Inoculum Potential and Soilborne Pathogens: The Essence of Every Model is Within the Frame

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Models relating inoculum density of soilborne pathogens to plant disease (7–9) have been "condemned" by Vanderplank (74), judged "not satisfactory" by Gilligan (29), deemed of "questionable validity" by Grogan et al (33), and declared mathematically invalid by Leonard (44). Without substantive discussion of these criticisms, the situation, like friction, is likely to generate more heat than progress.

Therefore, we contribute this rebuttal with a view to establishing: what the commotion is all about, how the models have been misapplied and misinterpreted, the logical biological bases for their construction, the probity of the basic mathematics, and the documentation for the experimental tests of their validity.

The roots of the controversy. To achieve perspective, we will review briefly the development of the mathematical models (7–9) designed to represent disease (D), which can be measured either as disease incidence (DI) or severity, as a function of inoculum density (ID), especially for models developed for rhizoplane and rhizosphere phenomena associated with fixed infection courts. Apparently they are at the heart of the controversy.

In an experiment in which an ID-D curve is developed, disease is plotted as a function of the amount of inoculum present in the system. Host and environmental parameters are held constant (the way differences in these parameters affect the relationships is treated later). The idealized form of the curve is presented in Fig. 1A, although all of its parts may not be manifested in every host-pathogen system. The models are designed to describe only that part of the curve labeled "true logarithmic scale." Certain conclusions regarding the role of synergism may be possible but the models do not function on the transitional or plateau portions (7–9) of the curves.

The assumptions underlying the models (9) are (i) that prepenetration processes conform to the known ecological relationships for plant pathogens in soil (eg, the operation of soil fungistasis); (ii) that there is a random distribution of inoculum and occurrence of events (infection); (iii) that the probability of infection occurring from units of inoculum is <1 although one propagule is capable of infecting and inducing symptoms; (iv) that all members of the host population are equally (genetically) susceptible and disease ratings are taken at the same time after inoculation; and (v) that infection results in symptom expression by the host (disease is not masked).

Consider a fixed infection court in soil, such as a hypocotyl or seed, subjected to increasing amounts of inoculum. If each propagule under the influence of the host in the rhizosphere germinates perfectly, one propagule introduced into this volume germinates, penetrates, and induces one infection (Fig. 2A). Two propagules induce two infections and so on. If this relationship is plotted as in Fig. 2B, line a, a straight line is generated with a slope value of one which indicates one infection per inoculum unit. Obviously, this perfect system does not reflect reality; inoculum is not 100% efficient. Assuming 50% infection efficiency of the inoculum, two propagules would be required to produce one infection, four to produce two, etc., as plotted in Fig. 2B, line b. With 10% efficiency, line c would be generated as in Fig. 2B. Thus, ID-infection relationships are affected by differences in efficiency of inoculum. When plotted, these generate families of straight lines with different slope values that depend upon efficiency values.

Data collected from an experiment reflecting this situation may be interesting, but will have little biological or quantitative meaning. Efficiencies are an unknown quantity for almost all pathogens and, without them, the volume of the rhizosphere and the quantity of inoculum participating in infection cannot be calculated. Even so, the family of straight lines in Fig. 2B, transformed to log infections-log ID results in a family of lines with different positions or intercepts, all with slope values of 1 (Fig. 2C). This suggests that a research worker can do an ID-D experiment involving a particular soilborne host-pathogen system and infer that the pathogen has activity (leading to infections) in the rhizosphere if the slope value of the relationship between log infections and log ID is near 1—a conclusion that does have biological meaning.

What if propagules are distributed in the three-dimensional volume of soil, but only those propagules touching the surface of the infection court can germinate, penetrate, and infect? As increasing amounts of inoculum are applied to the three-dimensional system, the question that arises is: what proportion of these units touch the rhizoplane? As evidenced by Leonard's letter (45), there is more than one approach to the problem, but the correct solution depends upon proper concepts and assumptions employed in constructing the model. In modeling this relationship, we followed the methods employed in the mathematics of packings and solids. The number of propagules touching the rhizoplane per unit area was considered to be inversely proportional to the square of the distance between them (9). The distance between propagules as a function of inoculum density is not a straight line as might be assumed; *ninium ne crede colori* (Vergil). McCoy and Powelson (49) demonstrated that this relationship was a curve. Thus, the

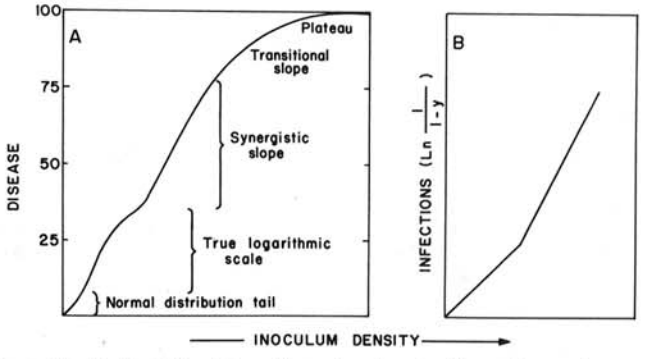

Fig. 1. Idealized relationships of inoculum density-disease interactions. **A,** Complexities that may be involved in the curve. The synergistic slope may only be present in certain systems and the normal distribution tail is hypothesized, but has not been demonstrated conclusively with experimentation. While 100% disease is indicated on the ordinate, infection sites may become limiting before this level is reached in some systems. **B,** Application of the semilog transformation which corrects for multiple infections to the ascending portion of the curve in **A.**

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slope of the curved line (Fig. 2D) resulting when the number of propagules touching the rhizoplane is plotted against inoculum density at any point is (9):

$$\text{slope value} = \frac{2k}{3(ID)^{1/3}}$$

in which k is a constant. When log infections (propagules on the rhizoplane penetrating and infecting the host) are plotted as a function of the log ID, the result is a straight line with a slope value of 0.67 (Fig. 2E). This slope value also is predicted for disease systems involving moving infection courts; in these, rhizosphere-rhizoplane relationships are not considered; this will be detailed later.

Note that the ID-infection (nontransformed) relationship is a straight line when there is a rhizosphere relationship (Fig. 2B); the plotted relationship is a curve when successful infections result only from propagules touching the rhizoplane (Fig. 2D). This is the heart of the controversy. Grogan et al (33,34) assume the ID-infection relationship to be always a straight line. Leonard (45) contends that the mathematics generating the curved line, interpreted as a rhizoplane relationship, are faulty. Gilligan's (29) equations 2 and 5 for both the rhizoplane and spermoplane generate straight lines for infections ("hits") as a function of ID.

It must be emphasized again that the log-log transformations are modeled for infections, not disease. Practically all experiments give host response data in terms of disease incidence or severity. Thus, the multiple infection correction (30) of the form ln 1/(1-y), with y being disease incidence (11) or severity per unit (35), is used for this parameter (Fig. 1B).

