

Calculating the Dimensions of the Rhizosphere—A Response

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Journal Series Paper 83-11-14 of the Kentucky Agricultural Experiment Station, Lexington 40546.

I thank R. J. Kryscio for statistical consultation.

Accepted for publication 13 April 1983.

EDITOR'S NOTE: This Letter to the Editor should be read in conjunction with Drury et al (3).

Drury et al (3) present two major points in their letter to the editor: the numerous assumptions on which the rhizosphere (and spermosphere) width calculations are based result in uncertainty about the accuracy of the calculated values; and decisions as to whether a rhizoplane or rhizosphere effect is operating should be based on a specific type of regression analysis of inoculum density (*ID*)-infection data proposed by Baker and co-workers (1,2), rather than on rhizosphere width calculations such as I have proposed (4). Each of these two points is based on a number of different assumptions and lines of reasoning. In this response, I will address points raised by Drury et al (3). Additionally, I will attempt to show that our disagreement is based on differences in fundamental assumptions about representation of the physical configuration of soil-root-pathogen systems, and that a critical evaluation of these assumptions and their implications indicates that my point of view is the more realistic one.

THE ACCURACY OF RHIZOSPHERE WIDTH CALCULATIONS

All models simplify reality. Consequently, all models are inaccurate to some extent. A decision as to whether the inaccuracy of a particular model is tolerable must be based on its intended purpose. The equations for the calculation of rhizosphere and spermosphere widths were envisioned as being used in two types of investigations: determination of whether the distance from a plant part to a propagule that can infect it is related to any other factor (such as propagule size, control measures, type of pathogen, etc), and determination of whether discrimination of rhizosphere and rhizoplane effects on the basis of the regression models proposed by Baker and co-workers (1,2) is valid in all situations (4,5). For the first type of investigation, the tolerable degree of accuracy is unknown at present. However, for the second type of investigation, both the direction and magnitude of tolerable inaccuracy can be known. In order to use the rhizosphere width calculation to show that a rhizoplane effect is not operating in a particular situation (ie, to show that propagules must be initiating infections from beyond what can be reasonably considered to be the rhizoplane), it is more important that rhizosphere size not be overestimated than that it not be underestimated. If the "true" rhizosphere is larger than a calculated value which indicates a rhizosphere effect (ie, rhizosphere width has been underestimated), it is still definitely a rhizosphere. If the "true" rhizosphere is smaller than the calculated value, then a rhizoplane effect may be present, and thus an incorrect decision could be made. Thus, the rhizosphere width calculation may be used to show that a rhizoplane effect does not exist as long as a minimum width can be calculated with some degree of certainty. The tolerable amount of overestimation can be considered to be that which allows reasonable certainty that it is not only propagules that actually touch the root that initiate infections. If a rhizoplane effect is considered to exist when the calculated

rhizosphere width is less than the radius of a pathogen propagule (ie, a propagule must touch the root in order to initiate an infection), then certainty of the existence of a rhizosphere effect can be based on the certainty of the accuracy of the values used in the calculation of rhizosphere width (ie, *ID*, infections, and the physical dimensions of the plant part concerned). If reasonable variation in the independent variables could not result in the calculation of a rhizoplane effect, then a rhizosphere effect can be assumed. So, a discussion of the tolerable accuracy of rhizosphere and spermosphere width calculations must center on whether or not a reasonably accurate minimum width can be calculated, not on the absolute accuracy of the calculations.

Drury et al (3) discuss several factors that reduce the accuracy of rhizosphere width calculations. Most of their points are valid. In particular, the term "propagule efficiency" appears to be more suited to the model than does the term "propagule competence," and should replace it. However, it is important to note that all but one of the factors which they discuss (as well as other factors discussed in the paper in which the rhizosphere width equations were presented [4]) result in an underestimation, not overestimation, of rhizosphere width. The one factor for which inaccuracy of estimation can be expected to sometimes result in overestimation of rhizosphere width is the *ID* of the pathogen. If pathogen *ID* is underestimated, the rhizosphere width will be overestimated. Thus, the question of whether a minimum rhizosphere width can be calculated is a question of whether the maximum probable *ID* of the pathogen used can be estimated. Drury et al (3) correctly point out that population assays based on the use of selective media may greatly underestimate natural populations of some propagules of some pathogens (such as oospores of *Phytophthora* spp.). However, for some other pathogens, recovery of 80–100% of the propagules added to nontreated soil has been reported (10,11). If propagules are counted visually, added to soil at specific *ID*s, and the population added is used in a rhizosphere width calculation, the rhizosphere width will be overestimated only if the pathogen population increases in the absence of the host. Such population increases may occur in soils treated by some methods, such as autoclaving. However, if nontreated soil or soil treated with a relatively mild method is used, and if assays performed after infestation do not indicate a population increase, then the *ID* at which the soil was infested should be able to be used for rhizosphere size calculations with reasonable certainty that *ID* has not been underestimated.

