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NEW FRONTIERS IN THE STUDY OF DISPERSAL AND SPATIAL ANALYSIS OF EPIDEMICS CAUSED BY SPECIES IN THE GENUS *PHYTOPHTHORA*

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■ **Abstract** Diseases caused by species in the genus *Phytophthora* are responsible for significant economic losses on a wide range of host plants. Spatial pattern is one of the most characteristic ecological properties of a species, and reflects environmental and genetic heterogeneity and reproductive population growth acting on the processes of reproduction, dispersal, and mortality. Species of *Phytophthora* can be dispersed either in soil, via surface water movement down rows, from rain splash dispersal, by air, or via movement by humans or invertebrate activity. Dispersal results in patchiness in patterns of disease or inoculum in soil. In this chapter we discuss the mechanisms of dispersal of members of this important genus and describe several methods that can be used to statistically analyze data for which spatial coordinates are known. The methods include testing spatial autocorrelation for binary data or continuous data, semivariograms, and regression models for spatial data. The goal of spatial pattern analysis is to gain an understanding of the mechanisms of dispersal of propagules and to sort out the physical and biological factors that are important for spread of plant pathogens and ultimately, for disease management.

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INTRODUCTION

Plant diseases caused by species in the genus *Phytophthora* are responsible for significant economic losses on many important food, fiber, and ornamental crops (30). *Phytophthora* species are classified in the Kingdom Stramenopila and the phylum Oomycota and are actually more closely related to the golden brown algae than the true fungi (20, 48). *Phytophthora* species have a diploid life cycle and contain cellulose in their cell walls unlike the true fungi. These water molds are capable of forming flagellated zoospores that are easily dispersed by water. *Phytophthora* species are motile microorganisms and dispersal processes play a major role in the spatial patterns that are observed within fields and globally.

Our goal in this chapter is to address several questions. First, why study spatial patterns? Second, what are the primary mechanisms of dispersal of *Phytophthora* species and how do these mechanisms relate to the spatial patterns of disease or propagules that have been observed in fields? Third, what kinds of experimental and statistical procedures are available and how can they be used to document spatial heterogeneity and the underlying ecological processes? Fourth, what new frontiers await us in the use of spatial pattern analysis for management of Phytophthora diseases? Are there potential new applications of spatial pattern analysis in the areas of population genetics, precision agriculture, or global climate change research? Our ultimate goal is to stimulate new research and encourage use of the best statistical, experimental, and biological tools available to address important questions on the spatial dynamics of Phytophthora diseases.

WHY STUDY SPATIAL PATTERNS?

Epidemics caused by *Phytophthora* species are often initially patchy in appearance. The patchiness can manifest itself over a range of hierarchical scales, from the individual host plant to regional or global scales. When this patchiness has

a certain amount of predictability so that it can be described quantitatively, we call it spatial pattern (25). Spatial pattern in plant pathology has been described as the arrangement of disease entities relative to each other and to the architecture of the host crop (39). Patterns of diseased plants or propagules in soil arise from the interaction of physical, chemical, and biological factors that influence pathogen dispersal and infection processes. Dispersal can be defined as the process of movement of individuals or their propagules into or out of the population or population area (94). The term dispersion refers to the spatial pattern that results from dispersal processes (16, 97). Spatial pattern is one of the most characteristic ecological properties of a species, and reflects environmental and genetic heterogeneity and population growth acting on the processes of reproduction, dispersal, and mortality (75, 116).

The biological and ecological characteristics of pathogen life cycles and diseases are the primary determinants of the spatial pattern of epidemics (14). In some cases, there is a direct relationship between past process and the spatial patterns that are observed. For species of *Phytophthora* that primarily cause root disease, the spatial pattern of initial inoculum in a field can have a large effect on the subsequent pattern of diseased plants that are observed. In other cases, spatial pattern analysis can be used to generate hypotheses about underlying ecological processes or suggest mechanisms that give rise to them (25). Spatial pattern analysis can provide quantitative information on population dynamics of pathogens and is essential for modeling and simulation activities. In addition, analysis of spatial patterns can aid in the design of experiments in epidemiological research, development of sampling programs for disease or pathogen monitoring, and for development of management strategies (16). Even data from the new field of genomics may lend itself to spatial pattern analysis. The spatial and temporal expression patterns of thousands of genes can now be elucidated using microscopic DNA microarrays (3). Statistical analysis of these complex spatial patterns within individuals will be needed to elucidate underlying function.

In recent years more researchers have begun to study and quantify Phytophthora disease epidemics using spatial analyses in order to relate the observed characteristics of epidemics to the underlying ecological processes including reproduction, dispersal, competition, and survival. These kinds of field studies are very labor intensive. For this reason there are still relatively few good examples in the literature of quantitative analysis of spatial pattern of Phytophthora epidemics over time (54, 62, 65, 100, 103, 104, 136). The paucity of information on the spatial dynamics of inoculum dispersal and disease spread has hindered our ability to develop more sustainable management strategies for many Phytophthora diseases. Phytophthora diseases continue to cause large losses on a wide range of important crops because management practices, including the use of genetic resistance, are not complete and in many cases, sole reliance on fungicides has led to pathogen resistance in the field for many species in the genus (10). Ecologically based approaches that include spatial analysis of epidemic development could clearly lead to more sustainable methods of disease management for a number of

Phytophthora diseases. An important overall goal of our research with the *Phytophthora capsici*–pepper pathosystem has been to move beyond the simple description of pattern of inoculum or disease during Phytophthora epidemics in the field and into the explanatory phase of spatial pattern analysis. We have identified operative mechanisms of dispersal that lead to the spatial patterns observed in naturally infested fields (103, 104). Once operative mechanisms of dispersal are identified, novel management strategies can be developed to block dispersal (103, 104). We have also dissected individual components of dispersal processes in the *P. capsici*–pepper pathosystem and determined their relative contributions to temporal and spatial components of epidemic development (106, 115).

DISEASE CYCLES CAUSED BY *PHYTOPHTHORA* SPECIES

We use the disease cycle of *P. capsici*, a typical *Phytophthora* species (Figure 1; see color insert) to frame much of our discussion in this chapter. The disease cycle of a pathogen includes the life history of the pathogen in association with its host. For many diseases caused by *Phytophthora* species, oospores provide the primary inoculum for epidemic development and these propagules reside in soil (30, 56). Other species of *Phytophthora* reside as chlamydospores in soil (98) or the pathogen survives primarily as mycelia or sporangia in infected plant parts such as tubers or infected roots that are buried in soil (126, 130). For many species, a component of their life cycle is soilborne. For heterothallic species, both an A1 and A2 mating type are required for sexual reproduction. These mating types are actually compatibility types and do not correspond to dimorphic forms. Other species in the genus are homothallic and do not require two mating types to produce sexual oospores (91). In most cases, sporangia or zoospores released from direct germination of sporangia are the primary infective units that are dispersed from overwintering inoculum and are responsible for primary infections. In the case of *P. capsici*, oospores germinate predominately by the production of sporangia but germination via a germ tube also is possible (Figure 1) (55). Repeated asexual cycles of sporangium production and dispersal are involved in secondary spread within and between fields for many species of *Phytophthora*. Many species produce dehiscent sporangia that are easily dispersed from infected foliage in wind-driven rain (30, 52).

WHAT ARE THE MECHANISMS OF DISPERSAL OF *PHYTOPHTHORA* SPECIES?

Dispersal processes have major effects on both the spatial and temporal components of epidemic development. To provide a conceptual framework, we have considered that diseases caused by *Phytophthora* species can be dispersed by several distinct mechanisms (Table 1). Dispersal processes from root-to-root in soil

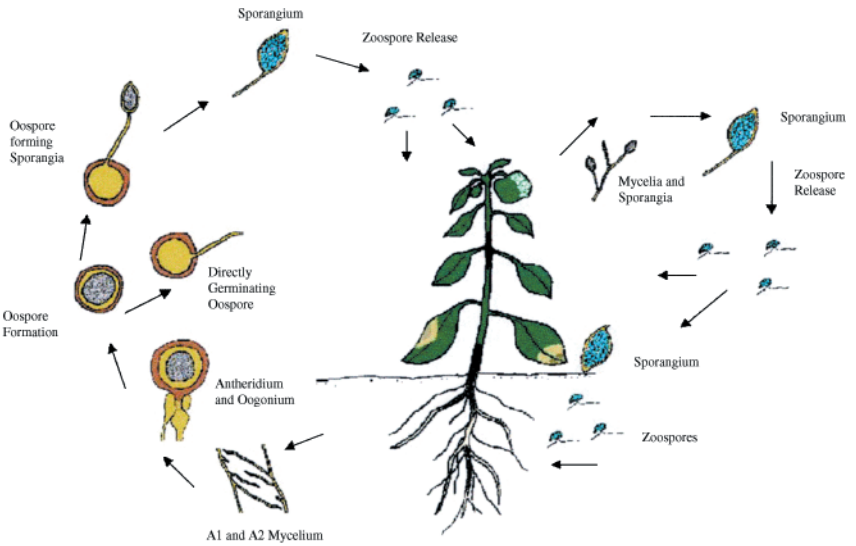


Figure 1 Disease cycle of Phytophthora blight on bell pepper caused by *Phytophthora capsic*. This disease cycle is typical of many diseases caused by species of *Phytophthora*. Most species contain a soilborne phase in which the sexual oospore or asexual chlaymydospores survive in soil or plant debris and provide the primary inoculum (usually sporangia and zoospores) for subsequent epidemics. Repeated cycles of sporangium formation, dispersal, and zoospore release are responsible for secondary cycles of inoculum in the asexual phase of disease.