Whatever slope values are generated, different environmental or host (susceptibility) parameters applied to each of the ID-infection relationships alter the slope values of the individual curves or straight lines just as does propagule efficiency in Fig. 2B. If log ID-log infections are plotted from these, the influence of the parameters of environment and/or host are reflected in each ID-D relationship and parallel straight lines are generated as in Fig. 2C. The positions of these lines reflect the impact of the environmental and/or host variables and thus have a relative value in terms of ID per unit of infection which is useful in quantitative analysis. Published examples of this kind are available (12,13,35,37,41,60, 68,78). These variables, thus, reflect changes in position rather than slope values in most cases; however, with appropriate manipulation of the soil environment, a spermosphere influence may be reduced to a spermoplane influence which reduces the slope value for 1 to near 0.67 (60).

Is there a rhizoplane? Grogan et al (34) suggest that convincing biological evidence has not been presented to demonstrate the existence of a rhizoplane effect in host-pathogen interactions. They reason that "in all known cases, fungus propagules can bridge a finite gap by some mechanism such as the production of a germ tube or by motility."

Clark (19,20) coined the term "rhizoplane" as a result of his research indicating that the bulk of the bacterial rhizosphere population of cotton plants was present near the root surface. This conceptualization was confirmed by many researchers, including Starkey (67) and Linford (46), from both cultural data and direct observation. Can this be true also for some fungi?

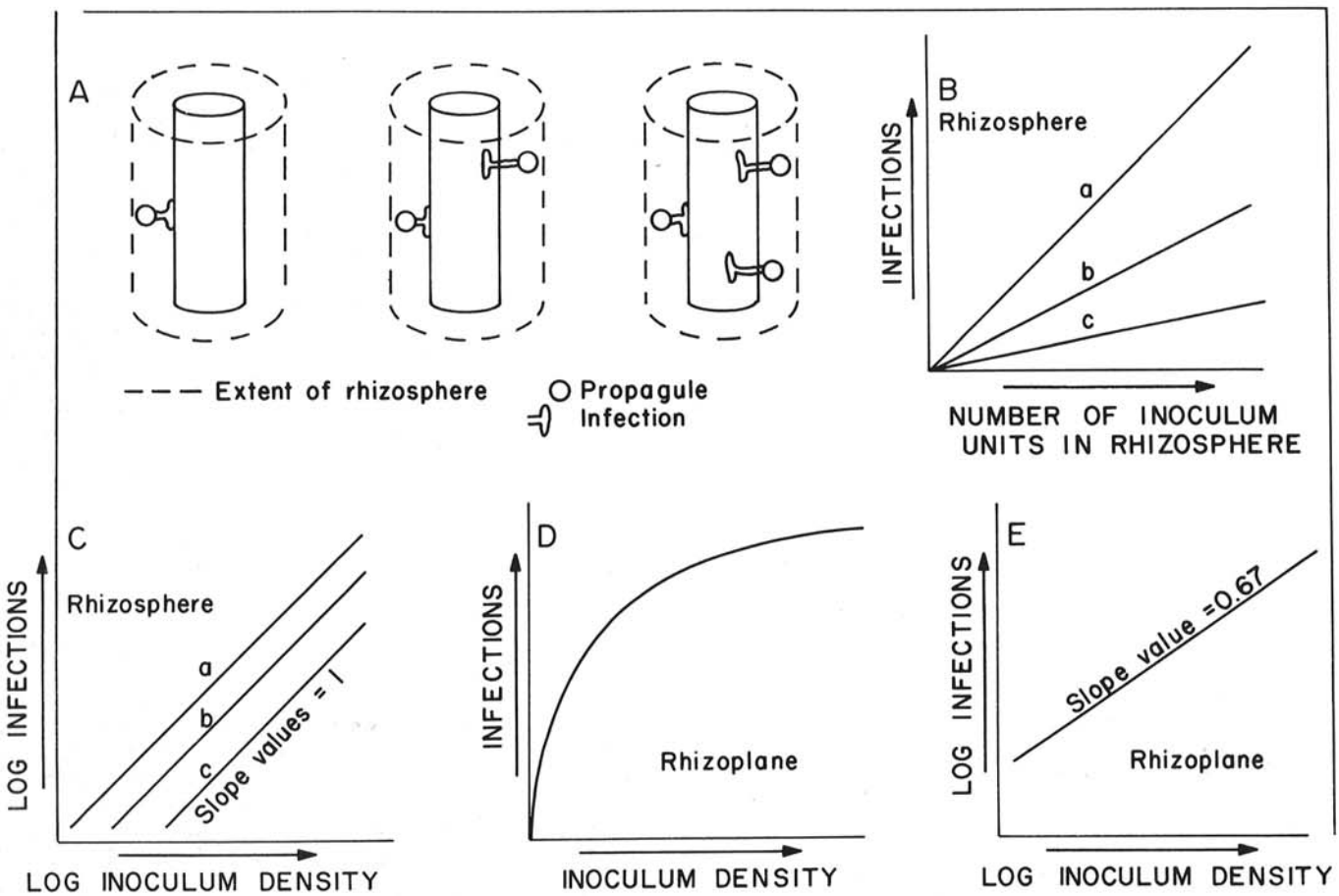

Fig. 2. Diagrammatic representation of the basic biological and mathematical relationships involved in models of rhizosphere and rhizoplane relationships. **A,** Section of a subsurface organ of a host in which propagules in the rhizosphere germinate, penetrate, and infect in a "perfectly" efficient system. **B,** The relationship in A transferred graphically to line a and the graphic results when 50% (line b) and 10% (line c) efficiencies are incorporated in the graph. **C,** Transformation of lines a, b, and c in B to log-log values resulting in three parallel straight lines all with a slope value of 1 log inoculum density unit per log infection unit. **D,** Graphic relationship in which inoculum density (in a volume of soil) increases and only those propagules touching the plane of the host surface are capable of germinating, penetrating, and infecting. **E,** Transformation of the curve in D to log-log values resulting in a straight line with a slope value of 0.67 log infection unit per log inoculum density unit.

There is a perennial shortage of available nutrients or energy sources in soil (21) and propagules of pathogenic fungi usually do not germinate in soil unless provided with some external nutritive stimulus (47). For host-pathogen relationships, this stimulus is provided by exudations from a plant organ in the vicinity of the propagules (61). Thus, propagules have been observed germinating at distances up to 20 mm from host organs (eg, 20,22,23,55, 62,63,66). No rhizosphere effect, however, has been found in other systems in which soilborne pathogens are involved (eg, 26,54,65). In the examples of research cited above, stimulation of germination at relatively long distances from host substrates is observed most frequently near plant organs that are rich in exudates such as seeds, although environmental factors, such as soil matric potential (eg, 66), also influence the extent of the effect. In contrast, other organs such as stems (below the surface of the soil) or roots exude nutrients to a lesser extent and, indeed, none may be detected on some plant surfaces (61). In such cases germination should occur only at or very near the surface and Griffin (31) concludes, after precise experimentation, that germination of *Fusarium oxysporum* chlamydospores in the rhizosphere of young peanut plants is primarily a rhizoplane effect. Even less germination was detected in the same system for conidia of *Aspergillus flavus,* because these require an exogenous source of nutrients to germinate even in pure culture (32). Thus, from observations of germination alone, there is evidence that only those propagules that touch the plant surface participate in the subsequent infection process in some systems.

