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Population Biology of Plant Pathogens

The Synthesis of Plant Disease Epidemiology and Population Genetics

It is no secret that much of the effort in plant pathology has shifted over the last 50 years from practical problem-solving to studies of more specialized academic interest (74,81). Our fundamental knowledge of plant disease and host–pathogen interactions has increased enormously, while at the same time the shift in emphasis has not been so extreme that practical disease management is ignored entirely. Granted, some practical problems still remain unsolved, but not always because of lack of attention by problem-solvers; some plant diseases are intractable, given current constraints, despite the relatively large inputs of energy and research activity (e.g., potato late blight, rice blast). Equally important, new disease problems continue to arise as old ones are solved (or not), in an ongoing dynamic. One needs only to look at the attention given recently to emerging pathogens, antimicrobial resistance, risks of biotechnology, and most recently, crop bioterrorism (Crop biosecurity and countering agricultural bioterrorism: Responses of the American Phytopathological Society. October 2002. Published online.) to see examples of the ever-changing problems facing plant pathologists.

One of the first things a student learns in plant pathology is that this is a multidisciplinary field that deals with all levels of biological organization, from molecules to ecosystems. Plant pathologists pursuing academic careers may have the luxury of specializing in a narrow niche (e.g., molecular genetics, systematics, micrometeorology, etc.). The rise of population genetics in plant pathology is characteristic of the specialization that has occurred in all of plant pathology. While suppression of plant disease remains the raison d'être for plant pathology, the utility of more basic disciplines is being actively investigated. However, the luxury of specialization is not often afforded to the practical problem-solvers. For solving practical plant disease problems, disciplinary boxes, or the labels attached to the various specializations within plant pathology, may actually constrain the creative process of problem solving. Each subdiscipline in plant pathology is circumscribed by finite, often disjunct, sets of concepts and methods. The solution to any particular problem may require a multidisciplinary team sharing ideas across specializations. Alternatively, but not mutually exclusively, solutions to real problems require broader training and appreciation of a variety of disciplines in plant pathology and other areas of science. In short, this is the classic problem of specialists and generalists (8,70,78).

The aim of this article is to attempt to bridge some of the differences between two related specializations in plant pathology, epidemiology and population genetics, for the purpose of solving disease problems. In earlier times, these two subdisciplines were considered part and parcel of the same field. Although not identified explicitly as "population genetics," a major theme in Vanderplank's (76) seminal book on epidemiology was the evolution of races overcoming host plant resistance. During the last two decades, the two fields have diverged, with few exceptions (53,54), leaving little overlap between them. Our thesis in this article is that integrating these two areas of study into a more unified approach is advantageous to solving practical plant pathology problems. Many practical plant pathologists are already operating without distinguishing between these two fields (and others); these are typically the plant pathologists in the trenches who are solving problems using the best available tools, whether spore traps or thermal cyclers. Thus, practitioners of plant pathology have already broadened their views beyond the currently narrow—and all too exclusive—perspectives of population genetics and epidemiology, and have taken a general biological perspective.

We begin this article by comparing and contrasting the aims and questions addressed in epidemiology and population genetics in plant pathology. This is followed by some thoughts on how the two fields can be synthesized into an area of study better referred to as population biology. Finally, we highlight the potential value of this synthesis by showing examples in which disease problems are being solved based on a broader population biology context.

Synthesis of Epidemiology and Population Genetics

Epidemiological concepts. Plant disease epidemiology is a discipline concerned with understanding the dynamics of disease in time and space (32,34). It is a holistic science in terms of being concerned simultaneously with populations of pathogens and host plants within an environmental context, i.e., the classic disease triangle. The interdisciplinary nature of epidemiology extends further because of the need to understand environmental complexity, including a variety of abiotic and biotic factors. Moreover, epidemics must often be analyzed within an environment strongly shaped by human activity, especially disease management (81). Among the temporal aspects of epidemiology, one might ask questions such as whether pathogens are monocyclic or polycyclic; and disease progress curves are analyzed to quantify the temporal development of epidemics (77). Spatial aspects are typically focused on the patterns of inoculum and disease, and the processes that form patterns, especially dispersal. Analysis of the dynamic changes in spatial patterns, e.g., focus expansion, is the attempt to integrate temporal and spatial aspects of epidemics simultaneously (77,81).

One of the major goals in epidemiology has been to establish a theoretical basis for understanding epidemics in time and space

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(33). Although many plant pathologists engage in epidemiological research in one form or another, few would call themselves epidemiologists. Plant disease epidemiologists are concerned with elucidating the general principles underlying epidemic development, with an emphasis on being quantitative and predictive. However, epidemiologists also engage directly in problem-solving, for example with disease forecasting (29) or crop loss assessment (26,49), where epidemiological concepts and methods are applied for improving disease management. These latter activities are at the interface of the theory and biology of plant disease epidemics and have a long history of being firmly rooted in problem solving.

Population genetics concepts. The major focus of population genetics is to understand the evolutionary processes shaping and maintaining genetic variation within and among populations. Changes in genotype or allele frequencies in populations are considered evolutionary changes, albeit, they often occur on microevolutionary time scales. Although this description of population genetics sounds almost arcane, the evolution of races (as mentioned above) in response to selection by deployment of resistant host plants and the evolution of fungicide resistance in response to fungicide applications are perfect examples of population genetics problems in plant pathology. In these cases, the emphasis is on understanding how natural selection (an evolutionary process) affects the frequencies of different races or different fungicide resistance phenotypes. From a practical perspective, this translates into whether host plant resistance or particular fungicides will remain effective for managing disease.