TABLE 1 Five potential mechanisms of dispersal of *Phytophthora* species in the field and effects of dispersal mechanisms on components of epidemic development

Dispersal mechanism	Mode of dispersal of inoculum	Effects on spatial pattern and rate of disease increase (r_*)
Mechanism I	Within soil a. Root growth to inoculum b. Inoculum movement to roots c. Root to root contact	Limited spread within rows New foci rare Slow elongation of clusters Rate (r_*) low
Mechanism II	Inoculum dispersal in surface water	Spread within and across rows New foci local or distant Rapid cluster expansion Rate (r_*) high
Mechanism III	Splash dispersal from soil to leaves, stems, and fruit	Spread within and across rows New foci local Moderate cluster expansion Rate (r_*) moderate
Mechanism IV	Aerial dispersal from sporulating lesions on leaves, stems, or fruit to other aerial parts of plants	Spread long distance New foci overlap quickly Rapid cluster expansion Rate (r_*) high
Mechanism V	Dispersal by human or invertebrate activity, including movement of soil, plants, or propagules	Spread either long distance globally or local within fields New foci local or distant in previously noninfested areas

can involve either root growth to inoculum, inoculum movement to roots, or root-to-root contact (mechanism I) (15, 111). The primary change in spatial pattern will occur within rows, the occurrence of new foci may be rare, and the degree of aggregation of disease will change little with time (Table 1). However, movement of inoculum down rows with surface rain water or furrow irrigation (mechanism II) will result in increased rates of disease and spread may be detected within many rows for long distances in a similar direction (7, 88, 111) (Table 1). Rapid rates of disease increase will occur if splash dispersal of inoculum from soil to aerial parts of plants occurs with rainfall or overhead irrigation events (mechanism III). There may be two-dimensional expansion of disease foci both within and across rows and new foci will occur in the field close to existing foci (99). Aerial dispersal of inoculum from sporulating lesions on leaves, stems, or fruit to other aerial parts of plants within and between fields is a major mechanism of dispersal for *Phytophthora* species that infect primarily above-ground portions of plants (mechanism IV). The rate of disease increase will be very rapid and the degree of aggregation of disease will decrease rapidly as focal expansion occurs. New foci will develop and overlap both within and across many rows or fields (66, 136)

TABLE 2 Some major species of *Phytophthora* classified based on their mechanism of dispersal and host crop infected

Dispersal mechanism	Species of *Phytophthora*	Host crop	Reference
I. Within soil: roots to inoculum, inoculum movement to roots, root to root	*P. sojae*	Soybean	131
	P. parasitica	Tobacco	111
	P. cinnamomi	Banksia	53
		Fraser fir	98
	P. citrophthora	Citrus	57
II. Inoculum spread in surface water	*P. parasitica*	Tomato	88
	P. capsici	Peppers, squash	103
	P. cinnamomi	Eucalyptus	109, 129
		Banksia	53
III. Splash dispersal from soil to leaves, stems, and fruit	*P. cactorum*	Strawberries	78
	P. palmivora, P. megakarya	Cocoa	43
	P. syringae	Apple	30
	P. botryosa	Rubber	30
IV. Aerial dispersal from sporulating lesions on above-ground plant parts	*P. infestans*	Potato, tomato	136
	P. palmivora	Cocoa	43
	P. parasitica	Rhododendron	4
V. Dispersal by humans or invertebrate activity including movement of soil, plants of propagules	*P. infestans*	Potato	37
	P. palmivora	Cocoa	31
	P. fragariae	Strawberry	28
	P. citricola	Citrus	51, 57
	P. cinnamomi	Eucalyptus	61

(Table 1). *Phytophthora* species also can be dispersed by human or invertebrate activities (mechanism V) including the movement of equipment with infested soil, movement of plant materials containing the pathogen, or by insect or animal vectors (26, 31, 38, 51, 61). This mechanism of dispersal can result in spread over very long distances including globally or on more local scales (51). Some major species of *Phytophthora* classified by their mechanisms of dispersal are shown in Table 2 for illustration.

Dispersal of Inoculum in Soil: Mechanism I

Because initial inoculum in soil provides the reservoir for subsequent epidemic development for species of *Phytophthora* with a soilborne phase, characterization of changes in spatial patterns of inoculum over time can provide fundamental information about inoculum dispersal mechanisms (16, 17). Several studies have documented aggregated spatial patterns of initial inoculum of populations of *Phytophthora* species in soil and related them to dispersal mechanisms (4, 19, 57, 103, 118). Initial inoculum of *P. parasitica* was aggregated in tobacco soils in Florida

and aggregation decreased with depth (34). Aggregation of inoculum has also been described for *P. parasitica* on pineapple and *P. parasitica* on citrus (19, 118). Propagules of *P. sojae* are aggregated near the soil surface and are higher in no-till versus conventional tilled fields of soybean since inversion of the debris with tillage does not occur in the no-till system (131).

Patterns of inoculum of *Phytophthora* species in soil have been mapped for perennial crops such as citrus or apple, and often reflect the distribution of the host roots in soil (57, 63, 71, 118). The spatial pattern of inoculum of *P. cactorum* in soils from apple orchards was strongly associated with distance from the tree. Propagule densities decreased with increased distance from the tree, with increased depth in soil, and with increased distance up slopes (57). Interestingly, earthworms castings contained propagules of *P. cactorum* and earthworms were implicated in dispersal of propagules (mechanism V) to the soil surface where densities were highest (57). Aerial photography has been used to document the spatial patterns of disease caused by *P. cinnamomi* over a 30-year period in a Banksia forest ecosystem and the rate of disease front extension was calculated (53). *Phytophthora cinnamomi* spread at faster rates in the low-lying downhill slope positions than in the upper slopes and the main mechanism of dispersal in the uphill direction was determined to be via root-to-root spread (mechanism I) (53).

Population densities of *Phytophthora* species in soil are dynamic and can be influenced by soil physical and chemical factors in addition to the presence of actively growing, susceptible roots. Soil physical and chemical characteristics are spatially heterogeneous within field soils (29, 85, 119, 128). There is little published information relating the heterogeneity of physical and chemical factors of soil with the heterogeneity of initial spatial patterns of propagules of *Phytophthora* species in soil. Soil clay content, sodium, and copper concentrations were useful in explaining the spatial variation among densities of several plant parasitic nematodes in field soils (92). Soil chemical factors are known to affect diseases caused by soilborne pathogens including *Phytophthora* species (73, 81, 86). Soil salinity may affect the spatial pattern of propagules and dispersal of *Phytophthora* species in soil, since high concentrations of soluble salts can predispose plant roots to more severe Phytophthora root rots (73). Methods of regressing spatially correlated variables including geostatistical analysis (5, 21, 41, 50, 113) or multivariate statistical procedures (59) can be used to describe the correlations among soil variables, pathogen propagules, and disease incidence. These analytical techniques can provide tools for a more fundamental understanding of spatial heterogeneity of *Phytophthora* species in soil and the relationship of initial inoculum to subsequent disease.

Primary inoculum of *P. capsici* in the soil causes root infections that progress to crown infections in pepper (104). Wilting almost always precedes crown lesion development in naturally infested fields, which suggests the importance of root infections (mechanism I) and the soilborne phase of the disease (104). Spatial pattern mapping and two-dimensional distance class analysis were used to map the focal expansion of disease symptom types over time in several fields that

were naturally infested with *P. capsici* (104). In one field, wilted plants appeared in a circular area surrounding a large aggregation of plants with crown lesions, whereas the incidence of plants with above-ground stem lesions (from splash dispersal, mechanism III) was low. The disease focus expanded both within and across rows for long distances in this field and was associated with water drainage patterns in the field. In this example, spatial pattern analysis of symptom classes was used to determine that inoculum dispersal within soil (mechanism I) and in surface water down rows (mechanism II) were operative dispersal mechanisms (104).

We characterized the changes over time in the spatial pattern of disease, soil propagule levels of *P. capsici*, and soil water content during naturally occurring epidemics of Phytophthora blight in three commercial bell pepper fields in two consecutive years (46, 65, 103, 104). In three of four fields examined, initial disease occurrence in fields was highly spatially correlated with areas of fields where high soil moisture content occurred early in the season (65) (Figure 2*A*,*C*). Early season propagule levels of *P. capsici*, as estimated by a leaf disk bioassay, did not consistently demonstrate strong spatial dependence and were not closely associated with the initial occurrence or severity of Phytophthora root and crown rot disease (Figure 2*A*) but demonstrated stronger spatial dependence with disease later in the season (Figure 2*B*,*D*). Propagules of the pathogen may have been distributed widely in soil in a given field but disease developed initially where pathogen propagules were present and conditions of soil moisture were conducive for disease (65). These results emphasized the importance of soil water in the development and spread of disease.

The components of primary dispersal of *P. capsici* in soil were examined in field experiments. The pathogen can be dispersed within the soil by one of several mechanisms (mechanism I; Table 1), including inoculum movement to roots, root growth to inoculum, and root-to-root spread (115). In controlled field tests, an infected plant containing sporangia and zoospore inoculum was placed in PVC tubes on the soil surface and rainfall spread inoculum to roots in soil. In other treatments, inoculum was buried in wax-encased peat pots that contained either sporangia and mycelia or infected roots. The use of wax-encased inoculum buried in soil allowed root growth into the inoculum source but prevented inoculum movement out of the wax-encased pots so that dispersal mechanisms could be compartmentalized. Infection of the roots and crowns of adjacent plants in the plot was consistently more rapid when inoculum moved to roots via water than when healthy roots grew to inoculum or roots that were buried in soil (115).