What about host-pathogen systems in which germination is observed at a distance? Cook and Schroth (23) have documented the vicissitudes that may affect the germinating propagule, which may be lysed in the rich substrates surrounding a seed. Baker and Cook (6, page 192) state: "As the lag period for growth of the microorganisms passes, and as cell division and nutrient utilization accelerate, the lowest nutrient concentrations disappear, the concentration gradient away from the source deepens, and the most distant cells from the source are left without nutrients. Within a few hours only those cells in actual contact with the source will receive sufficient nutrients for continued growth."

The considerations above involve only germination. A plant pathogen must not only germinate, but it also must breach the host barrier. Obviously, this host-pathogen interaction requires more energy (eg, 70,72) than does the relatively simple process of germination, especially in an intact infection court. Cook and Snyder (24) warn "that caution should be exercised in attempting to correlate a high degree of spore germination around underground plant parts with subsequent disease development. Spore germination is only the first step in the sequence of events leading to infection...if not lysed...the germlings in the presence of a host plant may not be able to infect because of inadequate nutrients for pathogenesis."

Efficiency must be taken into consideration in any attempt to model ID-D relationships. One method of measuring efficiency is to determine the probability of success in infection from single spores (7, page 1283). Wastie (77) reported 13% success in single conidium inoculation on broad bean leaves for *Botrytis fabae* and 0.9% for *B. cinerea*. This was an experimental laboratory system with foliage pathogens that encounter less antagonism on the leaf surface than do soilborne fungi. Thus, the conclusion is inescapable that even propagules touching the rhizoplane, with all of the advantages of being served first with nutrients and escaping much of the antagonism present at greater distances from the host surface, are not 100% successful in initiating infection. Although there are no comparable studies involving soilborne pathogens reported, Griffin (*personal communication*) is accumulating data that suggest a 16.4% efficiency for microsclerotia of *Cylindrocladium crotalariae* in the colonization of peanut roots.

From the foregoing, we suggest that convincing biological evidence has been presented that demonstrates the existence of host-pathogen combinations that have a rhizoplane association. This conclusion at least permits the construction of models that hypothetically could describe such associations.

Correct applications of transformations. An example of misapplication of transformations and inappropriate use of models is found in a paper by Grogan et al (33). They determined the incidence of *Verticillium* wilt in tomato fields in the Sacramento and San Joaquin valleys of California. Ten plants were assayed in each of 30 sites from each field. The inoculum densities of *Verticillium dahliae* were determined from soil samples collected from each site. Subsequently, the data originating from plots in which less than 100% disease incidence occurred was illustrated graphically as an ID-D curve (Fig. 1A in reference 33). The total number of these data points was five, one of which included a plot with a disease incidence of 98%: this is a tenuous value for use in transformations aimed at correcting for multiple infections (eg, 7,11,25).

The first question that arises is whether the data collected can be legitimately organized into an ID-D curve (Fig. 1). The relationship between inoculum density and disease holds only when other factors are held constant. When 46 commercial tomato fields were surveyed in two valleys (33), it is not likely that conditions were constant among the sampled sites.

Environment profoundly affects symptom expression in vascular wilt diseases (10,53). For example, the concentration of nitrogen and potassium in soil affected the incidence of disease caused by four Verticillium vascular-wilt pathogens (1). Again, Grogan is the coauthor of a paper (4) in which copper-induced soil fungistasis was reported to influence incidence of Verticillium wilt in the field. If these factors are kept in mind, it is apparent that the justification for using such diversely collected data points by stating that "environmental factors usually are not sufficiently limiting to prevent the common occurrence of 100% Verticillium-diseased plants in California fields" (33) is irrelevant or is of questionable value for fulfilling assumption v (above). The position of data points collected by this process, thus, may be profoundly influenced by environmental and/or cultural practices and regression analyses applied to such systems are not valid.

The next consideration in applying data to an ID-D relationship is that of determining statistical prerequisites that properly describe the entities and events of populations as they occur in time or space to conform to assumption ii. Kranz (48) states this inherent constraint simply: "...do not apply a transformation model blindly to any disease, check suitability first by verification of the underlying distribution." Early on, then, distribution of inoculum and host disease expression must be assessed. While random distribution of inoculum in cultivated fields was suggested (52), there is no overwhelming evidence that such a spatial distribution can be assumed. Further, application of the Poisson distribution (7,25) for determining the relative number of infections resulting from this inoculum (30) dictates that plants with symptoms also be randomly distributed within the area sampled (assumption ii). No test for random distribution of such plants is reported (33), and indeed, Christensen et al (18) suggest that hosts with symptoms of Verticillium wilt are not distributed at random in the field.

Further, the limitations of the ID-D relationship in the pure form dictate that readings for the amount of disease be recorded for plants of uniform age (assumption iv). In a host population, expression of symptoms in a single season over time may take the form of the simple interest increase as suggested by Vanderplank (75). Apropos of these considerations, Grogan et al (33) state the "incidence of Verticillium wilt...was determined at the same time as the soil samples [near the end of the growing season] usually within the last month of harvest," which suggests variation in ages of plants among fields at sampling time, even if they were planted at the same time. Also, did the soil samples taken at this time necessarily contain the initial inoculum density that induced disease?

Assumption iv also dictates uniform varietal susceptibility. Race l-resistant cultivars were grown, cultivar VF 145-B7879 being the "most common" (33). This suggests that other race l-resistant cultivars, potentially with varying responses, also were rated.

These factors alone, involving applicability and underlying distribution (discussed above), preclude application of the collected data points (33) to the models. The single variable influencing host response in an ID-D curve by definition is inoculum density. Thus, the illustration of data points in Fig. 1 of Grogan et al (33) more properly should be in the form of a bar

graph (2) because variables other than ID influence the position of these points.

After performing regression analysis of the five data points in their Fig. 1B, Grogan et al (33) assume "a good fit to a straight line...(r = 0.877)..." and later conclude that "...the ID-D relationship, when inoculum is within a limiting range, is arithmetically linear." To make the latter conclusion, they cite their Fig. 1B (which plots data points corrected for multiple infections), which must mean that ID related to infections (not DI) is a straight line. With this assumption, they convert the entire straight line intact to their Fig. 1C and transform, again intact, to their Fig. 1D (which is log infections vs log ID).

Is the assumption of a straight line relationship valid from a mathematical and/or statistical standpoint? The value of r, as calculated (by the authors) by the first-order linear regression equation, is 0.88. The r value, when calculated according to the second-order linear regression equation which describes a curve, is 0.89. The residual values (mean square) are 0.75 and 0.67, respectively, which suggests that neither the first- nor second-order models can be verified. These two alternatives are illustrated in Fig. 3A.