Since the 1980s, studies of genetic variation (using neutral genetic markers, i.e., markers not under selection per se) of plant pathogens have become very common (4,47,52). Under various models, it is possible to infer the evolutionary processes acting on populations from descriptions of population structure, i.e., the patterns of genetic variation within and between populations (45,50). For example, the extent and patterns of genotypic diversity within populations can be used to infer whether populations are clonal or have experienced recombination (5,15,51). Inferences about restricted migration and/or selection are sometimes made from differences in allele frequencies between subpopulations, because without recurrent migration, populations eventually diverge due to mutation and random changes (genetic drift) (55,60). Recent studies involving phylogenies and gene genealogies are blurring the distinction between population genetics and systematics in exciting ways (27,58,63), with similar goals of making inferences about evolutionary processes and history of various populations. In contrast, the exploitation of genetic variation for diagnostics or species identification is not considered population genetics because the focus is not on evolution (52,79).

What is "population biology?" We propose the use of the term population biology to describe a relatively holistic perspective of the ecological and evolutionary dynamics of plant and pathogen populations—and their interactions. Despite previous claims that epidemiology alone is a holistic discipline (80), population biology explicitly integrates ecological, genetic, and evolutionary principles within a population context (Fig. 1). As such, it is even broader and more encompassing than either epidemiology or population genetics alone.

Both epidemiology and population genetics are integral parts of population biology, and they share many concepts (Table 1). Although epidemiology and population genetics have been described as separate disciplines with distinct sets of questions, the merging (or re-merging) of the two is already quietly in process among the problem-solvers in plant pathology. How often have we heard plant pathologists use terms like "population studies" or "population analyses" to describe their research? In our experience, many of these studies would be categorized as neither purely epidemiology nor population genetics, but a hybrid of the two.

A closer comparison of the two disciplines shows that many of the concepts are actually shared (Table 1), although the vocabulary may differ because of their historically independent origins. For example, the epidemiological concepts of source of inoculum and dispersal could be considered in terms of migration (also called gene flow) by population geneticists. Similarly, the type of inoculum contributing to an epidemic may be a function of the pathogen's mating system, and answering this question may involve either epidemiological or genetic methods. In other words, the two disciplines may address similar questions within different conceptual frameworks and employing different tools. In the dispersal/migration example, an epidemiologist may collect data on spore densities, disease gradients, spatial patterns, and so on; the population geneticist may analyze multilocus genotypes, allele frequencies, gene diversities, and so on. Both approaches, however, could conceivably arrive at similar and/or complementary conclusions. Additional corollaries between the two fields abound. Further comparison of the concepts in Table 1 might include: host specialization and selection (and migration), resistance gene deployment and selection, competition and fitness, and more. Not all of the concepts have corollaries in both disciplines, but the extent of overlap clearly signals the commonalities and foreshadows the benefits that might be gained by combining the two fields rather than splitting them into separate disciplines. Therefore, we advocate taking a step back and seeing epidemiology and population genetics as part of a larger unified discipline (Fig. 1). Whether one is working purely in one field or the other, it

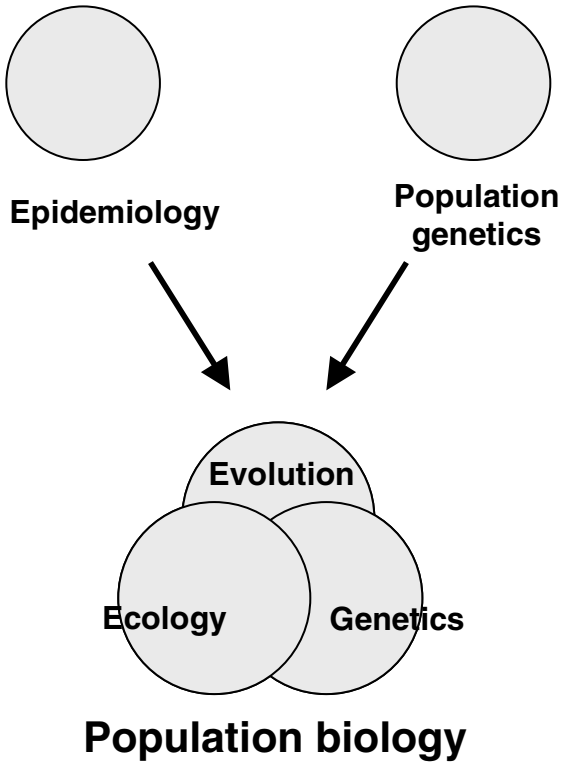

Fig. 1. The synthesis of epidemiology and population genetics: population biology. Adapted from Milgroom (53), and used with permission from the *Journal of Plant Pathology*.

is part of population biology; defining the relative mix of ecology, genetics, and evolution is less important than recognizing the broader context.

From the strategic perspective of addressing challenges in disease management, the two fields have a long history of being naturally joined. For example, breeding for resistance and resistance gene deployment are population genetics problems—with epidemiological consequences. Breeding for resistance requires an understanding of the diversity of pathogen populations and ensuring that early-generation breeding lines are screened against a wide range of genotypes in the pathogen population (61). Selectively neutral molecular markers can provide a good assessment of population structure of the pathogen but do not necessarily tell us anything about pathotype variation. Ideally, we need a combination of approaches using both neutral genetic markers and pathogenicity assays to guide resistance breeding and deployment (59,61). Durability of host plant resistance is an evolutionary process that depends on pathogen fitness, recombination, mutation, and so on (41), and was recently shown to correlate negatively with the evolutionary potential of target pathogens (46). Perhaps one of the best examples demonstrating the integration of epidemiology and population genetics is the use of cultivar mixtures in gene-for-gene systems. Long-term stable reduction of disease in cultivar mixtures depends inextricably on a combination of epidemiological and population genetic factors (20,25,56,57).