Inoculum Dispersal in Irrigation and Surface Water: Mechanism II

Phytophthora species have been isolated from surface water sources used for irrigation of major crops in many regions of the world (27, 64, 74, 79, 112, 114, 117, 127). *Phytophthora cinnamomi* was detected in headwaters of several major rivers in

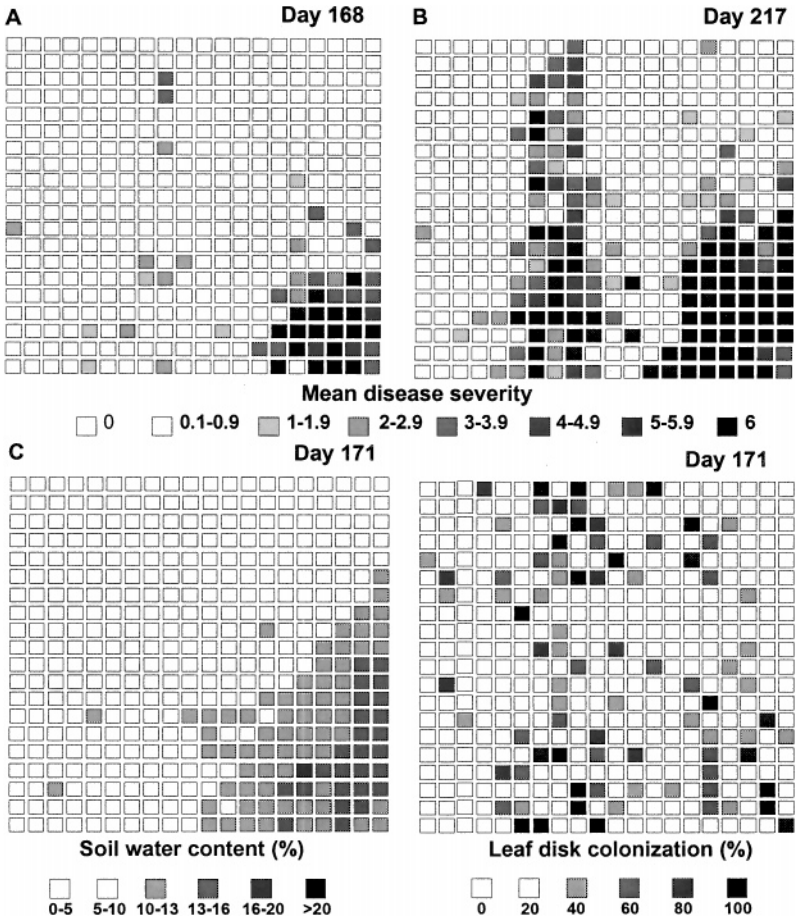


Figure 2 Spatial pattern map of disease severity caused by *Phytophthora capsici* on bell pepper at (*A*) Julian day 168 and (*B*) day 217 in a naturally infested field. Spatial pattern map of (*C*) gravimetric soil water content and (*D*) propagule levels of the pathogen in soil at day 171 in the same field.

South Africa over a geographically large area (127). Irrigation with infested surface water sources can result in rapid disease increase and is probably a major means by which species in the genus are dispersed regionally over longer distances between fields.

Inoculum movement down rows with surface water (mechanism II) is an important mechanism of dispersal for many polycyclic Phytophthora diseases (28, 88) and this is the predominant mechanism of dispersal of *P. capsici* in naturally infested fields (7, 11–13). *P. capsici* can spread from plant to plant within rows from initial point sources of inoculum and across rows from primary foci of disease

(7, 104, 108). Runs analysis, two-dimensional distance class analysis, and geostatistical analyses were used to quantify changes in spatial patterns of symptom development and inoculum in soil over time (Figures 2, 3) (65, 103, 104). Symptom expression was nonrandom in each field and was clearly aggregated. Aggregation of pairs of quadrats containing plants with wilt symptoms or dead plants was also greater within than across rows. Spread of disease occurred unidirectionally within many rows for long distances greater than 15 m and disease foci increased in size over time (Figure 2*A*,*B*) (103, 104). Inoculum of *P. capsici* was dispersed up to 70 m from point sources of inoculum in furrow-irrigated fields in California (13). In contrast, inoculum dispersal of *P. parasitica* on tomato from point sources of inoculum to roots in soil (mechanism I) was limited to less than 2 m. However, spread of *P. parasitica* to fruit on the surface of saturated soil (mechanism II) was extensive and the pathogen occurred in free water to distances of 68 m downstream from infestation sites during furrow irrigation (88). Path coefficient analysis was used to demonstrate the spread of *P. capsici* from point sources of inoculum in the field; the cumulative amount of rainfall had the largest direct effect on the progress of disease (7). All these examples suggest the importance of surface water movement within rows in spread of disease.

Movement of *Phytophthora* species in surface water sources in forest ecosystems also has been well documented (26, 63, 109). *P. cinnamomi* has been recovered from lateral subsurface water flowing at the base of lateritic soils to depths of 1 m in the Jarrah forests of Australia (63, 109, 129). Zoospores have been recovered in subsurface water and these propagules are responsible for infection of roots of Eucalyptus, Banksia, and many other species of trees (109, 129). Uphill disease spread has been attributed to inoculum movement from root to root in soil (mechanism I), whereas downhill disease spread has been attributed to drainage of surface (mechanism II) and subsurface water (53, 129) Aerial photography and quadrat mapping have been used to delineate differences in the boundary margins between diseased and healthy trees (26). Surface drainage water movement on logging roads and movement of soil (mechanism V) from logging and recreational vehicle traffic through National Parks has also been associated with pathogen spread in this system (26). Restricted access of recreational vehicles to areas infested with *P. cinnamomi* has been suggested to slow the spread of disease (26).

Splash Dispersal from Soil to Leaves, Stems, and Fruit: Mechanism III

Splash dispersal of *Phytophthora* species from soil to above-ground portions of plants with rainfall events (mechanism III) is a major means of dispersal of a number of species in the genus (9, 43, 76, 122). This mechanism has been clearly documented for the *P. cactorum*–leather rot pathogen of strawberry (77). Splash dispersal has been examined at three hierarchical scales for *P. cactorum* including splash from single water-drop impactions (133), spore transport with simulated

rain over small areas (78), and disease spread in the field with naturally occurring rain (78, 99).

Splash dispersal from single water-drop impactions was characterized using water-sensitive paper, fluorescent dyes, and *P. cactorum* infected strawberry fruit (133). Numbers of sporangia per droplet were described by a negative binomial distribution. Drop size, but not fall height, significantly affected sporangium dispersal in *P. cactorum* and between 9 and 56 sporangia were dispersed by a single impaction (133). Numbers of sporangia per impaction were linearly related to the impact velocity of the rainfall.

Dispersal of spores of *P. cactorum* and *Colletotrichum acutatum* from infected strawberry plants to other plants over small areas was investigated with a rain simulator (44, 78). Disease incidence increased with increasing rain duration and the incidence of leather rot increased from 1% with a 2 min rainfall event to 26% after a 16 min rainfall event. Secondary splash dispersal to noninfected plants after removal of the infected source plant also was documented in this pathosystem. Sporangia washed from fruit by a 4–8 min pre-rain event were subsequently dispersed to additional plants with subsequent rainfalls after the source inoculum was removed. These data indicate that secondary dispersal by rain splashing resulted in significant levels of disease.

Splash dispersal of inoculum with rainfall and disease spread in the field has been documented for many species of *Phytophthora* (Table 2). *P. cactorum* is dispersed with rainfall events and within-row spread has been observed and characterized using spatio-temporal autocorrelation analysis (99, 100). Strong barrier effects were operating across rows in the *P. cactorum*–leather rot pathosystem and a lack of spatial dependence beyond the first spatial lag was observed (100). Thus moderate expansion of disease foci occurred with this splash-dispersed *Phytophthora* species and new foci tended to be local either within or across rows. In contrast, secondary splash drops from rainfall impacting on cocoa leaves can carry sporangia of *P. palmivora* up to heights of 70 cm in cocoa trees and aerosol droplets can disperse inoculum to even great heights in the plant canopy (43).

Aerial Dispersal from Sporulating Lesions on Leaves, Stems, or Fruit: Mechanism IV

Aerial dispersal of inoculum from sporulating lesions on leaves, stems or fruit to other parts of plants within and between fields is a major mechanism of dispersal for *Phytophthora* species that infect primarily above ground portions of plants (mechanism IV). For *P. infestans* on potato, new foci will develop and overlap both within and across many rows and the rate of disease increase can be rapid (136). Sporangia of *P. infestans* do not survive under conditions of long-distance dispersal since sporangia viability is decreased under dry conditions. Short-range aerial dispersal has been reported for sporangia of *P. infestans.*

Evidence for two distinct dispersal processes were described for *P. infestans.* using spatial mapping of disease symptom classes (54). Late blight epidemics can

be initiated when infected tubers are planted in soil (mechanism V). Early season lesions on above-ground portions of plants were initiated close to the infected tuber source in replicated field trials (54). Subsequent focal expansion occurred from these infected plants and their aerial dispersal (mechanisms IV) was observed over longer distances and to adjacent crops (54).

Quantitative models of disease spread from a point source of inoculum on more local scales have been developed to describe disease gradients caused by *P. infestans* (83, 84). The gradient parameter, g, defined as the rate at which the logit of disease severity declines with distance, was determined. The gradient parameter is equal to the apparent infection rate divided by the wave velocity ($g = r/v$). Treatments such as rate-reducing resistance and fungicide use flattened disease gradients but did not affect the velocity of spread, which appeared constant. The velocity of spread of *P. infestans* was estimated to be 3.7 m/day in this study and was quite low (84). The authors suggested that the scale of the plot size affected results and that as the scale of the epidemic increases, the velocity should increase (84).

