Principles and Practice of
Managing Soilborne Plant Pathogens

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Chapter 2

CHALLENGES, OPPORTUNITIES, AND OBLIGATIONS IN ROOT DISEASE EPIDEMIOLOGY AND MANAGEMENT

C.L. Campbell and D.A. Neher

Root diseases cause extensive damage to crops and forest trees and result in significant losses in the production of food, forage and fiber worldwide. This damage is often not recognized because of the unseen nature of many root diseases and the fact that the visible foliar symptoms often belie the true nature of the damage that occurs below ground. Also, perhaps due to the circumstance that root diseases often remain the "hidden enemy" and because of the complexity and challenges of working with such diseases, losses in potential production due to root diseases remain largely unquantified.

The habitat in which roots grow is quite complex and provides significant challenges to researchers who delve into the mysteries of soilborne pathogens and root diseases. Within the physically and chemically variable milieu of soil, potential root pathogens survive and grow in competition with the myriad other microbes of the soil food web. Competition for nutrients, especially nitrogen, is intense and as roots grow through soil, they provide a primary source of nutrients through exudation, damage to fragile epidermal cells, and the sloughing of dead cells. Root turnover also contributes to the pool of scarcely available nutrients. Parasitism, predation, and omnivory are common in the soil food web. Some microbes survive well as saprophytes, only becoming root pathogens when specific opportunities arise. The soil environment, although seemingly well buffered, shifts continuously to favor one group of organisms or another within microsites on and between soil particles.

Given the complexity of soil ecosystems and root disease epidemics, substantial progress has been achieved in understanding the epidemiology of root diseases and in providing effective strategies and practices for their management (22). There remains, however, much to be accomplished,

and, as the true significance of roots diseases in reducing food, forage and fiber production is recognized, there will be an even greater demand for practical, economical and environmentally safe management options for root diseases. If epidemiology is to set the strategy for disease management (106), root disease epidemiologists are faced with a number of challenges, opportunities and obligations.

Our goal in this chapter is to identify some of these challenges, opportunities and obligations that face root disease epidemiologists. Through this process, we seek to compel our colleagues (and ourselves!) to continue to think about root diseases with a critical but innovative view. We also hope to entice new researchers to join the discipline of root disease epidemiology as they perceive the possibilities for advancing the science and solving meaningful problems.

CHALLENGES

Researchers continue to be challenged by many aspects of the ecology of soilborne pathogens and the epidemiology of root diseases because each pathosystem presents its unique challenges. In this section, we concentrate on three challenges applicable to many root disease systems: quantifying inoculum, assessing disease, and designing effective studies. Others will certainly wish to add to the list; however, if researchers will accept and resolve the challenges presented, we will have made tremendous progress!

Quantifying Inoculum

Propagules of soilborne pathogens are associated intimately with soil particles, organic residues, and other organisms in soil. Successful quantification of inoculum requires the initial separation, isolation or selection of propagules that will be effective in infecting host roots from the associated soil, organic matter and soil biota. The propagules must then be captured physically and identified. Propagules can be identified and quantified visually, or through a species-specific assay, or by growth on a culture medium by morphological, biochemical, or microscopic criteria. Finally, to quantify effective inoculum density in the soil ecosystem in which the propagules reside, there must be some mechanism to determine what proportion of the propagules obtained are viable and capable of infecting host roots. Ideally, there should also be means of determining what proportion of the potentially effective propagules have been recovered from the soil and how representative the soil sample is for

the area (e.g. plot, field, county or state) of interest. The process is not a simple task and as Benson (12) indicates, a large proportion of a research budget must often be expended for materials and time to assay inoculum in soil.

Many methods for quantifying inoculum of soilborne plant pathogens are available (12,98). The primary methods include direct counts, often after soil sieving (9,10,65), bioassays (10,89), and soil assays with baits or selective or semiselective media (25,51,98), enzyme-linked immunosorbent assay (ELISA) in commercial kits (3,62,67,96,101), and ELISA with monoclonal antibodies (44,100). Presence of a fungal pathogen in soil can also be determined through substrate colonization (68,85,102) or through the use of amplification procedures and species-specific DNA probes (19,20,43).

Which method is "best" for quantifying inoculum must be judged in relation to the original purpose for which it was developed and the purpose for which the method is to be used. Regardless of the method selected, there should be a critical assessment of the efficacy of that method prior to its use. Some comparative studies on the reliability (i.e. relative ability to identify the target pathogen correctly), precision (i.e. relative ability to provide the same result when the assay is performed repeatedly for a given soil sample), accuracy (i.e. the closeness of the value obtained to the true value) and efficiency (i.e. the cost per unit of information obtained) of some assays for quantifying inoculum have been performed (28,58,80,86,90,99). Data related to inoculum quantification is important in understanding the epidemiology of root diseases; more studies on inoculum quantification are needed.

The statistical issues of sampling, coupled with the biological reality of spatially aggregated propagules, often pose an apparent dilemma in quantifying the inoculum of soilborne pathogens. Often an investigator cannot obtain and assay a sufficient number of samples to obtain data with the desired degree of precision, especially for inoculum that is highly aggregated or clustered. Aggregation generally increases sample variance compared to a situation where propagules have a random or uniform spatial pattern. Yet, funds are often not available to conduct all of the assays required to obtain the desired degree of precision. Reasons for this include: (i) the relative expense (in terms of material and personnel costs) of most assays; (ii) the overwhelming desire to use a small number of samples to represent a relatively large area; and (iii) the physical, chemical and biological variation that occurs naturally in soil. One resolution to the dilemma, which is invoked all too often, is for the investigator simply to decide how much time and money can be spent on

assays, calculate the time and cost per assay and use the quotient of the two quantities to provide the sample number without due consideration of the statistical consequences. A better solution is to perform the needed preliminary studies with the target organism and intended assay to quantify the components of variance associated with the various sampling and assay procedures and then to calculate an optimum sample allocation plan and sample size (28,79). Even with this procedure, it may not be possible to obtain the optimum number of samples; however, at least an informed decision on resource allocation can be made prior to the actual study to quantify propagules.

The degree of success we achieve in relating data on inoculum density to other components of the epidemic depends primarily on the quality of the data obtained (21). As a result, a quality assurance plan should be developed for each study that will involve inoculum quantification. A quality assurance plan will probably include, whenever possible, the inclusion of the assay of a certain number of "known" samples (or calibration standards) during the actual performance of the assay to insure that all procedures are being followed and performed correctly. If a sample from a reservoir of soil with a known inoculum density is included in each "batch" of soil samples, the same propagule number (within whatever limits of measurement error that are established) should be obtained each time.

To meet the challenges of quantifying inoculum from soil, answers to a series of questions should be obtained prior to the collection of data with a particular method. Such questions include:

- How reliable, precise, accurate and efficient is the proposed assay?
- What are the critical steps and likely sources of error in performing the proposed assay?
- What allocation of resources during sampling and assay performance will provide the best quality data?
- What quality assurance procedures are in place to insure that the data are of the best quality possible?

Assessing Disease

Disease assessment is one of the most important and often most challenging tasks in the study of plant diseases. With root diseases, assessment is even more of a challenge than with most foliar diseases, because the host parts on which we desire to assess disease, i.e. roots and other subterranean plant parts, are "hidden" in the soil. This means that

symptoms, and even the extent of the host tissue to be assessed, can not be evaluated readily on roots. Additional or alternative steps must be taken to complete disease assessment.

Whether roots or shoots are the appropriate host part to be sampled and evaluated will often depend on the purpose of the assessment. If the purpose is to describe temporal progress of the disease on roots, then roots are the logical plant part for evaluation. If the purpose is to relate disease severity or incidence to yield, then the portion of the plant harvested for yield will be a determining factor in selecting the plant part to be evaluated. For example, with soil rot or pox (*Streptomyces ipomoea*) of sweet potato, severity of symptoms on the fleshy storage roots will be related directly to yield quality and quantity, whereas severity of symptoms on fibrous roots may be related only indirectly to yield (92). In contrast, when fruit are harvested from the above-ground plant parts, the severity of symptoms on shoots may influence yield more than the severity of root symptoms as in the case of Phytophthora root rot of processing tomato (78).