The valid method of transformation (used by all others in appropriately related research) is to convert each data point individually, not collectively. When this is done, the slope value (log-log) is not 1 as in their Fig. 1D but 1.54 as illustrated in Fig. 3B. That this value is not near 0.67 (9) is of no concern since, as established earlier, the five data points do not describe an ID-D relationship; wide fluctuations in the position of individual data points, not necessarily correlated with the amount of inoculum, can occur.

When Grogan et al (33) convert semilog (8,25) plots (their Fig. 1B) of points with various slope values as straight lines, they predictably obtain parallel straight lines (their Fig. 1D) when transformed to log infections–log ID whose slope values are always 1 and whose position is determined by slope values in the semilog plot. This is the rationale underlying Model 1 (rhizosphere) described above. Their Fig. 1C and D are comparable to our Fig. 2B and C.

Although we have doubts about the use of a data point with a DI value of 98%, we have incorporated it into our calculations since Grogan et al (33) included it. Not only is its multiple infection correction value suspect, but it should lie on the transitional portion (Fig. 1) of the ID-DI curve. Grogan et al draw a curve in Fig. 1A but they also point out that there is a good correlation with a straight line (r = 0.816) when this point is used in an ID-DI

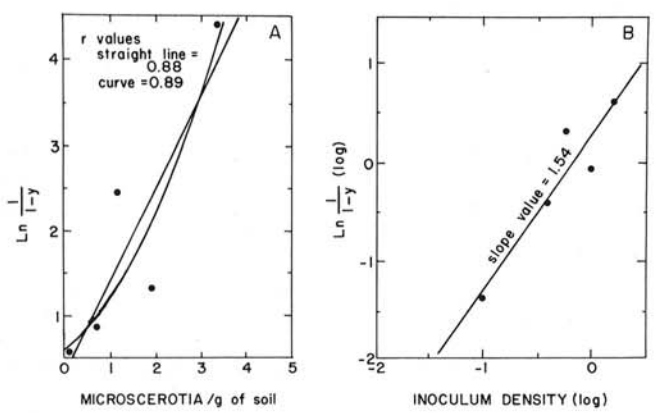

Fig. 3. A, The five data points (below 100% disease incidence) used by Grogan et al (33) for the relationship of inoculum density of *Verticillium dahliae* and ln (1/[1−y]) (in which y equals disease incidence) plotted by the computer as best fit for a straight line and a curve. The values of r (the correlation coefficient) are almost equal so that it is not possible to determine whether the true relationship is statistically a straight line or a curve. Residual (mean square) values are 0.75 and 0.67, respectively, also suggesting that neither the first or second order models can be verified. **B,** The values in A converted as individual points with regression analysis to the log-log transformation.

relationship. This phenomenon is not typical of other examples derived from experiments involving ID-DI curves where ID is the only dependent variable.

The crooked made straight and correlation coefficients in epidemiology. Grogan et al (34) question interpretations applied to linear slope values of DI versus inoculum density in transformations. They present three curves (their Fig. 1) stating that "arithmetic plots of most DI-ID data conform to lines A and B" and that "numerous plots of DI-ID data" conform to line C.

Line A cannot be found in any legitimate ID-D relationship in the literature.

Line B, when corrected for multiple infections (in their Fig. 2), is similar to the curve generated by Petersen (56) for infection foci resulting from various ID of *Puccinia graminis*. Vanderplank shows that such a curve can be interpreted as synergism (60;61; 74, page 91; 75, page 5). The only soilborne pathogen system in which experimentation suggests the operation of synergism involves *Rhizoctonia solani* (7,8,12,13,60,78). Thus, their line B is not typical of most host-pathogen interactions reported in the literature.

Line C is the only curve that Grogan et al (34) document with literature citations and, in these, slope values correlate with those predicted by the models (35,50,51). In such a relationship, DI at 100% is not reached even though high levels of inoculum are applied. Another example of this is furnished by Hanounik et al (37). They induced black rot of peanut with microsclerotia of *Cylindrocladium crotalariae* at various inoculum densities. In soil not treated with a fungicide, disease severity in the resistant cultivar Spancross only reached 31.5–34.5% at inoculum densities of 1,280–5,120 sclerotia per gram of soil. As pointed out by these authors and by us (7–9), it may be difficult to determine what points lie on the log-log portion of the ID-D curve (Fig. 1A); however, when the four lowest levels of inoculum density are used in the log-log transformation, the slope value for Spancross is 0.771; for five points, it is 0.691; for six points, it is 0.583 (37, Table 2). These are quite different from the slope value of 1.06 for the curve presented by Grogan et al (34) in their line C of Fig. 3 when all their points are used. Why?

Grogan et al (34) use data points in all transformations in Figs. 2–6 for values nearly equivalent to zero ID and zero or 99% disease. It is not mathematically or logically possible to obtain a multiple infection value for 100% disease. High values approaching 100%, while given in tables (eg, 74), may not be reliable (11). Similarly, there is no $\log_{10}$ value for zero, and probit values for zero and 100% are infinitely near ±5 standard deviations from the mean. Use of such extreme parameters skews curves and relationships to such an extent as to render them useless. For example, line B in Grogan et al (34, Fig. 2) suggests the operation of synergism according to the system of Vanderplank (74,75) explained above. When using the minimum value for a data point for probits and log ID in Fig. 6, Grogan et al (34) obtain a slope value of 1.65 (their Table 1) which is well below >2 which is predicted for synergism by the log-probit transformation (25,57). When line B is properly transformed with data points other than log −1 for ID and a minimum probit value of 1.910 (<0.1% disease) for DI, the slope value becomes 3.74 which suggests the operation of synergism. These skewed values are evident in all the transformations used by them (34).

Grogan et al (34) do not provide citations for the sources of their curves. As documented above, it is doubtful whether the curves were obtained from experiments reported in the literature. Their curves resemble those provided by Vanderplank (75) in his Fig. 1.7, except that number of infections rather than the DI is used by him on the ordinate.

With these factors in mind, it is difficult to pin down precisely the issues that Grogan et al (34) advance in support of their condemnations of transformations used in analyses of ID-D curves in epidemiology (7). The heart of the argument appears to be that "the apparent linearity...(from use of transformations) is a mathematical artifact with no intrinsic biological significance." To demonstrate this, they apply transformations appropriate for conversion of ID-D data, such as the semilog, log-probit, and log infections-log ID to their apparently phantasmic curves and show

that R^2 values are equally high when inappropriate transformations are used (logit and log ID-log D). In other words, almost any transformation, whether its assumptions are valid or not, "can stand a lot of abuse from natural systems without responding badly" (11).

