Letter to the Editor

Calculating Rhizosphere Size

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Accepted for publication 25 February 1981.

In a letter to the editor of this journal, C. A. Gilligan (3) proposed equations for calculating the width of rhizospheres and spermospheres. I recently attempted to use the published equations for such calculations and sometimes obtained negative values for rhizosphere and spermosphere widths.

The purposes of this letter is to describe this situation, and to present an alternative model.

PROBLEMS WITH THE MODEL

Gilligan (3) proposes a model of rhizosphere infection based on the prediction of the number of infections or "hits" H on a root system which consists of M roots of mean length L and mean radius r. A number of pathogen propagules N of radius r_i are distributed in a volume of soil V, and can infect if they are closer than a certain distance from the root surface W. The proposed equation is:

$$H = \frac{\pi\, LMN\,(r_r + W + r_i)^2}{V}. \qquad (1)$$

Equation 1 is also presented in a rearranged form that permits the calculation of W, the width of the rhizosphere. The proposed equation (after correction of a typographical error) is:

$$W = \left(\frac{HV}{\pi\, LMN}\right)^{1/2} - \left(r_r + r_i\right) \qquad (2)$$

A similar equation is presented for the calculation of a spermosphere around a seed. The proposed equation (after correction of a typographical error) is:

$$W = \left(\frac{3\,HV}{4\pi MN}\right)^{1/3} - \left(r_s + r_i\right) \qquad (3)$$

in which M is the number of seeds, and r_s is the mean radius of a seed. Equations 2 and 3 may be used to calculate values of rhizosphere and spermosphere widths; however, when certain values of the parameters are used, the calculated value of W is less than zero. This situation can be illustrated with equation 3. Rearrangement of equation 3 indicates that W is less than zero when:

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

Thus, equation 4 predicts that for any particular seed (r_s) and inoculum (r_i) sizes, there exists a ratio of hits per seed (H/M) to inoculum density (N/V) below which the width of the spermosphere is a negative number. The treatment of such negative spermosphere widths is not mentioned by Gilligan (3). However, the value for the spermosphere width presented for chitin-amended soil in Table 1 of his paper can only be obtained if calculated negative spermosphere widths are treated as being equal to zero. Although such a protocol would be mathematically correct if it were specified in the model, its existence would make the equation of questionable value for the calculation of spermosphere widths from experimental data.

PROPOSED MODEL

The equations developed herein are intended to be used in the calculation of theoretical rhizosphere or spermosphere widths from experimental data. Because of this objective, an attempt has been made to base the model on a few simple assumptions about the physical configuration of the soil-microorganism-plant system. The basic concept of the rhizosphere as a volume of soil that surrounds a plant part has been used by a number of authors (2,5,6,8); however, this concept has not been used in the development of specific equations for the calculation of rhizosphere or spermosphere size.

If every competent propagule (sensu Grogan et al [5]) in a certain volume of soil can initiate an infection, then the number of infections or "hits" H is equal to the number of competent propagules in that volume, and can be expressed as a function of pathogen inoculum density I, the volume of soil S, and the competence C of the propagule population:

$$H = ICS \qquad (5)$$

in which $0 < C \leqslant 1$. If both sides of equation 5 are divided by the number of infectable units (seeds, root pieces, plants, etc.) M, then the equation may be rearranged to express the volume of the spermosphere or rhizosphere for each unit S/M as a function of I, C, and the number of infections per unit H/M:

$$S/M = \frac{H/M}{IC}. \qquad (6)$$

Note that as C becomes smaller, H/M becomes larger, and when $C = 1$, then

$$\mathrm{S/M} = \frac{\mathrm{H/M}}{\mathrm{I}}.$$

Thus, a "minimum volume" for a rhizosphere or spermosphere (ie, when $C = 1$) can be calculated on the basis of pathogen inoculum density and the number of infections per unit. If actual infection data are available, and it can be assumed that propagules are randomly distributed in soil, then the theoretical number of infections per unit may be calculated with the multiple infection transformation (4). If only disease incidence data are available, a minimum volume may be calculated by substituting D/M, the multiple infection transformation of disease incidence, for H/M. Because a minimum of one infection must initiate each case of disease, H/M will always be greater than or equal to D/M. Thus, substitution of D/M for H/M can result in the underestimation, but not overestimation, of the value of S/M.