Various procedures or alternatives are available for the assessment of root or shoot symptoms associated with root diseases. We have presented specific methods for estimating severity and incidence of root diseases previously (27) and will not further discuss those methods here. Rather, the challenge to be considered here is how to choose and observe the most representative and meaningful sample of plant material for disease assessment while causing the least possible disturbance to the epidemic and the pathosystem. Three specific options have been utilized by root disease epidemiologists as a surrogate for assessment of root disease: in situ observation of roots; removal of plants (and roots) from soil; and assessment of foliar or shoot symptoms.

Rhizotrons or root observations boxes or tubes can be constructed and placed in the field (17,52,60,61,103,104). This option can be quite effective for a small sample of plants but is impractical and cost-prohibitive for large areas of fields or with many fields. Additionally, there is some disturbance of the soil system with placement of the observation ports and, if glass or plastic surfaces are used for observation ports, a modified environment is created for those roots and organisms being observed.

Plants can be excavated and excess soil removed so that the roots are exposed for assessment (26,29,52,77,93). This option requires that the sample unit be destroyed during the assessment, thereby eliminating the possibility of repeated observation on the same sample unit or plant. Also, it is labor intensive (particularly for larger annual plants and

certainly for many large perennials!), causes significant disturbances in the soil ecosystem and limits the number of times assessments can be made, particularly if yield data are required from the same study. Another challenge with root excavation for disease assessment is that those roots which have the greatest amount of disease may actually be sloughed prior to sampling or lost during the removal process due to their weakened physical structure. Because of this likely loss of severely diseased roots and the possibility that an important symptom is the stunting of the root system, disease assessments should be made in comparison to a healthy root system. Assessment of root area or volume in relation to healthy plants may be as significant a portion of a disease assessment as the determination or estimation of the area or volume of root tissue occupied by lesions.

A third option is to assess shoot symptoms that develop as a result of the root disease. This option is the least labor intensive and results in the least disturbance to the soil ecosystem. Although this approach has been used successfully in some pathosystems (24,31,50,53,94,95), it also results in assessment data that are not necessarily representative of the true progress of the root disease. Rather, root disease probably develops to a certain stage, which may be dependent on weather conditions, before any visible shoot symptoms are apparent (78). Inference of the extent of root disease by observing only foliar symptoms such as yellowing, epinasty, chlorosis or wilting should be done with caution (47). In some diseases for which foliar or shoot wilting occurs rapidly after root infection, e.g. Phymatotrichum root rot of cotton (50), evaluation of foliar symptoms may be quite appropriate. With other host plants, however, foliar symptoms may not be sufficiently sensitive to reflect actual damage occurring on roots or the appearance and severity of symptoms may be confounded with factors such as weather and host genotype.

Another challenging aspect of assessing disease severity on excavated roots is the relative location of the disease within the root system. In order to determine the physiological effects of disease on plants, the position and depth of lesions on roots should be evaluated (47). Morphometric root analysis systems are available (33–36) for the specification of root order (first, second or third) and type (lateral, tap). Lesions on tap roots would likely reduce water flow to stems more than lesions on lateral roots such that shoot symptoms would be present when the tap root has lesions but not when lesions are restricted to lateral roots (52,73).

Finally, the challenge of selecting the most appropriate number of samples to quantify how much disease is present enters into disease

assessment in much the same way it did for quantifying inoculum of soilborne pathogens. The resolution of this issue can be achieved with the procedures identified previously. An added challenge is that if plants are dug so that roots can be examined, adjacent plants must be excluded from future assessments because of the extensive disturbance to the soil ecosystem and possible alteration of plant competition. Thus, studies must be planned so that the number of specified, randomly selected samples can be chosen within the constraint of missing plants and a shrinking population from which to select future samples.

Designing Effective Field Studies

Root diseases pose a special challenge in designing effective field studies, because the initial inoculum is usually present in soil prior to the initiation of host growth or is introduced with the host. This is certainly true for most annual crops and for some perennials. Although the influx of inoculum during the course of a growing season from sources outside a field or adjacent area should not be discounted, compared to the situation with most foliar diseases, the continuous or regular influx of inoculum is a relatively rare event for root diseases. These factors imply that the spatial dimension of inoculum pattern is of primary importance in selecting the initial experimental design for field studies of root disease epidemiology.

Spatial scale is a factor that should receive more critical consideration in the design of field studies for root diseases. The spatial scale of concern often occurs in the horizontal plane of a field. However, the vertical pattern of propagule occurrence, which involves propagule distribution and root growth within the soil profile, has been considered for several pathogens (1,16,18,63,66,72) and should not be ignored.

Ecologists partition spatial scale into extent, the overall area encompassed by a study, and grain, the size of individual units of observation such as quadrats (2,108). Extent and grain define the upper and lower limits of resolution of a study, because inferences about patterns or processes of events cannot be made legitimately beyond the extent or below the grain of a study. Also, because our ability to discern and interpret biological and environmental effects on spatial processes is dependent on extent and grain of the study (83,108), investigators should consider these items in designing field studies carefully.

With agronomic and horticultural crops, field size or farm size may influence root disease epidemics and thus, determine the extent of the epidemic. However, a wide range of extent values have been used by

researchers working with soilborne pathogens and root diseases (23). Other biological and ecological criteria for determining extent will have to be identified for forests, riparian areas, or other natural ecosystems where no specific management practices, such as cultivation, define the boundaries of the ecosystem. The determination of an appropriate grain size for studying a root disease may be more challenging than the determination of an appropriate extent. Investigators have used grain sizes from < 1 to > 100 m^2 (23). Actual grain selection should be based upon: (i) biological factors such as cluster size and potential dispersal distance for a soilborne pathogen; (ii) soil factors such as soil map unit, soil type or obvious physical attributes of an area; and (iii) cultural factors such as row spacing and pattern of cultivation. The determination of grain, therefore, requires a fair amount of prior knowledge about the pathogen, host and the experimental site, including climatic influences.

Another factor of importance in designing field studies for root diseases is that the soil has a successional status and a degree of ecosystem stability that extends among seasons and from year to year. A complex and integrated food web of organisms is present in virtually every soil and soilborne pathogens are only one component in that food web (69). The interactions among pathogens and the other living residents of agricultural, forest, wetland, and prairie soils, particularly the species composition and function of microbes, play a vital role in determining the suppressiveness or conduciveness of soils for the development of root diseases and should be considered in designing field studies (105).

A third factor of importance in designing field studies concerns the physical and chemical properties of the soil itself. These compositional factors can significantly influence the environment to which roots are exposed. Differences in soils within fields and among fields, even in the absence of cultural and biological factors, thus represent a potential variation in environment that can affect the development of root disease epidemics.

A fourth factor of importance is the temporal scale at which the soil environment changes. Because of the chemical buffering capacity and the biological complexity and ecosystem stability of most soils, the soil environment changes rather slowly. However, cultural practices and weather patterns can affect the soil ecosystem among seasons or years. The addition of specific soil amendments and use of crop rotations can influence the soil environment and, thus, conditions for root disease development from year to year (105). In some cases, multiple year studies may be required to characterize the effects of specific treatments

or factors on root disease epidemics.

* * * * *

The challenges we have discussed are fundamental to the science of root disease epidemiology. These challenges in quantifying inoculum, assessing disease and designing effective field studies can be resolved and become successes. In doing so, they will serve to advance both the practical application and the theoretical framework of root disease epidemiology.

These challenges should not be viewed as impediments to research with soilborne pathogens and root diseases. Rather, they should be embraced as aspects of our experimental science that we are attempting to improve continuously. They are aspects of the discipline of root disease epidemiology that should be considered when any study is being planned and implemented. No single study will resolve all the challenges; however, even the consideration of the challenges accompanied with a serious consideration of the question "How can I quantify inoculum, assess disease and design experiments better?" must be viewed as a success.