Conversion of parameters and data points to straight lines is a common practice in epidemiology, but it has no significance itself. Vanderplank (74), with characteristic syntax, echoes our thinking: "...it is no part of the argument for the use of log (x/[1-x]) to suggest that regression lines are usually straight." Again, Kranz (43) states that "transformation of data is a well-known procedure in statistics mainly employed to straighten curves, convert percentages, and so on...(but) it is not always linearity that necessitates transformations." If one applies lack of fit tests to regression equations, as Wijetunga and Baker (78) did for Vanderplank's transformations (74) applied to a disease analogue, no model may demonstrate a better degree of fit than regression plots of nontransformed data. As a result of this, we did not reject Vanderplank's transformations. Rather, we agree with Waggoner (76) that in such cases in which the data fit any model, "the logical course is to choose a differential equation that is both biologically appealing and simple and then to use it if it fits the curve...reasonably well."

It shall be the task of the next section to test whether the models are "biologically appealing."

Are the assumptions used in the models valid? Gilligan (29) questions some of the assumptions of the models reiterating criticisms of the models listed by Vanderplank (75).

According to Vanderplank (75) "there is no known evidence that disease/inoculum curves for roots are in any way unique." To support this, he states that the ID-D curves generated by Last and Hamley (44) for lesions induced by *Botrytis fabae* have a slope value (log-log) not significantly different from 0.67. Similarly, in "...Wastie's [77] experiments with spores of *B. cinerea* applied to the surface of leaves of *Vicia faba* the log-log slope was approximately 2/3." The slope value generated by Last and Hamley (44) is 0.64; those by Wastie (77) are 0.43 (*B. cinerea*) and 0.27 (*B. fabae*). These wide ranges in slope values are predictable. First, as Vanderplank (75) suggests, slope values in systems involving both foliage and soilborne pathogens may differ depending on the location and number of data points used from the transitional or plateau portions of the ID-D curve. For application of data to the models, points from the transitional or plateau portions of the curve are not valid (7–9). Second, slope values for ID-infection relationships of propagules deposited on a plane surface (such as a leaf) would be a function of their efficiency in initiating infection (77, Fig. 2B). These two factors suggest that slope values of ID-infection curves for foliate pathogens should vary widely and this is indeed the case. A further defense of the "condemnation" is found in Vanderplank's (75) use of relationships plotting log ID as a function of log lesions. The mathematics of the models (7–9) dictate plots involving log ID vs log infections (not lesions or disease). Therefore, these criticisms are based on misapplications of the elements of the models.

When propagules are thoroughly mixed in the soil, a random distribution of inoculum occurs. Random distribution also may be present in cultivated field soils (52). Calculations for construction of the models assume a tetrahedral arrangement of propagules in the soil. Is this perfect distribution of inoculum in the models compatible with the random distribution found in mixed soil or in the field? The models are based on equations in which the average distance between propagules as a function of inoculum density is used (9). Thus, the key question is: how many measurements are required before the average distance among randomly distributed propagules can be computed with confidence?

In Fig. 4A, 60 points are placed in an area. The positions of the points were determined from a table of random numbers. Again, using this table, points were selected and the distance to the nearest adjacent point was measured. Kershaw (40) suggested a simple subjective analysis to determine the effect of the size of a sample on variation in the value of a mean which has application in this case. The method consists of increasing the number of samples several times, calculating a new mean each time, and plotting the values obtained against number of samples taken. The point at which the mean value ceases to fluctuate is easily determined. In Fig. 4B, the measurements between the randomly selected points in Fig. 4A are plotted in this way. The mean value is reached in six-to-eight measurements. Thus, the average distances between propagules used in the models is valid when hundreds, thousands, millions, and billions of propagules are randomly distributed in a typical experiment.

Gilligan (29) suggests that inoculum in our experiments (12,13,60,78) was not randomly distributed. In all cases, it was indicated that a twin-shell blender was used to mix the soil. The manufacturer of the instrument and soil scientists have used radioactive tracers to determine the distribution of particles mixed by the instrument. Mixing for under 1 min is required to reach random distribution.

Another assumption of the models, questioned by Vanderplank (75) and Gilligan (29), is that propagules are mathematical points in soil. For modeling purposes, there is no flaw in this assumption. First, points are used universally by modelers and mathematicians when the dimensions of the object represented by the points are small in relation to the volume of containment. The equations, developed by McCoy and Powelson (49), for determining the spatial distribution of soilborne propagules can be used to obtain a

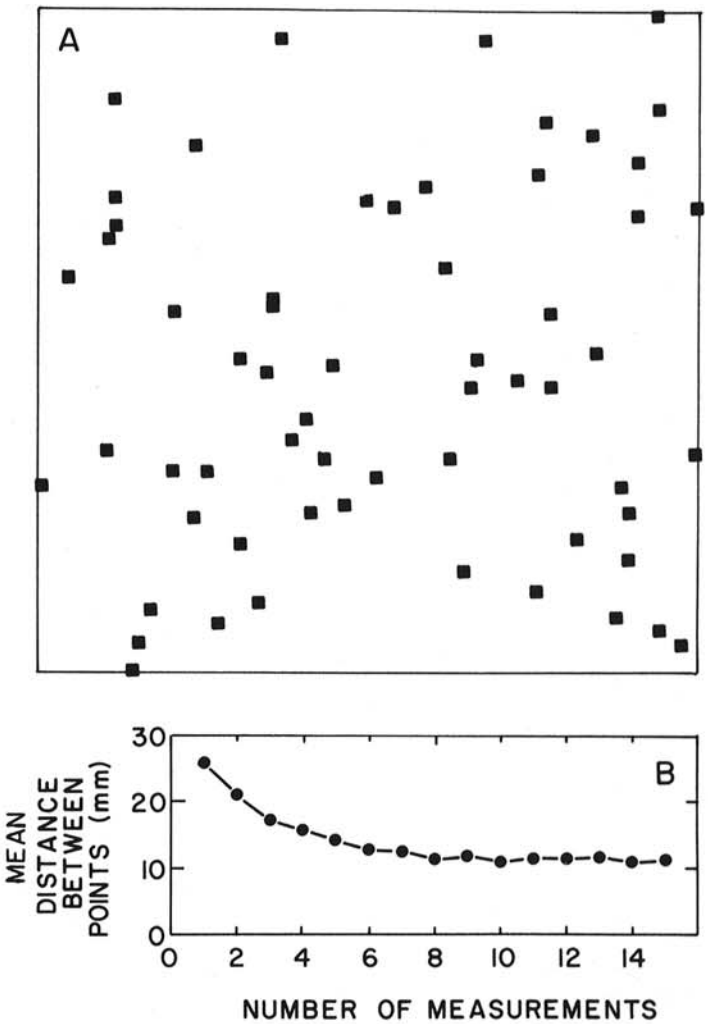

Fig. 4. The validity of using average distance between propagules as a component in calculations describing natural systems where distribution is at random. **A,** Location of 60 points whose positions in a grid was determined from a table of random numbers. **B,** Effect of increasing the number of measurements of distance between adjacent points on the values of the average distance between propagules according to the method of Kershaw (40). Relatively stable average values are obtained in six-to-eight measurements.

relative idea of the average distance between propagules in relation to their size.

