Letter to the Editor

A Reinterpretation of the Mathematical Analysis of Rhizoplane and Rhizosphere Effects

K. J. Leonard

Plant pathologist, U. S. Department of Agriculture, Science and Education Administration, Agricultural Research, Department of Plant Pathology, North Carolina State University, Raleigh 27650.
Accepted for publication 4 April 1980.

A large body of literature has accumulated relative to the concepts of rhizoplane and rhizosphere effects on the incidence of root diseases in relation to inoculum density in soil. The distinction between rhizoplane and rhizosphere effects is based on the mathematical interpretation of Baker et al (1). They concluded that in graphs plotting log of number of infections vs. log of inoculum density, a slope of 1.0 indicates a rhizosphere effect (ie, the pathogen can attack the root from a distance), whereas a slope of 0.67 indicates a rhizoplane effect (ie, the pathogen propagule must come in contact with the root for infection to take place).

Vanderplank (4), Gilligan (2), and Grogan et al (3) disputed this conclusion on the basis that the assumptions of Baker's model are not valid, and they proposed alternative models. Vanderplank (4) and Grogan et al (3) treated the rhizoplane as a very small volume adjacent to the root surface. Gilligan (2) argued that the number of propagules at the rhizoplane should include all of the propagules that were originally within the volume occupied by the host root. These propagules should have come into contact with the root as it displaced them. Both Gilligan's interpretation and that of Vanderplank and Grogan et al are based on the assumption that the rhizoplane effect applies to the propagules within a small volume that is quantitatively, but not qualitatively, different from the larger rhizosphere. Accordingly, the slopes of log-log plots of infections vs. inoculum density should not differ for rhizosphere and rhizoplane effects.

The difficulty in reconciling the alternative models of Vanderplank (4), Grogan et al (3), and Gilligan (2) with the model of Baker et al (1) is that Baker's model deals with a surface phenomenon for the rhizoplane effect, whereas the alternative models treat the rhizoplane effect as a volume phenomenon. Furthermore, the experimental evidence shows that in many cases the slopes of the log-log plots are near 0.67 which is predicted by Baker et al (1) but not by any of the alternative models. On this basis, it might appear that the biological evidence supports Baker's interpretation and that the disputed assumptions in his model either are appropriate or can be replaced by more valid assumptions without changing the logical conclusions of the model.

In their model, Baker et al (1) state that for points arranged in a regular tetrahedral lattice, the number of points per unit volume is inversely proportional to the cube of the distance between those points and their nearest neighbors. That is, $I = K_1/D_1^3$, where I is inoculum density, K_1 is a constant, and D_1 is the distance between a point and its nearest neighbor. The number of points at a surface (rhizoplane) is inversely proportional to the square of the distance between points and their nearest neighbors on the surface. That is, $S = K_2/D_2^2$, where S is the number of points on the surface, K_2 is a constant, and D_2 is the distance between a point and its nearest neighbor on the surface. Baker's model is based on the implicit assumption that $D_1 = D_2$, so that $D_2 = (K_2/S)^{1/2} = D_1 = (K_1/I)^{1/3}$ or $S = KI^{2/3}$ for the rhizoplane effect. Hence, $\log S = 2/3 \log I$.

The assumption that $D_1 = D_2$ in Baker's model is valid only if the surface in question conforms to the location of the points, but not if the surface exists independent of the points, as a root or seed surface does. This can be seen in the case of a spherical seed placed in a hole and covered with soil. In this example, Gilligan's alternative model does not apply, because the volume occupied by the seed was not previously occupied by pathogen propagules. If the propagules are arranged in a regular tetrahedral lattice, they occur in straight lines, but the surface of the seed is curved. Therefore, it is extremely unlikely for any pair of adjacent points in the tetrahedral lattice to both occur on the seed surface. Even if they did, the distance between them along the curved surface would be greater than the straight line distance between adjacent points. Thus, $D_1 < D_2$, and Baker's model is mathematically incorrect, because it is based on an assumption shown to be invalid.

It might be argued that the assumption $D_1 = D_2$ is not essential to arrive at the solution of Baker et al (1) that $S = KI^{2/3}$. For instance, if the ratio D_1/D_2 were constant over a range of inoculum densities, the model would give the result $S = KI^{2/3}$. There is, however, no justification for assuming that the ratio D_1/D_2 will be constant. In the above example, the nearest distance between points on the curved surface of a seed is measured along an arc, whereas the straight line distance between the nearest points is measured along the chord of that arc. For a spherical seed, an arc of 60° represents a distance of $\pi r/3$ in which r is the radius of the seed. The chord of the 60° arc represents a distance equal to r, and the ratio D_1/D_2 equals $3/\pi$. An arc of 90° represents a distance of $\pi r/2$, whereas the length of the chord is $(2r^2)^{1/2}$ or approximately $1.4r$, and the ratio D_1/D_2 is approximately $2.8/\pi$. Thus, as inoculum density changes, D_1 changes and the ratio D_1/D_2 changes. Therefore, the model of Baker et al (1) and their solution that $S = KI^{2/3}$ are both mathematically incorrect for this example.