More recently, several new models have been proposed to measure the velocity of epidemic waves from an infection focus (32, 110, 135). The assumption of constant frontal velocity (traveling dispersive wave) has been questioned and it has been proposed that epidemics, such as those caused by *P. infestans*, should proceed with increasing frontal velocity (dispersive epidemic wave) as the scale of the focus increases and thus, velocity is scale dependent (110). These models have been used to predict the velocity of expansion of late blight epidemics occurring over different spatial scales including: within a field (zero order–30 m), across fields (first order–300–800 km), or over several seasons (second order–1500 km) (110, 124, 125). If the velocity of focal expansion increases with the scale of an epidemic, then regional management strategies need to be in place that contain epidemics to zero order epidemics to slow the spread of disease. Further experiments need to be done to validate the proposed models with actual data collected on different spatial and temporal scales since data used to validate the models were taken from epidemics that progressed at different rates (110).

Spatial patterns of late blight epidemics on local and regional scales were studied using a combination of spatially referenced data and genetic markers to make inferences concerning dispersal of the pathogen from primary inoculum sources (2, 89, 136). In one study in the Netherlands, inoculum sources were identified using a combination of disease gradient analyses, spatially referenced mapping, and DNA fingerprinting of isolates (136). Most of the sources of inoculum in conventional potato fields originated from nearby refuse piles that were upwind of conventional fields (136). Aerial dispersal of *P. infestans* from 34 to over 3000 m was documented from refuse piles to fields. Infested organic potato fields also were implicated as a source of inoculum for aerial dispersal of *P. infestans* into nearby conventional fields in one season (136). A region gradient was monitored across fields and data indicated that genotype-specific sources of inoculum that differed in aggressiveness were dispersed among fields. Additional research using a combination of spatially referenced samples at different scales and DNA markers could be used to track local and regional migrations of other

aerially dispersed *Phytophthora* species and could lead to improved management strategies.

The spatial and temporal development of late blight epidemics were mapped to determine if host-related differences in genotypic composition of *P. infestans* populations were a consequence of differential pathogenicity to potato and tomato (66). Competition experiments were conducted with fields of potato by simultaneous inoculation with point sources of either a potato or a tomato isolate in different locations in the field. Disease spread was monitored on a spatial scale in contiguous quadrats and the genotype of the pathogen was determined after isolation from infected plants. The experiments clearly demonstrated greater competitive fitness of a potato genotype than a tomato genotype on potato in the field (66). Differences in the genotypic composition of populations of *P. infestans* on tomato and potato have been observed in North Carolina fields (36).

Dispersal by Human or Invertebrate Activity: Mechanism V

Dispersal of *Phytophthora* species by human activity, including movement of plant materials, or soils containing the pathogen, is responsible for migrations on a global scale. *P. infestans* can be dispersed in infected potato tubers or tomato fruit (mechanism V). Movement of infected tubers from Mexico was responsible for initiation of epidemics in Europe and the United States in the 1970s (38). Dispersal of *P. infestans* in infected plant material was probably responsible for initial introductions of the pathogen into the United States and subsequently to Europe in the nineteenth century from its ancestral home (38, 107). Many other *Phytophthora* species including *P. cinnamomi* in Australia and the southeastern United States, *P. capsici* into the southwestern United States, and *P. palmivora* and *P. megakarya* into Africa are believed to have been introduced into previously noninfested areas in infected plant materials (30). Invertebrate vectors including tent building ant species have been documented to move inoculum of *P. palmivora* vertically through infected cocoa trees (31). Chlamydospores of *P. cinnamomi* can be vectored by species of birds and termites (61). Snails and ants can vector the avocado pathogen *P. citricola* between trees. In these examples, dispersal via insects is generally on a local scale. The spatial patterns of Phytophthora disease caused by human- or insect-vectored dispersal mechanisms have not been well documented. Historical records and herbarium materials could be used to map global movement of pathogens on a spatial scale (107).

WHAT STATISTICAL PROCEDURES CAN BE USED FOR MEASURING AND DOCUMENTING SPATIAL HETEROGENEITY?

What kinds of statistical methods can be used for spatially referenced data; that is, data for which spatial coordinates are known? Some methods for nonspatially referenced data are presented elsewhere (14, 16). Three general categories

of methods are presented here including: (*a*) methods for evaluating the shape and size of clusters and for testing spatial autocorrelation on a lattice; (*b*) methods for estimating and modeling spatial autocorrelation as a function of between point distance; and (*c*) methods for relating disease to possible explanatory variables such as initial inoculum level and soil properties. We demonstrate several of these methods on data from an experiment studying an epidemic caused by *P. capsici* in a commercial bell pepper field (103).

Testing Spatial Autocorrelation of Binary Data and Describing Cluster Size and Shape

The positions of plants or quadrats in an agricultural field make up what is called a lattice, a discrete set of points in some sort of spatial arrangement. The methods in this section are concerned with the associations between points in the lattice and points that are in specified neighborhoods relative to these points. By defining neighborhoods in appropriate ways, it is possible to estimate and test associations between points at any distance and spatial orientation in the lattice and to determine the size and shape of clusters of similar points. For instance, we might be interested in the correlation between adjacent plants within a row in the field. In this case, the neighborhood for a given plant consists of the two plants immediately adjacent to it in the same row. If we are interested in the association between two quadrats in adjacent rows, we define the neighborhood of a quadrat to be the nearest quadrat in the two adjacent rows. To test the association between one plant and another plant three spaces down and two rows over, we define the neighborhood for each plant in the field to be all plants that are three spaces down from it and two rows over. Neighborhoods can also be defined across time periods, so that tests for associations across space and time can be constructed in exactly the same way.

Testing Spatial Autocorrelation: BB and BW Statistics for Binary Data The type of statistic used to test spatial association is called a cross product statistic. This is a general class of statistics that includes the familiar Pearson correlation coefficient and, for spatial data, Moran's *I*, Geary's *c*, the *BB* and *BW* join-count statistics (21, 123), and the 2DCLASS (42, 90) and 2DCORR (33) statistics. The spatio-temporal autocorrelation coefficient described in Reynolds & Madden (99) is also a member of this class. Binary spatial data, such as presence or absence of disease, can be analyzed with the join-count statistics known as the *BB* and *BW* statistics. These statistics measure association by counting up the number of pairs of neighbors that are both diseased (*BB*) or the number of pairs in which one is diseased and one is not (*BW*). The observed count is then compared to a reference distribution to determine whether the association is statistically significant. The *BB* statistic is also the basis for two-dimensional distance class (2DCLASS) analysis and the closely related 2DCORR analysis, which are designed to detect the presence of clusters in binary data. The join-count statistics can be generalized to provide tests for qualitative data that take on more than two values.

The data needed to compute a join-count statistic are presence or absence of disease (or inoculum) recorded on each plant (or quadrat) in a field and the spatial coordinates for each measurement. The *BW* statistic, for a particular definition of neighborhoods, is simply the number of pairs of neighbors in which one member is diseased (black) and the other is healthy (white); in other words, the number of black-white pairs. Positive correlation corresponds to low *BW* values and negative correlation corresponds to high *BW* values. This can be compared to the 95th percentile of a theoretical reference distribution or to a distribution simulated from the observed data by randomly permuting the locations of the diseased plants. If a field has $n = b + w$ locations on which measurements are taken and disease is measured at b of the locations, a simulated field with the same dimensions and the same amount of disease is generated by randomly allocating disease to b of the n locations. The procedure is to generate a large number of simulated fields in this fashion. The *BW* statistic is then computed for each simulated field, and the set of all the *BW* statistics from the simulated fields and the observed field make up the reference distribution. The hypothesis of no spatial autocorrelation is rejected if the observed *BW* statistic is smaller than 95% of the values in the reference distribution (for a significance level of 5%). The alternative hypothesis in this test is that there is positive spatial autocorrelation, so it is a one-sided test. Figure 3 (103) shows the pattern of disease in quadrats at two different dates in a commercial bell pepper field in North Carolina naturally infested with *P. capsici* (103). Treating each quadrat as either containing disease or not, we can test for autocorrelation between adjacent quadrats within rows. The number of adjacent pairs of quadrats in which one has disease and the other does not at day 217 is $BW = 72$. *BW* values for 99 simulated fields ranged from 161 to 198 (45). We conclude that there is significant positive autocorrelation between adjacent quadrats within a row because BW is smaller than 99% of the reference distribution.

Instead of comparing *BW* to a simulated reference distribution, an alternative is to use a normal approximation for the distribution of *BW* values. This approximation is appropriate as long as the lattice has a regular shape (i.e. not star-shaped or other such odd shape), the number of points in the lattice is large, and the proportion of quadrats containing disease is not close to zero or one. The cross product statistics have the general form

$$r = \sum_{i=1}^{n} \sum_{j \neq i} w_{ij} y_{ij}, \qquad 1.$$

where w_{ij} is a measure of the "proximity" between sites i and j and y_{ij} is a measure of how alike or different the responses are at sites i and j. In the case of join-count statistics, w_{ij} takes on the value 1 if site j is in the neighborhood of site i and 0 otherwise. However, other measures of proximity can be used in cross product statistics; the inverse of the distance between the points is one commonly used proximity measure. For the *BW* statistic, y_{ij} is one if the sites have different responses and zero otherwise; i.e. it is the squared difference of the responses.

