Monocyclic and Polycyclic Root Diseases: Distinguishing Between the Nature of the Disease Cycle and the Shape of the Disease Progress Curve

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In the recent literature on the quantitative epidemiology of plant diseases incited by soilborne pathogens, considerable discussion centers on the analysis of disease progress curves. Primary to those discussions is the choice of appropriate models to describe disease progress, and the significance attached to them (7,8,11). Madden (11) notes that models describing disease progress curves can be "statistical" or "biological." In the former, a mathematical expression which adequately describes the observed disease progress data is formulated, but no clear-cut biological interpretations are drawn from or integrated into the model. In biological models, the mathematical expression describing disease progress is formulated to reflect biological processes which are theorized to have predictable effects on disease progress. Although each type of model has inherent advantages and drawbacks (11), biological models may have a particular pitfall which has sometimes been overlooked in the recent literature, especially that on root diseases: the inappropriate use of them to infer specific biological processes from disease progress data. Investigators have sometimes failed to recognize that knowledge about certain biological processes is to be used as a *basis* for constructing the biological models used to analyze disease progress data; these biological processes cannot be properly *deduced* from observed disease progress data by reference to a biological model. This inappropriate use of biological disease progress models appears in some recent literature concerning diseases incited by soilborne pathogens. The problem centers on the use of Vanderplank's (15) terms "simple interest disease" (SID) and "compound interest disease" (CID).

In Vanderplank's treatment of epidemics (15), he distinguishes between two types of diseases, based on the disease cycle involved. He uses CID to describe diseases in which infected plants serve as inoculum sources for additional infections during a single growing season (the "pathogen moves from lesion to lesion or plant to plant"); in such an epidemic, the rate of disease increase is dependent on the proportion of disease present. He designated as SID those diseases in which the only source of inoculum in a single season is some reservoir other than the plants in the population ("one plant does not infect another in the same season"); in this type of epidemic, the rate of disease increase is independent of the proportion of disease present. In both cases, the rate of disease increase slows as the availability of healthy tissue decreases during the course of the epidemic. Thus, the SID and CID models are biological models of disease progress, describing monocyclic and polycyclic diseases, respectively. The mathematical statement of the CID model is $dx/dt = rx(1-x)$; the statement of the SID model is $dx/dt = r'(1-x)$. In these expressions, r and r' = apparent infection rates (15), x = proportion of disease, and t = time. These two expressions are known as the logistic and monomolecular equations, respectively. Both models describe disease progressions for which curved graphs represent the proportion of disease plotted against time. After integration of the above expressions with respect to time, the resulting equations may be transformed to the following linear expressions: $\ln [x/(1-x)] = rt + C$ for the CID model, and $\ln [1/(1-x)] = r't + C$ for the SID model (17), in which C denotes the integration constant. Thus, if the apparent infection rate is constant during the course of an epidemic, the plot of $\ln [x/(1-x)]$ vs time is linear when $dx/dt = rx(1-x)$, and the plot of $\ln [1/(1-x)]$ vs time is linear when $dx/dt = r'(1-x)$. The slope of the line is the apparent infection rate in either case. In Vanderplank's treatment, then, the apparent infection rate of a polycyclic disease (CID) can be determined by plotting $\ln [x/(1-x)]$ vs time, and the apparent infection rate of a monocyclic disease (SID) can be determined by plotting $\ln [1/(1-x)]$ vs time. Note that the choice of model (CID or SID) is based on prior knowledge of the disease cycle. Vanderplank (15) points out that the apparent infection rate does not necessarily remain constant over time; therefore, the line formed by plotting $\ln [x/(1-x)]$ vs time for a CID, or the line formed by plotting $\ln [1/(1-x)]$ vs time for an SID, will not necessarily be linear, but will change in slope as the infection rate changes during the growing season in response to such variable factors as environmental conditions and host susceptibility. In other words, the observed disease progress curve, with its varying infection rate, will not necessarily be described by the monomolecular or logistic equations (which have a constant r) for monocyclic and polycyclic diseases, respectively.

The notion that diseases incited by soilborne pathogens are SID (monocyclic) has become established in the literature, as noted by Campbell and Powell (4). This is probably the result of Vanderplank's use of Fusarium wilt as an example of an SID (15). Vanderplank did not, however, state a general correlation between SID and soilborne pathogens. (He also used tomato spotted wilt and common bunt of wheat as examples of SID.) Bald (1) stated that "During one experiment, one season, or the growth of an annual crop, most soilborne pathogens cause 'simple interest' disease," but he offered no experimental evidence to support this statement. In recent years, several investigators have examined the relationship between SID and soilborne pathogens. Often the approach has been to infer the nature of the disease cycle, CID (polycyclic) or SID (monocyclic), from the observed disease progress data by reference to one of Vanderplank's models. In some cases, investigators have measured the increase in disease over time, then determined whether the disease progress data are described by the logistic equation or the monomolecular equation. This determination is made by one of several methods: comparison of the observed data with curves fitting one of the two equations (16), significant linear fit of the data to one of Vanderplank's transformations (4,14), and/or value of the "shape" parameter of the Weibull distribution function fitted to the data (3,4,14). With this approach, some diseases incited by soilborne pathogens have been labeled SID (16) while others have been labeled CID (14), both (4), or neither (3).