OPPORTUNITIES

There is a growing cadre of knowledge about the population dynamics and ecology of soilborne pathogens and the epidemiology of root diseases. The epidemiological approach has been used to investigate the temporal and spatial aspects of a range of pathosystems, primarily for annual crops in agronomic and horticultural settings. A cohesive and innovative approach to the theoretical aspects of the temporal and spatial dynamics of soilborne pathogens and root disease epidemics is also developing (37,39,40,42,49).

With the increasing interest among root disease epidemiologists, the knowledge base concerning these diseases and recognition of losses in potential yield caused by root diseases has expanded. Thus, new opportunities for significant contributions to the fundamental and practical understanding of root disease epidemiology become available to researchers. Specific opportunities for innovative approaches arise in subjects such as the modeling of the components of root disease epidemics, forging the linkage between soil ecology and the ecology of root pathogens, comparing and classifying epidemics and setting strategies for disease management. Researchers can take full advantage of such

opportunities, in part, by maintaining a keen awareness of research progress in similar areas with foliar pathosystems and in microbial ecology. However, the greatest progress will be made through the development of innovative approaches and the realization that root disease epidemics may not have the same fundamental components and developmental pathways as foliar epidemics. Because the soil ecosystem is more complex than, and quite different from, the ambient ecosystem of the phyllosphere, and because of the distinct differences in structure and function between roots and leaves or shoots, there may be little reason to expect epidemics of diseases of roots and shoots to develop similarly. If researchers will avail themselves of the opportunities identified for root disease epidemics, we will continue to make progress toward understanding and managing these epidemics.

Modeling Root Disease Components

As Jeger (49) noted, "the modeling of root diseases has received rather less attention than that of foliar diseases". There has, however, been significant progress since that time in defining and modeling the components of root diseases and, in all probability, the seminal work of Gilligan (37–40,42) and Jeger (49) has provided a foundation in modeling and understanding of root disease epidemics that is even more complete and cohesive than that available for foliar diseases. As a result, the opportunities now exist to further explore, via modeling and empirical studies, the roles of primary and secondary infection, of root growth and of inoculum dynamics, including the survival of inoculum and the interactions of pathogens and other microorganisms in soil ecosystems. The empirical data can be evaluated with respect to current models and will serve as a basis for further modeling efforts. The understanding provided by these further modeling and empirical explorations will also present the opportunity to develop more rational and comprehensive strategies for managing root diseases.

Idealized disease progress and inoculum dynamics curves have provided the starting point for much of the mathematical analysis of root disease epidemics. The modeling efforts have been based on the pathogen or the host. Probability models have been proposed for the pathogen-based approach which relate infection to inoculum density in soil (38). Models based strictly on symptom expression on aboveground plant parts have also been proposed (21) (although the possible hazards of employing such models has been alluded to in the section on Challenges – Assessing Disease). For either the pathogen- or host-based approaches, monotonic

curves, in which disease increases progressively toward some upper, asymptotic level, have dominated work in the epidemiology of root diseases (42).

The shapes of cumulative curves of disease intensity for root diseases have been a source of interest to many researchers and have provided insight into the interrelations among the biological components of these diseases. Although the increase of plant disease intensity during epidemics caused by root pathogens often appears sigmoid over time, the expectation is for a monomolecular curve (25,64) to describe an epidemic which is monocyclic or "simple interest" sensu Vanderplank (106). The conclusion of mechanisms of disease increase based solely on the shape of the disease progress curve is inappropriate (25,71,88); however, the hypothesis of mechanisms that result in sigmoidal curves for disease development over time has been the basis of much of the recent mathematical or analytical modeling for root disease systems. Some unusual disease progress forms, such as double sigmoidal (4,45), have been described for foliar diseases but have not yet been reported for root diseases.

Models of cumulative disease curves and essential disease components have been proposed. Jeger (49) explored models based upon symptom expression by above-ground plant parts and then proposed detailed models to combine root growth and increase of lesions on roots with and without lesion expansion on roots. In an elegant and masterful sequence of publications, Gilligan (38,40–42) examined the interactive relationship between host and pathogen for components such as host infection by soilborne fungi, rate of contact of inoculum and roots, the dynamics of inoculum production and survival, the growth of roots, the occurrence of primary infections, the transmission of infection by root-to-root spread of pathogens, the role of root density, death of roots, latent and infectious periods for root pathogens, and antagonistic interactions between pathogens and other microbes in soil.

Presently, the shortage of empirical data imposes practical constraints on the analysis of root disease epidemics. For example, more experimental work is needed on the dynamics of inoculum survival in soil. How long does inoculum survive in soil, how long is it infective, and what do the curves of inoculum density and of infective inoculum density (i.e. those propagules that are actually infective) actually look like—e.g. are they monotonic or cyclic? More studies are needed on the interactive effects of pathogens and roots during epidemics. The density of roots certainly affects the dynamics of infection, but little is known about the effects of disease on root growth (42). Also, the dynamics of infection

of roots is only one component of the overall disease cycle; more information is needed on the length of time between root infection and when the roots become infectious (i.e. the latent period) and how long the roots remain infectious (i.e. the infectious period). The models proposed by Gilligan and Jeger are, thus, an excellent starting point for determining the mechanisms of root disease epidemics; however, there are many excellent opportunities for additional modeling studies and for empirical studies to evaluate the validity of currently proposed models and to serve as the stimulus for new, more comprehensive models.

Forging The Linkage Between Soil Ecology And The Ecology Of Root Pathogens

The beneficial role of nonpathogenic soil invertebrates is largely unexplored by plant pathologists, who usually just consider plant-pathogenic bacteria, fungi, and nematodes in soils. However, the nonpathogenic organisms in soil far outnumber the pathogenic ones! There are, for example, an average of 10^6 to 10^7 free-living nematodes that feed on bacteria, fungi, algae, and other nematodes, 10^4 to 10^5 enchytraeids (pot worms), 10^3 to 10^4 mollusks (slugs, snails), 10^2 to 10^3 myriapods (millipedes, centipedes), 10^2 isopods (wood lice), 10^2 Araneidae (spiders), 10^4 Collembola (springtails), and 10^5 Acranaria (mites) per square meter of soil (87). Microfauna and mesofauna in soils play important roles in decomposition of organic matter and nutrient cycling. Microinvertebrates, such as nematodes (7) and protozoa (109), contribute directly to nitrogen cycling by excreting nitrogenous wastes, which are released mostly as ammonium ions (48). Microinvertebrates also enhance soil fertility directly by depositing feces and existing as a reservoir of nutrients, which are released when they die. Microarthropods contribute to decomposition of organic matter indirectly by fragmenting detritus and increasing surface area for further microbial attack (13). Subsequently, soil invertebrates graze upon microbes, and thereby alter nutrient availability, affect microbial growth and metabolic activities by selective grazing, and alter the composition of microbial communities. In addition, soil fauna also transport bacteria, fungi, and protozoa (in gut or on the cuticle) across regions of soil impenetrable by microbiota, and thus enhance microbial colonization of organic matter (70).