Observe a period on this page and consider it relative in size to a chlamydospore 10 μm in diameter in soil. Relative to its size, at 250 propagules per gram, the next chlamydospore (period) would be almost 6 cm across this page. At 3,000 propagules per gram the distance would be approximately 3.7 cm. Microsclerotia of *Verticillium albo-atrum* (250 μm in diameter) would be over 1 cm distant from each other at three propagules per gram of soil (5). Clearly, propagules can be considered as points distributed in soil in a relative sense. Modelers who wish to describe phenomena below the soil surface must conform their thinking to the dimensional scale of the objects that they wish to describe mathematically!

Even if the above perspective betokens the validity of considering the location of propagules in soil as mathematical points, it is difficult to discern the leap of reasoning required to allow for their volume in the calculations by those most adamant on this point. Vanderplank's (75) argument, which is endorsed by Gilligan (29) and Leonard (45), is that the spores occupy a significant portion of the soil near the root. The fungus with the chlamydospore 10 μm in diameter, mentioned above, would occupy only 2.25×10^{-5} of the volume even at 3,000 propagules per cubic centimeter.

Gilligan (29) suggests that the sclerotia of *R. solani* that we used (60) were 2 mm in size since they passed a 2 mm screen. We screened to provide a soil-inoculum mix of texture and particle size suitable for mixing uniformly into similarly screened raw soil without inoculum. Screening does not determine the size of the propagules; their dimensions are determined by the genes of *R. solani*. There were large and small propagules (38,39), certainly all well below the size of 2 mm in diameter.

All these considerations fade, however, when the modeler again descends into the soil to determine the dimensions of the biological interactions in the infection court. Calculations in the model involving fixed infection court and fixed inoculum attempted to determine what proportion of the inoculum touched the infection court at any given ID. Obviously, propagules do not touch a plane with their entire surface—they only touch with a portion of their surface at a point on the rhizoplane. This point of contact must have dimension, but relative to the area of the rhizoplane, it is exceedingly small. However, the critical question, in a biological sense, is: does the small area of contact, assumed in the models as a point, conform to the actual situation where the host barrier is breached by the soilborne pathogen?

It is well established in plant pathology that propagules breaching intact host tissue do so by producing germination hyphae capable of penetration. The diameters of these penetration hyphae are typically 2–5 μm or less. This is the morphological unit assumed in the models as the point. It is difficult to see how the volume of a propagule, from which the infection hypha is produced, has anything to do with the geometry of the infection process—propagules do not breach host barriers like cannon balls!

Gilligan (29) and Leonard (45) contend that the expansion of a seed or root will have a significant effect on model construction. Certainly the root of a beet as it expands must be disruptive to all the carefully laid geometry of a modeler. Such a situation was not within the scope of the proposed models at their conception (9). Nevertheless, lesser alterations in the geometry of other infection courts occur and the key question is whether they are significant enough to be taken into consideration. The magnitude of change would not seem great for seed used in our investigations. For example, the increase in surface area of a radish seed due to imbibition during the 20- to 30-hr period during which it may be damped-off (13) is less than 2×10^{-1}.

Some situations in soil require that the growth of the host must be taken into consideration. The models (9) describe this situation in the systems involving moving infection courts. These are complicated systems involving rate of root growth, placement of inoculum in relation to the host, speed of germination, environment, as well as inoculum density as demonstrated by Griffin (31). The models for moving infection courts (9) simplify this situation by treating only the points of penetration. Thus, alterations of host geometry are automatically accounted for.

As developed later, the reasoning used by us and/or by the critics (29,33,34,45,75) to decide whether to treat the penetration of a host barrier as a point or whether the geometry is altered significantly by slight changes in dimension in modeling is empirical. In biology, such intuitive processes must be tested by experimentation. The passage concerning points (above), however, provides a basis for this assumption as a first approach.

Vanderplank (75) and Gilligan (29) also state that the models do not allow for competition between propagules for a restricted number of susceptible sites on target organs. This is true. Competition for susceptible sites occurs in the transitional and plateau portions of the ID-D curve (Fig. 1). It has been emphasized repeatedly (eg, 7,8) that the models only describe the ascending portion of the curve when independent action of propagules (27) operates. As developed later, data points used by us were carefully chosen to insure that they were not on the transitional or plateau portion of the curves.

The conceptual model and the value of *w*. It is not the purpose of this letter to provide a critical review of the alternative models of Grogen et al (33) and Gilligan (29). Their validity will be tested through experimentation by research workers in the future. Here, we discuss ecological and epidemiological considerations necessary for their development in theory expansion.

During the last three decades, the conceptions of ecological interactions between soilborne pathogens and infection courts of their hosts have been developed through massive research contributions. These concepts have been reviewed and described in many books, symposium papers, and review articles (eg, 16,21,28,48,71). The principles developed suggest that soil alone cannot provide carbon substrates necessary for germination and penetration of a host (70) by most soilborne pathogens. Substrates are provided by host organs developing below the soil surface. Thus, the concept of competent distance or volume (34), which appears to apply only to sclerotial units, has limited application.

In the systems of Grogan et al (34) and Gilligan (29), all the propagules in a volume of soil are considered as participating and are successful in the infection process. In this simple form, as inoculum density increases, the number of infections increase in direct and equal increments. This takes the form of the relationship illustrated in Fig. 2B, line a. Grogan et al (34), however, do not consider the efficiency of the propagules in inducing infection and Gilligan (29), without citing evidence, arbitrarily assigns a 50% value. Our preliminary calculations from the data provided by Benson and Baker (13) suggest that efficiency of propagules of *R. solani* is below 10%. Without knowledge of efficiency, w (width of the rhizosphere) cannot be calculated.

Simplifying assumptions may be valuable in modeling such relationships but it is also necessary to consider the dimensions and configuration of the infection court. For example, Gilligan (29) considers the entire root cylinder as susceptible to penetration; this may or may not be true depending on the disease system (9). Even if the entire root is susceptible, the growth of the organ as it moves through the soil triggers germination of propagules, resulting in successful infection, lysis, or other alterations. Thus, there may be none or a lower number of propagules under the influence of the more mature sections of the root which often exude lower amounts of exudates than do root tips (61). The concepts of Grogan et al (34) and Gilligan (29) were involved early on in the construction of the models (9) but were considered inadequate due to these and other biological and geometrical factors (including displacement of the propagules as they contact the root). Thus, the concept of the moving infection court was developed. In this, only the points at which infection will occur on the root were considered from a mathematical standpoint. This has been modeled by Bloomberg (15). This certainly conforms to the biology of the situation and allows for the efficiency component. Efficiency is measured by the position of the log ID-log infections relationship as determined graphically in regression analysis.