This approach, however, suffers from the error mentioned above, namely, that the nature of the disease cycle is being inferred from the disease progress curve. The nature of the disease cycle must be determined before an appropriate model can be chosen. For example, whereas it is true that the $\ln [1/(1-x)]$ transformation would linearize data from an SID (monocyclic disease) under specific circumstances (apparent infection rate remains constant over time), it is not true that field data which are linearized by this transformation are necessarily a product of a disease with an

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SID type biology. In fact, it is likely that rate of progress of an SID in a field will be modified by environment (2,10), host susceptibility (13), or other factors over the course of the season and will thus be poorly described by the monomolecular equation. Changes in environment and host susceptibility may be particularly important in determining the shape of the disease progress curve if "amount of disease" is measured as the number of plants showing advanced symptoms of disease, such as wilting or death (4,9), which may be even more subject to environment and host factors than is infection alone. Hence, when field disease progress data are analyzed to determine whether the transformation $\ln[1/(1-x)]$ or $\ln[x/(1-x)]$ gives the best linear fit, and on that basis the disease is concluded to be SID or CID, the variable influence of environment, host susceptibility, and other factors on infection rate and disease development over the course of the season are in effect integrated with and ascribed to basic disease cycle attributes (presence or absence of secondary infection cycles). Such conclusions may be incorrect. For example, Crowe and Hall (5) determined by direct observation that plant-to-plant spread of *Sclerotium cepivorum* occurs in stands of garlic. Thus, white rot of garlic is a CID, sensu Vanderplank. Yet linear regression analysis (*unpublished*) of the tabulated data of Crowe et al (6) indicates that disease progress data were better linearized by the SID transformation than by the CID transformation at the inoculum level of 6×10^{-2} sclerotia per gram. At the lower inoculum level of 7×10^{-3} sclerotia per gram, R^2 values and the pattern of residuals indicate that the CID transformation gives a slightly better linear fit than does the SID transformation. Inoculum level was an important determinant of disease progress curve shape, perhaps because most plants in a highly infested soil were infected before roots had grown enough to permit root-to-root spread of the pathogen. Thus, if conclusions had been drawn from the disease progress data concerning the type of disease cycle, they would have been equivocal or misleading. In a recent report on common root rot of wheat, Stack (14) presented data concerning the increase in disease severity (proportion of crown area showing symptoms) over time. By analyzing the disease progress data for conformity to one of Vanderplank's two models, he concluded that this disease is CID; ie, that secondary infection cycles occur. He discussed the possibility that the shape of disease progress curves can be determined by environmental or host factors, but concluded that these factors were not operative in the disease progress curves he observed, since the curves for different cultivars were not similar in the timing of the curves' inflection points. From the data presented, however, it is not possible to rule out that an environment-host genotype interaction had influenced the point of upward inflection of the disease progress curve, which he concludes to be an indication of secondary infection cycles. If common root rot is an SID, this observed increase in the rate of disease progress partway through the season could result from an increase in host susceptibility whose timing may be uniquely determined for each host genotype under the particular environmental conditions of the test.

Observation of the rate of disease increase in a field is important as basic information and as a tool in evaluating disease control strategies. Numerous quantitative summaries or expressions of this rate, certainly not limited to monomolecular and logistic (4,7,8,11), are available to facilitate comparison among epidemics. Such expressions may arise from either statistical or biological models. But to draw conclusions concerning the underlying biology of the disease cycle from such expressions is not tenable. Pielou (12, pages 46–53), concluding a discussion of population growth curves, notes that "chance variations inevitably cause discrepancies between what actually happens and what had been expected, on theoretical grounds, to happen. Consequently. . .it is rarely, if ever, possible to argue backward from a growth curve to a model."

Knowledge about the ability of a soilborne pathogen to spread from plant to plant during the season, ie, knowledge about the nature of a disease cycle, is useful. This information can best be gained, however, from experiments designed specifically to investigate the existence of secondary infection cycles, rather than from observations of disease progress over time, in which the effects of the disease cycle are confounded with those of such factors as environment and host susceptibility on infection rate over the season. Once information concerning the biology of the disease cycle as such is obtained, it can be used properly in constructing biological models for the analysis of disease progress data.

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