Plant pathologists often look at the negative effects of soil invertebrate mesofauna (14) and do not fully consider the beneficial aspects of these organisms (8). The rhizosphere-inhabiting collembolans, *Proisotoma minuta* and *Onychiurus encarpatus*, graze preferentially upon

the root pathogen, *Rhizoctonia solani*, on cotton seedlings in the presence of three well known biological control fungi, *Laetisaria arvalis, Trichoderma harzianum* and *Gliocladium virens* (32). Giant amoebae of the Vampyrellidae (*Arachnula, Thecamoeba, Saccamoeba, Vampyrella*) perforate conidia of *Cochliobolus sativus*, a fungus causing root rot of barley (82). Orabatid mites prefer feeding on pigmented fungi over nonpigmented fungi, which implies their potential for destroying pigmented pathogens such as *R. solani* and *C. sativus*. Larvae of *Bradysia coprophila* (dark-winged fungus gnat) prefer sclerotia of *Sclerotinia sclerotiorum,* the cause of lettuce drop in muck soils of Quebec, as a food source (5). *Bradysia coprophila* secretes chitinase in its saliva while feeding on sclerotia of *S. sclerotiorum*, thereby disrupting germination of the pathogen (6). The larvae failed to survive when provided the mycoparasitic fungus, *Trichoderma viride*, as a food source (6). Microarthropods create problems as pests usually because a preferred food source is absent. For example, root-grazing injury by species of the collembolan *Onychiurus* on sugar beet is caused by the rubbing of their bristled bodies against the root tissue. However, if certain types of weed species and certain kinds and amounts of organic matter are present providing the preferred microbial food supply, root injury decreases (32). These examples provide evidence that common species of small-animal communities can consume sufficient pathogen inoculum to lower disease incidence and suggest the need for a new look at the mechanisms underlying biological control and soils suppressive to root diseases. A better understanding of the interactions among soil flora and fauna would complement our understanding of root disease epidemics and should lead to a better understanding of the mechanisms of disease management strategies, particularly biological control.

Species composition and function may be more important than species diversity in determining disease suppression (105). Importance of functional groups in relation to disease suppression is exemplified by a positive correlation between suppression of corky root of tomato and the Shannon-Weaver diversity index (97) for functional groups of Actinomycetes isolated from rhizospheres of tomato seedlings grown in organically and conventionally managed soils (110). Disease suppression may depend on communities of microorganisms associated with a specific substrate of a certain quality under certain environmental and management conditions. For example, disease suppression is often enhanced by incorporation of organic amendments in soil. The effectiveness depends on the specific material used, the time elapsed since incorporation, and the pathogen under study. Fresh debris sometimes increases plant disease by

providing a food base for facultative saprophytic pathogens. For example, *R. solani* can utilize cellulose as a sole source of carbon (11), and thus thrives in fresh or immature compost material relatively high in cellulose content. A biological control agent, *Trichoderma* sp., degrades cellulose rapidly (46). However, if cellulose levels are high, free glucose concentrations accumulate and may repress synthesis of chitinase involved in hyperparasitism of *R. solani* by *Trichoderma* sp. (30).

Forging the linkage between soil ecology and the ecology of root pathogens will require that root disease researchers expand their view of the interactions between pathogens and roots. It will necessitate the examination of soil food webs to ascertain the expected or "normal" composition of the soil microflora and micro- and mesofauna for certain types of soil ecosystems in specific areas. It may also require that reference sites of reference systems be defined for comparison. For example, soil ecosystems with perennial hosts such as pasture species may be appropriate reference sites in a region for comparison of the diversity and abundance of soil organisms with those sites with annual hosts such as many agricultural crops (76). Such examinations will be time-consuming and expensive; however, the result of this expanded view will be a much improved understanding of the ecology of soilborne pathogens. This, in turn, will allow a more complete examination of the epidemiology of root diseases.

Comparing And Classifying Epidemics

Epidemiologists compare the progress curves of root diseases over time and the patterns of root disease in space in order to gain fundamental knowledge about the factors that influence the course of disease progress in time and space and, ultimately, to establish strategies for management of plant diseases. Vanderplank (106) identified the need for comparison of disease progress curves and provided an initial, simple framework for making such comparisons over time. He proposed the comparison of disease progress curves based upon two parameters associated with several disease progress models—initial disease and the apparent infection rate. Kranz (54–57) recognized the greater wealth of information contained in disease progress curves and proposed a multivariate approach in establishing the subdiscipline of comparative epidemiology. The recognition of a larger number of parameters available for comparison and the use of multivariate, statistical techniques for epidemic analysis and classification provides a more realistic view of the factors involved in disease progress over time than the simpler, two-parameter approach

proposed by Vanderplank. The goals of comparative epidemiology are thus twofold: (i) to examine the interrelationships among descriptors of epidemics and ascertain a minimum set of descriptors needed to characterize an epidemic; and (ii) to classify epidemics into a number of meaningful and interpretable classes or categories.

Although a few studies are available that apply the methodology of comparative epidemiology to the temporal (24,26,29) and spatial (81) aspects of epidemics caused by soilborne pathogens, there is much more to be learned in this area. Comparative epidemiological studies are needed that address both the temporal and spatial aspects of root diseases. The major challenge in making progress with such studies is the lack of data sets with information on a relatively large number (6 to 10) of observations and characteristics for epidemics of root diseases. The requisite data sets will be relatively expensive and difficult to assemble; however, the opportunities available for real advancement of our knowledge of root disease epidemiology through comparative studies will more than compensate for the costs associated with data acquisition.

An initial benefit of such comparative studies will be a better understanding of the parameters that are needed to characterize root disease epidemics. For example, there should be some degree of similarity in the things which need to be measured to characterize epidemics of wilt diseases caused by soilborne species of *Phytophthora* or *Fusarium* on annual hosts. Once a relatively small number of essential parameters is established for the characterization of certain types of epidemics, the cost and difficulty of data collection should be reduced. The set of essential parameters will, of necessity, contain elements that describe, or are surrogates for, host or root growth, inoculum dynamics, and disease development. Other elements may be needed to describe key ecological and environmental factors in the soil.

Once a fundamental understanding is obtained concerning the parameters that are needed to describe root disease epidemics, specific fundamental and immediately practical questions concerning the development and management of root diseases can be addressed. Questions to be answered might include: are there a limited number of types or categories of root disease epidemics that occur in agricultural ecosystems, and is the epidemic category determined primarily by the type of pathogen, host or soil characteristics? Do epidemics caused by species of *Phytophthora* and *Pythium* on agronomic or horticultural crops have more in common than epidemics caused by species of *Phytophthora* and *Rhizoctonia*? Do epidemics caused by fungi (e.g. *Aphanomyces*) differ from epidemics caused by prokaryotes (e.g. *S. ipomoea*), nematodes (e.g. *Meloidogyne incognita*), or viruses (e.g. soilborne wheat mosaic virus)? Are root disease epidemics of annually harvested, herbaceous crops more similar to each other than to epidemics of perennial crops? Is the type of host plant (e.g. legumes or grasses) more a determinant of epidemic behavior than pathogen type? Do epidemics of annual and perennial crops differ only in the relative time or spatial scale or in other fundamental ways?

Setting Strategies For Disease Management

With regard to disease management, Gilligan (40,42) has proposed a series of three equations, one for infected roots, one for total roots, and one for inoculum, that account for primary and secondary infection of soilborne pathogens with allowance for root and inoculum dynamics. The parameters of the models suggest alternative epidemiological strategies for disease management that can be evaluated and compared among pathosystems. These include (42, p. 158): (i) reduction of inoculum by "removal" of initial inoculum and/or increase in the rate of decay of inoculum; (ii) reduction in the rate of primary infection; (iii) reduction of the rate of secondary infection; (iv) alteration of host density by change in initial host density and/or change in asymptotic root density and/or change in the rate of production of roots.

From his theoretical analysis of the influence of root growth and inoculum density on the dynamics of root disease epidemics, Jeger (49) was able to provide several specific suggestions for root disease management strategies. These suggestions were to reduce pathogen density, to maintain a low rate of root extension relative to root infection, and to restrict lesion expansion. These recommendations are compatible with the epidemiological methods proposed by Gilligan (40,42) and provide a starting point for the empirical evaluation of strategies for root disease management.

Thus, the opportunity exists to evaluate which of these strategies or combination of strategies may be best for managing specific types of root diseases. Comparative epidemiology may allow the extension of such an evaluation to determine if specific strategies or combinations work best for categories of root disease epidemics. The result would be the ability to provide at least an initial prescription of the type of management strategy that may be expected to work the best for a "new" disease without extensive and costly empirical evaluation of each management option. Such a prescription should be derived from the knowledge of the basic set of parameters needed to characterize disease progress temporally and/or

spatially in specific categories of epidemics.