Population Biology and Problem Solving

Concepts of population biology have been successfully applied to problems at the strategic level, as described above for gene deployment. What potential is there for also applying them at a more tactical level for disease management? The value of this type of approach for solving problems may be best understood from a series of examples. These examples are examined from a disease management perspective, requiring solutions independent of disciplinary labels. Some of the problems highlighted below use genetic markers to assist in epidemiological analyses; others are based more on evolutionary concepts within an epidemiological context. The relative amount of input from either epidemiology or population genetics varies for every situation (Fig. 1); the primary consideration is the use of the concepts and approaches most appropriate for each individual problem.

Distinctly lacking in this section are examples of diagnostic methods for detecting or quantifying particular pathogens or genotypes. The literature is filled with examples of the latest techniques for taxonomy, diagnosis, and detection; just browse any of the latest plant pathology journals. These tools are often applicable for disease management or population biology, but by themselves, they are methods that need to be applied for solving problems. Many of the examples we cite herein rely on the use of genetic markers; the choice and development of markers for population genetics have been extensively discussed (4,73).

We divide our examples into four categories. First, we show examples in which specific genotypes of pathogens can be tracked in nature to show their dispersal. Second, we describe examples where the genetic diversity and spatial patterns of genotypes are used for inferring the types of inoculum responsible for epidemic development. Third, concepts of population genetics are applied alongside pathogenicity testing to address questions about host and tissue specialization and therefore potential for movement of inoculum. Finally, we highlight the interplay between epidemiology and genetics concerning the evolution of virulence (or "aggressiveness", see below).

Tracking genotypes. The first two examples highlight a relatively simple application of genetic markers to track specific genotypes in space and time. This type of study does not depend on an understanding of evolutionary or genetic concepts beyond those needed to develop markers for distinguishing among different genotypes. Nonetheless, this simple use of genetic markers can greatly enhance epidemiological studies. In these examples, the epidemiological problem was to determine the sources of primary inoculum and was accomplished by combining epidemiology and the use of genetic markers.

Source of primary inoculum of potato late blight. Zwankhuizen et al. (83) searched for the sources of primary inoculum of *Phytophthora infestans* that caused late blight epidemics in potato fields in The Netherlands. They combined a traditional epidemiological approach of studying disease gradients and the locations of disease foci in relation to potential sources (cull piles, organic farms, or volunteers) with a population genetics approach of studying the spatial distribution of pathogen genotypes. The obvious application of this information in disease management is to eliminate the source of inoculum, if possible, by sanitation. By asking which pathogen genotypes (DNA fingerprints) were found in commercial fields and comparing them to genotypes found in potential inoculum sources, these researchers were able to infer possible inoculum sources with a relatively high degree of confidence. Equally important, they could exclude other sources of inoculum because the genotypes did not match. A population biology approach that combined traditional epidemiological methods with identification of pathogen genotypes allowed them to make more definitive inferences about the sources of inoculum than could be made by either method alone.

This application of tracking genotypes was possible because the population biology of *P. infestans* had been studied in depth (23,28). Besides having genetic markers available from these previous studies, it was known that *P. infestans* often exists clonally in relatively few discrete genotypes. Thus, the background information available on population genetics greatly facilitated this problem-solving effort.

Source of primary inoculum in Stagonospora nodorum blotch of wheat. Stagonospora nodorum blotch is a major foliar disease of wheat worldwide. Despite decades of epidemiological research and recent population genetic analyses, the dominant source of primary inoculum has yet to be definitively determined (68,69). Identifying and targeting the source of initial inoculum is necessary because other management strategies, such as foliar fungicides aimed at secondary cycles, may not be feasible in certain situations. The potential sources of inoculum include infected seeds, debris, and other grasses, against which fungicidal seed treatment, crop rotation, and debris and weed management (tillage) are among some of the disease management approaches that might be applied.

Several lines of evidence support the potential for seedborne inoculum as a major

Table 1. Major concepts in epidemiology and population genetics in plant pathology[a]

Epidemiology	Population genetics
Source of inoculum	Population structure
Dispersal	Migration
Types of inoculum	Recombination
Host specialization	Mating systems
Gene deployment	Selection
Fungicide resistance	Fitness
Competition	Genetic drift
Disease progress	Mutation
Forecasting	Coevolution
Crop losses	Phylogenetics

[a] Adapted from Milgroom (53), and used with permission from the *Journal of Plant Pathology*.

source for foliar epidemics, including the demonstrated ability of *S. nodorum* to infect seed (12), high incidence of seed infection in commercial wheat seed lots (64), and efficient transmission of *S. nodorum* from infected seeds to seedlings (64,66). From an epidemiological perspective, the percentage of infected seed sown in the field correlated with the amount of foliar disease subsequently observed (Fig. 2A) (2,43,65). The most direct evidence for infected seed being an important source of inoculum, however, comes from a study combining the use of molecular genetic markers and epidemiological analysis of disease progress. Shah et al. (65) tracked the genotypes of *S. nodorum* from inoculated seed to foliar epidemics, and then to the next generation of infected seeds in the field. This study demonstrated conclusively that seeds were a source of at least some inoculum in the experimental fields, and exemplifies how genetic markers can be used directly to address epidemiological questions essential for sound disease management.

