## Letter to the Editor

# Modeling Rhizosphere Infection

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Mathematical models are increasingly being employed in the description, forecasting, and analysis of epidemics of aerial pathogens (8,12) but their application to the study of soilborne pathogens is rare. Quantitative studies of these pathogens are beset by three main problems: (i) observation of the pathogen in its subterranean habitat, (ii) identification of natural and often variable inoculum, and (iii) measurement of the indeterminate mycelial spread of the pathogen. Nevertheless, some work seeking to model the infection of subaerial host organs has been published in which attention has focused on the influence of rhizospheres on infection. Foremost in this field have been R. Baker and his colleagues, who proposed a model to distinguish between a *rhizosphere* effect, in which infection is caused by the germination of propagules and their growth to the host under a directional stimulus, and a *rhizoplane* effect, in which only those propagules in contact with the host can germinate and infect (2).

Here, I intend first to consider some limitations of this model and then to propose an alternative model.

## Limitations of the Model

Baker et al (2) defined the rhizosphere as a hollow cylinder of constant diameter which enveloped roots and proposed that, given a fixed infection court (or target organ) and nonmotile inoculum, an increase in inoculum density in the bulk soil and hence in the rhizosphere would increase infection in the host in direct proportion to the change in overall inoculum density. Hence, disease incidence plotted against inoculum density would have a slope of 1. If inoculum is added to soil, however, the proportional increase in the inoculum density in the rhizoplane is less than that in the rhizosphere (2). Therefore, Baker et al (2) proposed that the number of propagules per unit area rather than propagules per unit volume was critical in determining the number of potential infections. They assumed a tetrahedral arrangement of propagules in soil, so that $I = K_1/D^3$, where I is the inoculum density, D is the distance between propagules, and $K_1$ is a constant. The number of propagules per unit area in contact with the rhizosphere, ie, the number of successful infections (S), is given by $S = K_2/D^2$. Dividing and rearranging these expressions gives $S = K(I)^{2/3}$, where $K = K_2/(K_1)^{2/3}$ and taking logarithms yields a linear equation with slope 2/3:

$$\text{Log } S = 2/3 \log I + C \qquad (1)$$

Hence log-log plots of inoculum density-disease incidence (ID-DI) curves with slopes of 0.67 indicate rhizoplane infection (1,2).

Baker (1) applied this model to examples from the literature and Benson and Baker (3,4) and Rouse and Baker (10) used the model to interpret their own experimental data. Hence, Rouse and Baker (10), investigating preemergence damping-off of radish (*Raphanus sativus*) by *Rhizoctonia solani* Kühn, recorded slope values for log-log ID-DI curves that were not significantly different from 1.0 and 0.67 in nonamended soil and cellulose-amended soil, respectively. They ascribed the effect of biological control on *R. solani* by addition of cellulose to a shrinking of the rhizosphere to the rhizoplane. Addition of chitin, in contrast, did not significantly alter the slope of the ID-DI curve from 1.0.

Some implicit assumptions of the model described above are questionable. Baker et al (2) assumed regular tetrahedral arrangement of inoculum in soil, whereas, even with thorough mixing of soil, a nonuniform or, at best, random distribution of nonmotile inoculum is likely to obtain. In applications of the model, random distributions of inoculum and infections have been assumed (1,3,4,10), but it is not clear whether the authors first tested for randomness. Gregory's (7) multiple infection transformation of $y = \log_e (1/1 - x)$, derived from the Poisson series, was used to calculate the number of independent infections (y) from the proportion of diseased hosts (x). Where there are sufficient degrees of freedom, agreement of ID-DI response with the Poisson distribution may be checked by a $\chi^2$ test of the observed and expected numbers of healthy roots (5). However, even if conditions for randomness are satisfied, the log transformation for multiple infection is itself subject to logarithmic transformation, which, although mathematically acceptable, removes the data far from the observations.

A further assumption of the model is the zero volume attributed to inoculum. Although this simplification may be applicable to spores so small in relation to the size of soil particles that they might be pushed out of the way by an advancing root without touching it, this is certainly not the case for sclerotized inoculum of *R. solani* sieved to 2 mm before infesting soil (10).

Similar objections to the rhizosphere model have also been rehearsed by Van der Plank (11), who also pointed out that the model does not allow for competition between propagules for a restricted number of susceptible sites on target organs. Van der Plank (11) also reported that a slope of 2/3 for a log-log plot of ID-DI data is not unique to soil pathogens but may obtain for infection of *Vicia faba* by *Botrytis* spp. (9, 13) and, with appropriate selection of inoculum densities, for infection of *Nicotiana glutinosa* leaves by tobacco mosaic virus (11).