Gilligan (29) uses the data of Rouse and Baker (60) to calculate the value of w assuming that the spermosphere is a sphere encircling the seed with an equal radius value in all planes. The pathogen, *R.*

solani, grows through soil approximately twice as fast in a horizontal as in a vertical plane (14). Thus, the spermosphere should be in the shape of an ellipse in cross section. We have confirmed this geometrical configuration through observations of propagules of *R. solani* growing in the vicinity of a radish seed. Thus, w is different for every given plane about a seed for this type of host-pathogen interaction.

The principle is clear: conceptualizations provide a starting point, but the concrete biology and efficiencies of the host-pathogen interaction must be integrated into the system for construction of a valid model. "The more the marble wastes, the more the statue grows" (Michelangelo).

Criticisms of the mathematics developed by Leonard. Leonard (45) endorses the empirical attacks on the models by Vanderplank (75), Gilligan (29), and Grogan et al (34) as adequate "common sense approaches" if it were "not for the mathematical problem."

If the rhizoplane has volume, there is no question regarding the slope value of the log ID-log infections relationship; its value is 1.0 as developed in Fig. 2A–C. If infection on a rhizoplane (in a concrete natural system) occurs at a point, as developed above, then the log ID-log infection relationships slope value should be 0.67 (9). Although these calculations were used in physical chemistry for similar relationships on a molecular basis (36), Leonard (45) contends that the mathematics we used (9) are invalid. Our calculations were not new, only an application of the basic relationships discovered in antiquity between volume and surface area. Arrhenius (3) used these relationships in his idea of panspermia and more recently it has been used in modeling fungal growth (58,59,73). The basic mathematical approach, therefore, is well established in the physical and biological sciences and it is surprising that it is questioned. However, we contribute a defense of our application of the principle in detail:

The number of spores, N, in a cylinder of radius, x, surrounding a root of radius, r, and length, L, is as Leonard (45) stated, $I(\pi x^2 L - \pi r^2 L)$ in which I is the spore density. The result of differentiating this expression may be considered to be either the derivative, $dN/dx = 2I\pi xL$, or the differential, $dN = 2I\pi xLdx$. Leonard gave the result as $2I\pi xL$ (ie, the derivative). The expression, dN/dx, represents the rate of accumulation of spores into a theoretically expanding cylinder around the root (the rhizosphere) at a radial distance of x per unit increase of this radial distance. At $x = r$, the expression represents the rate of accumulation of spores into the rhizosphere at the root surface per unit of increase of the radius of the rhizosphere. For example, $x = r$ the rate could be 1,000 spores per centimeter, which is 100,000 spores per meter and 100 spores per millimeter. Clearly, this rate (number of spores per unit length) is not the number of spores impinging on the root surface. In contrast, the differential, $dN = 2I\pi xLdx$, represents the number of spores in a cylindrical shell at a radial distance of x and having an infinitesimal thickness of dx. However, dN is infinitesimal in the same sense that dx is infinitesimal. As dx approaches zero, so does dN. Thus, the limit of $dN = 2I\pi xLdx$, as dx approaches zero, is zero and not $2I\pi xL$. The limit of the differential, dN, at $x = r$ as dx nears zero does not represent the number of spores impinging on the root surface. If it did, the number of such spores would be zero. For these reasons, therefore, Leonard did not demonstrate that the number of spores impinging on the root surface of a cylindrical root is proportional to the spore density, I.

Leonard (45) pointed out that the demonstration by Baker et al (9) that surface density, S, is proportional to the 2/3 power of volume density, I, was based solely on the spatial distance between the points (inoculum propagules) of a tetrahedral density distribution and thus omitted, at least explicitly, a consideration of the surface dimensions of the root.

The 2/3 power relationship between surface density and volume density is a characteristic of density per se and not peculiar to a tetrahedral or other distribution. That surface density and volume density are inversely related to the square and the cube of the distance between density points, respectively, holds for a regular as well as a random distribution and, consequently, can be applied to the average distance between density points.

In concept, volume density is expressed as a ratio based on a uniform distribution such that the density of a medium (a number per unit cube) times any volume of the medium (in unit cubes) equals the total number for that volume. This implies that the surface (area) density of the medium (a number per unit square) is equal to the 2/3 power of the volume density. In other words, the number of particles in the surface array on the face of a cube is equal to the 2/3 power of the number of particles in a uniform cubic array within the cube. This is simply a corollary of our concepts of area and volume which are, respectively, concepts of standard squares and standard cubes.

A distribution need only be approximately, not absolutely, uniform and cubic for application of the concepts of volume and surface densities. Otherwise, such concepts would have no application to the physical world. Furthermore, models in epidemiology deal with populations, not individuals.

An example of a volume:surface density relationship in which the surface contour is clearly not the determining factor is that of a metal coat hanger or other shaped surface dipped into a can of paint. The volume density of the pigment particles and not the shape of the hanger determines the number of pigment particles per surface area (ie, the surface density) on the hanger.

This example is appropriate because the root surface is relatively large in comparison to the distance between spores, and the average distance between randomly distributed spores becomes constant with only a few measurements (Fig. 4). Also, since the concept of density is applied in relationship to whole plants and not to sections of roots, the surface considered is the aggregate host surface exposed to soil and the volume considered is that surrounding the system and not that of a single section.

In a somewhat similar solution of a problem that confirms our mathematics, Gyani (36) has proposed that the 1, 2/3, and 1/3 power relationships observed in adsorption phenomena reflect volume:volume, surface:volume, and linear:volume relationships, respectively, at the molecular level. Analogously, if the 1 and 2/3 power relationships of infection and inoculum density represent a rhizosphere and a rhizoplane, respectively, then one could conjecture that a 1/3 power relationship would be found if the pathogen required a wound to penetrate and linear fissures were present on the root surface; eg, due to mechanical injury.

Leonard's argument that the arc of a curved figure conforms to the distance between density points only at infinitesimally small values of arc and density interpoint distance is contrary to the applicability of the concept of density to curved figures in general and not peculiar to our application of it to plant pathology. It is not an argument apropos to the 2/3 power relationship. The conclusion of the argument, as presented, would be that the concept of density cannot be applied to curved figures unless the density field is a continuum rather than a distribution of points. A distribution of 1 mm distance between points corresponds to a surface density of 10^6 points per square meter and 10^9 points per cubic meter. If his argument is accepted, one would conclude for this density field that a cube 1 m on a side would have 6×10^6 points on its surface, but one could not conclude that a sphere having the same surface area of 6 m^2 would have the same number of points (6×10^6) on its surface. There is no such thing in the real world as an absolutely continuous density. Air and lead, for example, are not absolute continua. Thus, only noncurved figures could be considered in density phenomena.

The ultimate test of experimentation. Kranz (42) provides an excellent summary of the ultimate criteria for testing the validity of models developed for epidemiology: "a model is, in any case, an abstraction of the real world, a simplified approximation to reality (or parts of it) but by no means the reality itself or its replica. This implies that a model is rarely complete, final and an objective in itself. Every model is based on previous experience or experiments and must be verified again and improved by experimentation." Thus, all the empirical reasonings used by the critics (29,33,34,45,75) or by us are trifles if not experimentally verified. One who models to the exclusion of all else can only dream of the natural world.