OBLIGATIONS

Root disease epidemiologists have the principal obligation of making the results of their research defensible theoretically and biologically, understandable to others with a reasonable knowledge of ecology and epidemiology, and useful to those who seek to manage root diseases in the real world. The obligations of root disease epidemiologists can be met, in part, through accepting the challenges and taking advantage of the opportunities discussed earlier. These obligations are not met easily and require not only scientific knowledge, insight and integrity, but also perseverance and perhaps some luck.

We propose the following specific obligations or goals for root disease epidemiologists.

1. Establish a sound theoretical framework for understanding root disease epidemics.
2. Present possible strategies for root disease management inherent in an ecologically based framework.
3. Provide a practical, useful framework for describing and analyzing epidemics of root diseases.

Establishing A Sound Theoretical Framework

The mathematical models proposed by Gilligan (38–40,42) and Jeger (49), combined with simulation models such as those of Bloomberg (15) and Reynolds et al. (91), have provided the basis for meeting the first goal. Further empirical work is needed, as noted by Gilligan (42), to discern how infection by soilborne pathogens, and, more importantly, disease, affects root growth so that such effects can be incorporated into mathematical models. Additional empirical and modeling efforts also need to be devoted to the dynamics of survival of soilborne pathogens in soil ecosystems. Studies that examine the survival dynamics of specific propagules of individual pathogens alone will be useful. Studies that examine the dynamics of inoculum in a range of soil ecosystems would, however, be more useful in the long term, because methods to incorporate effects of antagonistic or competitive microorganisms, which also reside in the soil ecosystem with pathogens and roots, must be developed.

There is a need to incorporate spatial aspects of pathogen dynamics and root disease development into the overall theoretical framework for root disease epidemics before it can be considered complete. Spatial

attributes, at several scales from the rhizosphere of a root or whole plant to a focus of disease or a field and even a region, will be important (23). Although the modeling of the temporal attributes of root disease development is a significant challenge in itself, the incorporation of the spatial dimension into root disease models, along with the temporal dimension, will be even more challenging and may even require new approaches.

Presenting Strategies For Root Disease Management

As researchers and growers come to recognize the true significance of root diseases in reducing the yield of plants producing food, forage and fiber, there will be an even greater demand for strategies and practices to manage root diseases effectively and efficiently. Host resistance will answer part of the demand for better management of root diseases; however, there will be a significant need for additional strategies that are effective, economically feasible, and environmentally friendly to promote ecological sustainability in agricultural systems (74). This challenges root disease epidemiologists to broaden their view of soil ecosystems to encompass the view held by soil ecologists. As the ecological "world view" of root disease epidemiologists expands to encompass the many organisms that compose the soil food web, we will become aware of many more natural mechanisms for root disease control accounted for by various nonpathogenic microbes and invertebrates who graze preferentially on pathogenic fungi, bacteria, and nematodes (32,59).

In nature, there is a balance within soil communities between relative abundance of pathogens and nonpathogenic organisms. If the balance is disrupted, for example by use of general biocidal soil fumigants, it is difficult, if not impossible, for soil to regain its original diversity (111). We may benefit from comparative studies of soil communities between natural and agricultural ecosystems for identification of natural enemies, biopesticides, and other means of classical biological control (107). A more comprehensive understanding of the spatio-temporal distribution of microarthropods and microorganisms in soil and of the mechanisms of their interactions (59) is necessary so we can capitalize on these biological mechanisms in disease management. Biologically- and culturally-based strategies need to be explored further for control of root diseases. For example, cultural practices such as cultivation, fertilization, and irrigation influence the abundance and diversity of soil communities (75). The quantity and quality of fertilizer both affect soil communities. For example, composted manure and other organic materials increase the

biodiversity of soils and reduce nutrient leaching more than mineral fertilizers (30,84). We need to understand the effect of various management practices on the balance between beneficial and pathogenic organisms in soils.

Providing A Practical Framework For Monitoring Root Disease Epidemics

The development of a cohesive theory of root disease epidemics and the prescription of management strategies will be significant accomplishments and will require the efforts of many dedicated and innovative scientists over the course of many years. The results can potentially have long-lasting and significant impacts in reducing crop losses to root diseases and in increasing crop yields as part of an approach to developing sustainable agriculture in both the temperate and tropical regions of the world. However, if these results are to have the greatest impact possible, they must be translated into a series of practical, user-friendly steps that will provide an overall procedure to monitor populations of soilborne pathogens and epidemics of root diseases, to analyze the data obtained, and to apply the results to practical management measures.

There are at least five components that epidemiologists are obliged to include in a practical system for analyzing root disease epidemics. Each component adds to the overall completeness of the practical system and serves to provide a sound theoretical basis for the system.

First, protocols must be developed and evaluated for the sampling of soil and roots and the assay of such samples for determining the inoculum density of each of the major soilborne pathogens. Such protocols must include procedures for estimating variability within and among samples and means of assuring the quality of the data obtained. Such procedures should include, as far as possible, mechanisms of determining efficacy of propagules in the particular soil environment and information on specific strains or genotypes of the pathogens.

Second, optimum sampling plans need to be developed for assessing intensity of root diseases during the course of epidemics. Potential benefits and risks of protocols and plans proposed should be presented with regard to factors such as destructive versus nondestructive sampling, assessment of foliar compared to root symptoms, estimation of root biomass or healthy area (volume) duration of roots, and so on. Factors such as costs and variability associated with various sampling components should be included in such plans.

Third, data quality objectives and quality assurance plans should be specified for all data collected while studying root disease epidemics and the inoculum dynamics of associated pathogens. Data quality objectives provide preset limits that researchers seek to achieve during assays for pathogen propagule densities and estimation of disease intensity. Quality assurance plans provide opportunities to insert specific steps in assays and estimation protocols to assess whether such procedures are performed reliably.

Fourth, practical methods need to be developed and publicized for analyzing temporal and spatial pattern data for inoculum dynamics and disease progress. The methods required for such analyses should be made as simple statistically and analytically as possible. The conditions and assumptions under which these analyses can be used should be specified clearly, and any possible hazards associated with the analyses stated clearly and concisely. The availability of such methods would encourage many more researchers to monitor and investigate epidemics of root diseases and would add considerably to the databases available for modeling and for making management decisions.

Finally, practical methods, such as a series of templates or a classification scheme, need to be developed and publicized for comparing epidemics of root disease. Classes of epidemics based upon host or pathogen type would aid researchers and others in prescribing possible management options based upon what was known about the specific class of epidemics.

CONCLUSIONS

Considerable progress has been made in the last 10 years in the understanding of root disease epidemics, yet many relatively basic questions remain unanswered. We have attempted to identify challenges, opportunities and obligations for epidemiologists that will allow our understanding of root diseases to continue to increase in the decades ahead and will allow for the development and implementation of environmentally sound management procedures for root diseases.

The advent of molecular techniques such as PCR and DNA probes provides exciting new opportunities for identifying pathogens in soil; however, the application of such techniques is to date mostly qualitative and must become quantitative and less expensive to be utilized fully in monitoring pathogen dynamics in soils. The use of such techniques which may be much more specific in identifying pathogens than assays on semiselective media, for example, will not, however, eliminate the

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challenges of obtaining representative samples in the field and accounting for the complexities occurring in soil ecosystems.

The availability of faster microcomputers and more powerful software that can handle larger and more complex data sets provides enticing and encouraging challenges and opportunities for root disease epidemiologists. The enticement is to assemble and analyze larger and larger data sets of increasing complexity for the sake of recognition. The encouragement comes in knowing that data sets containing essential data can be analyzed. The hazard, of course, is that in the complexity of the analyses and with the ability to analyze larger and larger data sets, epidemiologists may lose themselves in the analysis, itself, and forget that the original purpose was to provide a simple and understandable interpretation of the epidemiological data.