However, an independent population genetics approach to this pathosystem (in different locations) yielded different interpretations, i.e., that ascospores are a dominant source of inoculum. These conclusions were based on finding high levels of gene and genotypic diversity, random associations among alleles at different loci (gametic equilibrium), and lack of subdivision in *S. nodorum* populations, all of which are consistent with sexual reproduction and long-distance dispersal of ascospores (39,40,48). Additional epidemiological and population genetic analysis showed that the early stages of epidemics before tillering were characterized by discrete disease foci, mostly comprising single pathogen genotypes, indicative of localized dispersal of asexual secondary inoculum (Fig. 2B and C) (67). Therefore, if ascospores do contribute significantly to primary inoculum, they must arrive at an early stage in the development of wheat. Additional studies to address these competing hypotheses are currently underway (G. C. Bergstrom, *personal communication*), using the type of mark-recapture techniques advocated recently (6,82).

Diversity and spatial patterns of genotypes. The combination of assessing pathogen genetic diversity and spatial patterns of genotypes and disease has proven to be a powerful combination for inferring the reproductive biology of pathogens in the field. For many organisms, all else being equal, sexual populations have more genotypic diversity because of recombination (5,51). Moreover, for many fungal plant pathogens, sexual and asexual inoculum differ in their dispersal characteristics, e.g., wind versus splash dispersal, which often affect the spatial patterns of disease and genotypes (see the *S. nodorum* example above [67]). Therefore, the combination of spatial patterns and genotypic diversity can be useful for inferring the type of inoculum most significant in an epidemic. The examples below highlight this combination.

Dispersal of secondary inoculum in two diseases of grapevines. Two grapevine diseases, esca and Eutypa dieback, have been the subjects of recent studies of the dispersal of secondary inoculum within vineyards using a combination of epidemiological and genetic methods (10,11). For both diseases, one important management question was whether pruning was a means of disease spread. In this case, an aggregated pattern of disease and genotypes would be expected as pruning operations typically proceed along rows; for esca, an aggregated pattern might also be expected if the pathogen is spread by root-to-root contact. For both these diseases, spatial patterns of disease (symptomatic vines) were analyzed using an epidemiological approach. No significant aggregation of disease was observed (Fig. 3), making pruning (and/or root-to-root contact for esca) an unlikely means of secondary disease spread. After eliminating the localized spread of asexual inoculum, the reproductive biology of the two pathogens was studied by first examining vines for fruiting bodies. Basidiocarps of *Fomitiporia punctata* (one of the pathogens associated with esca) (10) and perithecia of *Eutypa lata* (11,62) were found associated with diseased grapevines, indicating that sexual reproduction was a potential source of secondary inoculum (Figs. 3 and 4). The contribution of sexual reproduction was investigated by determining the genotypic diversity of the pathogens isolated from vines. For both pathogens, genotypic diversity was very high, with each vine colonized by different genotypes. This latter result strengthens the argument against the hypothesis that inoculum is spread clonally via pruning or roots; otherwise the same

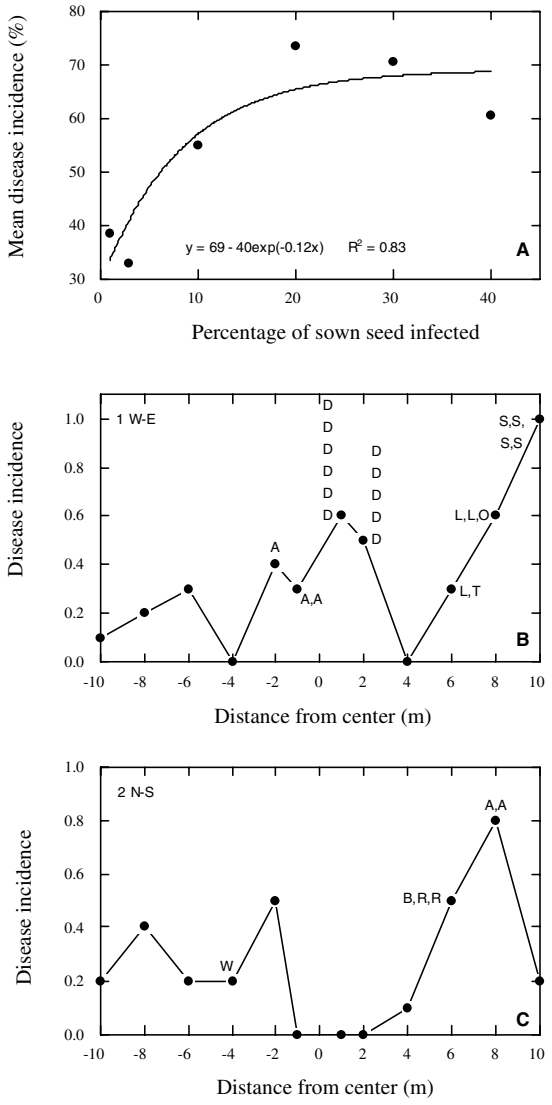

Fig. 2. Epidemiology and genotype tracking of *Stagonospora nodorum* on wheat. **A,** Disease incidence in foliar epidemics as a function of percentage of infected seed sown (65). **B and C,** Disease incidence and multilocus restriction fragment length polymorphism (RFLP) genotypes in two perpendicular transects (67). Genotypes of recovered isolates from each sampling location in the transect are shown by letters. Adapted from Shah et al. (65, 67).

genotypes would occur on multiple vines. Furthermore, ascospore progeny from perithecia of *E. lata* segregated for different genotypes, indicating that this fungus outcrosses under field conditions (11,62) (parallel studies could not be done with *F. punctata* because it is not possible to germinate basidiospores of this species in the lab). Taken together, random spatial patterns of disease and genotypes, high genotypic diversity, and the presence of sexual fruiting bodies are consistent with ascospore and basidiospore inoculum contributing significantly to secondary cycles of these diseases.