The above arguments can be applied to cylindrical roots as well as to spherical seeds. First, roots are not perfectly straight, so distances between points on their surface represent arcs rather than straight lines. Second, even if the roots were perfectly straight, it would be unreasonable to assume that they would grow in perfect alignment with the assumed tetrahedral lattice arrangement of propagules in the soil. Therefore, even in the unlikely event that the root grew so that it contacted adjacent propagules in the lattice, the angle between propagules along the circumference of the root would not be 0. Consequently, the distance between propagules at the surface would still be measured along an arc. A little reflection will show that for any type of arc, as the distance between adjacent points is reduced, the ratio of D_1/D_2 increases until a limit of 1 is reached at infinitesimal values of D_1.

The above arguments were applied to the biologically unrealistic assumption of a regular tetrahedral lattice arrangement of propagules in the soil, but they also would apply to a random distribution. Distances between points on a root surface would still be measured along arcs, and distances between points in the soil would still be measured along straight lines.

If we assume, as Baker et al (1) did, that the rhizoplane effect is a surface phenomenon rather than a volume phenomenon of the types envisioned by Vanderplank (4), Grogan et al (3), or Gilligan (2), we can use a different method to derive the relationship between S, the number of propagules at the root surface, and I, the inoculum density. First, consider a rhizosphere that extends to a distance b cm from the center of a root L cm long and r cm in radius. If there are I spores per cubic centimeter of soil, there will be $I(\pi b^2 L - \pi r^2 L)$ spores in the rhizosphere. These spores can be

thought of as points that occur in a series of nested, cylindrical shells of infinitesimal width around the root surface. The number of points per shell at a distance x from the center of the root can be found by substituting x for b and differentiating the expression $I(\pi x^2 L - \pi r^2 L)$ where x is the variable and all other parameters are constants. This gives the solution $2I\pi xL$ points at a distance x cm from the root center where $x \geqslant r$. That is, as the volume of the shell at x cm from the root center approaches 0, the number of points in the shell approaches $2I\pi xL$. For a rhizoplane effect, let $x = r$, and the number of spores in contact with the root surface is $2I\pi rL$. Notice that both the number of spores in the rhizosphere and the number in the rhizoplane are directly proportional to I, the inoculum density. Therefore, theoretically a plot of log S vs. log I should have a slope of 1 regardless of whether there is a rhizosphere or rhizoplane effect. In actual experiments, however, the value of S is not determined directly. Instead, the numbers of infections per plant are estimated from the multiple infection transformation, log $[1/(1-y)]$, in which y is the proportion of diseased plants. This transformation is based on the assumptions that propagules are randomly distributed, that the host plants are all equally susceptible, that all infections are equally likely to result in recognizable disease symptoms, and that disease symptoms are as likely to result from a single infection as from multiple infections. For many diseases caused by soilborne pathogens, one or more of these assumptions may not be valid, so the slopes calculated from plotting log $\log_e [1/(1-y)]$ vs. log I may deviate from the predicted value of 1.

The above analysis is not intended as an alternative model for disease intensity–inoculum density relationships for soilborne pathogens. The purpose is to show that these relationships would not be qualitatively different for rhizoplane and rhizosphere effects even if the rhizoplane effect were purely a surface phenomenon. I used this approach because I believe that so long as the mathematics of the model of Baker et al (1) appeared to be valid, there would always be doubts about the interpretation of any biological evidence contrary to their model. In retrospect, the biological and common sense approaches of Vanderplank (4), Gilligan (2), and Grogan et al (3) would have been sufficient had it not been for the mathematical problem. Logically, rhizoplane and rhizosphere effects could not be qualitatively distinct without any transition between them. Roots growing through the soil will contact spores in a volume, not along a plane (2). The distinction between a spore at 0 distance from a root and one at an infinitesimal distance ϵ from the root is biologically meaningless. Slight vibrations in the soil, expansions and contractions of roots and spores, and slight elasticity of root and spore surfaces would cause spores and roots to alternately touch and separate by very small distances. Increases in root diameter with growth or in seed diameter with imbibition of water would bring them into contact with spores not previously at their surface.

Baker's interpretation of the rhizoplane effect was an explanation of why plots of estimated numbers of infections vs. inoculum density often deviate from a straight line. In seeking a better explanation, we have available a large and valuable body of information about disease incidence–inoculum density relationships. Much of this information is available primarily because of the stimulation supplied by the modeling work of Baker et al (1). In science, innovative thinking is not always correct, but it is nearly always valuable.

LITERATURE CITED

1. BAKER, R., C. L. MAURER, and R. A. MAURER. 1967. Ecology of plant pathogens in soil. VII. Mathematical models and inoculum density. Phytopathology 57:662-666.
2. GILLIGAN, C. A. 1979. Modeling rhizosphere infection. Phytopathology 69:782-784.
3. GROGAN, R. G., M. A. SALL, and Z. K. PUNJA. 1980. Concepts for modeling root infection by soilborne fungi. Phytopathology 70:361-363.
4. VANDERPLANK, J. E. 1975. Principles of Plant Infection. Academic Press, New York. 216 pp.