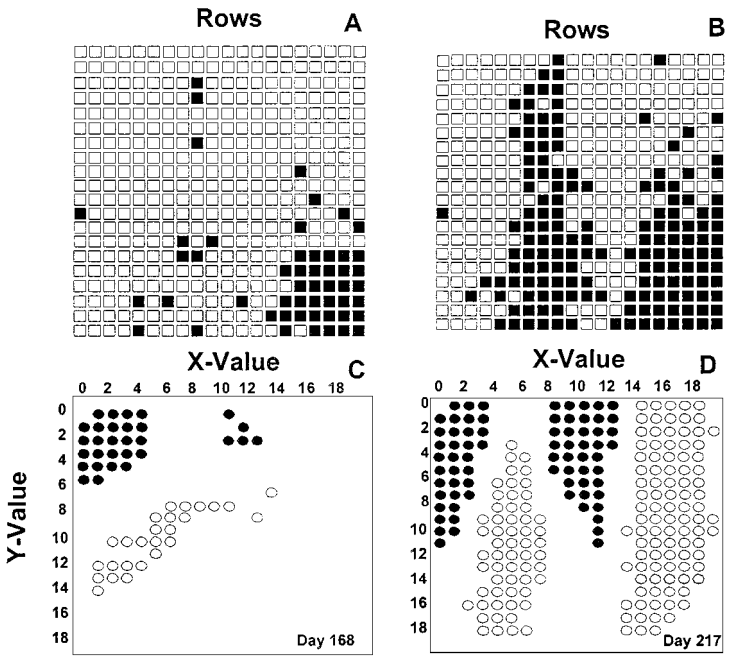


Figure 3 Spatial pattern of *Phytophthora* epidemics caused by *Phytophthora capsici* in a 20 by 20 lattice of contiguous quadrats at (*A*) day 168 and (*B*) day 217, and two-dimensional distance class array of the spatial pattern data at (*C*) day 168 and (*D*) day 217. Solid squares = quadrats that contain at least one diseased plant; open squares = quadrats that contain healthy plants. Solid circles = $[X, Y]$ distance classes with standardized count frequencies (SCF) greater than expected at $P < 0.05$; open circles = a SCF less than expected at $P > 0.95$.

For the *BB* statistic, y_{ij} is the product of the responses of the two sites, giving a one if both sites are diseased and zero otherwise. The expectations and variances of cross product statistics, and *BB* and *BW* statistics in particular, have been derived (21) and are given in several places (21, 45, 123). For the bell pepper field of Figure 3*B* the expectation and standard error of the *BW* statistic were calculated to be $\mathrm{E}(BW) = 179.9$ and $\mathrm{s}(BW) = 10.2$. The test statistic using the normal approximation (with a continuity correction) is then

$$\frac{BW - E(BW) + .5}{s(BW)} = \frac{72 - 179.9 + .5}{10.2} = -10.6.$$

This is far below the .0001 quantile of a standard normal distribution, so the p-value is close to zero. As before, we conclude that there is significant correlation between adjacent quadrats within the same row.

Description of Clusters: 2DCLASS, 2DCORR The aims of 2DCLASS analysis are to answer the question of whether the inoculum or disease forms clusters and to describe the size and shape of clusters (42, 90). The procedure is to compute the *BB* statistic for every distinct distance class in the lattice, where a distinct distance class is defined for each combination of within- and across-row distances. The distance classes are displayed in an array with different symbols to indicate significantly large *BB* statistics (Figure 3*C,D*). The 2DCLASS analysis of the disease incidence data at day 217 (Figure 3*B*) is shown in Figure 3*D*. Plants up to 11 quadrats apart within a row and three rows apart form clusters of correlated quadrats, forming "core clusters" that are longer down rows than across rows. When there is more than one cluster in a field, quadrats in one cluster are automatically correlated with quadrats in the other clusters. In this particular field, quadrats are significantly correlated with quadrats 8 to 12 rows away, with an average distance of 4 rows between clusters. The 2DCLASS analysis was done several times over the season (103). Comparison over time reveals that the season started with two clusters of disease, which then spread along the rows.

All of the methods discussed so far are based on the assumption that observations at different locations in the field are drawn from the same population and fluctuate randomly about some mean that is stationary across the field. An example of nonstationary means would be a large-scale trend from one side of the field to another. Recently, a modification to the 2DCLASS analysis called 2DCORR has been proposed (33) that assumes that the levels of disease across the field are not necessarily drawn from the same population. In 2DCLASS analysis, it is useful to label points by moving across the field in one direction, labeling a plant response z_i, as the head of a vector and its lag-h neighbor z_{i+h}, as the tail of the vector. Because a field has finite extent, the set of head sites is somewhat different than the set of tail sites. The 2DCORR procedure assumes that the means are different in the head and the tail regions of the field and depends on the observed levels of disease in the two parts of the field. The 2DCORR test asks the question, "Given the observed levels of disease in the head and the tail regions and the sizes of these regions, what is the probability of observing *BB* pairs of diseased plants h units apart?" The 2DCORR procedure tends to find larger core clusters than the 2DCLASS procedure. It does not tend to show correlations between clusters because the mean levels of disease in the two regions have been subtracted out.

Testing Regularity: Spatial Analysis by Distance Indices There are many methods of analyzing count data (e.g. number of propagules, number of diseased plants in a quadrat) to determine whether the distribution of counts is spatially random, clumped, or regular (14). We describe just one test here, called spatial analysis by distance indices (SADIE), because it explicitly uses spatial coordinates (96). This test was developed to describe the spatial pattern of insects. In this test a quantity called the distance to regularity is measured for the observed counts. Distance to regularity is defined as the minimum number of moves it would take to make the counts at all locations equal. Each "move" is a movement of one insect

(or propagule) over one unit of distance. A transportation algorithm (96) is used for computing the number of moves to regularity from any observed pattern of counts. The more highly aggregated a pattern of counts is, the higher the number of moves required to achieve regularity. To construct a reference distribution for the test, the observed counts are randomly permuted to the locations in the lattice many times, and the number of moves to regularity is computed for each permutation. The observed distance to regularity is then compared with the reference distribution. Turechek & Madden (121) demonstrate the use of this test on the pattern of strawberry leaf blight incidence in commercial fields.

Spatial Autocorrelation and Semivariograms of Continuous Variables

Tests for Autocorrelation: Moran's I and Geary's c Spatial autocorrelation for continuous variables, as opposed to binary variables, can be tested using Moran's *I* statistic or Geary's *c* ratio. Moran's *I* is closely related to the spatial autocorrelation coefficient and to the *BB* statistic; thus, Moran's *I* is large if there is high positive spatial autocorrelation. On the other hand, Geary's *c* is closely related to the semivariogram and the *BW* statistic, and a *negative* value of Geary's *c* indicates a positive spatial autocorrelation. The equations for these statistics are

$$I = \frac{\frac{1}{N_h}\sum_{i=1}^{N_h}(z_i - \overline{z})(z_{i+h} - \overline{z})}{\frac{1}{n}\sum_{i=1}^{n}(z_i - \overline{z})^2} \qquad 2.$$

$$c = \frac{\frac{1}{2N_h}\sum_{i=1}^{N_h}(z_i - z_{i+h})^2}{\frac{1}{n}\sum_{i=1}^{n}(z_i - \overline{z})^2}, \qquad 3.$$

where z_i is the response at the i^{th} location and z_{i+h} is the response h units away, N_h is the number of pairs of points that are h units away from each other, and n is the total number of points in the lattice. The numerator of each of these statistics is a cross-product statistic where $w_{ij} = 1$ if $j = i + h$, i.e. if point j is h units away from point i, and $Y_{i,i+h} = (z_i - \overline{z})(z_{i+h} - \overline{z})$ for Moran's *I*, or $Y_{i,i+h} = (z_i - z_{i+h})^2$ for Geary's *c*.

To test for spatial autocorrelation using either Moran's *I* or Geary's *c*, standardize the statistic by subtracting off its mean and dividing by its standard error, and compare to the quantiles of a standard normal distribution, Φ^{-1}. The hypothesis of no positive spatial correlation at lag h (a one-sided test) is rejected at the 5% significance level if

$$\text{Moran's } I\text{: } \frac{I - E(I)}{\sqrt{\text{Var}(I)}} > \Phi^{-1}_{.95}$$

$$\text{Geary's } c\text{: } \frac{c - E(c)}{\sqrt{\text{Var}(c)}} < \Phi^{-1}_{.05}.$$

The means are $E(I) = -\frac{1}{n}$ and $E(c) = 1$. The variances of Moran's I and Geary's c are given in Upton & Fingleton (123) and Cliff & Ord (21). This normal approximation is appropriate if the responses in the lattice are all drawn from the same distribution so that they all have the same mean and variance. In addition, the approximation requires the lattice to be relatively large and shaped such that points do not have very small numbers of neighbors.

The cross-product statistics are tremendously flexible. The lattice does not have to be rectangular; it can be irregularly shaped as long as there are a large number of neighbors for each point. Missing data do not cause any problem, although some computer software is written just for rectangular lattices without missing data. It is also possible to remove the assumption of constant mean at all points of the lattice. This is done by removing spatial variation in the mean before testing for spatial autocorrelation, either by regressing on covariates or by median polish (21). The expectation and variance for Moran's I_k, which is Moran's I computed on regression residuals, are given in Cliff & Ord (21).