In summary, the value that can be calculated by using the rhizosphere width equation is not very accurate; however, it is accurate enough for its intended purpose of allowing decisions to be made about whether or not a rhizoplane effect exists in a particular situation.

VOLUME AND SURFACE PHENOMENA

Drury et al (3) correctly point out that the rhizosphere and spermosphere width equations are based on an assumption that the relationship between infection and *ID* is a volume phenomenon—ie, it is assumed that a rhizoplane is merely a very small rhizosphere, not a qualitatively different phenomenon. They suggest that the

rhizosphere size equations should be used only if it has been established by the method of Baker and co-workers (1,2) that a rhizosphere effect exists. The interaction of their argument with the intended use of the rhizosphere width equation for differentiation of rhizosphere and rhizoplane effects produces two mutually-exclusive hypotheses: A) the theory of Baker and co-workers is valid, and calculations of rhizosphere width cannot be used to differentiate rhizosphere and rhizoplane effects; and B) the theory of Baker and co-workers is not valid. In order to show that hypothesis B is the correct alternative, it will be shown that the rhizoplane model of Baker and co-workers predicts results which are untenable when applied to *ID*-infection relationships for soilborne plant pathogens, and that these predictions result from a misrepresentation of volume-surface phenomena.

The interaction of suggested models of *ID*-infection relationships with the rhizosphere and spermosphere width equations was examined in the paper in which the rhizosphere width equations were presented (4). This was done because *ID*-infection data is used for calculation of rhizosphere width. Furthermore, since calculated rhizosphere width depends on the ratio of infections to *ID*, it was suggested that if the number of infections per unit of inoculum varies with *ID* (ie, a nonlinear relationship exists), then the most accurate estimate of rhizosphere size should be obtained by fitting a regression equation tied to the origin to *ID*-infection data, and then calculating rhizosphere width on the basis of the ratio of infections to *ID* predicted by the fitted equation at very low *ID*s (ie, as *ID* approaches zero). When this was done for a quadratic equation tied to the origin ($y = k_1ID + k_2ID^2$) or the limiting site equation ($y = N(1 - e^{-aID})$) proposed by Vanderplank (13), a finite value of rhizosphere width was calculated. However, when an equation of the form proposed by Baker and co-workers ($y = kID^b$) was used, unrealistic values for rhizosphere width were produced. If b was greater than 1, calculated rhizosphere width was 0 as *ID* approached 0. If b was less than 1, calculated rhizosphere width was infinity as *ID* approached zero. Only if $b = 1$ was a finite rhizosphere width calculated. Thus, if $b = 2/3$ (in which case a rhizoplane effect would be predicted according to the theory of Baker and co-workers [1,2]), the calculated rhizosphere width would be infinity. It was noted that the existence of this paradox makes the use of equations of the form proposed by Baker and co-workers (1,2) inappropriate for use in the calculation of rhizosphere width, and that the paradox may indicate that calculation of rhizosphere width for a rhizoplane situation is inherently impossible, and/or that either the rhizosphere width model or the model of Baker and co-workers (1,2) is invalid. Thus, the incompatibility of the rhizosphere width model and the theory of Baker and co-workers (1,2) has been noted by both Drury et al (3) and the author. To decide which of these alternatives is correct, it is necessary to examine the implications and assumptions of the theory of Baker and co-workers (1,2).

The theory of Baker and co-workers (1,2) distinguishes two distinct situations: rhizosphere effect and rhizoplane effect. The concept of the rhizosphere effect is widely accepted (7,8,13), and as

is shown below, it is compatible with the *ID*-infection theory developed by Vanderplank (13). In contrast, the concept of a qualitatively different rhizoplane effect has been criticized by a number of authors (7,8,13), and it is the source of the incompatibility of the theory of Baker and co-workers (1,2) and the rhizosphere and spermosphere size equations. For a rhizoplane effect, Baker and co-workers (1,2) proposed that

$$S = kID^{2/3} \quad (1)$$

in which S = number of successful infections, and k = a constant. Drury et al (3) refer to k as the "apparent surface area of the phenomenon." However, an examination of the tetrahedron space model used by Baker and co-workers (1,2) in the development of the theory indicates that it would be more proper to say that " k is proportional to surface area." If the tetrahedron space model, as corrected by McCoy and Powelson (9), is used to develop the equation, k is equal to a constant (0.9164) multiplied by surface area. In either case, k may be accepted as being either equal to, or close to and proportional to surface area. If equation 1 is a valid representation of the relationship between number of infections and *ID*, then it should be possible to use it to predict the number of infections which should result at specific *ID*s for a plant part of surface area = k . In particular, the equation can be used to predict numbers of infections in hypothetical experimental systems such as the following one.