It is interesting to note that for both of these pathogens, genotypic variation in vegetative (or somatic) incompatibility (Fig. 4) was great enough that these relatively simple genetic markers were sufficient to answer the questions being addressed (10,11). Although these markers may be perceived by some as old-fashioned (or low-technology), data with as much power as molecular methods for addressing this question could be obtained easily and cheaply. These are examples where the simplest methods were used to address specific questions directly. Estimation of population genetic parameters was not necessary for these studies.

Dispersal of inoculum in Ascochyta blight of chickpeas. Ascochyta blight of

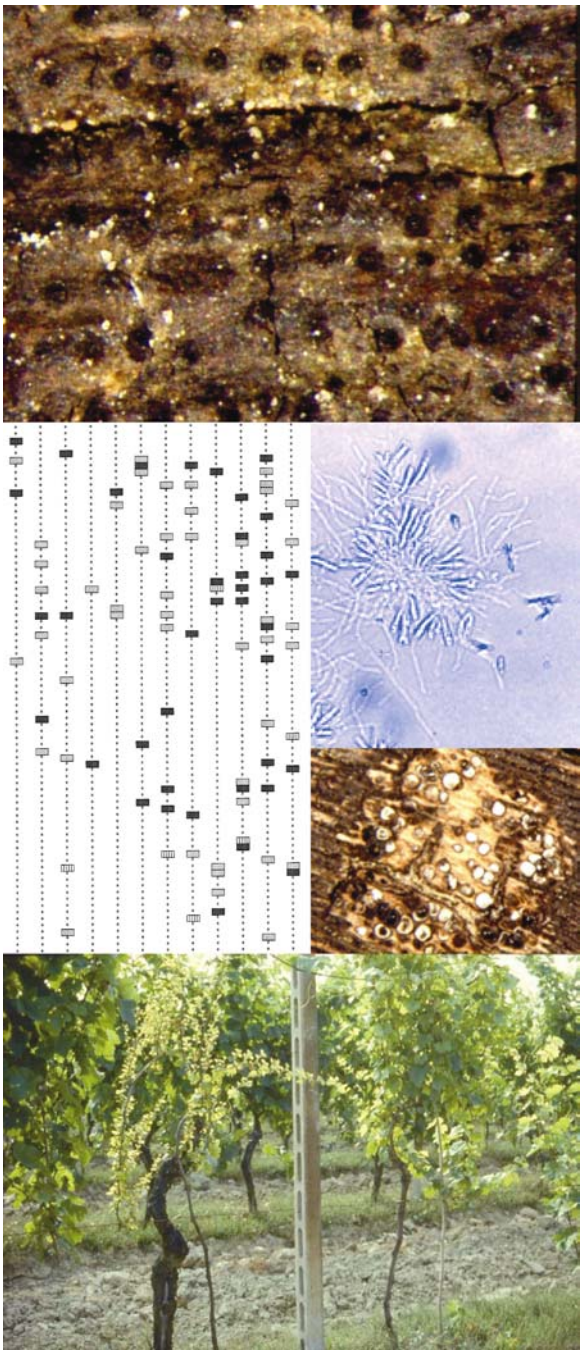

Fig. 3. Signs, symptoms, and spatial pattern of Eutypa dieback of grapevine. Clockwise from the top: stromata of *Eutypa lata* in grapevine bark; asci and ascospores of *E. lata* from squashed perithecium; section of stromata of *E. lata* showing perithecia; symptoms of dieback on grapevines; and spatial pattern of diseased vines showing symptoms, stromata, and/or perithecia of *E. lata* (11). Spatial pattern adapted from Cortesi and Milgroom (11), and used with permission from the *Journal of Plant Pathology*. Photographs courtesy of Paolo Cortesi.