To calculate the distance from a plant part from which infections have taken place (ie, the width of the rhizosphere or spermosphere)

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0031-949X/81/12122903/$03.00/0

it is necessary that the volume calculated in equation 6 be associated with a physical configuration appropriate to the plant part. For a seed, the volume defined by two concentric spheres might be appropriate:

$$S/M = V_o - V_s = \frac{4\pi r_o^3}{3} - \frac{4\pi r_s^3}{3} \quad (7)$$

in which V_o is the volume of the sphere that delimits the outer limit of the spermosphere and V_s is the volume of the sphere that delimits the inner limit of the spermosphere (ie, the surface of seed), and r_o and r_s are radii of the outer and inner spheres, respectively. The width of the spermosphere is $W = r_o - r_s$. This value may be obtained by combining equations 5 and 7:

$$\frac{H/M}{IC} = \frac{4\pi r_o^3}{3} - \frac{4\pi r_s^3}{3} \quad (8)$$

rearranging to make r_o the dependent variable:

$$r_o = \left(\frac{3\,H/M}{4\pi IC} + r_s^3\right)^{1/3} \quad (9)$$

and then subtracting r_s from both sides of equation 9:

$$W = r_o - r_s\left(\frac{3\,H/M}{4\pi IC} + r_s^3\right)^{1/3} - r_s \quad (10)$$

A similar equation may be derived for M root pieces of length L and radius r_r:

$$W = r_o - r_r = \left(\frac{H/M}{\pi LIC} + r_r^2\right)^{1/2} - r_s \quad (11)$$

Rearrangement of equation 10 indicates that W is less than zero only when:

$$\frac{3\,H/M}{4\pi IC} < 0 \quad (12)$$

Because an experimentally determined H must be greater than or equal to zero, all calculated values of W must thus be greater than or equal to zero.

LIMITATIONS OF THE PROPOSED MODEL

Equations 6, 10, and 11 may be used to calculate parameters for rhizosphere and spermosphere size in a variety of situations; however, their limitations should be recognized. In particular, it should be noted that the finite value of W that can be calculated using equations 10 or 11 is only a gross approximation of the relationship between the probability that a propagule can infect and its distance from the host. In a real-world situation, the probability of infection would decrease gradually with distance, rather than abruptly decrease to zero at distances greater than W. It should also be noted that a number of factors can influence the value of C, the competence of the pathogen propagule population. Host resistance, environmental variables, the presence of other microorganisms, and a nonrandom distribution of inoculum would all serve to reduce the value of C below one, and thus cause the calculated values of S/M and W to underestimate the true values. Similarly, the use of disease incidence, rather than infection data for the calculations can result in underestimation of S/M and W if more than one infection is necessary for disease expression. It should also be noted that the size of the plant part assayed (the seed or root segment) is not necessarily the same as the size of the actual infection site. If only a portion of the plant part is actually infectable, then the calculated value of W would be an underestimation of its real value. The accuracy of estimation of S/M or W also is dependent on the accuracy with which I has been measured. If I has been underestimated (such as is usually the case when I is determined by soil assay), then S/M and W will be overestimated.

Two aspects of the soil-microorganism-plant system which were intentionally excluded from the model are the effect of propagule size on rhizosphere size, and a qualitative distinction between rhizosphere and rhizoplane effects. The model may be useful for investigating both of these phenomena, however. If calculated values of rhizosphere size were compared with propagule size for a number of different pathogen-plant systems, some overall relationship between rhizosphere size and propagule size might be established. Similarly, comparison of rhizosphere size and propagule size in a single system may allow a decision as to whether a rhizosphere or rhizoplane effect is in operation. If the calculated rhizosphere size is close to the propagule diameter, it is possible that a rhizoplane effect could be operating (ie, only propagules very close to the plant cause infections); however, it is also possible that rhizosphere size has been underestimated.

