In this letter we question the existence of a strict rhizoplane association as defined by Baker et al (1-5), and extend the rhizosphere concept to include larger propagules that can infect from greater distances; ie., from outside the rhizosphere influence. Further, we question interpretations that have been applied to linear slope values of disease incidence (DI) versus inoculum density (ID) after $\log_{10} \ln - \log_{10}$ and other transformations. Also, we will examine the relationship and interpretation of transformed data with respect to the interpretation of arithmetic plots of the same data.

**Rhizoplane versus rhizosphere.** The strict rhizoplane concept assumes that only propagules in direct contact with the root surface can cause infection. However, convincing biological evidence has not been presented that demonstrates the existence of host-pathogen combinations with such a strict association. In all known cases, fungus propagules can bridge a finite gap by some mechanisms such as the production of a germ tube or by motility. Rhizoplane infection theoretically is indicated by the Baker models when the slope of the linear relationship between $\log (1-DI)^{-1}$ and log ID is 0.67 (3). This slope is the mathematical consequence of the assumption that propagules can be considered as points without volume distributed uniformly in the soil at the vertices of tetrahedra (5).

We agree with Vanderplank (13) and Gilligan (6) that the assumptions used for the derivation of the rhizoplane model are of questionable validity. Obviously, propagules have volume, and their distribution in soil approaches random. Therefore, the number of propagules in any unit volume should be directly proportional to the number in the whole volume. If the propagules are randomly distributed, some would touch the rhizoplane and others would be located in the soil surrounding the root surface, but only those within a limited distance from the root surface would be able to cause infection. Additional inoculum in the soil should result in a proportional increase in infection whether the propagules can infect from some limited distance or must be very near the root surface. Therefore, we maintain that the so-called rhizoplane association is merely a rhizosphere association with a small radius of influence.

**Plotting and interpreting data.** Vanderplank (13) has demonstrated with numerous examples of simple-interest type soilborne diseases that arithmetic DI-ID plots commence at the origin and are linear within the lower DI-ID range. If the availability of susceptible sites is not limiting and competition among propagules in the higher ID range is not significant, the linearity will continue until DI 100% is attained (line A, Fig. 1). Usually, however, the plot becomes curvilinear to the right in the higher DI-ID range because of competition among propagules for susceptible sites or because multiple infections are not distinguishable from single infections. If 100% of the host population is susceptible, DI 100% should be attained with successive increments of inoculum (line B, Fig. 1). Arithmetical plots of most DI-ID data conform to lines A and B and Vanderplank argued convincingly that these curves are "fundamental" and the basis for his "law of the origin": "When disease is plotted against inoculum, both on arithmetic scales, the curve starts at the origin."

In numerous plots of DI-ID data in current literature (10-12), DI 100% is not attained even though very high levels of inoculum are indicated on the x-axis; the curve approaches a plateau and additional inoculum produces little or no increase in DI (line C, Fig. 1). This leveling off of DI indicates that some factors other than ID are limiting DI increase; for example, a portion of the population may be highly resistant or some plants have escaped because of nonrandom distribution of inoculum.

As noted by Baker (3), transformations usually are done to facilitate interpolation and extrapolation of data, and also are used to test the validity of various assumptions regarding the DI-ID relationship. To illustrate the effect of various transformations on distribution of data points in DI-ID plots, and to demonstrate the potential for error in the biological interpretation of data after transformation, we have assigned DI-ID values to the three arithmetic plots in Fig 1, plotted the transformed points (Figs. 2-6), and used regression analysis to find the parameters of the straight lines of best fit (Table 1).

The Gregory (7) multiple infection correction transformation $[\log \ln (1-DI)^{-1} \text{ versus ID}]$ was derived from the Poisson distribution and is based on assumptions that one propagule can cause an infection, there are no significant differences in aggressiveness and virulence of inoculum units nor in host susceptibility, distribution of inoculum and infections is random, and the disease is of a "simple interest" type (13). This transformation also is used to convert DI% into estimated number of infections per unit, but the estimate is valid only in the lower DI-ID range where ID is limiting. The logit transformation of percent infected plants, also is derived from the Poisson distribution with the same assumptions, but assumes in addition that the disease is a "compound interest" type due to the logarithmic multiplication and spread of the inoculum. In contrast, when the log-probit transformation is used, assumptions are that relative susceptibility of the host population units is normally distributed and infections develop in proportion to logarithm of the inoculum dosage. Thus, inasmuch as the assumptions made for the Poisson distribution are quite different from those for the log-probit transformation, it follows that one or the other, but not both should be appropriate for transformation of ID-DI data from any given host-pathogen combination.