With the ever growing need to limit losses due to root diseases in order to provide food, forage and fiber for human needs, the need to provide environmentally and ecologically sound management practices for these diseases becomes paramount. Agricultural practices for managing root diseases must be designed to optimize crop productivity and minimize the impacts on beneficial soil organisms and the environment. The success that epidemiologists have in setting the strategies for successful root disease management will depend on their understanding of ecology of soilborne pathogens and the epidemiology of the diseases they cause.

LITERATURE CITED

1. Adams, P.B. 1981. Forecasting onion white rot disease. Phytopathology 71:1178–1181.
2. Addicot, J.F., Aho, J.M., Antolin, M.F., Padilla, D.K., Richardson, J.S., and Soluk, D.A. 1987. Ecological neighborhoods: scaling environmental patterns. Oikos 49:340–346.
3. Ali-Shtayeh, M.S., MacDonald, J.D., and Kabashima, J. 1991. A method for using commercial ELISA tests to detect zoospores of *Phytophthora* and *Pythium* species in irrigation water. Plant Dis. 75:305–311.
4. Amorim, L., Filho, A.B., and Hau, B. 1993. Analysis of progress curves of sugarcane smut on different cultivars using functions of double sigmoid pattern. Phytopathology 83:933–936.

5. Anas, O., Alli, I., and Reeleder, R.D. 1989. Inhibition of germination of sclerotia of *Sclerotinia sclerotiorum* by salivary gland secretions of *Bradysia coprophila*. Soil Biol. Biochem. 21:47–52.
6. Anas, O., and Reeleder, R.D. 1988. Feeding habits of larvae of *Bradysia coprophila* on fungi and plant tissue. Phytoprotection 69:73–78.
7. Anderson, R.V., Gould, W.D., Woods, L.E., Cambardella, C., Ingham, R.E., and Coleman, D.C. 1983. Organic and inorganic nitrogenous losses by microbivorous nematodes in soil. Oikos 40:75–80.
8. Andrén, O., and Lagerlöf, J. 1983. Soil fauna (microarthropods, enchytraeids, nematodes) in Swedish agricultural cropping systems. Acta Agriculturae Scandinavica 33:33–52.
9. Barker, K.R. 1985. Nematode extraction and bioassays. Pages 19–35 in: An Advanced Treatise on Meloidogyne, Vol. 2: Methodology. K.R. Barker, C.C. Carter, and J.N. Sasser, eds. North Carolina State University Graphics, Raleigh, NC.
10. Barker, K.R., Townshend, J.L., Bird, G.W., Thomason, I.J., and Dickson, D.W. 1986. Determining nematode population responses to control agents. Pages 283–296 in: Methods for Evaluating Pesticides for Control of Plant Pathogens. K.D. Hickey, ed. APS Press, St. Paul, MN.
11. Bateman, D.F. 1964. Cellulase and the *Rhizoctonia* disease of bean. Phytopathology 54:1372–1377.
12. Benson, D.M. 1994. Inoculum. Pages 1–33 in: Epidemiology and Management of Root Diseases. C.L. Campbell and D.M. Benson, eds. Springer-Verlag, Berlin, Germany.
13. Berg, N.W., and Pawluk, S. 1984. Soil mesofaunal studies under different vegetative regimes in north central Alberta. Can. J. Soil Sci. 64:209–223.
14. Beute, M.K., and Benson, D.M. 1979. Relation of small soil fauna to plant disease. Annu. Rev. Phytopathol. 17:485–502.
15. Bloomberg, W.J. 1979. A model of damping-off and root rot of Douglas-fir seedlings caused by *Fusarium oxysporum*. Phytopathology 69:74–81.
16. Boag, B., Brown, D.J.F., and Topham, P.B. 1987. Vertical and horizontal distribution of virus-vector nematodes and implications for sampling procedures. Nematologica 33:83–96.
17. Böhm, W. 1979. Methods of Studying Root Systems. Springer-Verlag, Berlin, Germany. 188 pp.

18. Bruton, B.D., and Reuveni, R. 1985. Vertical distribution of microsclerotia of *Macrophomina phaseolina* under various soil types and host crops. Agric. Ecosyst. Environ. 12:165–169.
19. Cahill, D.M., and Hardham, A.R. 1994. Exploitation of zoospore taxis in the development of a novel dipstick immunoassay for the specific detection of *Phytophthora cinnamomi*. Phytopathology 84:193–200.
20. Cahill, D.M., and Hardham, A.R. 1994. A dipstick immunoassay for the specific detection of *Phytophthora cinnamomi* in soils. Phytopathology 84:1284–1292.
21. Campbell, C.L. 1986. Interpretation and uses of disease progress curves for root diseases. Pages 38–54 in: Plant Disease Epidemiology, Vol. 1: Population Dynamics and Management. K.J. Leonard and W.E. Fry, eds. Macmillan, New York, NY.
22. Campbell, C.L., and Benson, D.M., eds. 1994. Epidemiology and Management of Root Diseases. Springer-Verlag, Berlin, Germany. 344 pp.
23. Campbell, C.L., and Benson, D.M. 1994. Spatial aspects of the development of root disease epidemics. Pages 195–243 in: Epidemiology and Management of Root Diseases. C.L. Campbell and D.M. Benson, eds. Springer-Verlag, Berlin, Germany.
24. Campbell, C.L., Jacobi, W.R., Powell, N.T., and Main, C.E. 1984. Analysis of disease progression and the randomness of occurrence of infected plants during tobacco black shank epidemics. Phytopathology 74:230–235.
25. Campbell, C.L., and Madden, L.V. 1990. Introduction to Plant Disease Epidemiology. John Wiley & Sons, New York, NY. 532 pp.
26. Campbell, C.L., Madden, L.V., and Pennypacker, S.P. 1980. Structural characterization of bean root rot epidemics. Phytopathology 70:152–155.
27. Campbell, C.L., and Neher, D.A. 1994. Estimating disease severity and incidence. Pages 117–147 in: Epidemiology and Management of Root Diseases. C.L. Campbell and D.M. Benson, eds. Springer-Verlag, Berlin, Germany.
28. Campbell, C.L., and Nelson, L.A. 1986. Evaluation of an assay for quantifying populations of sclerotia of *Macrophomina phaseolina* from soil. Plant Dis. 70:645–647.
29. Campbell, C.L., Pennypacker, S.P., and Madden, L.V. 1980. Progression dynamics of hypocotyl rot of snapbean. Phytopathology 70:487–494.

30. Chung, Y.R., Hoitink, H.A.J., and Lipps, P.E. 1988. Interactions between organic-matter decomposition level and soilborne disease severity. Agric. Ecosyst. Environ. 24:183–193.
31. Culbreath, A.K., Beute, M.K., and Campbell, C.L. 1991. Spatial and temporal aspects of epidemics of Cylindrocladium black rot in resistant and susceptible peanut genotypes. Phytopathology 81:144–150.
32. Curl, E.A., Lartey, R., and Peterson, C.M. 1988. Interactions between root pathogens and soil microarthropods. Agric. Ecosyst. Environ. 24:249–261.
33. English, J.T., and Mitchell, D.J. 1989. Use of morphometric analysis for characterization of tobacco root growth in relation to infection by *Phytophthora parasitica* var. *nicotianae*. Plant Soil 113:243–249.
34. English, J.T., and Mitchell, D.J. 1994. Host roots. Pages 34–64 in: Epidemiology and Management of Root Diseases. C.L. Campbell and D.M. Benson, eds. Springer-Verlag, Berlin, Germany.
35. Fitter, A.H. 1982. Morphometric analysis of root systems: application of the technique and influence of soil fertility on root system development in two herbaceous species. Plant Cell Environ. 5:313–322.
36. Fitter, A.H. 1987. An architectural approach to the comparative ecology of plant root systems. New Phytol. 106 (Suppl.):61–77.
37. Gilligan, C.A. 1983. Modeling of soilborne pathogens. Annu. Rev. Phytopathol. 21:45–64.
38. Gilligan, C.A. 1985. Probability models for host infection by soilborne fungi. Phytopathology 75:61–67.
39. Gilligan, C.A. 1985. Construction of temporal models: III. Disease progress of soil-borne pathogens. Pages 67–105 in: Advances in Plant Pathology, Vol. 3. C.A. Gilligan, ed. Academic Press, London, UK.
40. Gilligan, C.A. 1990. Mathematical modeling and analysis of soilborne pathogens. Pages 96–142 in: Epidemics of Plant Diseases: Mathematical Analysis and Modeling, 2nd ed. J. Kranz, ed. Springer-Verlag, Berlin, Germany.
41. Gilligan, C.A. 1990. Antagonistic interactions involving plant pathogens: fitting and analysis of models to non-monotonic curves for population and disease dynamics. New Phytol. 115:649–665.