Alternative approaches to the quantification of root infection have been proposed. For example, Van der Plank (11) suggested that if the concentration of propagules in the rhizosphere is varied by serial dilution of the test soil, the relationship between the relative inoculum densities in the rhizosphere and disease incidence could be plotted. In common with the models discussed above (1,2), however, he did not allow for the volume occupied by the host. Moreover, Van der Plank's approach does not distinguish between infection via the rhizosphere and the rhizoplane. Because the existing situation is not satisfactory, I propose a different model.

## Proposed Alternative Model

The width of the rhizosphere can be estimated from the observed number of target organs and the probability of contact between root and inoculum. When infection is confined to propagules in the rhizoplane and contact between host and inoculum is essential for infection, then the effective target is a cylinder of radius $(r_r + r_i)$, where $r_r$ is the radius of the root and $r_i$ the radius of a single propagule. This is easy to visualize if the radius of the cylinder is considered to delimit the area including and surrounding a root in which the center of a propagule must occur if the propagule is to

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touch the root. When, however, the pathogen can grow through the rhizosphere to infect the host, the effective target volume remains a cylinder but its radius is enlarged by the width of the rhizosphere (w). When the total volume of soil containing inoculum and the length of root passing through this soil are known, the probability of a single propagule infecting a single root can be calculated from the ratio of target volume:total volume. With a total of M roots of mean length (L) and N propagules in a volume (V), the expected number of infections or "hits" (H) is given by:

$$H = \frac{L.\ M.\ N.\ \pi\ (r_r + r_i)^2}{V} \quad (2)$$

for rhizoplane infection and

$$H = \frac{L.\ M.\ N.\ \pi\ (r_r + w + r_i)^2}{V} \quad (3)$$

for rhizosphere infection. Rearranging equation 3 permits calculation of w:

$$w = \left(\frac{H.\ V}{L.\ M.\ N.\ \pi}\right)^{1/2} - (r_r + r_i) \quad (4)$$

This approach to modeling rhizosphere infection was developed to interpret infection by relatively large propagules, of the size of sclerotia (6). It has not yet been tested for infection of roots by spores. It would seem likely that equations 3 and 4 would hold for such situations, however, when the radius of a single propagule ($r_i$) is so small in relation to $r_r$ and w that it may be treated as zero.

There is a further advantage to this model. Unless the pathogen is capable of markedly faster growth than the host, target organs will have grown past the pathogen before infection is initiated and will be, in effect, stationary when attacked. Unlike Baker's (1,2) model, it is not necessary to distinguish between motile infection courts (or targets). Moreover, it is unnecessary to distinguish between motile and nonmotile inocula, because propagules capable of hyphal growth or propulsion across the rhizosphere and those requiring contact to cause infection may be regarded as members of the same continuum, differing only in the maximum distance across which they can respond to host propinquity.

The model, therefore, is both simpler and more sensitive than those of Baker et al (2), since it can detect differences in the widths of rhizospheres. By using slopes of inoculum density-disease incidence curves, Baker et al (2) were restricted to making qualitative distinctions between rhizosphere and rhizoplane infections. This limitation led Rouse and Baker (10) to deduce that the mechanism whereby addition of chitin to soil infested with *R. solani* inhibited preemergence damping-off of radish seedlings did not influence rhizosphere-rhizoplane relationships. Since infection resulting in preemergence damping-off was limited in these experiments to the seed (10), we are, in effect, considering "spermosphere" and "spermoplane" infections. Accordingly, if the radish seed is considered to be a sphere of radius $r_s$, equations 2 and 4 may be adjusted to give:

$$H = \frac{M.\ N.\ 4/3\ \pi\ (r_s + r_i)^3}{V} \quad (5)$$

for spermoplane infection and

$$w = \left(\frac{H.\ V}{M.\ N.\ 4/3\ \pi}\right)^{1/3} - (r_s + r_i) \quad (6)$$

for spermosphere infection.

Analysis of some of the data of Rouse and Baker (10) presented in their Fig. 3A using equations 5 and 6 (Table 1) permits more detailed comparison of the effects of nonamended and chitin-amended soil on infection of radish by *R. solani*. To minimize the occurrence of multiple infections, only data derived from low inoculum densities have been used. The parameters used in the calculations of expected numbers of infections and mean widths of spermospheres are detailed in Table 2.