Although it is nonsensical to transform data for curve A which is arithmetically linear, we, nevertheless, have done so to illustrate the effect of the transformations on all three data sets. Note that all of the transformations result in curvature to the left of lines A and B.

The Gregory (7) multiple infection transformation usually results in the linearization of data for a curve such as B that bends to the right, but in this case it resulted in over correction (Fig. 2) because the data were too near linear before transformation ($R^2 = 0.898$). This transformation failed to linearize data for line C, but if only data points wherein ID is limiting (0-46 propagules) had been transformed, a higher $R^2$ value would result ($R^2 = 94.0$).

The Baker (3) transformation $[\log \ln (1-DI)^{-1} \text{ versus log ID}]$ nearly always produces a higher $R^2$ value than does the Gregory transformation. This log-log conversion damps variation, and results in clumping of data points, thereby producing a better linear fit. Note that the assigned data points in the untransformed plot (Fig. 1) are nearly evenly spaced. In contrast, the Baker (Fig. 3) and straight log-log transformations (Fig. 4) bunch the points in the upper right quadrant and, as a result, $R^2$ values for all three data sets are highly significant (Table 1).

As noted by Grogan et al (9), the slope of 0.67 that Baker et al (5) equated with a rhizoplane association is based on assumptions of questionable validity. If the Gregory transformation results in
linearity, slope of the line in the Baker-transformation plot equals 1.0 and efficiency of inoculum is indicated by the y-intercept value. If slope values in the Baker plot are not equal to 1.0, the only valid interpretation is that the Gregory transformation failed to produce a straight line. This may occur when experimental error or variability results in a significant departure from the origin in the Gregory plot or if the transformation fails to produce linearity as for example with curve C in Fig. 1. In this case, slope of line C in the Baker plot actually was 1.06 (Table 1), but if additional increments of inoculum from the plateau portion of the curve were included in the regression analyses the slope value would decrease to 0.67 or lower (authors, unpublished).

As noted previously, curve C represents a situation in which inoculum is a limiting factor in the lower DI-ID range, but a plateau is reached at ID of about 45 propagules; additional inoculum produces no effect on DI indicating that other factors are limiting. This might be due to nonrandom distribution of inoculum if data pertained to field sampling for ID, or to variability in susceptibility of the host population. Note, however, that all of the transformations except Gregory's resulted in highly significant R² values (Table 1). For the Baker and straight log-log transformations, this is due to clumping of the data points (Figs. 3 and 4). Thus we consider the apparent linearity to be a mathematical artifact with no intrinsic biological significance. Similarly, both the log-logit and log-probit transformations resulted in highly significant R² values (Table 1), but this is not proof that relative susceptibility was normally distributed (probit) or that the disease was a compound interest type (logit). Thus, it seems obvious that the R² parameter is not an adequate test for the validity of assumptions made prior to the selection of a particular transformation. In our opinion, biological interpretations should be based on biological observations and not on tests for linearity of transformed data. Transformations are useful for interpolation, but as Vanderplank (13) so aptly argued, they often obscure the real facts that can be visualized best from straight arithmetic data plots.

Components of our conceptual model. The root surface, although in the shape of a cylinder, is a plane surface with respect to fungus propagules that are close enough to contact the surface after germination. Thus, the factor that determines how many infections can occur is not the distance between propagules, but the number of randomly distributed propagules within a finite layer of soil adjacent to the root surface. Because of random distribution, some of the propagules will be on the rhizoplane and others will be distributed at random within the soil volume adjacent to the root and all that are not too distant have some probability of causing infection.
To facilitate discussion, we will use three descriptive expressions, biologically competent, competence distance, and competence volume. The expression "biologically competent" pertains to whether propagules are capable of germination and infection if located near enough to the root surface, but more is inferred than by the term "viable" that usually is determined as ability to produce growth when incubated on a nutrient medium. Newly formed sclerotia of *Sclerotinia minor*, for example, will produce mycelial colonies from superficial growth if cultured on nutrient agar, but are not capable of infecting susceptible host tissue without prior colonization of an exogenous food base (8). However, after sufficient aging followed by drying (conditioning) they become capable of eruptive mycelial germination and utilization of stored food resources when rewetted and can infect directly without a food base (competent). Incidentally, we have observed that *S. rolfsii* (discussed later) also has a similar capability for direct infection initiated by hyphae from sclerotia that are conditioned to germinate eruptively (Z. K. Punja and R. G. Grogan, *unpublished*).