INTERACTIONS WITH OTHER MODELS

Because data for the calculation of rhizosphere or spermosphere widths must come from inoculum density–infection experiments, it is important to examine relationships between the width models (equations 10 and 11) and existing inoculum density-infection models. These models involve expressions of number of infections H, or number of infections per unit H/M, as a function of inoculum density I. For calculating rhizosphere width, a regression equation would be fitted to I-H/M data, the regression equation would be substituted for H/M in equations 10 or 11, and then W would be calculated as a function of I. If the ratio of H/M to I was constant over the entire range of Is used (ie, a linear relationship existed), then W would be the same for all values of I. If the ratio of H/M to I varied with I, then the calculated W would change with I. An appropriate I-H/M model should adequately describe both of these two situations, and not produce nonsensical values of W. Three models will be examined.

Baker and co-workers (1,2) proposed that I-(H/M) data be fitted to an equation of the form:

$$H/M = kI^b \quad (13)$$

where k and b are constants. In this theory, the value of b is taken to indicate whether a rhizosphere ($b = 1$) or rhizoplane ($b = 0.67$) effect is in operation. This equation can be used to describe a number of general relationships. However, when it is incorporated into equation 10 or 11, nonsensical values of W are usually generated at low values of I. It can be shown that:

$$\lim_{I\to 0^+}\left(\frac{kI^b}{I}\right) = \begin{cases} 0 \text{ if } b > 1 \\ k \text{ if } b = 1 \\ \infty \text{ if } b < 1 \end{cases}$$

and thus, for equation 10:

$$\lim_{I\to 0^+}\left(\frac{3kI^b}{4\pi IC} + r_s^3\right)^{1/3} - r_s = \begin{cases} \left(\frac{3k}{4\pi C} + r_s^3\right)^{1/3} & 0 \text{ if } b > 1 \\ & -r_s \text{ if } b = 1 \\ & \infty \text{ if } b < 1 \end{cases}$$

Thus, a constant nonzero value of W is calculated when I approaches zero only if $b = 1$. Such an exact value of b would be rare in a regression of real data. It should further be pointed out that if $b = 0.67$, the calculated limit for W as I approaches zero is infinity. Thus the "rhizoplane" situation generates an infinitely large value for the width of the rhizosphere. If equations 10 and 11 are valid, then the existence of this paradox would tend either to support criticisms (3,5,6,8) of the theory of Baker et al (1,2), or to indicate that the calculation of rhizosphere size for a rhizoplane situation is inherently impossible, and results in nonsense values of rhizosphere size. In either case, the use of equation 13 in

rhizosphere size calculations would be inappropriate.

Vanderplank (8) proposed an equation based on the Poisson distribution as an alternative to that of Baker et al (1,2). This equation includes parameters representing the number P and susceptibility A of potential infection sites:

$$H/M = (P/M)\ (1-e^{-AI}) \quad . \tag{14}$$

Equations of this form can be easily incorporated into equations 11 and 12. As I approaches zero, it can be shown that the ratio of H/M to I approaches a constant:

$$\lim_{I\to 0^+} (P/M)\ \frac{(1-e^{-AI})}{I} = (P/M)\ A$$

The use of equation 14 thus seems to be more appropriate than the use of equation 13. However, for certain sets of data, finite values of P and A cannot be determined. This occurs when the data describe a line which curves upward. This problem could be overcome by fitting a positive exponential equation; however, such an equation would not have the same theoretical relation to a biological situation that equation 14 does.

Several other regression equations could be fitted to I-infection data. In some situations it may be appropriate that a quadratic equation with no intercept be fitted:

$$H/M = k_1 I + k_2 I^2.$$

This equation does not correspond to a particular conceptual model, and should not be used to extrapolate beyond the range of the data that has been fitted. However, it can be used to describe a number of types of relationships and to test for a linear realtionship between H/M and I by determining if k_2 is significantly different from zero, and has a finite limit as I approaches zero:

$$\lim_{I\to 0^+} \frac{k_1 I + k_2 I^2}{I} = k_1.$$

It is hoped that the availability of the equations presented in this paper will both encourage the calculation of rhizosphere and spermosphere sizes, and stimulate the development of related theories.

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