Partial Autocorrelation Spatial correlations between points that are not immediately adjacent to each other are partly caused by the accumulation of correlations of neighbors between them. Partial autocorrelation measures the amount of correlation between z_i and z_{i+h} that is not accounted for by the intervening correlations. For example, the correlation between a plant in a quadrat of one row and a plant one quadrat up and one row over is partially explained by the lag-1 correlation within rows and the lag-1 correlation across rows. Any additional correlation, caused, say, by disease spreading in a diagonal direction, is called the partial autocorrelation. Computations of partial autocorrelations can help elucidate how far disease spreads in different directions. The partial autocorrelation between point 1 with coordinates (i, j) and point 3 with coordinates $(\mathrm{i}+1, \mathrm{j}+1)$, given point 2 with coordinates (i, $\mathrm{j}+1$) or $(\mathrm{i}+1$, j), may be estimated by

$$r_{13.2} = \frac{r(1,1) - r(1,0)r(0,1)}{\sqrt{(1 - r(1,0)^2)(1 - r(0,1)^2)}},$$

where $r(1,1)$ is Moran's I for points 1 row and 1 quadrat apart, $r(1,0)$ is Moran's I for points in the same quadrat of adjacent rows, and $r(1,0)$ is Moran's I for points in adjacent quadrats of the same row. The partial autocorrelation for points that are farther apart is given in Haining (50, p. 236).

Spatial Autocorrelation and Semivariograms Under the assumption that the mean and variance are constant across the field, spatial autocorrelation between two points h units apart may be estimated by a multiple of Moran's I,

$$r(h) = \frac{\sum_{i=1}^{N_h}(z_i - \overline{z})(z_{i+h} - \overline{z})}{\sum_{i=1}^{n}(z_i - \overline{z})^2} = \frac{N_h}{n} I \qquad 4.$$

(49, p. 119). Spatial autocorrelation coefficients are usually computed for several values of lag distance h and plotted against h. When viewed as a function of lag

distance, this is called the correlogram. Reynolds & Madden (99) discuss autocorrelation and partial autocorrelation parameters in a spatial-temporal autoregressive model and provide more sophisticated estimators.

The semivariogram is another function for quantifying spatial association. It has advantages for estimating and modeling spatial association, and is usually used in geostatistical applications, rather than the covariance function. The semivariogram is $\gamma(h) = \frac{1}{2}\text{Var}(z_i - z_{i+h})$ and the classical estimator is

$$\hat{\gamma}(h) = \frac{1}{2N_h}\sum_{i=1}^{N_h}(z_i - z_{i+h})^2, \qquad 5.$$

which is the numerator of Geary's *c*. If the variance is stationary across a field, the covariance function and the semivariogram are mirror images of each other since $\gamma(h) = \text{Var}(z) - \text{Cov}(z_i - z_{i+h})$. An idealized semivariogram shape is shown in Figure 4*A* (47). The range of spatial autocorrelation is the distance beyond which little spatial autocorrelation is evident. The range can be read off the semivariogram as the lag distance where the curve almost reaches a plateau or *sill*. Information about the trends and periodicities in the spatial pattern can also be seen from the semivariogram. Larkin et al (65) computed the semivariograms for four different directions for disease severity in the pepper field shown in Figure 2*B* (103) (Figure 4*B*). The semivariogram for variation within rows has a linear shape that never reaches a sill. This indicates that the correlation between one quadrat and another decreases with lag distance within a row, but correlation is still evident even 15 quadrats apart. The semivariogram for variation across rows shows an oscillating wave pattern. This is indicative of sets of rows with high disease severity alternating with sets of healthy rows. Together, the within-row and across-row semivariograms describe a pattern of disease consistent with more rapid spread down rows than across rows. The shapes of the two semivariograms for the diagonal directions are blends of the within- and across-row semivariograms and are more similar to the idealized picture in Figure 4*A* (47).

If estimates of the range, the variance, and the microscale variance are desired, parametric models may be fitted to the data, either by weighted nonlinear least squares (24, p. 99) or, preferably, by restricted maximum likelihood (72, p. 306). Equations for some of the most commonly used semivariogram models and information about fitting them can be found in Gumpertz et al (47).

Regression Models for Spatial Data

In this section we briefly describe some types of models that are useful for relating disease in one location to disease in other locations or to other explanatory variables. Software is currently available to fit some of these models, notably models for normally distributed data, but methods are still under development for most of them.

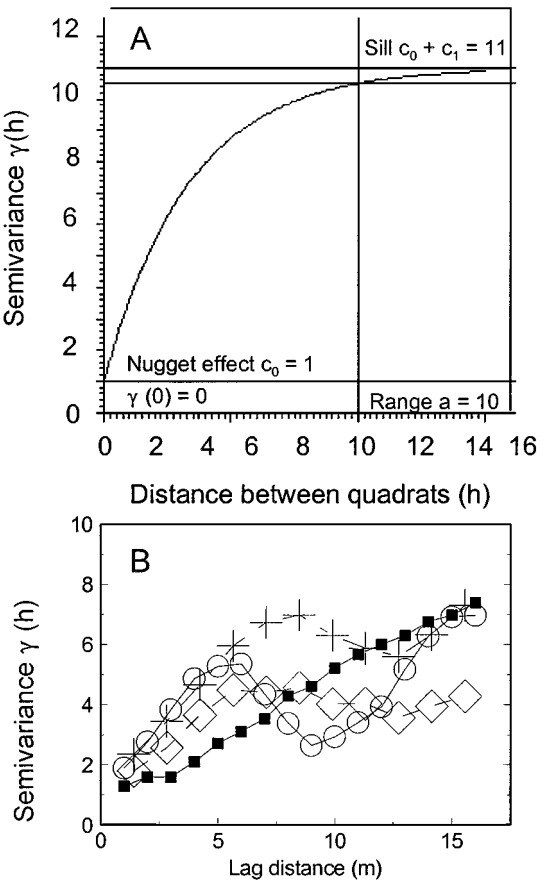


Figure 4 (*A*) An idealized exponential semivariogram model with nugget effect $c_0 = 1$, sill $c_0 + c_1 = 11$, and range $a + 10$, (*B*) Semivariogram of disease severity caused by *P. capsici* at day 217 from the field shown in Figure *2B*. Solid square = within rows, open circle = across rows, open diamond = 45 degrees, cross = 135 degrees.

Spatial Interaction Models In spatial interaction models, also known as Markov random field models (21, 24), the level of disease or the probability of disease depends on the level in the neighboring locations. Spatial interaction models are appropriate for data on a lattice of discrete points. This type of model is most useful when the aim is to describe the relationships among points in the lattice, such as the dispersal of propagules from one quadrat to another. These models are similar to autoregressive and moving average time series models, but the two-dimensional nature of spatial relationships introduces some special considerations. Cliff & Ord (21) and Cressie (24) present the details of simultaneous autoregressive models, conditional autoregressive models, and moving average models for normally distributed spatial data. These models can be fitted using commercial statistical software (60). This type of analysis requires specification of a neighborhood structure and a measure of proximity. Reynolds & Madden (99) discuss the extension of these types of models to spatial-temporal data, and also give some

recommendations about choosing neighborhood structures and proximity measures for modeling disease development. They demonstrate the use of spatial-temporal autoregressive integrated moving average models (STARIMA) for studying the development of an epidemic of leather rot of strawberry (99). In this example, the change in logit (disease incidence) from time t to time $t + 1$ was found to depend on disease at the previous time in the same plot and in the nearest neighbor plots, but larger lags in time or space did not seem to play a role, which is consistent with the known mechanism of splash-dispersal of this pathogen in strawberry. They found that a first-order moving average term was necessary, which they interpreted to indicate that external factors such as weather, which were not included in the model, also affected the incidence of disease (99).

Spatial interaction models have also been developed for spatial binary (presence/absence) data (5, 6). Software is not yet commercially available to fit this type of model, which is called an autologistic model. The autologistic model was recently used for studying the Phytophthora epidemic in bell pepper described previously (46). The logit of the probability of disease on Julian day 177 [note: $\text{logit}(p) = \log \text{ odds of } p = \log(\frac{p}{1-p})$] was modeled as a function of soil water content (%), pathogen population levels in the soil (number of leaf disks colonized out of five), and the numbers of diseased neighbors in each of four directions: adjacent quadrats within rows, adjacent quadrats in neighboring rows, diagonally up one quadrat and one row, and diagonally down one quadrat and up one row. There was a clear visual correspondence between soil water content and disease presence at day 168 (Figure 2*A*,*C*); (46). The fitted model picked up this relationship and also found a correspondence between soil propagule levels (Figure 2*D*) and disease at day 177 (data not shown) (46). The odds of disease were estimated to increase 337% with a 5-percentage point increase in soil water content. The odds of disease were estimated to be 47% higher if one leaf disk was colonized than if none were, and to be nearly six times higher if all five leaf disks were colonized than if none were colonized. Since the soil water content and pathogen propagule levels are also spatially correlated, it was difficult to distinguish between effects of the explanatory variables and effects of neighboring points, but disease presence in diagonally adjacent quadrats also appeared to be a significant factor. A similar model was fitted to disease and soil variables at two later dates in the season: on Julian days 195 and 217. The magnitude of the effect of early season soil water content (day 174) and leaf disk populations (day 171) on disease decreased throughout the season. Soil propagule levels of *P. capsici* measured at the second sampling time later in the season (day 218) were a good indicator of disease presence on the day 217 sampling date.

In a second field (data shown in Figure 1, Reference 46), soil water content and pathogen propagule levels in soil were not good predictors of disease presence. In that field, substantial within-row dependence of diseased plants between adjacent quadrats was seen. The estimated odds of disease were nearly four times higher if one neighbor was diseased than if none were diseased.

Linear, Nonlinear, and Generalized Linear Models for Spatial Data Geostatistical methods such as kriging, universal kriging, and cokriging use linear models with spatially correlated errors (67). This type of model is appropriate for continuous data, either normally distributed or transformed to normality, when the aim is either (*a*) to estimate the effects of explanatory variables (also known as covariates) or (*b*) to map or spatially interpolate to locations where the response variable was not actually measured. In this type of model, the explanatory variables describe the large-scale trends and the spatial correlation structure describes spatial pattern at smaller scales that is not explained by the covariates. Commercial software is currently available to fit both linear and nonlinear models with spatially correlated errors (72). A wealth of climate and environmental data are available on the Internet that can be incorporated into spatial models of disease patterns (8, 22).