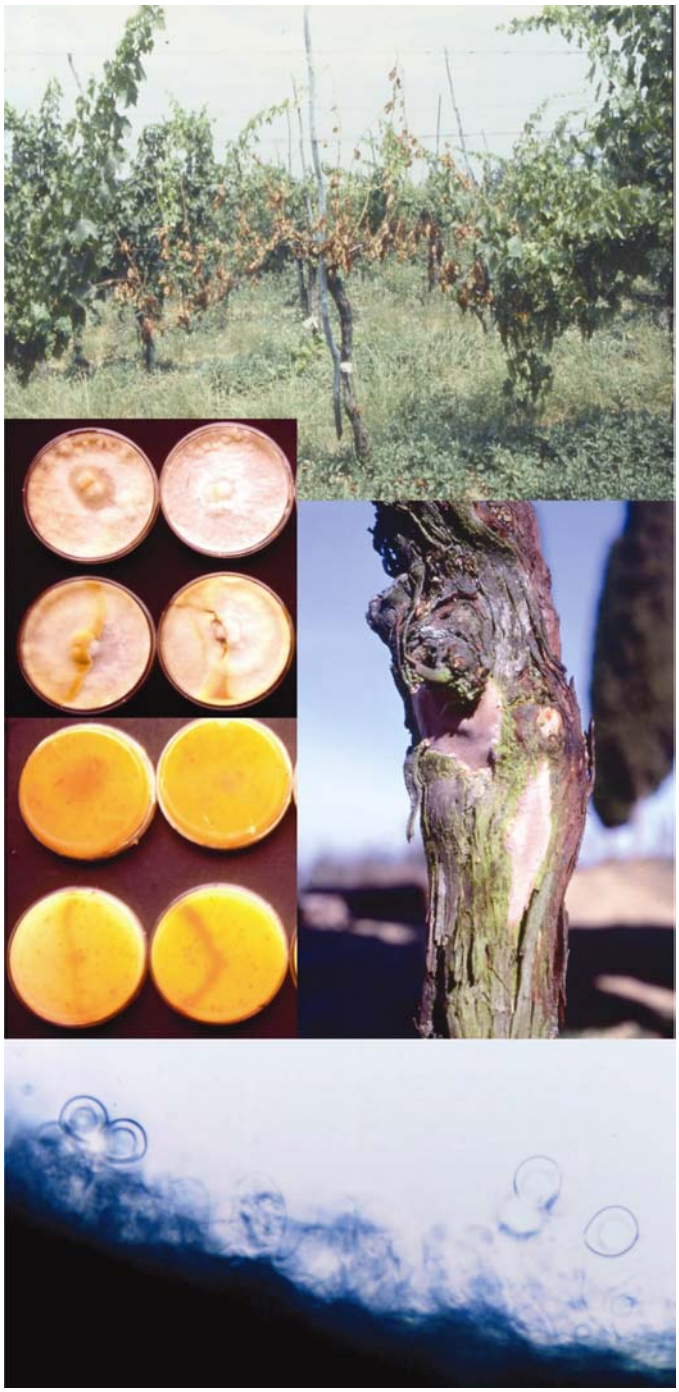

Fig. 4. Signs, symptoms, and somatic incompatibility assay in *Fomitiporia punctata*, a causal organism associated with esca disease of grapevine. Clockwise from the top: symptoms of esca in grapevines; fruiting bodies of *F. punctata* (resupinate hymenium) on grapevine; basidiospores from fruiting body of *F. punctata*; somatic incompatibility testing in *F. punctata* (10). Photographs courtesy of P. Cortesi and G. Minervini.

chickpea, caused by the loculoascomycete fungus *Ascochyta rabiei* (teleomorph: *Didymella rabiei*), can be initiated via conidia or ascospores (36). Conidia are produced by pycnidia in disease lesions on seeds, leaves, pods, and overwintered chickpea debris and are splash-dispersed short distances by rain. In contrast, wind-dispersed ascospores are produced from pseudothecia that only develop at low temperatures on senescent chickpea debris (37). Both conidia and ascospores are important epidemiologically in chickpea-growing areas where both mating types of the fungus occur (36,38). The pathogen can be readily isolated from chickpea seed, and seedborne conidia and mycelium are the primary means by which the pathogen has been introduced into new chickpea-growing areas worldwide (37). Seedborne inoculum results in heavily infected young plants with lesions concentrated on the hypocotyl (14,44). Ascochyta blight epidemics in the Pacific Northwest (PNW) are characterized by lightly infected plants with lesions that are randomly distributed in the upper canopy (T. L. Peever, *unpublished*). This pattern of disease, coupled with the observation that pseudothecia have been identified throughout the PNW (38), suggests that ascospores are the more important primary inoculum source in this region. Additional support for this hypothesis has come from population genetic studies with genetic markers. Microsatellite markers were found to be in gametic equilibrium in all PNW populations examined to date, and mating type ratios were not significantly different from 1:1, consistent with a model of primary infection by ascospores (T. L. Peever, *unpublished*).

To test the hypothesis that ascospores are the dominant type of primary inoculum for Ascochyta blight epidemics in the PNW, 19 Ascochyta blight foci (5 to 10 m diameter) were sampled in three PNW chickpea fields. The hypothesis was that each disease focus in a field was initiated by a single ascospore and the focus expanded through asexual conidial reproduction. The size of the foci and the timing of sampling suggested that several generations of asexual (clonal) reproduction had likely occurred following the initiation of

Fig. 5. Signs, symptoms, and life cycle of *Ascochyta rabiei* (teleomorph: *Didymella rabiei*) on chickpea. Clockwise from top left: cirrhi of conidia of *A. rabiei* produced from pycnidia on a chickpea stem; asci and ascospores of *D. rabiei*; seed infection and cultural morphology of *A. rabiei*; life cycle; pod infection showing concentric rings of pycnidia; symptoms on resistant and susceptible chickpea in an experimental field; pseudothecia of *D. rabiei* on chickpea stem; leaf symptoms; and Ascochyta blight focus in commercial chickpea field. Photographs courtesy of W. J. Kaiser, R. M. Hannnan, J. D. Rogers, F. J. Muehlbauer, and C. Armstrong.

each focus (Fig. 5). The resulting isolates were genotyped at four microsatellite loci and the mating type locus; their multilocus genotypes were then compared within and among foci (T. L. Peever, *unpublished*). Each disease focus was initiated by a different multilocus genotype, consistent with the hypothesis of ascospores as the primary inoculum source within a field. However, the majority of foci tested (12 of 19) contained more than one pathogen genotype, which may indicate (i) that more than one ascospore initiated the focus, (ii) that the foci were recolonized by another genotype after establishment, or (iii) that the focus was initiated by diverse asexual (seedborne) inoculum. These results are consistent with an epidemiological model of each infection focus being initiated by ascospores followed by several generations of conidial reproduction and expansion of the disease focus driven by rainfall. However, they are also consistent with a model of each disease focus being initiated by one or more genotypes of seedborne conidial inoculum.

Experiments designed to distinguish definitively among inoculum sources using genetically marked strains are currently underway. The practical significance of these results is that disease management strategies can be targeted specifically at ascospores if they are found to be the dominant inoculum source. Possible control measures aimed at eliminating or reducing ascospore inoculum could include chemical applications during the sexual phase to prevent ascospore release, or the application of biological control agents to chickpea debris. These approaches may reduce or eliminate ascospore inoculum, reducing the necessity for fungicide applications later in the season. Alternatively, if seedborne inoculum is shown to play a significant role, improved seed testing and seed treatment may be required and control measures can be specifically targeted at seed.