42. Gilligan, C.A. 1994. Temporal aspects of the development of root disease epidemics. Pages 148–194 in: Epidemiology and Management of Root Diseases. C.L. Campbell and D.M. Benson, eds. Springer-Verlag, Berlin, Germany.
43. Goodwin, P.H., English, J.T., Neher, D.A., Duniway, J.M., and Kirkpatrick, B.C. 1990. Detection of *Phytophthora parasitica* from soil and host tissue with a species-specific DNA probe. Phytopathology 80:277–281.
44. Harrison, J.G., Lowe, R., Wallace, A., and Williams, N.A. 1994. Detection of *Spongospora subterranea* by ELISA using monoclonal antibodies. Pages 23–27 in: Modern Assays for Plant Pathogenic Fungi: Identification, Detection and Quantification. A. Schots, F.M. Dewey, and R.P. Oliver, eds. CAB International, Wallingford, UK.
45. Hau, B., Amorium, L., and Filho, A.B. 1993. Mathematical functions to describe disease progress curves of double sigmoid pattern. Phytopathology 83:928–932.
46. Henrissat, B., Driguez, H., Viet, C., and Schülein, M. 1985. Synergism of cellulases from *Trichoderma reesei* in the degradation of cellulose. Bio/Technology 3:722–726.
47. Hornby, D., and Fitt, B.D.L. 1981. Effects of root-infecting fungi on structure and function of cereal roots. Pages 101–130 in: Effects of Diseases on the Physiology of the Growing Plant. P.G. Ayres, ed. Cambridge University Press, Cambridge, UK.
48. Hunt, H.W., Coleman, D.C., Ingham, E.R., Ingham, R.E., Elliot, E.T., Moore, J.C., Rose, S.L., Reid, C.P.P., and Morley, C.R. 1987. The detrital food web in a shortgrass prairie. Biol. Fertil. Soils 3:57–68.
49. Jeger, M.J. 1987. The influence of root growth and inoculum density on the dynamics of root disease epidemics: theoretical analysis. New Phytol. 107:459–478.
50. Jeger, M.J., and Lyda, S.D. 1986. Epidemics of Phymatotrichum root rot (*Phymatotrichum omnivorum*) in cotton: environmental correlates of final incidence and forecasting criteria. Ann. Appl Biol. 109:523–534.
51. Johnson, L.F. and Curl, E.A. 1972. Methods for Research on the Ecology of Soil-borne Plant Pathogens. Burgess, Minneapolis, MN. 247 pp.
52. Jones, K.J. 1990. Components of resistance in *Nicotiana tabacum* to *Phytophthora parasitica* var. *nicotianae*. Ph.D. thesis, North Carolina State University, Raleigh, NC.

53. Kenerley, C.M., Papke, K., and Bruck, R.I. 1984. Effect of flooding on development of Phytophthora root rot in Fraser fir seedlings. Phytopathology 74:401–404.
54. Kranz, J. 1968. Eine Analyse von annuellen Epidemien pilzlicher Parasiten. III. Über Korrelationen zwischen quantitativen Merkmalen von Befallskurven und Ähnlichkeiten von Epidemien. Phytopathol. Z. 61:205–217.
55. Kranz, J. 1974. Comparison of epidemics. Annu. Rev. Phytopathol. 12:355–374.
56. Kranz, J. 1978. Comparative anatomy of epidemics. Pages 33–62 in: Plant Disease: An Advanced Treatise, Vol. II: How Disease Develops in Populations. J.G. Horsfall and E.B. Cowling, eds. Academic Press, New York, NY.
57. Kranz, J. 1988. The methodology of comparative epidemiology. Pages 279–289 in: Experimental Techniques in Plant Disease Epidemiology. J. Kranz and J. Rotem, eds. Springer-Verlag, Berlin, Germany.
58. Larkin, R.P., Ristaino, J.B., and Campbell, C.L. 1995. Detection and quantification of *Phytophthora capsici* in soil. Phytopathology 85:1057–1063.
59. Lussenhop, J. 1992. Mechanisms of microarthropod-microbial interactions in soil. Pages 1–33 in: Advances in Ecological Research, Vol. 23. M. Begon and A.H. Fitter, eds. Academic Press, San Diego, CA.
60. Lussenhop, J., and Fogel, R. 1993. Observing soil biota in situ. Geoderma 56:25–36.
61. Lussenhop, J., Fogel, R., and Pregitzer, K. 1991. A new dawn for soil biology: video analysis of root-soil-microbial-faunal interactions. Agric. Ecosyst. Environ. 34:235–249.
62. MacDonald, J.D., Stites, J., and Kabashima, J. 1990. Comparison of serological and culture plate methods for detecting species of *Phytophthora*, *Pythium*, and *Rhizoctonia* in ornamental plants. Plant Dis. 74:655–659.
63. MacGuidwin, A.E., and Stanger, B.A. 1991. Changes in vertical distribution of *Pratylenchus scribneri* under potato and corn. J. Nematol. 23:73–81.
64. Madden, L.V. 1980. Quantification of disease progression. Prot. Ecol. 2:159–176.
65. Menzies, J.D. 1963. The direct assay of plant pathogen populations in soil. Annu. Rev. Phytopathol. 1:127–142.

66. Mihail, J.D., and Alcorn, S.M. 1982. Quantitative recovery of *Macrophomina phaseolina* sclerotia from soil. Plant Dis. 66:662–663.
67. Miller, S.A., Bhat, R.G., and Schmitthenner, A.F. 1994. Detection of *Phytophthora capsici* in pepper and cucurbit crops in Ohio with two commercial immunoassay kits. Plant Dis. 78:1042–1046.
68. Mitchell, D.J., Kannwischer-Mitchell, M.E., and Zentmyer, G.A. 1986. Isolating, identifying, and producing inoculum of *Phytophthora* spp. Pages 63–66 in: Methods for Evaluating Pesticides for Control of Plant Pathogens. K.D. Hickey, ed. APS Press, St. Paul, MN.
69. Moore, J.C., and de Ruiter, P.C. 1991. Temporal and spatial heterogeneity of trophic interactions within below-ground food webs. Agric. Ecosyst. Environ. 34:371–397.
70. Moore, J.C., Walter, D.E., and Hunt, H.W. 1988. Arthropod regulation of micro- and mesobiota in below-ground detrital food webs. Annu. Rev. Entomol. 33:419–439.
71. Morrall, R.A.A., and Verma, P.R. 1981. Disease progress curves, linear transformations and common root rot of cereals. Can. J. Plant Pathol. 3:182–183.
72. Nakai, T., and Ui, T. 1977. Population and distribution of sclerotia of *Rhizoctonia solani* Kühn in sugar beet field soil. Soil Biol. Biochem. 9:377–381.
73. Neher, D.A. 1990. Inoculum density, furrow irrigation and soil temperature effects on the epidemiology of Phytophthora root rot of processing tomato. Ph.D. thesis, University of California, Davis, CA.
74. Neher, D.A. 1992. Ecological sustainability in agricultural systems: definition and measurement. J. Sust. Ag. 2(3):51–61.
75. Neher, D.A. 1996. Biological diversity in soils of agricultural and natural ecosystems. Pages 55–72 in: Exploring the Role of Diversity in Sustainable Agriculture. R.K. Olson, C.A. Francis, and S. Kaffka, eds. ASA Press, Madison, WI.
76. Neher, D.A., and Campbell, C.L. 1994. Nematode communities and microbial biomass in soils with annual and perennial crops. Appl. Soil Ecol. 1:17–28.
77. Neher, D., and Duniway, J.M. 1991. Relationship between amount of *Phytophthora parasitica* added to field soil and the development of root rot in processing tomatoes. Phytopathology 81:1124–1129.