A single perfectly spherical seed of surface area = 1 cm^2 is placed in the exact center of a perfectly spherical container which will hold just the seed plus 1 cm^3 of soil. Thus, the radius of the seed will be 0.282 cm, the distance from the center of the seed to the edge of the container will be 0.639 cm, and the distance from the surface of the seed to the edge of the container will be 0.357 cm. A large number of these experimental units is constructed. Pathogen propagules are mixed randomly (or regularly) into the soil at specific *ID*s. It is assumed that *ID* and infection can be measured perfectly, that every propagule which "touches the seed" initiates an infection, that all seeds are identical in size and susceptibility, and that a spermiplane effect as defined by Baker and co-workers is present. According to the theory of Baker and co-workers (1,2), the number of infections may be calculated as a function of *ID* by equation 1. In Table 1, the predicted number of infections per seed for specific *ID*s and the ratio of infections to propagules are presented. Examination of the number of predicted infections per propagule indicates that for *ID*s less than one propagule per cubic centimeter of soil, more than one infection per propagule is predicted. For example, if the experiment were repeated a number of times with 100 experiment units and 0.01 propagules per cubic centimeter of soil in each performance, it would be predicted that there would be an average total of one propagule in the 100 experimental units, and 4.64 infections in those same 100 experimental units. This implies that a single propagule can initiate more than one infection, even though a propagule must touch a seed in order to initiate an infection (and thus one propagule can touch only one seed)—obviously an impossibility.

Rather than accept that hypothetical results such as those above indicate a deficiency in the theory of Baker and co-workers (1,2), Drury et al (3) propose that there is a minimum relevant distance for any particular *ID*, and correspondingly, a minimum relevant *ID* for any particular system size. They propose that the minimum relevant distance is equal to $ID^{1/3}$, and correspondingly, the minimum relevant *ID* for a particular distance is equal to the reciprocal of the distance cubed. For the hypothetical system described above, the minimum relevant *ID* would be the reciprocal of the distance from the seed surface to the edge of the container cubed: $(0.357)^{-3} = 22$ propagules per cubic centimeter of soil. Thus, they would consider the entire example above to be irrelevant. This viewpoint might be accepted if the minimum relevant distance concept were a widely accepted attribute of similar systems; however, no appropriate references to the development of this concept are cited by Drury et al (3), and in fact, the concept is irreconcilable with established statistical theory. The proper treatment of systems in which a random distribution of objects or

TABLE 1. Seed infections as a function of inoculum density as predicted for a hypothetical experimental system in which a spermiplane effect is in operation

Inoculum density (propagules per cm^3 of soil)	Successful infections per seed ^a	Infections per propagule ^b
0.0001	0.0022	22.0
0.001	0.01	10.0
0.01	0.0464	4.64
0.1	0.215	2.15
1.0	1.0	1.0
10.0	4.64	0.46

^aCalculated using the equation $S = kID^{2/3}$, in which S = successful infections per seed, ID = inoculum density, and k = surface area per seed (1 cm^2).

^bRatio of successful infections per seed to average number of propagules per seed in a system where each seed is surrounded by 1 cm^3 of infested soil.

points is overlaid by bracketing measures is applicable to any number of dimensions (eg, lines, planes, and volumes), and can be illustrated simply for one dimension as follows.

Assume a line of indeterminate length. On this line we randomly distribute points at some mean density d (units = number per unit length). Bracketing distances of a certain length l are now superimposed on the line in either a random or regular manner. This system is a Poisson process (12). The probability that a single bracketing distance will contain no points is e^{-dl} . Correspondingly, the probability that a bracketing distance will contain one or more points is $1 - e^{-dl}$. These probabilities (as well as the probability that a bracketing distance will contain any particular number of points) can be calculated for any finite positive values of d and l . No matter what the values of d and l , there will always be some probability ($0 < P < 1$) that a bracketing distance will (and similarly, will not) contain points. There is no minimum distance, and similarly, there is no minimum ID which must be used to test the theory of Baker and co-workers (1,2).