Host and tissue specialization. Plant pathogens display varying levels of host specificity, with some having highly specific interactions with their hosts (specialists) and others having broader host ranges (generalists). Many pathogens are also able to cause disease symptoms on more than one plant tissue or induce different symptoms at different times during the growing season (63,75). It has been widely assumed by plant pathologists that the same genotype of a pathogen causes disease on different hosts or tissues, but this hypothesis has been rarely tested. Hypotheses of host or tissue specificity can be rigorously tested using appropriate sampling strategies (e.g., sampling multiple hosts in the same locations) and the application of molecular markers, combined with conventional pathogenicity testing.

Alternaria alternata *on citrus.* The host specificity of *A. alternata* causing Alternaria brown spot on various citrus cultivars was tested using a combination of molecular markers and pathogenicity/virulence assays (60). Using pathogenicity tests, which measured disease incidence, no evidence of host specialization could be detected among *Alternaria* isolates infecting different tangerine × grapefruit hybrids (Fig. 6A). In contrast to pathogenicity testing, however, the same isolates were highly differentiated genetically (Fig. 6B), suggesting that there was restricted migration of pathogens among cultivars despite their close geographic proximity (meters apart). Based on the known ability of *Alternaria* to disperse aerially, an alternative explanation is that some degree of host specialization exists and the pathogen population is differentiated on different hosts because of selection. The lack of evidence for host specialization from pathogenicity tests may have been because the fitness assay used (disease incidence) was insufficiently sensitive to detect specialization and/or the wrong component of fitness was measured. If the authors had relied exclusively on traditional methods such as pathogenicity testing, no host specialization would have been detected (60).

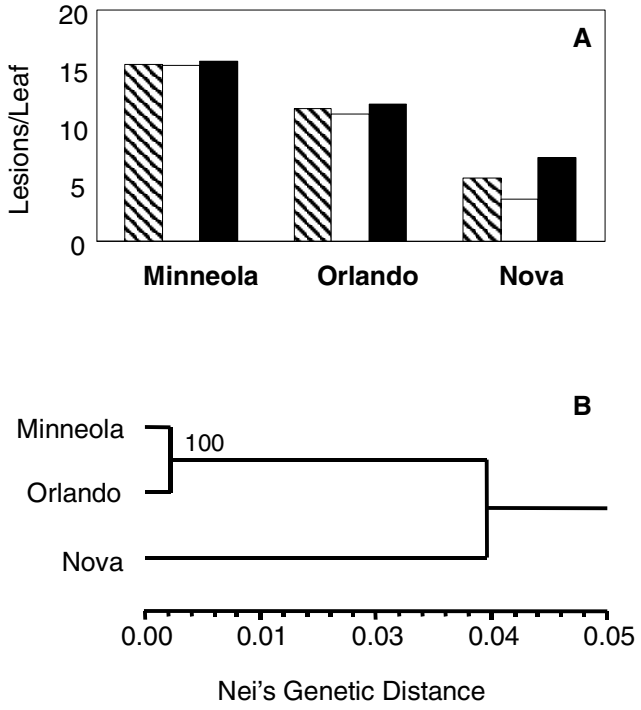

Fig. 6. Genetic differentiation and host specificity among *Alternaria alternata* isolates sampled from different tangerine × grapefruit hybrids (60). **A,** Virulence of isolates sampled from 'Minneola' (hatched bars), 'Orlando' (gray bars), and 'Nova' (solid bars) growing in close proximity in the same citrus grove inoculated on detached leaves of 'Minneola', 'Orlando', and 'Nova'. **B,** Phenogram estimated from random amplified polymorphic DNA (RAPD) allele frequencies showing genetic differentiation of pathogen subpopulations on 'Nova' from those on 'Minneola' and 'Orlando'. This branching pattern was found in all 1,000 bootstrapped phenograms. Adapted from Peever et al. (60).

Colletotrichum graminicola *on annual bluegrass and creeping bentgrass.* Another study that examined host specificity using molecular markers and pathogenicity assays involved *C. graminicola* causing anthracnose basal rot on annual bluegrass and creeping bentgrass (3). Similar to the *Alternaria*/citrus results, isolates from each host could be easily differentiated with random amplified polymorphic DNA (RAPD) markers but did not show strict host specificity when inoculated on both hosts. These examples illustrate that a population biology approach can augment pathogenicity studies on host specialization, providing new insights and different interpretations.

An important practical implication of host specialization is evaluating sources of inoculum for epidemics. Assessing genetic differentiation of pathogens among hosts is somewhat similar to tracking genotypes, as discussed above, although the analyses are more quantitative than qualitative. If pathogens are specialized on economically important plants, different genotypes may colonize weeds or other alternate hosts surrounding crop fields and may not be significant sources of inoculum. Conversely, lack of genetic differentiation is expected from pathogens with a wide host range (generalists), and weeds and other hosts may represent significant sources of inoculum for epidemics on commercial plants.

Symptom types in Sclerotinia sclerotiorum. The association of particular genotypes of *S. sclerotiorum* with different disease symptoms and tissue types on canola was recently analyzed using a phylogeographic approach (63). Isolates were sampled from atypical rosette infections that occurred early in the growing season

and from more typical stem infections that occurred later in the season in the southeastern United States. These isolates were tested for associations between lesion type (early rosette, late upper stem, and late basal stem) and multilocus haplotype (genotype) within a phylogenetic framework. Independent tests of association of haplotype and disease lesion type were performed for each locus separately. Lesion type was not significantly associated with haplotypes among an evolutionarily older phylogenetic lineage but was significantly associated with specific haplotypes in more recently derived lineages. This result suggests that the older haplotypes were better generalists (i.e., able to cause a wider range of disease symptoms) than the more recently derived lineages. The authors also hypothesized that the older lineages may have a wider host range, allowing them to cause disease on a broader range of hosts than the more derived specialist genotypes. Despite the fact that specific lesion types were significantly associated with certain haplotypes, other haplotypes were associated with all three lesions types. The association of distinct genotypes of a pathogen with specific lesion types or host ranges could have important practical implications for disease control. First, if growers know that specific fields are colonized with specialist genotypes, these fields could be rotated to other nonhost crops. Second, resistance screening procedures may need to be modified to include pathogen genotypes specialized on each tissue type or host, or may require modification to evaluate disease at different stages of plant growth or on different plant tissues.

