13 cm. **B.** Radius of outer limit of pathozone was 0.01 cm. The proportion of infected sclerotia was estimated from $1-(1-v/V)^{IV}$ in which V is the total volume of soil (10^3 cm^3) and I is the inoculum density per unit volume. For the shell model, v is the volume between two hollow spheres, an inner one with radius 0.05 cm and an outer one with radius equal to the outer limit of the pathozone. For the displacement model, v is a solid sphere with radius equal to the outer limit of the pathozone.

$$P\left[\begin{matrix}\text{propagule can infect}\\ \text{root from a distance } r\end{matrix}\right] = \beta e^{-\alpha r^2}\ r \leqslant R. \qquad (12)$$

That the probability of infection should decrease exponentially with distance is intuitively appealing. The power of the distance, as well as the parameters α and β, however, may vary with the host and pathogen. The function r^2 is proposed here because it gives a less rapid decline in probability of infection close to the root surface than would the first power of r. The probability density function, $\beta\exp(-\alpha r^2)$ is initially concave downward rather than concave upward as with $\beta\exp(-\alpha r)$ (Fig. 2). Both properties agree with the limited experimental data available for infection of bean seedlings by *Rhizoctonia solani* (23), of sugar beet petioles by *Sclerotium rolfsii* (32), and of wheat roots by *Gaeumannomyces graminis* (*unpublished*). The function r^3 would give an even slower rate of decline in probability of infection close to the root than r^2 (Fig. 2).

Estimates, $\hat{\alpha}$ and $\hat{\beta}$, of the parameters of equation 12 may be obtained from the logarithmic form of the equation, $\ln(p_r) = \ln\hat{\beta} - \hat{\alpha}r^2$, in which p_r is the observed proportion of propagules that can infect the host from a distance r, as obtained by experiment. The parameter R cannot be estimated from equation 12. It may instead be determined directly from the placement experiment, as the distance closest to the root from which no infection is observed to occur. Because retransformation from the logarithmic to the original form of equation 12 involves certain statistical difficulties concerning error structure, alternative methods of curve fitting may be preferred (37).

For the shell model, the probability that the sclerotium is capable of causing infection, if it occurs within the pathozone of the root, is given by combining equations 11 and 12:

$$\psi = \int_{r_r}^{R} [2r\beta/(R^2 - r_r^2)] \exp(-\alpha r^2)\mathrm{d}r. \qquad (13)$$

Integration by substitution (details available from author) gives:

$$\psi = \{\beta/[\alpha(R^2 - r_r^2)]\}[\exp(-\alpha r_r^2) - \exp(-\alpha R^2)]. \qquad (14)$$

A corresponding expression for the displacement model in which the constants of integration are 0 and R is given by $\psi = \beta[1 - \exp(-\alpha R^2)]/\alpha R^2$. This expression is simpler than equation 14, but no allowance was made in its derivation for changes in bulk density surrounding the growing root, within the infection zone.

The probability that a host unit remains uninfected when it occurs in soil of volume V, containing a total of N randomly distributed and potentially infective propagules is $(1 - \theta\psi)^N$. For a cylindrical pathozone, of length L, θ is estimated by $\pi R^2 L/V$ and:

$$P = \{1 - [\pi\beta R^2 L/\alpha V (R^2 - r_r^2)][\exp(-\alpha r_r^2) - \exp(-\alpha R^2)]\}^N. \qquad (15)$$