78. Neher, D.A., McKeen, C.D., and Duniway, J.D. 1993. Relationships among Phytophthora root rot development, *Phytophthora parasitica* populations in soil, and yield of tomatoes under commercial field conditions. Plant Dis. 77:1106–1111.
79. Neher, D.A., Peck, S.L., Rawlings, J.O., and Campbell, C.L. 1995. Measures of nematode community structure and sources of variability among and within agricultural fields. Plant Soil 170:167–181.
80. Nicot, P.C., and Rouse, D.I. 1987. Precision and bias of three quantitative soil assays for *Verticillium dahliae*. Phytopathology 77:875–881.
81. Noe, J.P. and Barker, K.R. 1985. Relation of within-field spatial variation of plant-parasitic nematode population densities and edaphic factors. Phytopathology 75:247–252.
82. Old, K.M. 1967. Effects of natural soil on survival of *Cochliobolus sativus*. Trans. Br. Mycol. Soc. 50:615–624.
83. O'Neill, R.V., DeAngelis, D.L., Waide, J.B., and Allen, T.F.H. 1986. A Hierarchical Concept of Ecosystems. Princeton University Press, Princeton, NJ. 253 pp.
84. Ott, P., Hansen, S., and Vogtmann, V. 1983. Nitrates in relation to composting and use of farmyard manures. Pages 145–154 in: Environmentally Sound Agriculture. W. Lockeretz, ed. Praeger, New York, NY.
85. Papavizas, G.C., and Davey, C.B. 1959. Isolation of *Rhizoctonia solani* Kuehn from naturally infested and artificially inoculated soils. Plant Dis. Rep. 43:404–410.
86. Papavizas, G.C., and Klag, N.G. 1975. Isolation and quantitative determination of *Macrophomina phaseolina* from soil. Phytopathology 65:182–187.
87. Paul, E.A., and Clark, F.E. 1989. Soil Microbiology and Biochemistry. Academic Press, San Diego, CA. 273 pp.
88. Pfender, W.F. 1982. Monocyclic and polycyclic root diseases: distinguishing between the nature of the disease cycle and the shape of the disease progress curve. Phytopathology 72:31–32.
89. Pfender, W.F., Rouse, D.I., and Hagedorn, D.J. 1981. A "most probable number" method for estimating inoculum density of *Aphanomyces euteiches* in naturally infested soil. Phytopathology 71:1169–1172.

90. Punja, Z.K., Smith, V.L., Campbell, C.L., and Jenkins, S.F. 1985. Sampling and extraction procedures to estimate numbers, spatial pattern, and temporal distribution of sclerotia of *Sclerotium rolfsii* in soil. Plant Dis. 69:469–474.

91. Reynolds, K.M., Gold, H.J., Bruck, R.I., Benson, D.M., and Campbell, C.L. 1986. Simulation of the spread of *Phytophthora cinnamomi* causing a root rot of Fraser fir in nursery beds. Phytopathology 76:1190–1201.

92. Ristaino, J.B., and Averre, C.W. 1992. Effects of irrigation, sulfur, and fumigation on Streptomyces soil rot and yield components in sweetpotato. Phytopathology 82:670–676.

93. Ristaino, J.B., Duniway, J.M., and Marois, J.J. 1989. Phytophthora root rot and irrigation schedule influence growth and phenology of processing tomatoes. J. Am. Soc. Hort. Sci. 114:556–561.

94. Ristaino, J.B., Larkin, R.P., and Campbell, C.L. 1993. Spatial and temporal dynamics of *Phytophthora* epidemics in commercial bell pepper fields. Phytopathology 83:1312–1320.

95. Ristaino, J.B., Larkin, R.P., and Campbell, C.L. 1994. Spatial dynamics of disease symptom expression during *Phytophthora* epidemics in bell pepper. Phytopathology 84:1015–1024.

96. Schmitthenner, A.F. 1988. ELISA detection of *Phytophthora* from soil. Phytopathology 78:1576. (Abstr.).

97. Shannon, C.E., and Weaver, W. 1949. The Mathematical Theory of Communication. University of Illinois Press, Urbana, IL. 117 pp.

98. Singleton, L.L., Mihail, J.D., and Rush, C.M. 1992. Methods for Research on Soilborne Phytopathogenic Fungi. APS Press, St. Paul, MN. 265 pp.

99. Sneh, B., Katan, J., Henis, Y., and Wahl, I. 1966. Methods for evaluating inoculum density of *Rhizoctonia* in naturally infested soil. Phytopathology 56:74–78.

100. Thornton, C.R., Dewey, F.M., and Gilligan, C.A. 1994. Development of monoclonal antibody-based immunological assays for the detection of live propagules of *Rhizoctonia solani* in soil. Pages 29–35 in: Modern Assays for Plant Pathogenic Fungi: Identification, Detection and Quantification. A. Schots, F.M. Dewey, and R.P. Oliver, eds. CAB International, Wallingford, UK.

101. Timmer, L.W., Menge, J.A., Zitko, S.E., Pond, E., Miller, S.A., and Johnson, E.L.V. 1993. Comparison of ELISA techniques and standard isolation methods for *Phytophthora* detection in citrus orchards in Florida and California. Plant Dis. 77:791–796.

102. Tsao, P.H. 1983. Factors affecting isolation and quantitation of *Phytophthora* from soil. Pages 219–236 in: *Phytophthora*: Its Biology, Taxonomy, Ecology, and Pathology. D.C. Erwin, S. Bartnicki-Garcia, and P.H. Tsao, eds. APS Press, St. Paul, MN.

103. Upchurch, D.R., and Ritchie, J.T. 1983. Root observations using a video recording system in mini-rhizotrons. Agron. J. 75:1009–1015.

104. Upchurch, D.R., and Ritchie, J.T. 1984. Battery-operated color video camera for root observations in mini-rhizotrons. Agron. J. 76:1015–1017.

105. Van Bruggen, A.H.C., and Grünwald, N.J. 1994. The need for a dual hierarchical approach to study plant disease suppression. Appl. Soil Ecol. 1:91–95.

106. Vanderplank, J.E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York, NY. 349 pp.

107. Waage, J.K. 1991. Biodiversity as a resource for biological control. Pages 149–163 in: The Biodiversity of Microorganisms and Invertebrates: Its Role in Sustainable Agriculture. D.L. Hawksworth, ed. CASAFA Report Series No. 4. CAB International, Wallingford, UK.

108. Wiens, J.A. 1989. Spatial scaling in ecology. Funct. Ecol. 3:385–397.

109. Woods, L.E., Cole, C.V., Elliott, E.T., Anderson, R.V., and Coleman, D.C. 1982. Nitrogen transformations in soil as affected by bacterial-microfaunal interactions. Soil Biol. Biochem. 14:93–98.

110. Workneh, F., and Van Bruggen, A.H.C. 1994. Suppression of corky root of tomatoes in soils from organic farms associated with soil microbial activity and nitrogen status of soil and tomato tissue. Phytopathology 84:688–694.

111. Yeates, G.W., Bamforth, S.S., Ross, D.J., Tate, K.R., and Sparling, G.P. 1991. Recolonization of methyl bromide sterilized soils under four different field conditions. Biol. Fertil. Soils 11:181–189.