Evolution of virulence (aggressiveness). Plant pathologists have long been interested in the appearance and increase in frequency of highly virulent, i.e., aggressive, genotypes in pathogen populations. (To be consistent with terminology in evolutionary biology (9,19,22,42), we use the term "virulence" to refer to mortality or decrease in fitness a pathogen causes its host because of infection; the analogous term in plant pathology is "aggressiveness" [31,76]). An understanding of the factors affecting the emergence of highly virulent pathogens is the first step in preventing their occurrence. Below we highlight an example in which the epidemiological and evolutionary factors have been described in detail.

The emergence of tomato necrosis in Spain. The severe outbreak of tomato necrosis in Spain, caused by *Cucumber mosaic virus* and its satellite RNAs (satRNA), in the late 1980s and early 1990s (35) has been analyzed in detail from a population biology perspective. Necrogenic satRNAs are hyperparasites of CMV and are responsible for the severe necrosis induced in tomatoes (24). The frequency of necrogenic satRNAs was shown to change over time, rising to high frequencies at a time when the disease was severe in the field, and later being replaced by non-necrogenic satRNA variants (1). The appearance (and subsequent disappearance) of a highly virulent pathogen in tomatoes was analyzed in terms of possible evolutionary dynamics. In fitness studies, the necrogenic and non-necrogenic satRNAs were similar for infectivity, accumulation levels, and encapsidation efficiency (16). However, in mixed infections, the necrogenic satRNAs accumulated to higher levels and were transmitted at higher frequencies than non-necrogenic satRNAs (18). Overall, however, the presence of satRNAs depresses the accumulation of CMV and therefore decreases the efficiency of both CMV and satRNA transmission by aphids (18). Modeling of this system (17) showed that it was theoretically possible for the necrogenic satRNAs to invade the CMV population and outcompete both the non-necrogenic satRNAs and satellite-free CMV isolates, but only under conditions of high aphid vector densities (resulting in high transmission rates between plants). As predicted, the years of outbreak of tomato necrosis in Spain correlated closely with years having high densities of aphids on tomato plants (17).

The implications of this study for disease management are profound because it implies that ecological conditions—those under at least some control by growers—can affect the emergence of virulent pathogens. This is an exciting context in which to examine the links between evolution and epidemiology because vector densities may determine the evolution of virulence of the pathogen. From a disease management perspective, it should be recognized that the outbreak of virulent pathogens is an evolutionary question, but one that is inexorably linked to ecological and epidemiological conditions. Management of vector populations is typically inefficient for managing virus diseases that are transmitted nonpersistently. However, in this case, manipulating vector populations and transmission rates may reduce the risk of highly virulent pathogen variants emerging.

Additional studies. Our choice of studies to illustrate the utility of population biology for addressing practical plant disease problems is by no means exhaustive; there are a number of other studies that could be described in more detail. For example, tracking pathogen genotypes recently has been considered on continental scales (7,30), with important implications for resistance breeding and gene deployment. Another pathosystem in which the tracking of genotypes could have enormous disease management benefits is blue mold of tobacco, caused by *Peronospora tabacina*. Primary inoculum is thought to migrate long distances, and with the recent development of genetic markers (71,72), it may now be possible to combine genetic and epidemiological techniques (13) to identify sources of primary inoculum. In the near future, more research using genetic markers in combination with epidemiology is likely to emerge. Finally, pathogen monitoring in the field is becoming easier and more accessible as new technologies are developed (21), with great potential benefit to population biology if applied to problems in the field.

Conclusions

All too often, technical trends or the perceptions of fashion determine the direction of scientific research. The number of papers using genetic markers and describing genetic variation in plant pathogen populations has increased dramatically in the past 10 to 15 years (53), but often with little contribution to solving real problems (54). This pattern may be reminiscent of the trends 20 years earlier when epidemiology went through a method-oriented stage (80). Research is ultimately dependent on methods,; and the availability of certain technologies makes it possible to address questions that might not otherwise have been feasible. The risk is that methods may drive the questions, not vice versa. Nonetheless, more recent developments are encouraging for population biology, if the kinds of examples described above are any indication of the future direction in this area. It appears that plant pathologists are going beyond the methods and are using genetic and epidemiological tools in the context of population biology to solve problems in agriculture. Our view is that these advances are possible because rigid disciplinary labels are not getting in the way. Success depends on recognizing that the most appropriate concepts and methods can be brought together from a variety of fields when problems need to be solved.

In the examples highlighted above, we discuss the synthesis of plant disease epidemiology and pathogen population genetics into a more holistic perspective of population biology. What about an even broader perspective? Plant pathology is an interdisciplinary field, and circumscribing population biology as a subfield within plant pathology is potentially as exclusionary as any other specialization! Labels are convenient for discussion, but they need to be pushed aside—one by one—so that future challenges are addressed without self-imposed disciplinary barriers.

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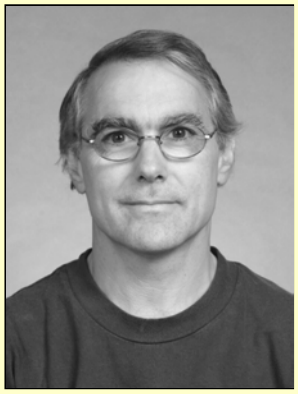

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