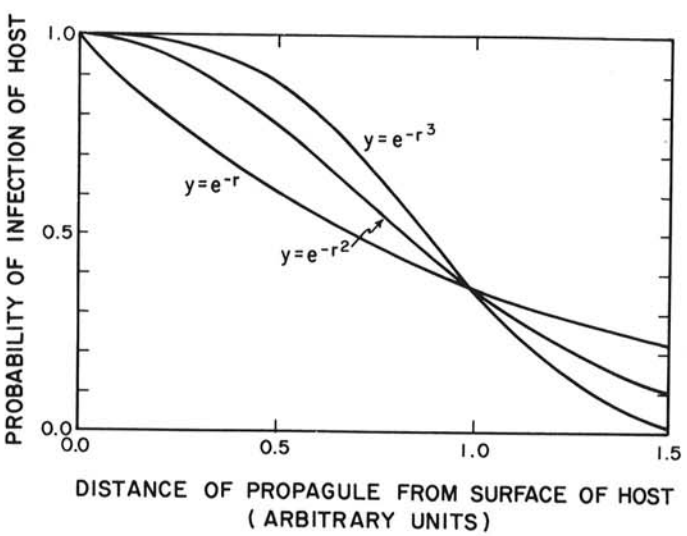

Fig. 2. Comparison of three functions, of the form $y = \beta\exp(-\alpha r^m)$ for the probability (y) that a propagule can infect a host organ at a distance r. The parameters α and β were set at 1.0.

APPLICATIONS AND LIMITATIONS OF THE MODELS

The simple binomial and negative binomial models may be used, as with the initial models of Gilligan (14) and Ferriss (10), to estimate the width of pathozones from population studies. Alternatively, if the extent of the pathozone is known, the models may be used to predict the proportion of uninfected or of diseased hosts. The original models (10,14) assume, however, that the probability of infection, ϕ, is constant for all propagules that occur in a pathozone. Variation in ϕ may arise due to: secondary infection from infected tissue rather than from soil inoculum (9), interaction between host and pathogen such that the probability of subsequent infection on a previously infected root may be altered (16), and variation in host density. If host density is high relative to propagule density, such that single propagules frequently occur in two or more pathozones, the inoculum potential of the propagules (sensu Garrett [12]) may not be sufficient to permit successful infection of all encountered hosts. If N is held constant and M (the number of hosts) is varied, plots of U against M should yield straight lines for both the binomial (for randomly distributed propagules) and the negative binomial models (for clumped propagules), if ϕ is constant.

There are three unknowns (α, β, and R) in equation 15, the more complex binomial model in which allowance is made for distance of the propagules within the pathozone from the host surface. Clearly, therefore, equation 15 cannot be solved, as was possible for equations 2 or 4 for the simple models, to estimate R and hence the width of the infection zone. The model is proposed instead as a basis for analytical modeling (26) of infection of subterranean plant organs after estimation of α, β, and R from experiments, as outlined above. The model can be adapted, with slight adjustment, for spherical infection zones such as might be expected to surround seeds or, in the case of hyperparasitism, sclerotia. The expression for the probability of a host remaining uninfected for a spherical infection zone of inner radius, r_{sd}, and outer radius, R, and shell model is:

$$\psi = \int_{r_{sd}}^{R} [r^3\beta/(R^3 - r_{sd}^3)] \exp(-\alpha r^2)\, dr \qquad (16)$$

Integration by substitution, followed by integration by parts (details available from author) gives:

$$\psi = \{\beta/[2\alpha^3(R^3 - r_{sd}^3)]\}\{[\exp(-\alpha r_{sd}^2)(1 + \alpha r_{sd}^2) - \exp(-\alpha R^2)(1 + \alpha R^2)]\}.$$

It is envisaged that further refinements may be made to the models. For example, the effect of aging of propagules may be incorporated into the model by the use of experimentally determined values of α, β, and R, over time. Alternatively, functions such as $\alpha = \gamma e^{-\lambda t}$, in which γ and λ are constants and t is age of propagules, may be used to approximate the change in the probability of propagules achieving infection. With increasing sophistication, however, it is likely that computer simulation, with allowance for stochastic variation, would be of more use to the experimenter than single analytical solutions.

There are two principal limitations to the application of the models presented above. The first is the need to estimate the proportion of the total soil population of propagules of a given pathogen, or parasite, that is infective. While such estimation is difficult for many spore-producing fungi (28), even after artificial infestation of soil, it is much less difficult for many sclerotia-

producing fungi, to which the models are primarily directed. The second limitation arises if only a portion of the host is susceptible to infection. If, however, the proportion of external surface of the host that is susceptible can be estimated, appropriate adjustment could be made for the volume of the pathozone.

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