Is the 0.67 (log-log) slope found in the literature for ID-D curves obtained by experimentation? A review of all appropriate data

published before the models were constructed (7) suggested slope values (log-log) were near 0.67 for club root of crucifers and Fusarium root rot of beans. Since the models were fashioned, slope values for log ID-log infections relationships near 0.67 have been reported by members of our group (35,60) and by others (17,37,41,50,51,68,69). Whereas we do not consider that the evidence for verification of the models predicting slope values of 0.67 is complete, actual experimentation suggests that the hypothesis should not be abandoned.

Leonard (45) admits that "the experimental evidence shows...in many cases the slopes of the log-log plots are near 0.67." The critics offer no explanation for this phenomenon except that "additional increments of inoculum from the plateau portion of the curve included in the regression analyses [would decrease the slope value] to 0.67 or lower" (33). As treated in this letter and in our other publications (eg, 7–9, 35,60), this concern is ever present.

Until recently there had been no objective means for determining whether data points lie on the transitional or plateau portions of the ID-D curve. Points usually are selected at relatively low values of disease incidence or severity to insure a high probability that they lie on the ascending portion of the curve and also to afford confidence in the successful operation of the multiple infection correction. At least two methods may now be advanced, however, for an objective analysis to determine whether data points derived from experimentation may be used with confidence: (i) regression analysis yielding slope values near those predicted by the models whether a few (near the origin) or many more experimentally derived data points are used, and (ii) demonstration that a slope value (log-log) of 1, which by definition should not include data points on the transitional or plateau portions of the ID-D curve, can be reduced in the same disease system with the same inoculum levels to a value of 0.67.

Regarding method i: articles with relatively high numbers of experimentally derived data points for ID-D curves are becoming available. For example, Stasz and Harman (68) exposed resistant or susceptible pea seeds to 15–35 inoculum levels of *Pythium ultimum*. Slopes of regression lines in all these tests did not differ significantly from 0.67 (log-log). The slope of 0.67 (log-log) predicts a curve when DI corrected for multiple infections ($1/[1-y]$) is plotted as a function of ID (nontransformed). A curve is generated no matter how many data points are used; slope values are near 0.67 (log-log) whether four, five, six, seven, or nine points are used in regression (Fig. 5). It is not likely that the slope value near 0.67 (log-log) was generated by incorporation of points from the transitional or plateau portions of the ID-D curve.

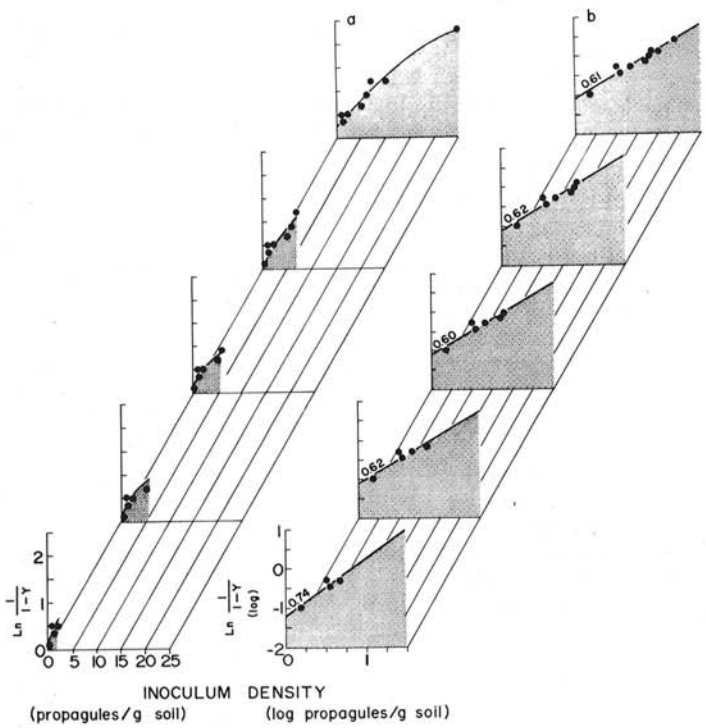

Fig. 5. Typical inoculum density-disease relationships accumulated by Stasz and Harman (68) for *Pythium ultimum* inducing rot of pea seeds using all points available below 90% disease. **A,** Curves resulting from best fit in the semilog transformation for the first four, five, six, seven, and total number (nine) of data points. **B,** Slope values (log-log transformation) using the data points in a are shown on the regression line.

Regarding method ii: Rouse and Baker (60) exposed radish seeds to various ID of *R. solani*. In unamended raw soil, slope values were near 1 (log-log) as has been the case in other published reports (7,78). This suggests that inoculum functions in the spermosphere. This is also the slope value that should be generated in the systems of Gilligan (29), Leonard (45), and Grogan et al (34). If data points had been included from the transitional or plateau portions of the ID-D curve, the slope value (log-log) should have been <1.0 whatever system was used. When cellulose was added to soil, the slope value of the log ID-log infection curve was reduced to 0.61—not significantly different from 0.67 (60, Fig. 2)—and the same inoculum levels were used as in raw soil. Also, at all inoculum levels, DI was reduced in cellulose-amended soil compared to raw soil. Thus, if anything, less inoculum participated in the infection process in the soil treated with cellulose. All these considerations demonstrate that data points generating the slope value of 0.61 could not have been on the transitional or the plateau portion of the ID-D curve obtained for soil amended with cellulose if points were not on this portion of the curve when the experiment was done in unamended soil.

The models discussed above are still considered by us to be in the hypothesis category, but considering the factors elaborated above we submit that the evidence does not permit the conclusion that the models should be abandoned. Rather, the experimental evidence suggests that they may be valid for a number of systems involving soilborne pathogens if the essence of experimental application is within the frame of the models. When they were first conceived (9), they were diffidently advanced as abstractions of the geometrical "considerations" encountered by soilborne pathogens and their hosts. They were unproved hypotheses which, if valid, might have some application in basic concepts. Since that time, however, they have been used for practical applications, for example, in the precise comparative quantitative analysis of relationships involving biological control through organic amendments to soil (35,60), mechanisms involving disease control using pathogen-suppressive soils (78), and survival and pathogenic activity of inoculum in storage (41).

The truth of the matter is that nothing that is modeled by humans is ever exactly right. It is partly our fate and partly our fault. There is only one consolation: whenever human construction falls short of reality, reality always wins out in the long run.

Over 80 yr ago, Erwin F. Smith (64) became embroiled in forensic biology. We advance one of his statements as appropriate even today: "The courteous reader is also requested to examine into these diseases and repeat my observations and experiments and those of seven other people whose writings are mentioned by me as worthy of consideration...."

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