A similar approach has been taken with generalized linear models for binary and count data (1, 41, 80). These types of models have not yet appeared in studies of root disease epidemics, but examples of spatial logistic regression are available in the entomology and forestry literature. Related types of models for regressing disease presence or inoculum levels on covariates while taking spatial correlation into account, such as generalized linear mixed models and hierarchical models, are currently under development.

NEW FRONTIERS FOR RESEARCH

Novel Methods of Disease Management

Research into the mechanisms of dispersal of *Phytophthora* species has led to the development of new methods of disease management using ecologically based approaches. For soilborne species in the genus that are dispersed from rain splashing or surface water movement, modifications in surface topography in fields by use of living mulches, no-till cover crops, straw mulch, or pine bark mulch on the soil surface can significantly reduce the spread of a number of splash-dispersed *Phytophthora* species (77, 106, 134). A rain simulator was used to quantify the effect of various types of ground covers including soil, straw, sand, or plastic mulch on dispersal of *P. cactorum* (77). There was a significant interaction between rain intensity and ground cover. Disease incidence exceeded 80% on black plastic, while incidence was only 15% on straw mulch (77). Incidence of Phytophthora blight on pigeon pea was greatly reduced by the presence of weeds in plots, which reduced the splash dispersal of the pathogen from soil to above-ground plant parts (18). Dispersal of *Colletotrichum acutatum* was significantly reduced on a living mulch of sudan grass compared to black plastic mulch (93).

The spatial dynamics of Phytophthora blight on bell pepper and the incidence of disease was modified dramatically by suppressing specific dispersal mechanisms (106). On bare soil plots, all mechanisms of dispersal are possible and final incidence of Phytophthora blight on bell pepper was 71–72% in bare soil plots when a

point source of initial inoculum was placed at one end of rows 4 and 5 (Figure 5*A*). Black plastic mulch can act as a barrier to dispersal of soilborne inoculum under the plastic to aerial parts of the plant (mechanisms III and IV) but can increase splash dispersal of surface inoculum (mechanism III) in free water on the surface of plastic (mechanism II). Disease onset occurred first and final disease incidence was 42–79% in peppers grown on black plastic mulch with a sporulating fruit placed on the plastic surface (Figure 5*D*). The presence of stubble from a cover crop of wheat effectively reduced dispersal by blocking surface water and splash dispersal mechanisms (mechanisms II and III). Final disease incidence was only 2.5–43% when stubble from a fall-grown cover crop was present and initial inoculum was placed in rows 4 and 5 (Figure 5*B*). Metalaxyl is transported upward in the plant, so protection of above-ground portions of the plant from splash-dispersed inoculum is probably more effective than protection of roots and crowns in soil from inoculum moving down the plastic in surface water (106). The fungicide metalaxyl prevented across-row disease spread from point source inoculations but did not suppress within-row spread of surface inoculum on plastic and root infection (Figure 5*C*). Thus, studies on the spatial dynamics of Phytophthora blight epidemics on bell pepper have also provided new information of the "mode of action" of metalaxyl in the field.

Changes in surface topography and inoculum source type dramatically affected disease gradients and within-row spread of *P. capsici* (Figure 6). Soil in rows that were either covered with black plastic, stubble from a rye-vetch cover crop or bare soil was noninfested or infested with sporangia or oospores buried in the transplant hole at the end of each row. Final disease incidence in bare soil plots was 100, 69, and 10% in rows infested with sporangia, oospores, or noninfested soil, respectively (Figure 6*A*). In contrast, disease incidence in plots planted in a rye-vetch cover crop was 11, 12, and 3% and in plants planted in plots covered with black plastic was 7, 20 and 18%, in rows infested with sporangia, oospores, or noninfested soil, respectively. Disease gradients changed little over time in plants grown in plots with stubble from a cover crop infested with sporangia inoculum since dispersal within rows by surface water was greatly reduced by the cover crop (Figure 6*C*). Changes in the disease gradient over time were most pronounced in plants in plots grown with bare soil and sporangial inoculum, demonstrating significant within-row spread on bare soil (Figure 6*B*). Note a delay in disease onset and lower disease incidence occurred in bare soil plots infested with oospore inoculum, since a lag time was required before oospores germinated and produced sporangia and zoospores that were available for dispersal. Further work is needed on the influence of inoculum source type on the spatial dynamics of epidemic development since it is often assumed that disease will be more severe with oospore inoculum.

Host genotype mixtures of potato were planted in a field study on late blight to determine if the aerial dispersal of the pathogen could be reduced (2). Either a susceptible cultivar was planted in a pure stand or two partially resistant cultivars were arranged alone or in alternate rows with the susceptible variety in the field.

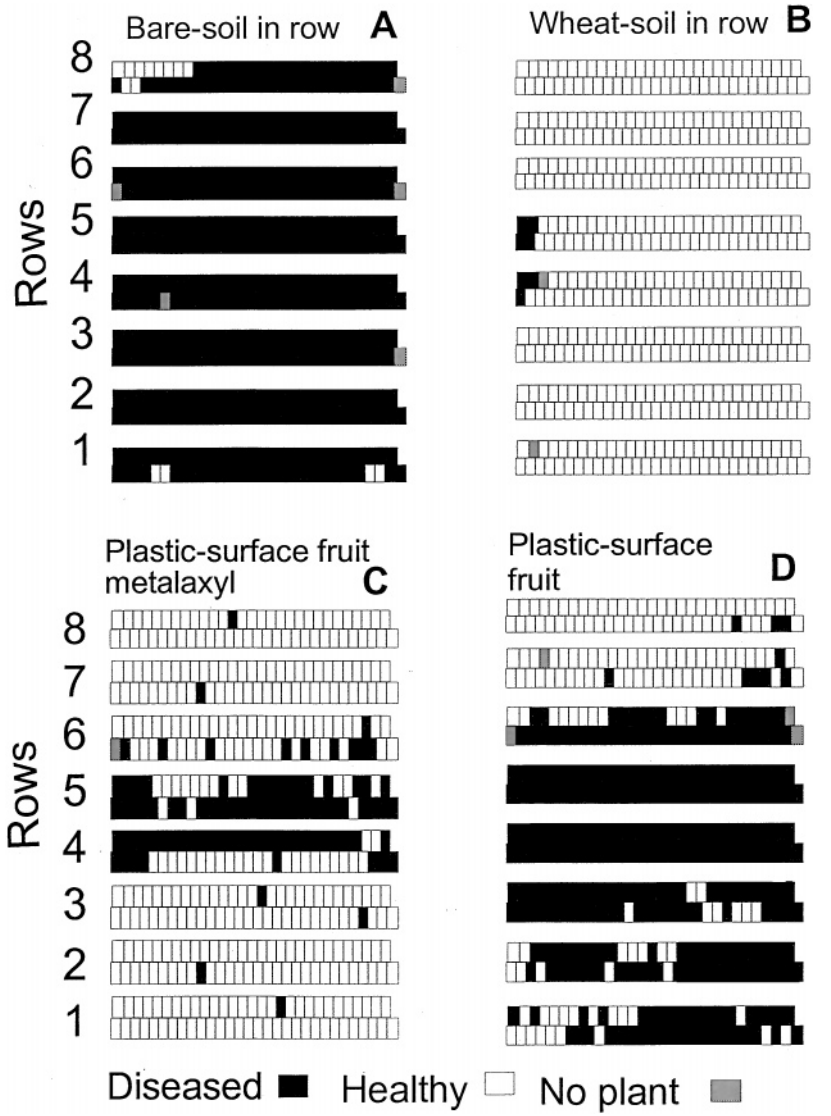
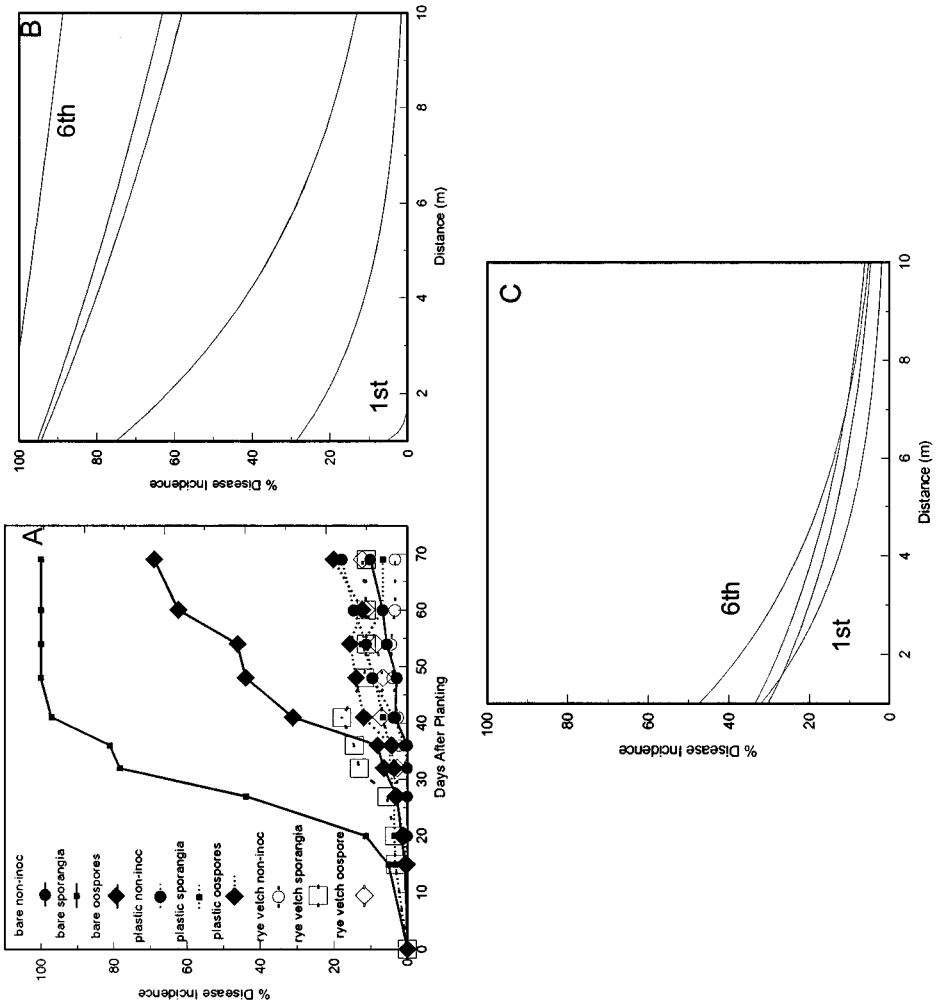