TABLE 1. Observed and expected numbers of infections and width of spermosphere for infection of *Raphanus sativus* by *Rhizoctonia solani* in nonamended and chitin-amended soil

| Treatment | Inoculum density[a] (propagules/g) | Total number of propagules (N) | Number of infections observed[a,b] | Number of infections expected[c] | Mean ± SE width of spermosphere (w)[d] (mm) |
|---|---|---|---|---|---|
| Nonamended | 3.39 | 678 | 42.0 | 15.5 | 0.74 ± 0.153 |
| | 2.00 | 400 | 15.9 | 9.1 | |
| | 1.43 | 286 | 14.2 | 6.5 | |
| | 0.75 | 150 | 5.2 | 3.4 | |
| Chitin-amended | 3.93 | 786 | 27.0 | 18.0 | 0.23 ± 0.091 |
| | 2.50 | 500 | 15.9 | 11.4 | |
| | 1.07 | 214 | 5.9 | 4.9 | |
| | 0.54 | 108 | 1.3 | 2.5 | |

[a] Data from Rouse and Baker (10).
[b] The observed number of infections (x) was derived from $x = N[\log_e N - \log_e (N-y)]$ where N is the number of hosts available and y is the average number of hosts infected, after Gregory (7).
[c] The expected numbers of infections were computed from equation 5, assuming that contact between propagule and main root axis was essential for successful infection.
[d] Calculated from equation 6.

TABLE 2. Parameters used in calculations of expected numbers of infections and mean widths of spermospheres given in Table 1

| Symbol | Value | Description | Source |
|---|---|---|---|
| M | 50 | Number of seeds | Rouse and Baker (10) |
| N | Varied with inoculum density | Number of propagules | Multiply inoculum density expressed as propagules/g (10) by total weight of infected soil |
| V | $210 \times 10^3$ mm$^3$ | Volume of 200 g of soil | Measurement |
| $r_s$ | 1.84 mm | Mean maximum radius of germinating seed | Measurement |
| $r_i$ | 1.00 mm | Maximum radius of propagule | Rouse and Baker (10) |

The results in Table 1 imply that infection occurred across a spermosphere, as suggested by Rouse and Baker (10). However, the results of Table 1 also show a significant reduction in the mean width of spermosphere in chitin-amended soil compared with nonamended soil ($P \leq 0.05$).

The values of w in Table 1 may underestimate the dimensions of the spermospheres, since equation 6 assumes "perfect" infection, ie, every propagule lying within or touching a spermosphere is capable of infecting the seed. If the probability of a propagule surviving long enough to infect the host is p, then corrected estimates of the width of spermosphere ($w^1$) may be obtained from the values of w in Table 1, using the following equation:

$$w^1 = (w + r_s + r_i)\,(1/p)^{1/3} - (r_s + r_i) \qquad (7)$$

Therefore, 50% loss in viability would yield corrected estimates for the width of the spermosphere as 1.7 mm in nonamended soil and 1.0 mm in chitin-amended soil. Similar corrections might also be made to w to allow for a hemispherical target instead of a spherical one, since it is not clear from Rouse and Baker's paper (10) whether seeds were covered with infested or uninfested soil. (Clearly, if uninfested soil was used, only the lower half of seeds may be considered to have been in contact with infested soil and the target reduces to a hemisphere with a consequent increase in estimates of w.)

Studying and modeling spermosphere infection of rapidly germinating seeds is, however, prone to error because the geometry of the infection court changes rapidly as the plumule and radicle emerge and the testa is dislodged. Consequently, models characterizing the zone of influence between host organ and pathogen propagules are perhaps better studied in relation to root infection. Even so, when roots are grown in infested soil, results may be confounded by infection of roots by more than one inoculum unit and cross infection of healthy roots by infected roots. These problems may be overcome, at least for inoculum consisting of relatively large propagules such as sclerotia, by restricting inoculum within soil to a single horizontal plane through which roots are allowed to grow (6). In this case, the effective target is a circle of area $\pi(r_r + w + r_i)^2$ and the probability of a single infection is given by the ratio of target area:total area of the inoculum plane. For a plane of radius R containing N propagules and through which M roots grow, the expected number of infections or "hits" (H) is given by:

$$H = \frac{M.\ N.\ (r_r + w + r_i)^2}{R^2} \qquad (8)$$

for rhizosphere infection.

Knowledge of the effective size of the rhizosphere surrounding hosts is but one phase in understanding the complexities of inoculum density-disease incidence responses. However, it does provide a starting point for systems analysis of root infections to which may be added information on the two succeeding phases of infection, namely, initiation of infection after contact and the rate of subsequent exploitation of this infection.

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