"Competence distance" refers to the maximum distance a propagule can lie from a root surface and still have a chance of causing infection. This differs from the rhizosphere concept in that the competence distances of propagules of various fungi differ and are influenced by size (energy for growth) of the propagules and other factors that comprise the biological and physical environment of the soil. For sclerotia of *S. minor* and *S. rolfsii*, for example, the maximum competence distances are about 1.0 and 3.0 cm, respectively, as determined by placing competent sclerotia on the surface of moist field soil at various distances from susceptible tissues. Competent sclerotia of both fungi can germinate without stimulation by root exudates. Thus, their competence distances are the maximum distance from which the sclerotia can germinate and grow and cause infection in the presence of biological competition and without an extraneous food base. If too far from the root, the sclerotia usually germinate and produce a spherical pattern of growth but after several days and failure to contact the root surface, secondary sclerotia are produced instead of an infection.

We do not have comparable data on the competence distances of much smaller soilborne propagules that may require stimulation from root exudates to alleviate fungistasis or dormancy. It seems reasonable, however, that they would have much smaller competence distances and volumes than the larger propagules, and may be limited to the distance of the rhizosphere influence which can be estimated by using Gilligan's model (6).

Increase in root volume in proportion to increase both in length and radius results in proportional increase in the competence volume. With some diseases, infections occurring anywhere on the root may be equally damaging, but with others (such as lettuce drop) only those infections initiated within about 5.0 cm of the soil surface usually result in stem-root girdling that kills the plant (8). Thus, the competence volume of soil is delimited by this depth from the surface and the increase in exposure is due to increase in root radius. For example, with a competence distance of 1.0 cm and a delimiting depth of 5.0 cm, a lettuce seedling with a 0.1-cm root radius would be exposed to 19.0 cm$^3$ of infected soil whereas a nearly mature plant with a 1.5-cm root radius would be exposed to 63.0 cm$^3$, a 3.3-fold increase. Even more striking is the increase in exposure to sclerotia of *S. rolfsii* that would result from the expansion of a sugarbeet seedling with a root radius of 0.1 cm to a radius of 9.0 cm. With the 3.0-cm competence distance of *S. rolfsii*, and a competence depth of 5.0 cm, the competence volume for the seedling is about 150 cm$^3$ and for the larger root it is 990 cm$^3$ or a 6.6-fold increase in exposure.

Our conceptual model also provides a rationale for sampling field soils to determine the levels of ID for predicting disease losses. As described for *S. minor*, the competence volume for a nearly mature lettuce plant is about 63 cm$^3$. Therefore, to sample for ID, random samples should be taken after the land has been prepared for planting, with a probe that when inserted in the soil to a depth of 5.0 cm will contain about 63 cm$^3$ of soil. Sclerotia are screened from the samples individually or after bulking. The percentage of 63-cm$^3$ samples with one or more competent sclerotia (tested for ability to germinate eruptively on quartz sand) provides an estimate of the potential maximum percent of infected plants.

A concept somewhat different from competence volume of soil adjacent to the root is that each competent propagule occupies the center of an infectious volume that comprises a part of the total volume of infested soil. Size of the infectious volume is proportional to competence distance of the propagule [radius of a sphere with volume = (4/3)πr$^3$]. Thus, a sclerotium of *S. minor* with a competence distance of 1.0 cm would have an infectious volume = 4.2 cm$^3$, whereas a *S. rolfsii* with a competence distance of 3.0 cm would have a volume = 113.0 cm$^3$. If 1,000 cm$^3$ of soil contained about 238 sclerotia of *S. minor* or 8.8 of *S. rolfsii*, essentially the whole volume (with allowance for random distribution and consequent overlapping) would be occupied and a root penetrating it would have little chance of escaping infection. Similarly, if as suggested by Gilligan (6), the propagules were located experimentally in a plane in a volume of soil contained in a 100-cm-diameter container, roots growing through the infested plane with an area of 314 cm$^2$ would encounter a near 100% infectious barrier if about 100 *S. minor* or 33 *S. rolfsii* were distributed randomly in the plane. Further, because more than one plant could encounter an infectious barrier or volume, a single sclerotiorum could kill more than a single plant.

To summarize, we emphasize two biological concepts pertinent to soilborne inoculum and diseases. The first is that although soilborne inoculum is distributed in three-dimensional space, the number of propagules in any volume of soil is directly proportional to ID, and only those propagules that are biologically competent and located in the competence volume of soil near the root surface have any chance of causing infections. Therefore, data plots and interpretations of DI–ID data should pertain only to this portion of the inoculum; otherwise results and interpretations will be based on inoculum that exerts no influence on DI. The second concept is that a single competent propagule located within the competence volume adjacent to the root surface potentially can cause an infection. Thus, ID within a limiting range is directly proportional to the number of infections (13). Finally, biological interpretations should be based on biological observations and not on tests for linearity of transformed data.

LITERATURE CITED

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