Figure 5 Spatial pattern of epidemics caused by *Phytophthora capsici* in bell pepper as affected by cultural and chemical control strategies. Each solid rectangle represents an infected plant within a row. Initial point sources of inoculum were at the ends of rows 4 and 5. Data shown are from one replication. (*A*) Bare soil in plots infested with inoculum placed in the row, (*B*) no-till wheat mulch plus soil inoculum in the row, (*C*) black plastic mulch plus surface inoculum and metalaxyl applied through the irrigation system, (*D*) black plastic mulch plus surface fruit inoculum.


A
B
C
bare non-inoc
bare sporangia
bare oospores
plastic non-inoc
plastic sporangia
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rye vetch non-inoc
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rye vetch oospore
% Disease Incidence
Days After Planting
Distance (m)
6th
1st

Changing the spatial arrangement of cultivars in the field had a large impact on disease. For the susceptible cultivar, disease progression was slower in the mixed stands than in the pure stands and the disease remained focal in all the plots at all the scoring dates. Disease severity was 30 to 50% lower in mixed than in pure stands for the susceptible cultivar and yields were increased. For the partially resistant cultivars, there was no advantage of planting in mixtures and yields were increased in one of two years for mixed versus pure stands (2). The impacts of planting of cultivar mixtures on the spatial dynamics of late blight epidemics needs further experimentation in the field on larger spatial scales and represents an excellent example of how a simple change in planting strategy can have a large impact on an aerially dispersed *Phytophthora* species.

Intersections of Population Genetics and Spatial Pattern Analysis: Molecular Epidemiology The term molecular epidemiology has been used to describe a new and exciting area in research in infectious diseases of humans (70). For plant disease, the goal of a molecular epidemiologist is to identify the microorganisms responsible for disease; determine their physical sources, their biological, evolutionary, and phylogenetic relationships; their routes of transmission; and those genes and accessory elements responsible for their virulence or pesticide resistance (70). It is clear from this definition that this new area of research represents a convergence of several traditional disciplines including population biology and genetics, ecology, host parasite interactions, taxonomy, and epidemiology. This new area of research will require disintegration of traditional disciplinal insularities and development of multidisciplinary teams if it is to move forward in our science.

The contributions of population genetics to plant disease epidemiology and management of a number of diseases have been reviewed recently (82). A wide range of neutral and nonneutral genetic markers based on isozyme analysis, serological assays, DNA probes, RFLP, and PCR methods have been used in phylogenetic studies and to measure genetic variation in populations of *Phytophthora* species (23, 37, 58, 95). The *P. infestans*–late blight example on potato has been studied more extensively than other Phytophthora diseases. Genetic markers have been used to monitor the population structure of isolates within and among fields and to track global migrations of the pathogen (37, 38, 40, 82). Most of the modern worldwide populations of *P. infestans* were dominated by a single clonal lineage until the early 1980s (40). These data led to the "genetic bottle" hypothesis that suggests that a single clonal genotype of *P. infestans* migrated from Mexico (where

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Figure 6 (*A*) Disease progress of Phytophthora blight on bell pepper over time as influenced by inoculum source type of either sporangia or oospores and cultural practice including bare soil, plastic mulch, or stubble from a rye-vetch cover crop, and disease gradients from an introduced point source of sporangia inoculum over six rating times in plots planted on (*B*) bare soil or (*C*) stubble from a rye-vetch cover crop.

most populations are very diverse and sexually reproducing) and was responsible for the late blight epidemics of the nineteenth century in Europe (40). The spatial structure of the population sampled in this study included isolates collected on a global basis from many different fields in relatively modern times. Molecular tools are now available that may enable us to discern migration patterns of historic epidemics caused by *P. infestans* using herbarium materials from the nineteenth century (107).

Geostatistics and geographical information systems software (GIS) were used to examine the range of spatial dependence of various genotypes of *P. infestans* (89). GIS software is used to store, manage, visualize, and analyze spatially referenced data. Most genotypes of *P. infestans* showed spatial autocorrelation in a range of 13,000 to 20,000 m and some epidemics were dominated by only one genotype in a given year, which suggests strong clonal reproduction (89). In other regions in Mexico, populations of the pathogen are known to be quite diverse within a given field, both mating types are found, and sexual reproduction is common (37). In many areas of the United States, new genotypes of *P. infestans* have displaced old genotypes. If sexual reproduction becomes more common in fields and the pathogen overwinters in plant debris, the initial spatial patterns of a given genotype in a field could have large impacts on subsequent epidemic development, particularly if the genotypes vary in sensitivity to fungicides or in fitness.

Pathogen genotype mapping using GIS could also be useful to track regional occurrences of new genotypes of the late blight pathogen and dispersal from seed producing regions to areas receiving potato seed tubers in order to forecast the future population structure of epidemics.

Precision Agriculture Applications Precision agriculture can be defined as a management strategy that uses information technologies to bring data from multiple sources to bear on decisions associated with crop production (87). Precision agriculture has three components: capture of data at an appropriate scale and frequency, interpretation and analysis of those data, and implementation of a management response at an appropriate scale and time (87). There are obvious applications of spatial pattern analysis in precision agriculture studies.

In precision agriculture applications, tractor mounted devices that are capable of collecting spatially referenced soil samples and delivering spatially referenced doses of a given fertilizer or pesticide are now possible (87). Ideally, one could use these technologies to reduce excessive fertilizer or pesticide use in environmentally sensitive areas. Currently, there is a dearth of spatially referenced data available for most soilborne *Phytophthora* species that could be useful for precision agriculture applications. The hypothesis that these new technologies will lead to reduced pesticide use has also not been adequately tested with data sets from soilborne pathogens (35). Development of spatially referenced data sets for *Phytophthora* species with a soilborne phase will require a shifting of research priorities from controlled studies at experiment stations to "in field" assessments of pathogen propagule densities, disease, and soil chemical and fertility factors at relevant spatial scales (103, 131). Many of our existing data sets for soilborne *Phytophthora*

species are two-dimensional and limited information is available on the occurrence of these pathogens at different depths in soil.

The potential of precision agriculture is currently limited by the lack of appropriate measurement and analysis techniques for agronomically important factors (87). Improved detection methods for specific soilborne pathogens that are limiting to crop production are needed. For precision IPM applications, soil assays need to be developed that enable quick qualitative or quantitative identification of target species of *Phytophthora* or other soilborne plant pathogens. Ideally, assays could be conducted in the field on tractor-mounted devices. Development of additional specific primers to accurately identify individual species of *Phytophthora* are needed and optimization of the PCR assays for detection of different propagule types in soil is important if these assays are to be used for precision agriculture applications (23, 68, 69, 105, 120). Few studies with soilborne *Phytophthora* species have addressed these needs.

GIS databases that contain spatially referenced data sets for a given field and regionally are needed to make pathogen management systems viable on a spatial scale. Multivariate analysis of soil physical, chemical, and biological components will be needed in order to develop management options for precision agriculture applications. Geostatistics and indicator kriging has been suggested to map the probability of exceeding a threshold in a pest population for precision IPM applications (35). At the present time, these data sets are lacking for most soilborne *Phytophthora* species. Unlike insect populations, often very low densities of inoculum can cause high levels of disease for species of *Phytophthora* (30, 101). A very extensive regional assessment of soybean fields infested with *P. sojae* was conducted in the midwest (132). *P. sojae* populations were greater in conservation till than conventional-till fields. Pathogen population data could be collected on a spatially referenced scale within fields and include genotypic and phenotypic information. Precision IPM may be possible and potentially additional management options could be deployed in addition to changes in tillage practices for control of *P. sojae* (132).

CONCLUSIONS

Phytophthora species can be dispersed by one of several mechanisms. An understanding of these mechanisms of dispersal has led to the development of novel management strategies in the field. Dispersal in soil, via surface water, by rain splash, by air, or via human or insect activity are the major dispersal mechanisms for *Phytophthora* species. Disease management strategies that rely on ecologically based approaches have the potential in the long term to be more sustainable than single input options (102).

Whether space is truly the final frontier is a matter of perspective. Certainly for diseases caused by species of *Phytophthora*, there are a number of new frontiers that can be explored in the coming years to study the basic biology of these devastating pathogens and develop new management options using spatial pattern

analysis. The development of new technologies in the area of molecular biology and information technology will enable us to gather more spatially referenced data on pathogen populations and disease more rapidly at many spatial scales from the individual cell, to the population in the field, to the ecoregion, or globally. Analysis of these data using some of the statistical procedures discussed in this paper may lead to improved disease management in the twenty-first century.

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