The question may now be asked: if the rhizoplane model of Baker and co-workers is not valid, how is it that it was derived from the apparently valid concept of propagules being represented by points in space? The answer is not that the points in space concept is invalid (the valid rhizosphere concept was also developed from it), but that the manner in which a plane (representing the root surface) is superimposed on the point space is inappropriate. The rhizoplane model starts with a volume in which points are regularly distributed, and for which the distance between points can be calculated (1,2). A plane is then superimposed onto the point space in such a manner that the plane exactly lines up with a layer of points. Since the distance between points is the same in the point space and on the plane, the density of points on the plane can be related to the density of points in the space (volume) by means of the distance between points. This procedure gives rise to the rhizoplane equation (equation 1). The problem with this procedure is that it ignores the fact that as the density of points in space is increased, not only does the density of points on the layer intersected by the plane increase, but also, the density of the layers of points increases—ie, the layers become closer together. As long as propagules are considered to be points, this third dimension cannot be incorporated into the model, since the probability that a plane (with no height) will intersect a point (with no finite dimensions at all) is zero. However, if the propagules are assumed to have some finite volume, then it can be shown that the probability that a plane inserted into a point space parallel to the layers of points will intersect a layer is proportional to the one-third power of the density of the points in space (ie, $P = kID^{1/3}$, where P = probability, ID = point density, and k = a constant which incorporates point size). Combining the probability that a layer will be intersected with the equation for the density of points per layer (ie, the rhizoplane equation) gives:

$$S = k_1 ID^{2/3} \cdot k_2 ID^{1/3} = k_3 ID.$$

This is the same form as the rhizosphere equation. Thus, the value of S that can be calculated using equation 1 is not predicted successful infections, but rather, an abstract entity with no correspondence to a measurable property of soil-plant-pathogen systems. Regardless of whether a rhizosphere or rhizoplane effect is operating, the basic form of the ID -infection equation is

$$S = VID$$

in which V = rhizosphere volume. This equation describes a straight line regardless of the values of V and ID .

RHIZOSPHERE SIZE AND LIMITING SITES

Drury et al (3) attempt to reconcile the rhizoplane effect equation of Baker and co-workers (1,2) with the Poisson one-hit equation proposed by Vanderplank (13) by proposing a combined equation (equation 9 in their paper [3]). However, because the combined equation incorporates the rhizoplane equation of Baker et al (1,2), it shares the deficiencies of that equation, and should be rejected. Although Vanderplank's equation does not explicitly incorporate rhizosphere size, an examination of its derivation for soilborne

pathogens can be used to show that rhizosphere size can be derived from it.

Vanderplank's equation described a Poisson process. For a soilborne pathogen, it may be considered to describe a situation in which propagules are distributed randomly in soil at some ID , and potential infection sites, each with a volume of soil V_s associated with it (the "sitesphere"), are superimposed on this distribution of propagules in soil. It is assumed that any propagule within a sitesphere initiates an infection. The expected proportion of sites that do not contain spores is e^{-IDV_s} . Thus, the expected proportion that contain spores is $1 - e^{-IDV_s}$. For a specific number of sites per unit (seed, root segment, etc) N , the expected number of infections per unit Y would be

$$Y = N(1 - e^{-IDV_s}). \quad (2)$$

This is essentially the same equation as that proposed by Vanderplank (13), except that he designated V_s as a site susceptibility parameter. Regression analysis of ID -infection data using equation 2 (after multiple infection transformation [6]) can be used to estimate N = sites per unit, V_s = sitesphere volume, and NV_s = rhizosphere or spermosphere volume per unit. The value of NV_s can be used to estimate rhizosphere or spermosphere width using the appropriate equations (4). Alternatively, a quadratic equation tied to the origin ($y = k_1 ID + k_2 ID^2$) can be used in the estimation of rhizosphere volume and width (4,5), and either a quadratic equation tied to the origin or the general equation proposed by Baker and co-workers ($y = kID^b$) can be used to determine whether limiting sites are present (5).

Although it must be recognized that the values calculated by using the above procedures are estimates, and are subject to the limitations discussed above, they still may be of use in evaluating the activities of soilborne pathogens. If sites are found to be limiting in a particular situation, then reasonable objectives of further research might be to ascertain the nature of the sites and to develop methods for the modification of site number and susceptibility. Additionally, comparison of calculated values of N and rhizosphere width for different pathogens may be of use in developing concepts of pathogen behavior; and an understanding of ID relationships is a prerequisite for the development of models which incorporate the effects of other factors on disease (eg, simulation and multiple regression models).

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